**POSTERS**

**DEVELOPMENTAL BIOLOGY**

**MICRORNA ANALYSIS OF GROWING KIDNEYS**

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**Aim:** To examine changes in miRNA expression during compensatory renal hypertrophy (CRH).

**Background:** CRH naturally occurs when one kidney is removed for kidney donation or cancer removal. The remaining kidney grows approximately 30% and this is dominated by an increase in individual cell size. MicroRNAs (miRs) have been described to play important post transcriptional roles in growth control. However, their role in CRH remains unknown.

**Method:** C57BL6 mice underwent a left kidney nephrectomy (unx) or sham operation. The remaining kidney was collected 24, 48 and 72 h post-surgery (n = 6 per group). RNA was extracted and underwent small RNA-Sequencing. QuickMIRSeq was used to identify and quantify miRs and edgeR was used for miR differential expression analysis.

**Results:** Significant differential expression of miRs were only detected at 48 h and 72 h timepoints. At 48 h, 24 miRs were differentially expressed (19 upregulated and 5 downregulated), of those upregulated, 6 belong to the Let-7 family (Let-7a,b,c,f,g,i). At 72 h only 5 miRs were differentially expressed of which 1 was from the Let-7 family (Let-7a). Subsequent enrichment analysis of Let-7 family miR gene targets at 48 h suggested a decrease in genes with roles in mitotic spindle assembly and inhibition of mTORC signalling.

**Conclusion:** Let-7 miR family members were upregulated at 48 h and 72 h post nephrectomy. These microRNAs have been previously implicated in cell cycle control and in nephrogenesis. Our enrichment analysis suggests that expression of Let-7 miRs may be involved in cell cycle and contribute to the cellular hypertrophy seen in CRH.

**EXPERIMENTAL TUBOLOINTERSTITIAL DISEASE**

**DEVELOPMENT OF A SPECTRAL FLOW CYTOMETRY APPROACH FOR DIAGNOSTIC PROFILING OF SINGLE URINARY EXTRACELLULAR VESICLES**

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**Aim:** To develop a workflow for purifying and profiling single urinary extracellular vesicles (uEVs) using spectral flow cytometry.

**Background:** uEVs contain molecules which participate in paracrine communication between tubules, immune function and fibrotic responses. Previous analyses of uEVs primarily examined bulk populations using labour-intensive methods (i.e., proteomics and transcriptional profiling). Here, we present a novel, high-throughput flow cytometric method of profiling single uEVs.

**Methods:** 50 ml urine from four healthy adults were centrifuged at 650g for 10mins to remove cells. Supernatants were treated with protease inhibitors followed by centrifugation at 2000g for 20 mins to remove cellular debris. uEVs were concentrated to 500 μl using serial centrifugation at 3500g for up to 40mins in an Amicon Centrifugal Filter unit, followed by centrifugation at 20000g for 30 mins. Flow cytometry quantitation was completed using 400 nm beads, with antibodies against markers of nephron source: podocin (podocyte), CD45 (leukocyte), CD31 (vascular), CD10 (tubular), CD13 (tubular) and MUC1 (tubular). FCMpass was used to calculate uEV size.

**Results:** Mean uEV concentration was 4.86 × 10⁵ particles/μl. uEVs ranged in size from 250 nm to 770 nm, with mean diameter of 354 nm. uEVs stained positive for proximal tubular markers CD10 (mean: 3.57 × 10⁴ particles/μl) and CD13 (mean: 4.82 × 10⁴ particles/μl) and distal tubular marker MUC1 (tubular). Comparatively, there were minimal uEVs positive for podocin (mean: 2.60 × 10³ particles/μl), CD45 (mean: 1.99 × 10³ particles/μl) and CD31 (mean: 1.96 × 10² particles/μl).
Conclusions: uEVs from healthy individuals are predominantly from the tubular compartment, with minimal uEVs from podocyte, leukocyte and vascular compartments. We aim to continue developing this novel diagnostic platform for translation to kidney disease patients.

LONG NON-CODING RNAs (lncRNAs) IN PRECLINICAL MODELS OF DIABETIC KIDNEY DISEASE (DKD): SYSTEMATIC REVIEW

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Aim: Delineate the mechanistic role of lncRNAs in preclinical models of DKD through systematic review.

Background: Exploring the role of long non-coding RNAs (>200 nt long), one of the least investigated novel epigenetic mechanisms, in gene-expression regulation of DKD may lead to insights into DKD-pathogenesis and establish potential treatment-targets.

Methods: Electronic search from five bibliography-databases (Ovid-Medline, Embase, PubMed, Scopus, Web of Science) was conducted and studies included based on a protocol-specified criteria of any published original-experimental-controlled studies in English using in-vivo murine and/or in-vitro murine or human cell-models of DKD that explored lncRNAs-mechanisms. Studies were excluded if model is non-DKD, gene-modulation intervention or lncRNA mechanism were not reported. Two independent investigators conducted the search/screening, key study-characteristics were extracted, and study-quality assessed by SYRCLE’s risk-of-bias-tool (in-vivo), or NTP-OHAT recommendations for in-vitro studies.

Results: This search yielded 2002 articles with 144 qualitative studies extracted involving 77 different individual lncRNAs in preclinical DKD-models. Majority of studies explored the role of lncRNAs in pathogenesis of DKD. The five most common mechanisms were fibrosis, cell-viability/proliferation, cell-apoptosis, inflammation, oxidative stress. Most commonly investigated lncRNAs were MALAT1, TUG1, NEAT1, PTEN, ASS1, GAS5, CASC2, MEG3, etc. KenC1,017T1, MIAT, ZEB-AS1, of which lncRNA TUG1, GAS5, CASC2, ZEB-AS1, MEG3 had potentially protective role. The role of competing endogenous RNA (ceRNA) was often explored.

Conclusion: LncRNAs are implicated in a wide variety of pathogenic processes in DKD, with individual lncRNAs playing either a detrimental or protective role in fibrosis, inflammation, apoptosis & other molecular processes. Gene-modulation-interventions can limit burden of DKD. This review highlighted need for more standardized reporting of methodology in preclinical studies. The role of lncRNAs in DKD is an emerging field, requiring more research to fully establish their role.

GENERAL NEPHROLOGY—ACUTE KIDNEY INJURY

REVERSIBLE ACUTE KIDNEY INJURY WITH HIGH GRADE PROTEINURIA ASSOCIATED WITH FAK AND MEK INHIBITION TREATMENT FOR METASTATIC MELANOMA

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Background: Focal adhesion kinase (FAK) and MEK inhibitor combination therapy is being trialled as treatment for metastatic Neuroblastoma-RAS (NRAS) mutant melanoma. FAK is a non-receptor tyrosine kinase implicated in tumorigenesis, promoting cell survival and proliferation. Proteinuria is a known dose limiting side effect of FAK inhibition therapy. We report a case of acute kidney injury following FAK and MEK inhibition therapy, necessitating treatment cessation.

Case Report: A 70-year-old female with metastatic NRAS-mutant melanoma was enrolled in a Phase I trial investigating FAK inhibitor, IN10018, and MEK inhibitor, cobimetinib, combination therapy. On routine monitoring there was nephrotic range proteinuria, with urine protein creatinine ratio of 1873 mg/mmol. Trial treatment was subsequently withheld. Two weeks later the patient presented with oliguric acute kidney injury. Serum creatinine was 279 μmol/L at the time of presentation and peaked at 501 μmol/L from a baseline of 98 μmol/L 1 month prior. Autoimmune serology and imaging were non-contributory in identifying a cause for renal impairment. Renal biopsy showed significant acute tubular necrosis, with mild interstitial inflammation and background changes of mild interstitial fibrosis. Electron microscopy revealed partial foot process effacement. Management was supportive only. Renal function improved with proteinuria resolving fully and creatinine decreasing to 110 μmol/L 2 months post the cessation of the FAK inhibitor.

Conclusions: FAK inhibition shows promise as anticancer therapy, particularly in combination with other agents. We present a case of heavily proteinuric acute kidney injury following IN10018 and cobimetinib combination treatment requiring withdrawal of trial therapy. This proposes acute glomerulonephropathy as an adverse effect from FAK and MEK inhibitor therapy.

ACUTE INTERSTITIAL NEPHRITIS WITH INFLAMMATORY BOWEL DISEASE

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Background: A plethora of case reports are published around the relationship between acute interstitial nephritis (AIN) and irritable bowel disease (IBD) management medications. A well-established relationship exists between Vedolizumab and AIN. There are also reports of Mesalazine and AIN, which could manifest months to years post
treatment. We present a case of AIN and the causative drug under contention.

Case Report: We present a case of a 19-year-old female with a history of ulcerative colitis for 1 year which was managed with Mesalazine since the initial diagnosis and trialled on Azathioprine and Infliximab with nil effect. She was commenced on Vedolizumab in February and bloods prior to this showed creatinine 60umol/L. A 2 months post the first infusion of Vedolizumab, she presented with calf pain and routine bloods showed creatinine 280umol/L. Urine dip showed blood and protein and a renal biopsy was consistent with florid acute interstitial nephritis. As Vedolizumab was the most recent addition to her medications, the initial blame was on this. However, her treating gastroenterologist connected Mesalazine to the AIN and hence this has been ceased together with commencement of high dose steroids. All these measures have improved creatinine to 160umol/L, but not back to baseline. The potential for AIN from Vedolizumab persists.

Conclusions: This case highlights the importance of careful history, especially medication history to work out the potential causative drugs for AIN. Our patient was on both Mesalazine and Vedolizumab, and potentially both are the reason behind her sudden acute kidney injury.

CHARACTERISTICS AND OUTCOMES OF INPATIENTS CONSULTED FOR ACUTE KIDNEY INJURY AT A TERTIARY METROPOLITAN HOSPITAL IN SYDNEY, AUSTRALIA

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Aim: To describe the characteristics and evaluate outcomes of inpatients consulted for acute kidney injury (AKI).

Background: AKI occurs in 7–18% of hospitalized patients and associated with significant morbidity and mortality. Follow-up rates are poor despite studies showing early nephrology review improves overall survival.

Methods: An audit was undertaken on inpatients consulted for AKI in 2021 at Prince of Wales Hospital (POWH). Demographic, laboratory and clinical data were retrieved from electronic medical records.

Results: Out of approximately 1020 patients with AKI detected yearly by the POWH AKI electronic alert, 104 were consulted by nephrology in 2021. ACE-I and ARB were withheld in all 40 patients and restarted in 9 out of 33 (27%) who survived the admission and in 3 outpatients. No patients recommenced on ACE-I or ARB died suggesting they can be safely used post AKI and likely protective. SGLT2-i was withheld in 6 out of 7 patients and recommenced in 3 patients (43%) on discharge and in 1 outpatient. Metformin was withheld in 19 out of 22 patients and recommenced in 13 out of 20 (65%) who survived the admission and in 3 outpatients. 36 (35%) patients had renal follow-up at POWH, 15 (14%) lived outside the POWH catchment area, 21 (20%) did not attend nephrology follow-up, 21 (20%) had poor prognosis or died in hospital and 11 (11%) had no renal follow-up organized.

Conclusion: ACE-I, ARB, SGLT2-i and metformin were withheld in most patients and recommenced in 27%, 43% and 65% of patients respectively on discharge, highlighting the importance of AKI follow-up to review timing of medication recommencement.

HIGH, HIGHER, HIGHEST: A PATIENT WITH AKI AND MARKEDLY ELEVATED CREATININE

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Background: The degree of elevation of creatinine is used as a marker to stage severity of acute kidney injury (AKI). Levels of creatinine greater than ≥353.6 umol/l constitute advanced, stage 3 AKI by
international criteria. The physiological effects or prognostic significance of progressively higher creatinine levels at diagnosis in AKI is unknown.

**Case Report:** A 66-year-old male with schizophrenia and alcohol dependence had been generally unwell for a few days. He presented to the emergency department with altered sensorium, ongoing haematemesis and a haemoglobin of 60 g/L. While being readied for urgent endoscopy, he suffered cardiac arrest and was resuscitated. Initial investigations revealed severe acute kidney injury with creatinine 2854 μmol/L, urea 107 mmol/L and potassium 8 mmol/L. He was intubated and underwent emergency dialysis with gradual improvement. Endoscopy showed diffuse gastritis. Subsequent investigations revealed bilateral hydronephrosis secondary to infiltrative bladder cancer. The duration of obstruction was unknown. With the placement of bilateral percutaneous nephrostomies, urine output was re-established. He was able to come off dialysis after a week, with partial renal recovery.

**Conclusions:** A serum creatinine of 2854 μmol/L (32 mg/dL) is a rare occurrence. The degree of elevation of creatinine may be related to AKI aetiology. Progressive obstructive uropathy may have allowed a creeping rise in urea and creatinine in our patient. In the community, the previous history of schizophrenia and poor self-care could have contributed to a relatively late recognition of encephalopathic features. Creatinine, a normal constituent of human plasma does not have known pathological effects by itself. In acute uraemia, elevations of other substances such as urea, potassium and metabolic acids are potentially more harmful.

**MICROEMBOLISATION OF POLYMER COATING INTO THE KIDNEY FOLLOWING CORONARY ANGIOGRAPHY**

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**Background:** Polymer coating of intravascular devices have been increasingly used since the 1980s to reduce friction and thus arterial spasm and pain during vascular access. There have been reports of polymer coating shearing off during access and embolising to organs including lungs, brain, feet, heart, skin, liver and kidney. A post-mortem study observed distal embolization of polymer coating in 10% of percutaneous coronary interventions. This is likely an under observed clinical event.

**Case Report:** Mr AW presented following an out-of-hospital cardiac arrest for which he was successfully resuscitated and diagnosed to have an ST-elevation myocardial infarction. He underwent successful percutaneous coronary intervention to the left anterior descending artery. His echocardiogram demonstrated a reduced ejection fraction and he was treated with optimal medical therapy.

Post angiography and discharge from hospital, his renal function deteriorated from a baseline creatinine of 90μmol/L to 200μmol/L over a ten-month period with associated proteinuria (albumin: creatinine ratio 142 mg/mmol) and microscopic haematuria. Imaging of the kidneys was unremarkable. He underwent a renal biopsy.

His renal biopsy showed microvascular embolisation with hyaline-like material. Occasionally this material is extruding out of the arteriolar walls into the interstitium causing his interstitial inflammation. His renal function began to improve following his biopsy and 16 months post angiography has returned to normal.

**Conclusions:** This is the first case report that demonstrates microvascular embolization of a polymer coating following coronary angiography into the renal vasculature with associated interstitial inflammation.

**REVERSIBLE WORSENING OF KIDNEY FUNCTION WITH HIGH PROTEIN DIETS: A CASE REPORT IN A PATIENT WITH CHRONIC KIDNEY DISEASE**

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**Background:** Deleterious effects of a high protein intake in chronic kidney disease (CKD) are well known, as are the acute increases in creatinine in those without kidney disease who consume certain protein supplements, especially those containing creatine esters. We report a patient with stable CKD stage 3 who presented with an acute kidney decline after taking large amounts of protein supplements.

**Case Report:** A 55-year-old male diabetic and hypertensive patient with a horseshoe kidney, not previously known to the renal service, presented with an unexplained abrupt rise in creatinine from his baseline of 130 to 176 μmol/L. In the month prior to attempting to lose weight, he had started a high protein supplement four times daily (an additional 112 g of whey and milk protein) along with a predominantly meat-based diet. He had no recent medication changes or known nephrotoxic insults. Urine was unremarkable. A glomerulonephritis screen was negative. Renal ultrasound revealed a horseshoe kidney with no other pathology. His new protein supplements were ceased. This resulted in an immediate, sustained reduction of creatinine to baseline levels. He was advised to continue a low-protein diet, and kidney function remained stable 4 months afterwards.

**Conclusion:** Our patient had a rise in creatinine temporally associated with a significant increase in protein intake, even though this was mainly whey protein, which has negligible creatine. A high protein diet is known to cause acute haemodynamic changes. This change was poorly tolerated in our patient with pre-existing CKD. The rapid recovery to baseline after withdrawal of supplements supports this hypothesis. This case illustrates the risk of acute renal worsening with even short-term high protein diets in those with chronic kidney disease.
VEDOLIZUMAB INDUCED ACUTE INTERSTITIAL NEPHRITIS: A CASE OF FAILED STEROID PROPHYLAXIS

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Background: Acute interstitial nephritis (AIN) is a frequent cause of acute kidney injury (AKI) and is commonly drug induced. Vedolizumab, a monoclonal antibody used in the treatment of refractory ulcerative colitis (UC), has been reported to cause AIN. We describe the first case of vedolizumab induced AIN in which pre-dose steroid prophylaxis failed to prevent a recurrence of AIN on rechallenging with vedolizumab.

Case Report: A 21-year-old male with a history of refractory UC presented with a history of malaise, fevers, and nausea. He had commenced vedolizumab 6 months prior and recently received his 3rd dose. Initial investigations showed pyuria (>500 leucocytes x106), AKI (serum creatinine 133 μmol/L, ref range: 60–100 μmol/L) and bilateral renal echogenicity. Following initial improvement with antibiotics and fluids (serum creatinine 100 μmol/L) he was discharged. Following a further dose of vedolizumab his renal function worsened (serum creatinine 185 μmol/L) and he underwent kidney biopsy. This demonstrated AIN with a 50% lymphocyte rich interstitial infiltrate. Vedolizumab was withheld and renal function spontaneously improved (serum creatinine 138 μmol/L). In view of his refractory UC and limited treatment options, vedolizumab was re-tried with high dose prednisone prophylaxis. This was unsuccessful, with further deterioration in his renal function (serum creatinine 186 μmol/L) requiring a prolonged course of high dose prednisone to treat which was weaned over 4 months. 12 months after cessation of vedolizumab his renal function remained impaired (serum creatinine 108 μmol/L).

Conclusions: We present the first case of vedolizumab induced AIN with failed steroid prophylaxis. This case highlights the importance of balancing the risk and benefits of continuing an effective treatment for a refractory disease when AIN is a complication of treatment.

AUDIT OF THE IDENTIFICATION AND MANAGEMENT OF METABOLIC ACIDOSIS IN A TERTIARY HOSPITAL KIDNEY UNIT

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Aim: To describe the identification and initial management of metabolic acidosis (MA) in an inpatient kidney service.

Background: There are few management guidelines for clinicians to follow for diagnosis and management of MA. Arterial blood gas (ABG) analysis is considered the gold-standard for diagnosing MA, but increasingly, clinicians use venous blood gas (VBG).

Methods: We retrospectively audited the inpatient management of MA in patients discharged from the Concord Hospital Kidney Department from June 2019 to June 2021. Patients were divided into groups: Severe-MA (SMA), pH <7.2 or serum bicarbonate (sHCO3) <15 mmol/L; Non-Severe-MA (NSMA), pH 7.2–7.34 or sHCO3 15–22 mmol/L; non-MA.

Clinical, laboratory and management data were collected from the hospital electronic medical records (eMR) under appropriate ethics approval.

Results: From 540 patients reviewed, 30 had results consistent with SMA and 40 NSMA. ABG utilization decreased as MA severity decreased (30% SMA, 12.5% NSMA). VBG use was higher for both groups (43.3% SMA, 40% NSMA). The remainder (26.7% SMA, 47.5% NSMA) were diagnosed purely on sHCO3. Considering initial management, nine patients were excluded as receiving kidney replacement therapy (5 SMA, 4 NSMA). 83.3% of SMA patients diagnosed using either an ABG or VBG received sodium bicarbonate (NaHCO3) compared to 42.9% diagnosed using sHCO3.

80% of NSMA patients diagnosed using an ABG received NaHCO3 compared to 50% diagnosed using a VBG, and 35.3% diagnosed using sHCO3.

Conclusions: Whilst the literature confirms strong correlation between blood pH from ABGs and VBGs in certain populations, a lack of standardized approach to blood pH estimation may lead to differences in MA management.

TRABECTEDIN CAUSING SEVERE RHABDOMYOLYSIS LEADING TO OLIGO-ANURIC ACUTE KIDNEY INJURY REQUIRING HAEMODIALYSIS

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Background: Trabectedin is an alkylating chemotherapy agent used as a second line treatment for unresectable or metastatic sarcoma. Rhabdomyolysis has been reported as a known but rare side effect of trabectedin. Rhabdomyolysis is the clinical syndrome resulting from elevated creatine kinase (CK) as a consequence of skeletal muscle injury, with clinical features ranging from benign myalgias to multi-organ failure including acute kidney injury (AKI).

Case Report: This case involves a 67-year-old male first diagnosed with myxoid liposarcoma in 2010. He was initially managed with surgical excision with adjuvant chemotherapy but was subsequently commenced on trabectedin due to disease progression in 2022. One month later, features of acute cholecystitis were incidentally noted on
a staging computer topography scan resulting in referral to the emergency department. Admission bloods demonstrated pancytopenia, a mixed liver injury and an AKI with a creatinine of 325 μmol/L (60–110 μmol/L), from a normal baseline. Trabectedin was ceased. He was initially treated with empiric antibiotics but despite this developed a progressive oliguric AKI. In investigating the underlying aetiology of the AKI, rhabdomyolysis secondary to trabectedin was queried and subsequently confirmed with a CK of 21,166 U/L. He failed to improve despite aggressive fluid resuscitation and required haemodialysis. He remained dialysis dependent for 1 month prior to renal recovery. His current creatinine is 127 μmol/L. 

Conclusions: Rhabdomyolysis is an important but rare and idiosyncratic side effect of trabectedin, with cases of severe disease resulting in anuric AKI requiring haemodialysis being exceedingly rare. Prompt recognition is imperative to prevent potentially irreversible consequences. This case highlights the need for judicious monitoring of CK levels prior to each cycle of trabectedin.

A CASE SERIES OF PATIENTS WITH CKD PRESENTING TO LIVERPOOL HOSPITAL

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Introduction: Diabetic myonecrosis is a rare microvascular complication of diabetes. It presents with acute or subacute pain and swelling to the affected limb secondary to ischaemic necrosis of skeletal muscle. It is commonly associated with other microangiopathic complications of diabetes including nephropathy, retinopathy and neuropathy. The most common site of involvement is the thigh, followed by the distal lower limb.

Aims: To describe a case series of patients presenting with diabetic myonecrosis to our tertiary centre.

Methods: A total of five patients were identified from renal department consultant records from the Renal Unit records with diabetic myonecrosis from 2017 to 2022. Patient demographics and details regarding diabetes and control (Hba1c), associated complications and region of diabetic myonecrosis was gathered.

Results: All five patients had type 2 diabetes mellitus with a Hba1c between 6–8% however had evidence of previously poor control with Hba1c levels greater than 10%. Two patients were on haemodialysis, one on peritoneal dialysis, one was a renal transplant recipient and the other a stage IV CKD patient. The most common complications of diabetes in addition to nephropathy were peripheral vascular disease (3 out of 5), and retinopathy (2 out of 5). Three patients were on insulin therapy in addition to oral glucose lowering therapy. 4 out of the 5 patients presented with acute/subacute unilateral thigh pain. Creatine kinase levels were only modestly elevated and only one patient had a muscle histology consistent with myonecrosis.

Conclusions: Patients had features of previously poorly controlled diabetes with significantly elevated Hba1c and other microvascular disease. Nearly all cases presented as unilateral thigh pain and creatine kinase as well as muscle biopsy were often non-contributory to the diagnosis.

BACKGROUND ON Nephrology—Diabetes/Cardiovascular/Hypertension

DIABETIC MYONECROSIS—A CASE SERIES OF PATIENTS WITH CKD PRESENTING TO LIVERPOOL HOSPITAL

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Background: Idiopathic nodular glomerulosclerosis (ING) is a rare condition typically seen in elderly Caucasian males and is associated with hypertension, smoking, obesity and hypercholesterolaemia. It classically presents with renal dysfunction and nephrotic range proteinuria. Histologically it’s virtually indistinguishable from diabetic glomerulosclerosis and exclusion of diabetes mellitus is required. We present an unusual case of ING with no history of smoking and rapid progression of renal impairment despite a paucity of predictors for end-stage disease.

Case Report: An 89-year-old man with a longstanding history of untreated hypertension and hypercholesterolaemia was referred for investigation of declining renal function over a 3-year period. He was subsequently found to have microscopic haematuria and nephrotic-range proteinuria. The blood pressure was 200/85 mmHg despite Telmisartan 80 mg and Indapamide 1.5 mg daily. Body mass index was 31.5 kg/m².

Investigations showed a serum creatinine (sCr) 193 μmol/L and estimated glomerular filtration rate (eGFR) 25 ml/min, from sCr 137 μmol/L, eGFR 39 ml/min 7 months prior. The 24-hour urinary protein excretion was 3596 mg. Computed tomography-intravenous pyelogram to investigate microhaematuria was normal. Autoimmune screen and serum electrophoresis were unremarkable. The haemoglobin A1c was 5.4% with a fasting blood sugar level of 4.8 mmol/L.

Thirteen glomeruli were seen on biopsy, 4 were globally sclerosed. Light microscopy showed nodular Periodic acid-Schiff positive expansion of mesangium with peripherally displaced nuclei—Kimmelstiel Wilson-like nodules. Moderate interstitial fibrosis and tubular atrophy was present. Immunofluorescence was negative. Electron microscopy showed thickened glomerular basement membranes with mild foot process effacement and mild arteriosclerosis.

Conclusions: ING is an uncommon condition; it is important that clinicians can recognize it and manage modifiable risk factors for progression. This atypical case of ING contributes to our limited knowledge of the disorder.
PRESCRIPTION OF SGLT2 INHIBITORS TO PATIENTS WITH CHRONIC KIDNEY DISEASE: A QUALITATIVE STUDY OF NEPHROLOGISTS AND CARDIOLOGISTS

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Aim: To explore with Nephrologists and Cardiologists their perspectives regarding prescribing SGLT2 inhibitors (SGLT2i) to patients with chronic kidney disease (CKD).

Background: SGLT2i have reno- and cardioprotective benefits in people with CKD independent of their anti-hyperglycaemic effects.

Methods: Semi-structured interviews were conducted with 12 Nephrologists and 12 Cardiologists working in diverse areas in New South Wales. Purposive and snowball approaches were used to recruit participants. Emergent themes were identified from transcripts. Interviews were conducted until thematic saturation was reached.

Results: Nephrologists were generally comfortable prescribing SGLT2i to patients with diabetes and CKD. If a patient was under the care of an Endocrinologist, many Nephrologists preferred to check the prescription with the Endocrinologist prior to initiation. Whilst several Nephrologists had prescribed SGLT2i to patients without diabetes, the lack of a Pharmaceutical Benefits Scheme (PBS) CKD indication for SGLT2i was a major barrier to prescribing SGLT2i to this patient group. There was variability among Nephrologists regarding the minimum estimated glomerular filtration rate that is acceptable for commencing SGLT2i, and the timing of renal function monitoring after SGLT2i initiation in patients with CKD. This latter point, in part, reflected differences in concern about the risk of volume depletion. Non-Heart Failure Subspecialist Cardiologists were less comfortable compared with Nephrologists prescribing SGLT2i to patients with advanced CKD.

Conclusions: The lack of a PBS CKD indication is an important factor preventing Nephrologists prescribing SGLT2i to patients without diabetes. Nephrologists have a role in reassuring Cardiologists and other specialists about prescribing SGLT2i to patients with more advanced CKD. Additionally, there is a need for clear, specific guidance about prescribing SGLT2i in patients with renal dysfunction and renal function monitoring after SGLT2i initiation.

RECRUTMENT FOR A DOUBLE-BLIND, RANDOMIZED, PLACEBO-CONTROLLED TRIAL TO DETERMINE THE EFFECT OF BEETROOT JUICE ON REDUCING BLOOD PRESSURE IN AUTOSOMAL DOMINANT POLYCYSTIC KIDNEY DISEASE (BEET-PKD)

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Aim: To evaluate the efficacy of beetroot juice (BRJ) on reducing blood pressure (BP) in hypertensive adults with autosomal dominant polycystic kidney disease (ADPKD).

Background: Impaired endothelial-derived nitric oxide (NO) synthesis due to dysfunction of polycystin-1 is a key contributor to the pathogenesis of hypertension in ADPKD. In previous studies of chronic kidney disease, BRJ increased plasma NO, reduced BP and improved endothelial function.

Methods: In this single-centre, investigator-initiated, randomized, double-blind, placebo-controlled study (Western Sydney Local Health District Human Research Ethics Committee approval 2020_ETH01718), ADPKD patients (n = 60; ages 17–80 years, eGFR>30 ml/min/1.73 m² and treated with at least one anti-hypertensive drug) will be allocated 1:1 to either 70 ml of nitrate-rich (400 mg nitrate/dose) or nitrate-depleted BRJ daily for 4 weeks. The primary outcome is change in the clinic BP from baseline. Secondary outcomes are changes in daily home BP, urinary albumin to creatinine ratio, serum NO metabolites and serum asymmetric dimethylarginine from baseline. Adherence will be monitored by responses to daily text messages with home BP values.

Results: Recruitment commenced in May 2022 and participants were identified from a PKD Database and direct communication with nephrologists. To date, 112 participants have been identified and 35 were eligible. Of these, 11 did not respond to invitation, 5 were excluded for other reasons and 19 were recruited. Ten participants have completed screening and 4 have been randomized. Based on the current recruitment rate of 4.5 patients/week, we expect that the trial will be completed in October 2022.

Conclusions: The good recruitment in this study suggests that people with ADPKD may favour participation in short-term clinical trial interventions perceived as having minimal adverse effects and endorsed by their nephrologist.
OUTCOMES OF TRANSCATHETER AORTIC VALVE REPLACEMENT (TAVR) IN PATIENTS RECEIVING CHRONIC DIALYSIS IN AUSTRALIA AND NEW ZEALAND USING DATA-LINKAGE

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ABSTRACT

Background: TAVR is increasingly being considered as an alternative to surgical aortic valve replacement in patients on chronic dialysis who are considered high operative risk. This bi-national data-linkage study describes the clinical outcomes following TAVR between 2008 and 2015.

Methods: Data-linkage between Australia and New Zealand Dialysis and Transplant (ANZDATA) Registry and jurisdictional hospital admission datasets was used to evaluate outcomes for all incident and prevalent patients receiving chronic dialysis between 2008 and 2015 who underwent a TAVR as defined by the Australian Classification of Health Interventions. Descriptive statistics were used to describe baseline characteristics and outcomes.

Results: Nineteen in-centre haemodialysis patients underwent TAVR with a median follow-up of 1.5 yrs [1.1–2.4], 14(74%) were male, median age, dialysis vintage and BMI were 76.4 yrs [70.4–80.2], 4.4 yrs [1.4–8.4] and 27.3 kg/m² [21.6–28.7], respectively. No patients were on peritoneal or home-haemodialysis, and no procedures were performed in New Zealand. Twelve of the procedures occurred in 2015. No patients had undergone previous balloon aortic valvuloplasty or cardiac surgery. The prevalence of ischaemic heart disease, diabetes mellitus, congestive cardiac failure and cerebrovascular disease were 63%, 89%, 32% and 16%, respectively. The median length of stay was 8 days [IQR4-15]. An intensive care admission was required in 14(74%) patients with a median stay of 45 hours [IQR27-51]. Thirty-day mortality was 5.3%. There were no perioperative myocardial infarctions, a single patient had a stroke. Seven patients (36.8%) required blood transfusions and two patients (11%) required a permanent pacemaker within 30-days. The 1-year mortality was 21.1% (n = 4).

Conclusions: TAVR was an uncommon but increasingly utilized procedure in patients receiving chronic dialysis and was associated with reasonable outcomes. Data linkage provides an efficient way of assessing utilization of surgeries and outcomes in this population.

STAGED RESECTION OF SUSPECTED BILATERAL PHAEOCHROMOCYTOMA IN A PATIENT WITH END-STAGE KIDNEY DISEASE ON HOME HAEMODIALYSIS

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Background: Phaeochromocytoma is a rare finding in the dialysis population. The usual complexities of its management are complicated by diagnostic considerations of anuria and catecholamine variability in relation to dialysis, as well as reduced margins of error in perioperative fluid balance. We describe the management of a haemodialysis patient with resistant hypertension secondary to suspected bilateral phaeochromocytoma. We report on the surgical outcomes of the initial staged resection as well as the effect of dialysis timing (pre- and post-dialysis) on laboratory plasma metanephrine measurements.

Case Report: A 73-year-old male with a background of hypertensive nephrosclerosis and home haemodialysis, presents with resistant hypertension. CT imaging for back pain incidentally revealed bilateral adrenal gland lesions, measuring 5.5 cm (Left) and 1.4 cm (Right). Subsequent PET Gallium Dotatate study was consistent with bilateral neuroendocrine tumours, favoured to represent phaeochromocytomas. Plasma normetanephrine levels were concordant at 6600 pmol/L (Ref <1079 pmol/L). Maximum alpha-receptor blockade was achieved with high-dose phenoxybenzamine (300 mg/day), followed by propranolol and nifedipine to attain optimal pre-operative blood pressure. Pre-operative fluid status optimization (via haemodialysis ultrafiltration) balanced the risk of intraoperative hypotension with anaesthetic considerations of potential pulmonary oedema.

Staged laparoscopic resection of the left adrenal lesion confirmed the diagnosis of phaeochromocytoma on histopathology. Intraoperative blood pressures were maintained with minimal vasopressor support, and postoperatively with no support, suggestive of suspected bilateral phaeochromocytomas. A right adrenalectomy is planned in 2 months.

Conclusion: We report a case of suspected bilateral phaeochromocytoma in a patient on home haemodialysis. Our case highlights the complexities of diagnosis and management of phaeochromocytoma in this high-risk population, and we demonstrate staged resection of bilateral lesions can be a safe approach.

GENERAL NEPHROLOGY—EPIDEMIOLOGY AND PUBLIC HEALTH

BEYOND ENDEMIC CHRONIC KIDNEY DISEASE(CKDU)—THE LANDSCAPE OF CKD IN SRI LANKA

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Aim: To identify the burden of CKD beyond CKDu in Sri Lanka. 

Background: CKD epidemiological patterns vary significantly across the globe. Understanding local patterns is crucial for prevention, early diagnosis, improved outcomes and optimum resource allocation. Sri Lanka is experiencing a rise in non-communicable diseases, including CKD. Media and geo-political institutions are focusing on CKDu in the region, overlooking the potentially bigger problems of CKD driven by particularly diabetes and hypertension. Rightly so CKDu has been heavily investigated. It is limited to certain regions in the country where its prevalence, even in the highest risk areas, is only 2.44%–4.35% of the population (Ministry of Health 2019 estimates). Very little is known about the rest of the country.

Methods: A pilot study will be conducted over 2 years in the Kandy district, using a three-armed mixed method design. Kandy is a non-CKDu endemic region, with approximate population of 1.5 million. Study arm one will establish a regional CKD registry and study CKD related disease patterns. Study arm two will describe community rates of CKD through the conduct of a prevalence study. Study arm three will profile prevalence of CKD within nephrology and non-nephrology medical settings in a selected group of nationally representative hospitals through a standard survey questionnaire of specialists.

Conclusions: This would be the first representative profile of all CKD in Sri Lanka. It will describe the burden of CKD and its load within the health system in Sri Lanka, giving a perspective of CKDu in the burden. The pilot is proof of concept of greater precision mapping of CKD and its resource utilization across the country.

AWARENESS OF RISK FACTORS FOR KIDNEY DISEASE AMONG AN URBAN POPULATION IN SRI LANKA

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Aim: Identify awareness of risk factors for kidney disease among public in an urban region in Sri Lanka.

Background: Awareness of common risk factors of kidney disease such as diabetes and hypertension among the general public leads to prevention, early detection and improved outcomes. Over the last three decades significant media and geo-political attention has been given to CKD of uncertain aetiology (CKDu) in Sri Lanka that public awareness has improved but skewed towards CKDu.

Methods: This study was undertaken as part of a CKD prevalence study. The sample was selected through a stratified random sampling method from urban Kandy District in Sri Lanka which is not a CKDu endemic region. Five screening questions were asked to evaluate participants’ understanding of risk factors for kidney disease.

Results: About 265 participants were studied. Mean age was 54.9 years (SD15.3). 55% were females, 86.7% were of Sinhalese ethnicity and 89.6% were educated beyond primary school. 28.3% and 29.5% had diagnosed diabetes and hypertension respectively. 49.4% stated that they are aware of risk factors for kidney disease, whereas 29.4% did not know and 21.1% were unsure. The majority (55%) stated water was the main risk factor followed by drug induced injury (17.3%) and diabetes (13.9%). On direct questioning 65% identified diabetes but only 31.3% identified hypertension as a risk factor.

Conclusions: Majority of participants identified water as the main risk factor for kidney disease in Sri Lanka. Attention given to CKDu has detracted the public’s awareness of the more common risk factors such as diabetes and hypertension. This highlights the need for increasing awareness among the public regarding the main risk factors for kidney disease.

GENERAL NEPHROLOGY—GLOMERULONEPHRITIS

IGG4 RELATED KIDNEY DISEASE: SPECIFIC RADIOLOGIC AND PATHOLOGIC FINDINGS

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Background: IgG4-related disease is a multi-system immune disorder characterized by formation of fibro-inflammatory lesions, commonly involving the pancreas, retroperitoneum, lymph nodes, salivary glands and kidneys. Diagnosis can be challenging, as clinical presentation and biochemical and radiologic investigations may be non-specific. We report a case of biopsy proven IgG4-related kidney disease with hallmark biochemical, radiologic and pathologic features.

Case Report: A 56-year-old man with IgG4-related disease was referred for investigation of worsening kidney function. IgG4-related disease was diagnosed 3 years earlier, following pancreatitis. Investigations demonstrated a serum creatinine (sCr) of 110umol/L, estimated glomerular filtration rate (eGFR) 63 ml/min. Twelve months
before, sCr was 65umol/L, eGFR>90 ml/min. Total serum IgG and IgG4 concentrations were markedly elevated at 41.58 g/L and 10.56 g/L, respectively. The C3 and C4 were low at 0.82 g/L and 0.10 g/L. Urine sediment was bland and 24-hour urine revealed 0.29 g of proteinuria.

Contrast computed tomography demonstrated enlarged kidneys with rounded hypodense lesions in the cortex. Magnetic resonance imaging identified regions of severe diffusion restriction in both kidneys. Positron emission tomography demonstrated normal kidneys and no regions of abnormal glucose metabolism. Kidney biopsy confirmed IgG4-related disease, with storiform fibrosis and lymphoplasmacytic infiltrate, with large numbers of IgG and IgG4 positive cells. Treatment with oral prednisolone 40 mg (0.6 mg/kg) resulted in immediate improvement in kidney function (sCr 92 umol/L, eGFR 79 ml/min) and total IgG and IgG4 concentrations (13 g/L and 2.0 g/L respectively).

Conclusion: We present a case of biopsy proven IgG4-related kidney disease demonstrating characteristic radiologic and pathologic appearances, which may not correlate with disease severity. It is important that clinicians recognize these features on imaging and pathology, and consider IgG4-related disease as a possible diagnosis.

THE OUTCOMES OF PATIENTS WITH KIDNEY FAILURE SECONDARY TO FOCAL SEGMENTAL GLomerulosclerosis (FSGS) IN AUSTRALIA AND NEW ZEALAND: AN ANZDATA STUDY

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Aim: To evaluate the characteristics, treatments, and outcomes of all patients with kidney failure due to FSGS in the Australian and New Zealand dialysis populations, using data from the ANZDATA Registry.

Background: The outcomes of patients with FSGS on kidney replacement therapy (KRT) have not been well described.

Methods: All adult patients with kidney failure who commenced KRT in Australia and New Zealand from 15th of May 1963 to 31st of December 2018 were retrospectively extracted from the ANZDATA Registry. Outcomes of patients with FSGS were compared to those with other causes of kidney failure (non-FSGS).

Results: 85052 patients commenced KRT during the study period, of whom 2991 (3.5%) were patients with FSGS. Compared to patients with non-FSGS, patients with FSGS experienced similar mortality on dialysis (adjusted hazard ratio [aHR] 0.98, 95% CI 0.90–1.06, p = 0.55) and following kidney transplantation (aHR 0.92, 95% CI 0.73–1.15, p = 0.466). The risk of first kidney allograft loss was higher in patients with FSGS (aHR 1.20, 95% CI 1.04–1.37, p = 0.010). However, when death was analysed as a competing risk, the survival in both groups was similar (sub-hazard ratio [SHR] 1.09, 95% CI 0.94–1.28, p = 0.26). Patients with FSGS had a longer waiting time for kidney transplantation (aHR 0.92, 95% CI 0.86–0.98, p = 0.015) and experienced an increased risk of disease recurrence in the allograft (aHR 1.73, 95% CI 1.35–2.21, p < 0.001). Compared to patients with other forms of glomerular disease, patients with FSGS experienced similar dialysis and transplant patient survival and death-censored rate of kidney transplantation and allograft loss but higher rates of primary kidney disease recurrence.

Conclusions: FSGS was associated with similar dialysis and transplant patient survival and death-censored first allograft loss compared to non-FSGS and other forms of glomerular disease.

ANTINEUTROPHIL CYTOPLASMIC ANTIBODY (ANCA) ASSOCIATED GLOMERULONEPHRITIS AND LUNG CANCER: DISEASE RESPONSE TO NOVEL IMMUNOCHEMOTHERAPY

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Background: Antineutrophil Cytoplasmic Antibody (ANCA) associated glomerulonephritis presenting with lung cancer has been reported in limited number of cases. Despite growing evidence of safety of immunotherapy in autoimmune diseases, there is no evidence to suggest this in ANCA associated glomerulonephritis. We here present a case of renal limited ANCA associated glomerulonephritis concurrently diagnosed with lung cancer managed with chemoimmunotherapy.

Case Report: A 74-year-old man presented with an acute kidney injury with eGFR of 8 ml/min², creatinine of 585 mmol/L and urea of 38.7 mmol/L requiring haemodialysis. His urine protein creatinine ratio was 426 g/mol of creatinine and urine microscopy showed >500 x 10⁶/L of leukocytes and dysmorphic erythrocytes. Investigations revealed elevated MPO-ANCA titres at 22 CU with negative anti-GBM, and renal biopsy confirmed the diagnosis of pauci-immune crescentic necrotising glomerulonephritis. He was started on IV methylprednisolone and cyclophosphamide with no signs of renal recovery. Incidentally, a computer tomography (CT) scan of the chest showed a lung lesion and further investigations revealed a stage IIib (cT4N1M0) squamous cell carcinoma of the lung. Subsequently, immunosuppressant treatment was ceased and chemoimmunotherapy (carboplatin, paclitaxel and pembrolizumab) was initiated to control the progression of the cancer while achieving sustained AAV remission. At 7-months post diagnosis, PR3-ANCA titres increased to 47 CU with negative MPO/C-ANCA without evidence of tumour growth or vasculitis. Subsequently, PR3-ANCA titres decreased further whilst the tumour increased in size following aggressive cancer treatment with second line agent.

Conclusion: When ANCA associated glomerulonephritis presents with lung cancer targeting the cancer first, while interrupting immunosuppression, can result in remission of cancer associated glomerulonephritis.
Our case also adds to the literature evidence concerning the safety of immunotherapy in ANCA associated glomerulonephritis.

**EPIDEMIOLOGY, CLINICOPATHOLOGICAL CORRELATION AND PROGNOSIS OF PATIENTS WITH FIBRILLARY AND IMMUNOTACTOID GLOMERULOPATHIES IN THE HUNTER REGION**

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**Background:** Fibrillary and immunotactoid glomerulonephritis are uncommon. Fibrillary glomerulonephritis (FGN) is often renal-limited, whereas immunotactoid glomerulopathy (IT) is more associated with haematological disorders. There is limited literature on the natural history of these glomerulopathies in Australia.

**Aims:** We aim to study the prevalence, clinicopathological correlation and outcomes of FGN and IT in our regional centre.

**Methods:** A local renal biopsy database was interrogated for cases of fibrillary and immunotactoid glomerulopathies from 2000 to 2020. Data collected includes demographics, serum creatinine, haematuria status, proteinuria, comorbidities, and histopathological findings.

**Results:** Out of 4083 native and transplant kidney biopsies, we had 14 cases of FGN (biopsy incidence 0.34%) and 2 cases of IT (biopsy incidence 0.04%). The mean age at presentation was 59.8 yrs. 42.9% were males. No patients with FGN had dysproteinemia, whereas both patients with IT had chronic lymphocytic leukaemia. 75% of patients with FGN and all patients with IT had haematuria. All patients had proteinuria. Mean presenting albuminuria was 254 mg/mmol for FGN and 604 mg/mmol for IT. Mean presenting serum creatinine and eGFR were 149 μmol/L and 48 ml/min respectively for FGN and 95 μmol/L and 72 ml/min for IT. The prognosis of FGN was poor with limited response to immunomodulatory therapy; six patients (46.2%) reached end-stage kidney disease (ESKD) after a median of 42 months (1-96 months). High-grade presenting proteinuria led to poor renal outcomes. Patients with IT had disease remission with treatment of their underlying haematological disease.

**Conclusions:** FGN is rare, with poor response to immunomodulatory therapy. It carries poor renal prognosis. Less proteinuria at diagnosis may predict a more benign disease course. IT is associated with haematological malignancy and carries better prognosis and response to treatment.

**MIXED IGA NEPHROPATHY AND MINIMAL CHANGE DISEASE**

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**Background:** IgA nephropathy with minimal change disease is a rare clinical entity in adults, with distinct clinicopathological features and unclear prognostic implications. Treatment involves pharmacotherapy directed towards minimal change disease. We report a case of mixed IgA nephropathy and minimal change disease, with characteristic clinical and pathological features of a dual glomerulopathy.

**Case Report:** A 41 year old gentleman was referred for investigation of new onset nephrotic syndrome. Background history was significant for psoriatic arthritis treated with certolizumab. On presentation, he was grossly oedematous, with evidence of ascites and pulmonary oedema. Blood pressure was 130/80 mmHg and there had been 5 kg weight gain over 2 weeks.

Investigations demonstrated serum creatinine (sCr) of 60 μmol/L, estimated glomerular filtration rate (eGFR) > 90 ml/min. Serum albumin was 17 g/L and total cholesterol 12.1 mmol/L. A 24-h urine protein was 17 g and protein creatinine ratio (PCR) was 1739 mg/mmol. Urine sediment demonstrated microhaematuria and 75% dysmorphic red cells. Kidney biopsy demonstrated increased mesangial cellularity with one fibrocellular crescent. Immunofluorescence was significant for IgA and lambda immune deposits. There was diffuse (approximately 80%) effacement of the podocyte foot processes on electron microscopy. Treatment with oral prednisone, 50 mg, resulted in reduced urine PCR to 12 mg/mmol. Serum albumin and total cholesterol normalized to 38 g/L and 3.2 mmol/L respectively. Urine microscopy normalized with no haematuria. The patient continues on 10 mg prednisone daily and remains in remission.

**Conclusion:** We present a case of biopsy proven concurrent IgA nephropathy and minimal change disease manifesting as florid nephrotic syndrome. Treatment is clear and directed towards minimal change disease, but prognostic implications including risk of relapse and progression to end-stage kidney disease remain unclear.

**A CASE OF GOODPASTURE DISEASE COMPLICATED BY COVID-19 INFECTION**

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**Background:** COVID-19-era has complicated the management of glomerulonephritis as both COVID-19 infection and its vaccines have been associated with de novo disease and relapses.

**Case Report:** We report a case of a 20-year-old Caucasian male, a diesel mechanic and a smoker who presented two-weeks after second dose of AstraZeneca vaccine with fever, cough and haemoptysis. Investigations included a CT-chest, showing pulmonary haemorrhage, positive Anti-GBM-antibody at 5.8, micro-haematuria and normal Creatinine at 75. A diagnosis of Goodpasture-disease was made and he was treated with pulsed Methylprednisolone (PM) and oral Cyclophosphamide. Plasmapheresis was considered but not given as
clinically well following PM. Repeat HRCT within four-weeks showed complete resolution of haemorrhage and down-trending antibodies at 4.3. Six weeks into treatment, he developed COVID-19 infection with transient macro-haematuria. Over the next month his antibody levels gradually rose to 7.7 with micro-haematuria but normal creatinine. A subsequent renal biopsy confirmed Goodpastures, PM, IV Cyclophosphamide and two doses of Rituximab. Three months after second induction and more than 30 plasmapheresis sessions, his antibody remained positive at 2.3 with normal creatinine, ongoing micro-haematuria and suppressed CD19. He was switched to Mycophenolate with ongoing prednisolone.

**Conclusions:** Our patient's refractory disease could be explained by COVID19 infection and ongoing exposure to hydrocarbons. Initial COVID-19 vaccine may have played a role, but there has been no case reports associating AstraZeneca vaccine to Goodpasture-disease. Evidence on management of refractory disease is limited, but Rituximab and mycophenolate have been used in several case reports and it can take up to 10 months for full clinical and immunological remission.

**A NOVEL CASE OF PROLIFERATIVE GLOMERULONEPHRITIS WITH MONOCLONAL IGG DEPOSITS (PGNMID) AND STRONG LINEAR STAINING OF GLOMERULAR CAPILLARY WALL**

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**Background:** Immunofluorescence (IF) in PGNMID most commonly demonstrates a granular texture of deposits along the glomerular capillary wall (GCW). Less frequently, semi-linear and smudgy appearances have also been reported. Here, we report a novel case of PGNMID with strong linear staining of the GCW with IgG kappa.

**Case Report:** 51-year-old male presented with a three-day history of dyspnoea, orthopnoea and oliguria. He had a 20 pack-year smoking history. He was hypertensive 206/104 mmHg and clinically volume overloaded. Biochemistry showed a serum creatinine of 504 umol/L, albumin of 29 g/L, urine protein-creatinine-ratio (U-PCR) of 557 g/mol with microscopic haematuria. Glomerulonephritis screen including anti-GBM was negative. A low C3 was noted—0.39 g/L [0.90–1.80] and normal C4. Worsening biochemistry and refractory fluid overload rendered the patient to haemodialysis. Light microscopy demonstrated diffuse endocapillary and mesangial hypercellularity, and <10% interstitial fibrosis. Immunofluorescence demonstrated heavy linear IgG and kappa deposition along the GCW, and granular deposition in the mesangium, with sparing of the tubular basement membrane. IF for lambda was negative. Electron microscopy showed amorphous deposits within the mesangium and subendothelial regions, with severe foot process effacement. Serum protein electrophoresis, serum free light chain ratio, flow cytometry on peripheral blood, and a bone marrow aspirate and trephine failed to identify a plasma cell clone. He was treated with 2 × 750 mg doses of rituximab 2 weeks apart, in addition to a weaning course of oral prednisolone. One month later, the patient became independent of dialysis.

**Conclusions:** This unique case shows that PGNMID can also present with a strong linear pattern. Notably, his anti-GBM serology and tests for diabetes were negative. Early recognition and treatment may confer a favourable response.

**ETHNICITY AND GLOMERULAR DISEASE: THE REPRESENTATION BIAS IN PUBLISHED RANDOMIZED CONTROL TRIALS**

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**Aim:** To examine the representation of ethnicity within randomized control trials (RCTs) on the management of glomerular disease.

**Background:** Ethnicity is a key determinant of clinical progression and outcomes of glomerular disease. However, the detail reporting of ethnicity in RCTs is unknown.

**Methods:** We conducted a meta-epidemiological study of RCTs on the management of glomerular disease. All RCTs identified in the KDIGO - Glomerular Diseases Guidelines published in 2021 were included. Dual data extraction was undertaken for reporting of ethnicity, subgroups undertaken and presence of effect modification. Differences in ethnicity reporting were analysed using a Chi-squared test or Fisher exact test. Linear association between the year of publication, locations, ethnicities, outcomes, ethnicity and ethnic groups was assessed using a Chi-squared test for linear-by-linear association.

**Results:** Overall, 500 RCTs were included, 88 (18%) trials reported ethnicity which increased over time (p = 0.016). Most trials were conducted in Asia (n = 212, 42%), however, trials conducted in North America or multinational RCTs were more likely to report ethnicity.

**Conclusions:** A minority of RCTs on glomerular disease reported ethnicity. Although the reporting of ethnicity has improved, less than half of the studies conducted after 2010 have reported ethnicity. The lack of reporting on ethnicity may impact the generalisability of the findings of RCTs to clinical practice to improve health outcomes of people living with glomerular disease.
A case series of elevated myeloperoxidase (MPO) antibody titres without evidence of ANCA-associated vasculitis (AAV) on renal biopsy in patients recently vaccinated with the ChAdOx1-S (Oxford/Astra Zeneca) vaccine

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Background: Coronavirus disease-19 (COVID-19) vaccines have been temporally associated with both de-novo and relapsed glomerulonephritis. We present a series of three patients with de-novo elevated myeloperoxidase (MPO) antibodies within 3 months of receiving the second ChAdOx1-S (Oxford/Astra Zeneca) vaccine. None had features of ANCA-associated vasculitis (AAV) on renal biopsy.

Case Report: Three female patients between the ages of 61 and 73 years presented with impaired renal function, new haematoproteinuria, anaemia and hypertension within 3 months of receiving their second ChAdOx1-S vaccine. Two patients had known, but quiescent, IgA nephropathy and lupus nephritis respectively. The third patient had no prior renal history. Creatinine ranged from 150 to 630 μmol/L, with urine protein to creatinine ratio between 485 and 1140 mg/mmol. MPO antibody titres were measured at >222 international unit/ml (IU/ml), >222 IU/mL and >80 IU/mL. The remainder of the vasculitic screens were negative. All three renal biopsies had no features of AAV. Two cases with prior renal disease demonstrated relapse despite prior bland urinalysis. The third case demonstrated features of focal and segmental glomerulosclerosis. Immunosuppression was used in one case with minimal response. The patient later passed away from an infected perforated diverticulum. The remaining two patients were managed conservatively due to chronic damage.

Conclusion: There may be a temporal relationship between COVID-19 vaccines and the de-novo development of MPO antibodies without features of AAV on renal biopsy. This case series demonstrated the importance of renal biopsy, especially in those with co-existent renal disease for accurate diagnosis. The decision to treat is dependent on the presence of salvageable kidney tissue and must be balanced with the risk of immunosuppression especially in the elderly.

Successful treatment of type II cryoglobulinaemia causing acute kidney injury with plasma exchange and rituximab in setting of Sjogren’s syndrome

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Background: Cryoglobulinaemia is a systemic inflammatory condition characterized by immune complex-mediated small-to-medium-sized vasculitis. Type II cryoglobulinaemia can be mixed polyclonal and monoclonal secondary to Hepatitis C or connective-tissue diseases such as Sjogren’s syndrome. We describe a case of type 2 mixed cryoglobulinaemia associated with Sjogren’s syndrome leading to acute kidney injury (AKI) required haemodialysis which was successfully treated with plasma exchange and rituximab.

Case Report: 55-year-old female with Sjogren’s and CANVAS (Cerebellar Ataxia with Neuropathy and bilateral Vestibular Areflexia Syndrome) presented with 2-weeks of right pleuritic chest pain and cough. She was diagnosed and treated for community acquired pneumonia. She was hypertensive. Further investigations revealed acute kidney injury (creatinine 186 μmol/L), microscopic haematuria, and proteinuria (2.8 g/day), urinary Bence Jones protein, raised serum free light chain ratio (5.46), low complements and persistently positive ANA, RhF, Anti-SSA/SSB consistent with Sjogren’s. Cryoglobulins were undetectable.

She represented 4 weeks later with abdominal pain, worsening renal function, fluid overloaded and respiratory failure requiring intubation and haemofiltration. Repeat glomerulonephritis screen revealed detectable cryoglobulins with 1 g/L IgM-kappa paraprotein consistent with Type II Cryoglobulinaemia. She was commenced on high dose prednisolone, plasma exchange (seven sessions) and weekly rituximab (four doses). Renal function improved and she became dialysis independent. Renal biopsy performed after therapy showed endocapillary hypercellularity and focal interposition of capillary loop; there was no cryoglobulin or vasculitis features. Her kidney function and complements normalized. Cryoglobulins remained undetectable on maintenance mycophenolate and prednisolone 5 months later.

Conclusion: Type II cryoglobulinaemia should be considered as a cause of renal impairment in patients with Sjogren’s syndrome. Our case highlights an alternative treatment modality with plasma exchange and rituximab which can be effective in severe cryoglobulinemic glomerulonephritis/ vasculitis.

Kadcyla induced nephrotic syndrome

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Background: Newer cancer treatments which include targeted therapy and immunotherapy agents are being increasingly recognized to cause kidney injury. Kadcyla (Trastuzumab emtansine) is a HER2-targeted antibody-drug conjugate. In current practise Kadcyla is used in the treatment of Her 2 positive breast cancer (both early and metastatic stages). Renal adverse effects are not commonly noted side effects of this drug. We describe a case of nephrotic syndrome related to Kadcyla use.

Case: 37 year old European woman with metastatic breast cancer was referred to renal clinic with anasarca and nephrotic range proteinuria. She was treated with neoadjuvant chemotherapy and mastectomy after initial diagnosis in 2017 and palliative chemotherapy. In 2020
she developed confusion and diagnosed with brain metastasis. There was no other significant medical comorbidities. Baseline renal function and albumin prior to initiation of Kadcyla was normal. In March 2021 she commenced 3 weekly Kadcyla and 3 monthly Zoledronic acid infusion (briefly paused Kadcyla while receiving radiotherapy in August). On initial review in renal clinic in November 2021 she reported weight gain of 30 kilograms since starting treatment. She was noted to have developed adrenal insufficiency and diabetes. She had 12 gm proteinuria, serum albumin 22 g/dL and eGFR 90 ml/min. Further investigations for causes of nephrotic syndrome. Kidney biopsy showed normal light microscopy and foot process effacement on electron microscopy indicating minimal change disease. Kadcyla and Zoledronic acid were discontinued. An expectant approach was taken and proteinuria and anasarca improved with cessation.

Conclusion: The trend of proteinuria and hypoalbuminemia coincided with timing of Kadcyla infusion. Hence we conclude Kadcyla is the likely cause for nephrotic syndrome. There are case reports of nephrotic syndrome related to Kadcyla.

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Aim: To describe the clinical characteristics and outcomes of patients with biopsy-proven ANCA-associated glomerulonephritis at a regional health service in Victoria.

Background: Rapidly progressive glomerulonephritis is a potentially devastating sequela of ANCA-associated vasculitis. There is currently limited Australian data on patients with ANCA-associated glomerulonephritis, particularly in a regional setting.

Methods: We conducted a retrospective review of all renal biopsies performed at Bendigo Health between 1/1/2013 and 31/12/2021. Cases of newly diagnosed ANCA-associated glomerulonephritis were included. ANCA-negative glomerulonephritis was excluded. Information about treatment and follow-up was extracted from clinical records. A catchment population of 320 878 people and the Poisson distribution were used to estimate regional disease incidence.

Results: 27 cases of new ANCA-associated glomerulonephritis were identified. The calculated disease incidence in our region was 9.3 cases per million person-years (95% CI 6.2–13.6). The median age at diagnosis was 67 years (IQR 56–77 years). 59% of patients (n = 16) were diagnosed with microscopic polyangiitis and 41% (n = 11) with granulomatosis with polyangiitis. Two patients had overlap Goodpasture syndrome. Treatment data was available for 25 patients. Most received intravenous methylprednisolone (96%) and cyclophosphamide (89%).

Two patients underwent plasma exchange. The median follow-up time was 38 months (IQR 21–65 months). Approximately one quarter of patients relapsed (n = 7) with a median time to relapse of 23 months (IQR 19–27 months). 19% of patients (n = 5) required ongoing renal replacement therapy. Three patients (11%) died.

Conclusions: Renal involvement in ANCA-associated vasculitis continues to carry significant morbidity and mortality. Our local disease incidence is comparable to that published by the handful of other Australian epidemiological studies.

ANCA ASSOCIATED VASCULITIS OR RENAL CELL CARCINOMA ASSOCIATED PARANEOPLASTIC VASCULITIS, WITH A MANAGEMENT ISSUE OF IMMUNOSUPPRESSION

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Background: Antineutrophil cytoplasmic antibody (ANCA) associated vasculitis is rare, but associated with significant mortality and morbidity, with 10% of patients requiring chronic dialysis. Treatment involves immunosuppression to preserve renal function and prevent life-threatening complications. Paraneoplastic ANCA has associations with gastrointestinal, pulmonary, and haematological malignancies. Here we describe a case associated with Renal Cell Carcinoma (RCC).

Case Report: We present a 78 year old female with subacute kidney failure (eGFR 22 ml/min/1.73 m2), on a background of hypertension and baseline eGFR of 55 ml/min/1.73 m2. She had 2.2 gm proteinuria and microhaematuria, MPO 98 CU and PR3 74 CU. Imaging revealed a 10 cm left RCC with local invasion and asymmetric renal differential on MAG3 (left kidney providing 62% function). She underwent nephrectomy, with non-malignant tissue demonstrating segmental glomerulosclerosis, adhesions, and disruption to Bowman's capsule, representing resolving crescents. Postoperatively her eGFR was 15 ml/min/1.73 m2, with increasing ANCA titres. The risk of immunosuppression to prevent ANCA progression versus the risk of metastatic malignancy was discussed with the patient. She initially declined immunosuppression but developed extrarenal manifestations and agreed to oral azathioprine and prednisolone. Subsequent disease progression manifested as fevers and worsening arthritis. She was treated for sepsis, and immunosuppression briefly withheld. Within 24 h her vasculitis rapidly progressed, requiring acute haemodialysis. The patient agreed to immunosuppression after extensive discussions and re-staging excluded RCC recurrence. After nearly 4 months, the patient received rituximab. Extrarenal manifestations resolved and her ANCA titres were suppressed, however she remains dialysis dependent.

Conclusions: This case highlights the risks of paraneoplastic ANCA vasculitis progressing to end stage renal failure. ANCA positivity can be associated with malignancy. Surgically curable malignancy should be pursued aggressively prior to immunosuppression.
A CASE OF SEVERE MEMBRANOUS NEPHROPATHY AFTER mRNA COVID-19 VACCINATION TREATED WITH COMBINATION RITUXIMAB AND TACROLIMUS

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Background: Primary Membranous nephropathy (pMN) is one of the most common causes of nephrotic syndrome in adults. The mRNA Coronavirus disease 2019 (COVID-19) vaccination is a new vaccine that has been one of the most effective public health measures to curb the pandemic. Reports regarding a link between COVID-19 mRNA vaccination and pMN are emerging. We report a case of pMN hypothesised to be trigged by the mRNA COVID-19 vaccination.

Case Report: A 39-year-old male with a history of asthma presented to his GP with peripheral oedema and hypertension. A month prior he had received two doses of the Pfizer-BioNTech mRNA COVID-19 vaccine on 01/09/2021 and 22/09/2021. On presentation a 24 hr urine demonstrated protein excretion of 7000 mg/24 h, a protein:creatinine ratio (PCR) of 455, and an albumin:creatinine ratio (ACR) of 320, his serum albumin was low (25 g/L). A diagnosis of pMN was made on renal biopsy which demonstrated phospholipase A2 receptor (PLA2R) antibody positivity on immunofluorescence with positive serum PLA2R antibody levels at 196 relative units (RU)/ml (<14, negative; >20, positive). His renal function was preserved (eGFR>90), his malignancy screening revealed no abnormalities. He was initially managed conservatively with antiproteinuric therapy and diuretics. Four months later his PLA2R levels were 122RU/ml, and he remained nephrotic. A decision was made to administer rituximab combined with tacrolimus. In light of the pandemic we administered his third vaccine on 15/03/2022 prior to immunosuppression however, this was changed to a protein-based vaccine (Novavax).

Conclusions: We postulate a temporal relationship between mRNA COVID-19 vaccination and pMN. Here we have demonstrated the utility of an alternative vaccine regime, along with a concurrent tacrolimus and rituximab therapy for safe treatment of pMN.

HAEMOPTYSIS IN A STABLE KIDNEY TRANSPLANT PATIENT PREVIOUSLY SEROPOSITIVE FOR ANTI-GBM AND ANCA GLOMERULONEPHRITIS

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Background: Double-seropositive vasculitis is defined as coexistence of both anti-neutrophil cytoplasmatic antibody (ANCA) and anti-glomerular basement membrane (anti-GBM) antibodies. Consistent with the low recurrence rate of anti-GBM disease, seropositive relapses are nearly always related to the ANCA-associated vasculitis (AAV). The role of plasmapheresis (PLEX) in the management of AAV remains controversial given the recent landmark PEXIVAS trial demonstrating no benefit of routine use in AAV patients. We describe a rare case of AAV relapse 5 years post-kidney transplant, which was successfully treated with PLEX.

Case Report: A 64-year-old Caucasian man presented to the emergency department with productive cough, haemoptysis and worsening dyspnoea. He had been diagnosed with double-seropositive anti-GBM and anti-myeloperoxidase (MPO) glomerulonephritis 7 years ago. He was dialysis-dependent then with 100% glomerular crescent formation on kidney biopsy but no lung involvement. Both PLEX and cyclophosphamide treatment were deemed futile and discontinued. Accordingly, he remained dialysis-dependent and subsequently had a deceased donor kidney transplant 2 years later.

On arrival, he was noted to be febrile and in respiratory distress. Initial investigations revealed severe anaemia, with elevated inflammatory markers and acute kidney injury. Chest imaging showed extensive pulmonary infiltrates suggestive of pulmonary haemorrhage, infection or inflammation. He was started on methylprednisolone, broad-spectrum antibiotics and PLEX. He required a short period of invasive ventilatory support following bronchoscopy, which confirmed diffuse alveolar haemorrhage. Anti-MPO antibodies were positive. He was commenced on cyclophosphamide and mycophenolate motefil was ceased. He completed 10 sessions of PLEX and was discharged home 1 week later with no further haemoptysis and improved kidney function.

Conclusions: Relapse of AAV post-kidney transplant is uncommon and a selective group of AAV patients might benefit from PLEX treatment.

A RETROSPECTIVE COHORT STUDY OF BIOPSY-PROVEN IMMUNOGLOBULIN A NEPHROPATHY (IGAN) IN AUCKLAND AND NORTHLAND—INCIDENCE, CLINICAL FEATURES, THERAPIES, AND OUTCOMES

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Aim: To improve the understanding of the incidence, clinical features, therapies, and outcomes for New Zealand (NZ) Māori and Pasifika populations with Immunoglobulin A Nephropathy (IgAN).

Background: IgAN is the most common glomerulonephritis and can lead to chronic kidney disease or end stage kidney disease. Overseas studies have identified that for IgAN ethnic differences exist. There is a paucity of studies within NZ assessing the incidence, clinical features, therapies, and outcomes of IgAN, and scant evidence looking at outcomes in Māori and Pasifika populations in NZ.

Methods: A biopsy database identified all patients with biopsy-proven IgA Nephropathy within three District Health Boards within the
Auckland and Northland region between 01/01/2003 and 31/12/2018. Data was collected from electronic records.

**Results:** A total of 167 patients with IgAN were included over a 16-year period. The incidence was 0.9 per 100,000 adults per year. 55.7% of patients were males. There were 44.3% NZ European, 10.8% NZ Māori, and 7.8% Pasifika. The mean weight was 81.1 kg with BMI 28.8. Mean blood pressure was 131 mmHg systolic and 79 mmHg diastolic with mean arterial pressure of 96 mmHg. Patients used a mean of two anti-hypertensive agents (range 0–4). Regarding therapies, 82.6% were on renin angiotensin system (RAS) blockade, 29.9% on corticosteroids, 6% on immunosuppressants, 12% on omega-3 fatty acids, and 36.5% on statins.

**Conclusion:** This is one of the largest cohorts of biopsy-proven IgAN with 18 cases in Māori and 13 cases in Pasifika. There was no statistically significant difference in weight, blood pressure, or anti-hypertensive medications between Māori compared to NZ European. Māori had fewer statins (p = 0.04) and Pasifika had lower blood pressure (systolic p = 0.001, diastolic p = 0.03) and fewer anti-hypertensive medications (p = 0.004).

**CRESVENTIC IMMUNOGLOBULIN A NEPHROPATHY WITH POSITIVE ANTI-NEUTROPHIL CYTOPLASMIC ANTIBODIES: A RARE COMBINATION**

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**Background:** Immunoglobulin A Nephropathy (IgAN) is generally a slowly progressive disease, with less than 10% of patients presenting with rapidly progressive glomerulonephritis (RPGN). We report a rare case of severe crescentic IgAN with positive circulating anti-neutrophil cytoplasmic autoantibodies (ANCA).

**Case Report:** A 64-year-old female presented with acute kidney injury (AKI) and peripheral oedema on a background of type 2 diabetes mellitus, hypertension, sarcoidosis, monoclonal gammopathy of undetermined significance (MGUS), and stage 3B chronic kidney disease (CKD).

Serum creatinine peaked at 404 μmol/L with an eGFR of 10 ml/min/1.73 m² during admission. Urine albumin/creatinine ratio (ACR) was 380 mg/mmol, with significant microhaematuria (660 × 106/L erythrocytes).

Nephrotic factors were withheld, and furosemide was commenced to manage fluid overload. Intravenous methylprednisolone pulse was given for suspected primary glomerular pathology. Renal biopsy showed 16 glomeruli, six with fibrocellular crescents. Immunofluorescence showed mesangial and capillary granular staining with IgA and C3. The diagnosis of IgAN was subsequently made.

She was commenced on prednisolone 80 mg daily and discharged. Subsequently, ANCA returned as positive with elevated anti-myeloperoxidase (MPO) titre of 7.8 AI. Autoimmune and viral screen was otherwise unremarkable. Four weeks later, creatinine remained elevated at 416 μmol/L, with urine ACR 398 mg/mmol. Based on poor steroid response, crescents on biopsy, and ANCA positivity, she was treated with intravenous cyclophosphamide for 6 months. Renal function improved rapidly and at 6 months creatinine was 211 μmol/L, urine ACR 79 mg/mmol, and ANCA was negative. She was maintained on low dose azathioprine and prednisolone.

**Conclusions:** ANCA-positive IgAN is a rare, severe disease phenotype which has greater response to immunosuppression than ANCA-negative IgAN. Clinicians should consider measurement of ANCA in IgAN patients with RPGN, as immunosuppression therapy in this patient cohort could vastly improve outcomes.

**NON-DERMATOLOGICAL NEOPLASMS IN RENAL TRANSPLANT RECIPIENTS AND PATIENTS WITH GLOMERULAR DISEASES**

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**Aims:** To compare the risk of non-skin cancers after exposure to immunosuppressants between renal transplant recipients (RTRs) and patients with glomerular disease (GD).

**Background:** To date, there is no previous comparison on the risk of non-skin cancers between RTRs and patients with GD treated with long-term immunosuppressants.

**Methods:** A retrospective cohort study was undertaken among adult RTRs with first-time kidney graft and GD patients treated with long-term immunosuppressants.

**Results:** The cohort comprised of 61 RTRs and 51 GD patients followed up over a median duration of 74.0 months (Interquartile range, IQR 44.5–110.5) and 52.5 months (IQR 29.8–81.8) after exposure to immunosuppressants, respectively (p = 0.088). Between two cohorts, no statistical differences were noted between baseline demographics except duration and intensity of immunosuppressants. RTRs were treated with more intense and lengthy immunosuppressive therapy compared to GD patients. Three renal cell carcinoma (RCC), two prostate carcinoma, two colorectal carcinoma (CRC), and one sarcoma of chest were found in RTRs whereas 2 RCCs and 1 CRC in GD group. The cumulative incidence of solid cancers in RTRs and GD and 11.5% (N = 7) and 5.9% (N = 3), respectively. One RTR developed post-
transplant lymphoproliferative disorder affecting the scalp. No haematological malignancy was reported in GD cohort. The mean age of patients at the time of cancer diagnosis and mean onset of cancer was 47 years (RTRs) versus 67 years (patients with GD), and 133 versus 147 months, respectively.

**Conclusions:** Immunosuppressants may increase the risk of solid cancers in both cohorts. Higher cancer risk is likely dependent on the intensity and duration of immunosuppressants.

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**AN INTEGRATED SKIN CANCER EDUCATION PROGRAM IN PATIENTS WITH GLOMERULAR DISEASES TREATED WITH LONG TERM IMMUNOSUPPRESSANTS**

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**Aims:** To determine the effect of an integrated skin cancer education program on skin cancer awareness and sun-protective behaviours in patients with glomerular disease (GD) treated with long-term immunosuppressants.

**Background:** Sun-protective strategies focusing on skin cancer awareness are needed in immunosuppressed patients at risk of skin cancers.

**Methods:** A pilot prospective cohort study in Central Queensland was undertaken among adult patients with GD, who completed survey questionnaires on skin cancer and sun-health knowledge (SCSK), sun-protection practices and skin examination pre- and post-education.

**Results:** 25 patients participated in the study. All participants completed questionnaires at pre-education, 3-month and 6-month post-education. The mean age of all participants was 61 years (standard deviation, SD = 15.3 years) and 52% were male. 88% of all participants were Caucasians and 36% had a history of skin cancer. There was a significant increase in SCSK scores from baseline at 3-months (19.0 [SD 3.5] vs. 20.2 [SD 3.4], p = 0.039) and 6-months post-intervention (19.0 [SD 3.5] vs. 20.9 [SD 1.9], p = 0.004). The frequency of outdoor activities and compliance with each sun protective practice did not change significantly pre- and post-education.

**Conclusion:** An integrated skin cancer education program improved knowledge of skin cancer and sun health, self-skin examination and formal skin assessments by general practitioners. However, improvement in skin cancer awareness of patients did not extend to compliance with sun-protective practices.

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**COMIRNaty (Biontech-Pfizer) COVID-19 Vaccine Associated IgA Nephropathy**

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**Background:** SARS-CoV-2 (Covid-19) mRNA vaccines such as the Comirnaty (BioNTech-Pfizer) and Spikevax (Moderna) have recently been associated with the onset of IgA Nephropathy. We describe a case of IgA nephropathy that developed following the administration of a second dose of the Comirnaty Covid-19 vaccine.

**Case Report:** A 37-year-old man presented to the Emergency Department 2 days after his second Comirnaty Covid-19 vaccine with a cola-coloured urine, 3+ proteinuria on urinalysis, hypertension, and a stage 1 acute kidney injury (serum creatinine of 124 μmol/L; normal: 60–110). He was provisionally diagnosed with rhabdomyolysis and managed with intravenous fluid with resolution of his hematuria. Planned follow up showed ongoing microscopic hematuria, proteinuria and persisting abnormal kidney function. A 24-h urine collection shows 1.8 g of proteinuria. ANA, ENA, ANCA, dsDNA, anti-GBM were negative. C3/C4 were normal. He proceeded to kidney biopsy which demonstrated mesangial hypercellularity on light microscopy and 2+ granular mesangial reactivity for IgA, kappa and lambda on immunofluorescence consistent with IgA Nephropathy. He was commenced on Irbesartan 150 mg daily and 10 mg dapagliflozin. Three months after commencing treatment his proteinuria had worsened and his serum creatinine remained elevated (urine protein/creatinine ratio 297 mg/mmol; serum creatinine 126 μmol/L). Prednisolone was added to his therapy. One month later, there was a marked improvement in his proteinuria (urine protein/creatinine ratio 182 mg/mmol) and normalization of his renal function (serum creatinine 89 μmol/L). He was continued on a 6-month weaning course of prednisolone.

**Conclusion:** Covid-19 vaccine induced IgA Nephropathy is an emerging entity that should be considered in patients with new onset hematuria and proteinuria following mRNA covid-19 vaccination. Further studies are needed to confirm the pathophysiology and best management for this condition.
A REVIEW OF RENAL BIOPSY BLEEDING COMPLICATIONS AND
PRACTICES IN A SINGLE CENTRE REGIONAL HOSPITAL

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Aim: To describe bleeding complications and practices of renal biopsies performed at a single centre regional hospital over a five-year period, and to discuss risk factors for bleeding as well as the use of desmopressin pre-procedure.

Background: Renal biopsies are conducted to aid in the diagnosis of renal pathologies and guide management. Whilst clearly beneficial, complications can be significant including haematuria and perinephric haematoma formation, at times requiring transfusion, embolization, or even nephrectomy. Use of desmopressin to reduce these complications remains controversial.

Methods: Retrospective medical record review.

Results: 153 percutaneous native renal biopsies were reviewed with bleeding complications occurring in 16.9% (n = 26). Minor complications (not requiring intervention) occurred in 13.7% (n = 21). Major complications requiring intervention occurred in 3.3% (n = 5), with 2.6% requiring transfusion. One patient required interventional embolization and subsequent nephrectomy. There was no discrepancy in complications when accounting for demographics, or inpatient versus outpatient procedures. Rates were higher in patients with reduced eGFR and elevated urea, and when performed under ultrasound (direct and indirect visualization) compared with computer tomography (5.9%, n = 1, vs. 18.4%, n = 25). Desmopressin use was associated with lower bleeding rates (13% vs. 19.2%). The most common diagnosis obtained was IgA nephropathy (19.6%, n = 30), followed by diabetic/hypertensive nephropathy (17.7%, n = 27) and pauci-immune glomerulonephritis in (9.2%, n = 14).

Conclusions: In a regional centre, native renal biopsy bleeding complications were higher when performed under ultrasound and in patients with impaired renal function. Demographics, presence of anaemia, thrombocytopenia, or hypoalbuminaemia did not affect complication rates. Minor complications and major complications requiring intervention were comparable in reported studies, however transfusion rates were higher in this study. Bleeding complications rates were lower in those who received desmopressin.

PATTERNS OF HOSPITAL ADMISSIONS AMONG NON-DIALYSIS CHRONIC KIDNEY DISEASE PATIENTS

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Aim: To analyse the patterns of hospitalisations among non-dialysis CKD patients.

Background: Chronic kidney disease (CKD) patients have higher hospital costs than non-CKD patients in Australia (average annualized cost: $1829 in 2012), but little is known about the diagnoses/patterns of hospitalisations before death or starting kidney replacement therapy.

Methods: We analysed hospitalisations among 7221 CKD patients enrolled in CKD.QLD registry from 10 public renal clinics in Queensland, Australia. They were followed for a total of 25 390 person-years, from enrolment, between May 2011 and until they started KRT or died, or the censor date of 30th June 2018. Queensland Health supplied data on public hospital admissions with ICD-10-AM codes, costs, and outcomes (death and KRT). For some conditions we grouped several ICD-10-AM codes together into a single “causal” category.

Results: About 81% patients were admitted once or more, contributing to 40 566 admissions, costing $226.3 million, with an average person year of 1.6 admissions and cost of $8915. 59% of admissions were 1-day admissions, although they constituted only 16% of total costs. Neoplasm/cancers constituted the greatest proportion of 1-day admissions (15.3%), followed by anaemia (9%) and kidney-related admissions (8.6%). 41% of admissions were >1-day admissions, which represented 84% of total costs. Leading causes were cardiovascular disease (CVD) (19%), followed by respiratory (11%) and kidney-related (8.4%). Readmissions within 30 days of discharge constituted 41.4% of all admissions and 45% of total costs. Of these readmissions, 46.2% were for the same primary diagnosis, while 56.4% were 1-day admissions.

Conclusion: The hospital burden is high reflecting great burden of morbidities in these CKD patients. One avenue for hospitalization minimisation is preventing readmissions for the same diagnosis following complex (>1 day) hospitalisations.
Methods: We analysed primary causes of hospitalisations among 7221 CKD patients enrolled in CKD.QLD registry from 10 public renal clinics in Queensland, Australia. Patients were followed from enrolment, between May 2011 until starting KRT or death, or the censor date of 30th June 2018. Queensland Health supplied data on hospital admissions with ICD-10-AM codes, hospital days and costs. ICD-10 AM chapters 14 (kidney/genitourinary), 21 (rehabilitation/waiting/pharmacotherapy/dialysis preparation) and four (endocrine/nutritional/metabolic disorders) contained some CKD-related codes. These were extracted/combined into CKD.QLD’s “CKD-related” category.

Results: In chapters 14, 4 and 21, 61%, 25% and 20% of admissions were CKD-related. They constituted 4.7%, 1.3% and 2.4% respectively of all admissions. CKD.QLD’s “CKD-related” category represented 8.5% of total admissions, 7.3% of hospital days, and 8.6% of total costs. ~60% of these admissions were one-day admissions. Admissions not directly related to CKD constituted 91.5% of total admissions, 92.7% of hospital days, and 91.5% of total costs. ~59% of these were one-day admissions, but most hospital days and costs were driven by longer admissions, with cardiovascular and respiratory diseases as leading causes. After annualising CKD-related vs non-CKD hospital episodes, the number of admissions were 0.1 versus 1.5, hospital days 0.4 versus 5.7 and costs $764 versus $8153.

Conclusion: The major burden of hospitalisations and costs of these patients is driven by their serious co-morbidities, which are not directly related to, although maybe conflated by, their CKD. Minimisation of these complications and progression is a powerful management challenge.

EPIDEMIOLOGY OF RENAL PATHOLOGIES IN NORTH BRISBANE OVER 17 YEARS—A SUB-STUDY OF THE QUEENSLAND RENAL BIOPSY REGISTRY

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Aim: To establish the changing epidemiology of biopsy confirmed glomerulonephritis (GN) via auditing renal biopsy activity across Metro North Hospital and Health Service from 2005 to 2021.

Background: Royal Brisbane and Women’s Hospital is the only public centre performing renal biopsies in North Brisbane and serves a population of 1 030 006. The Queensland Renal Biopsy Registry (QRBR) was established to provide details of patients with biopsy-proven kidney disease to improve disease understanding and tracking.

Methods: Pathology reports from the Pathology Queensland were reviewed between 01/01/2005 and 31/12/2021. Data was grouped by year and pathology.

Results: 1433 renal biopsies were reviewed. Biopsy activity has almost doubled from 5.92 biopsies per hundred thousand people per year (php/yr) to 11.36 php/yr across the study period. There were 1039 cases of primary or secondary GN. Of the 636 primary GN, IgA nephropathy remains the most dominant pathology affecting 1.04 php/yr, followed by Crescentic GN (0.88 php/yr) and Focal Segmental Glomerulosclerosis (FSGS) (0.78 php/yr).

Regarding patterns of individual pathologies, there was an increase in membranous nephropathy (MN) incidence from 0.33 php/yr before 2014 to 0.56 php/year after 2014. An incidence increase was also observed in FSGS (0.64 php/yr to 0.94 php/year before and after 2014).

Conclusion: Although biopsy activity in North Brisbane has steadily increased over the past 17 years, the average rate of biopsy was 9.22 php/yr as opposed to previously quoted Queensland average of 12.04 php/yr, both are significantly lower than 21.5 php/yr reported in state of Victoria. The finding of rising incidence of MN and FSGS has also been reported in Singapore and in the west. This data provides important clues for the trends in renal diagnoses in coming years.

GENERAL NEPHROLOGY—OTHER

PHARMACIST-LED PARTNERED PRESCRIBING: IMPROVING THE ACCURACY OF DISCHARGE MEDICATION DOCUMENTATION

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Aim: To determine if pharmacist-led partnered prescribing (PPP) on discharge reduced errors in documentation of medications in the discharge prescription and the discharge summary of people with kidney disease compared to medical prescribing (MP).

Background: Inaccurate medication documentation on discharge produces poorer patient outcomes and is costly to the healthcare system. Affected patients are 2.3 times more likely to be readmitted to hospital compared with those patients who have an accurate discharge summary.

Methods: This Interventional 2-phase study compared current workflow (MP) with the subsequent implementation of the interventional workflow (PPP). Patients were included if they were hospital inpatients discharged from the renal unit within pharmacy working hours and had a discharge prescription and discharge summary.

An error in documentation was defined as documentation (or no documentation) of a medication not in agreement with the documented treatment plan (for the discharge prescription) or the final, pharmacist reviewed discharge prescription (for the discharge summary).

Results: Data was collected from 185 discharged patients (95 in MP phase then 90 in PPP phase). Discharge prescriptions with at least one error reduced from 75.8% in the MP phase to 6.7% in PPP phase ($p < 0.001$). Discharge summaries with at least one error reduced from 53% in MP phase to 24% in PPP phase ($p < 0.001$). Improvements in electronic medical record workflow has the potential to further reduce these errors to 48% and 3% respectively. The most common error in both phases of the discharge prescription and discharge summary was omitted medication, with the most erroneous medications being paracetamol, vitamin D medications and erythropoiesis stimulating agents.
Conclusion: PPP reduces errors in the documentation of discharge medications of patients with kidney disease.

A STATEWIDE GLANCE AT TRENDS IN AETIOLOGY AND TREATMENT OF BIOPSY PROVEN INTERSTITIAL NEPHRITIS OVER THE LAST 10 YEARS

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Background: Interstitial nephritis (IN) is characterized by inflammatory infiltrate in the kidney interstitium. Consensus guidelines for treatment are not yet established.

Aim: Evaluate the aetiology and treatment of kidney biopsy-proven IN across a 10-year period in Tasmania.

Methods: Patients in Tasmania from 2011 to 2021 with biopsy-proven IN and adequate digital medical records were included. Aetiology, kidney function and steroid use were recorded.

Results: Of 1650 biopsies performed, 47 (12 female) met our inclusion criteria. Mean age was 56-years (range 12–80 years) and mean eGFR at time of biopsy was 30 ml/min/1.73 m² (range 12–96 ml/min/1.73 m², two patients on haemodialysis). Thirty-six (77%) were drug induced, 4 (8%) due to autoimmune/systemic disorders, 2 (4%) due to infection and in 5 (11%) the cause was not identified. Of drug induced IN, proton pump inhibitors (PPI) were the most common (35%). There were no cases of PPI induced IN in our dataset after 2019. NSAIDs were the second most common causative medication (4%), with 4 of 5 cases occurring in the second half of the study period. The first case of immune check-point inhibitor (ICI) IN was recorded in 2017, two subsequent cases in 2021. 19 (40%) patients were treated with varying doses and duration of steroids.

Conclusion: There is a wide range of aetiologies implicated in IN. Until 2019, PPIs accounted for 1/3 of drug induced IN, yet in our dataset there was no PPI induced IN after 2019, possibly reflecting increasing awareness and changes in PPI PBS prescribing. ICIs are an emerging cause of IN. There was great heterogeneity in the dosage and duration of steroids used in treatment of IN.

ECULIZUMAB THERAPY AND COMPLEMENT REGULATION IN CATASTROPHIC ANTIPHOSPHOLIPID SYNDROME

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Background: Catastrophic Antiphospholipid Syndrome (CAPS) is a rare, life-threatening form of antiphospholipid syndrome characterized by diffuse arterial and venous thrombosis, in the presence of positive antiphospholipid antibodies. The mechanism of CAPS involves antiphospholipid antibodies activating complement leading to a cascade of thrombosis and cytokine storm. Patients with CAPS have an increased rate of complement regulator gene mutation leading to the proposal that complement dysregulation is instrumental to the pathophysiology of CAPS.

Case Report: We report a case of an 18-year-old female with a history of miscarriages presenting with clinical and laboratory findings consistent with life-threatening CAPS, resistant to initial treatment, and finally responding to Eculizumab (C5-inhibitor monoclonal antibody). The clinical features included oligoanuric renal failure, profound thrombocytopenia, a large pulmonary embolus, upper limb deep venous thrombosis, and splenic/renal infaracts. The presumed trigger was an upper respiratory tract infection. The patient was supported with haemofiltration, heparin infusion, immunosuppressive therapy with methylprednisolone and cyclophosphamide, IVIg and plasma exchange. The patient developed gastrointestinal bleeding, and despite aggressive standard therapy as described, failed to improve. Eculizumab was commenced on the basis that complement dysregulation was a major contributor to the resistant CAPS, supported by laboratory evidence of profound hypocomplementaemia. Following the addition of Eculizumab, the patient clinically improved, transitioned off haemofiltration, and the heparin infusion changed to warfarin without further bleeding. Follow up care demonstrated impaired renal function (eGFR = 27) and genetic analysis for complement regulator mutations are in progress.

Conclusions: (1) Supports a central role for complement dysregulation in the pathophysiology of CAPS; (2) Eculizumab therapy and targeted complement regulation may be a life-saving therapy for resistant CAPS.

UTILITY OF POTASSIUM BINDERS: DATA FROM THE PATIROMER (VELTASSA) COMPASSIONATE ACCESS PROGRAM

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Aim: To illustrate the potential role of patiromer in managing hyperkalaemia in patients who have failed other potassium lowering therapies.

Background: Hyperkalaemia is a common complication of chronic kidney disease (CKD), heart failure and important treatments thereof, such as renin-angiotensin-aldosterone system inhibitors. Patiromer sorbitex calcium (Veltassa) binds potassium in the gastrointestinal tract and provides an alternative to the currently limited potassium lowering therapies available.

Methods: Vifor’s Compassionate Access Program for patiromer commenced in November 2020 and is ongoing. Inclusion criteria comprises: at least 1 episode of hyperkalaemia; CKD, heart failure or other
medical condition leading to hyperkalaemia, failure of other potassium lowering strategies. Data collected included: age, gender, location, underlying disease, previous hyperkalaemia therapies trialled and days on therapy.

Results: As of 3rd February 2022, 58 patients had been commenced on patiromer through Vifor’s Compassionate Access Program. About 86% had CKD and 14% heart failure as their primary diagnosis. About 58% of patients were male and 72% of patients over 55. Twenty-five participants (43%) had previously failed to manage their hyperkalaemia with a low potassium diet and 52 (89%) had trialled resnium. The average duration of treatment with patiromer was more than 170 days. Eight patients discontinued therapy—3 due to gastrointestinal side effects, 2 received kidney transplants, 1 commenced dialysis and 1 was lost to follow up.

Conclusions: Patiromer is a well-tolerated alternative to managing serum potassium in patients with hyperkalaemia who have failed other therapies.

FRAILTY IN PEOPLE WITH ADVANCED CHRONIC KIDNEY DISEASE (CKD) ATTENDING A KIDNEY SUPPORTIVE CARE (KSC) SERVICE

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Background: Frailty is a multidimensional syndrome where physiological decline leads to increased vulnerability to adverse health outcomes. Frailty can be difficult to assess, but is common in people with CKD. The Clinical Frailty Scale (CFS), is a simple, albeit, subjective tool with significance as a predictor for mortality in CKD.

Aim: To examine frailty, using the CFS, in people attending a KSC clinic over a 10-month period.

Methods: Retrospective analysis of people attending KSC clinics between 1st July 2021 and 30th April 2022. Demographics, disease data, Charlson Comorbidity index (CCI), Resource Utilization Group Activities Daily Living (RUG-ADL), Australia-modified Karnofsky Performance Status (AKPS), and treatment pathway, were extracted from clinical records. Medical and nursing staff rated frailty from 1 to 9 using the CFS. Results were analysed descriptively and group differences tested using the Mann–Whitney U test.

Results: The CFS was recorded for 240 people at 366 KSC attendances. Median age was 79.6 years (IQR 13.1 years), 43.8% were female, median eGFR was 16 ml/min/1.73 m² (IQR 9 ml/min/1.73 m²) and 26% were on kidney replacement therapy (KRT). Average CCI was 6.9 (SD 2.0), median RUG-ADL 4 (IQR 1) and median AKPS 60 (IQR 10). Median CFS was 4 (IQR 2), representing very mild frailty.

There was no significant difference in CFS between people on conservative and KRT pathways (p = 0.11), with median CFS of 5 (IQR 2) and 4 (IQR 2), respectively.

Conclusion: Surprisingly, this study cohort were, in general, only rated as having very mild frailty using the CFS. Frailty was similar in people on conservative and KRT pathways. Selection bias may have contributed to these findings. Further longitudinal studies are required to understand frailty in the KSC population.

AMYLOID POST COVID-19 VACCINE—ANY RELATION?

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Background: The coronavirus pandemic has changed lives forever. The effect of this virus on morbidity and mortality is evolving and devastating. Some diseases are triggered not only by the infection but also by the vaccine. There are well-established cases of glomerulonephritis, including minimal change disease and membranous nephropathy. We present a diagnosis of amyloid 1 week after the Pfizer-BioNTech COVID-19 vaccine.

Case Report: A 60-year-old male of Laos descent who has been in Australia since the 1980s presented with acute onset of peripheral oedema and weight gain 1 week after receiving the second dose of mRNA Pfizer-BioNTech COVID-19 vaccine. He had no past medical history and was not taking regular medications, including over the counter remedies. The laboratory investigation revealed serum creatinine 69µmol/L and serum albumin 16 g/L. 24-h urine yielded 5 g of protein. Renal biopsy showed Congo red staining of amyloid deposits. The myeloma work-up showed a serum free light chain ratio of 0.78, IgG kappa 3 g/L paraprotein and positive urine Bence Jones protein. The bone marrow biopsy demonstrated 10% plasma cell aggregates via CD138 and amyloid deposit within extramedullary tissue. He received bortezomib, cyclophosphamide and dexamethasone chemotherapy without haematological response and is currently being worked up for a stem cell transplant.

Conclusions: There are implications of S-protein in COVID-19 disease and vaccine, which are still under research in the literature. Our case highlights the importance of the prompt investigation, including biopsy in patients with life changing symptoms post vaccination.

ANCA-ASSOCIATED VASCULITIS AND INCIDENTAL MICROSCOPIC RENAL CELL CANCER

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Background: The relationship between anti-neutrophil cytoplasmic antibody (ANCA)-associated vasculitis (AAV) and malignancy is complex. Combined studies have identified a standardized incidence ratio of cancer in vasculitis between 1.6 and 2.0. This increased risk has been primarily attributed to the long-term effects of immunosuppression. Mitigating malignancy risk in both the short and long term remains one of the significant challenges in AAV treatment. The finding of an incidental neoplasm on renal biopsy occurs in approximately 0.2% of cases and can further complicate treatment options.

Case Report: A 52-year-old Cambodian man presented with a 2-month history of lethargy, sinusitis, arthralgia/myalgia, and microscopic haematuria with a recent diagnosis of hypertension. The investigations revealed creatinine 132 μmol/L, proteinuria 1.78 g/24 h and positive ANCA-MPO titre 62 IU/mL. Renal biopsy showed simultaneous AAV renal injury and an incidental finding of neoplasmic proliferation, positively staining for renal markers (PAX8 and CA-IX) but negatively staining for prostate markers (NKX3 and PSAP), consistent with clear cell renal cell carcinoma (RCC). Imaging showed PET avidity in the prostate but not in the kidneys. Subsequent prostate biopsy ruled out malignancy. He was treated with pulse methylprednisolone and maintained on prednisolone monotherapy. At 24-months, his surveillance CT did not show RCC progression with creatinine 122 μmol/L and ANCA-MPO titre 14 IU/mL.

Conclusion: Beyond staging and histopathological identification, there remains little evidence to guide the best course of therapy in these cases. In our patient, the decision to treat solely with corticosteroid was based on the concern of malignancy progression and the patient's clinical improvement without cyclophosphamide induction.

INCIDENCE OF HYPOGLYCAEMIA FOLLOWING INSULIN ADMINISTRATION FOR TREATMENT OF HYPERKALAEMIA

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Aim: The purpose of this study is to investigate the incidence of hypoglycaemia following the current insulin protocol for treatment of hyperkalaemia.

Background: Insulin is often used to treat hyperkalaemia in hospitalized patients. As a result, hypoglycaemia is a potential complication. The current protocol at our hospital includes the administration of 10 units insulin with 50 ml glucose 50% for the treatment of hyperkalaemia, similar to most other protocols. As part of the Hospital Acquired Complications (HACs) reduction exercise, we studied the incidence of hypoglycaemia following insulin administration and associated risk factors.

Methods: We conducted a retrospective audit of hospitalized patients with hyperkalaemia who received insulin as per hospital protocol between August 2020 and January 2021. About 207 patients were identified, of which 15 patients were excluded as their post-treatment blood glucose levels were not recorded. The remaining 192 patients were assessed for the incidence and risk factors of developing hypoglycaemia.

Results: Fifteen episodes of hypoglycaemia (7.8%) were identified among 192 hyperkalaemic patients treated with insulin. Ten patients had blood glucose levels (BGL) between 3 and 4 mmol/L (52%) whereas five developed clinically significant hypoglycaemia with BGL < 3 mmol/L (2.6%). Hypoglycaemia occurred on an average of 4 h following insulin administration. 165/192 patients had acute kidney injury or chronic kidney disease of whom 13 developed hypoglycaemia (7.9%). Among 59 patients with type 1 or 2 diabetes on regular insulin therapy, three developed hypoglycaemia (5%).

Conclusions: The incidence of hypoglycaemia following the current protocol of insulin administration is significant and the risk of exposure is elevated in patients with renal dysfunction. We need to consider a revised insulin protocol to treat hyperkalaemia to minimize the risk of hypoglycaemia.

SEVERE AND PROLONGED HYPOCALCAEMIA POST ZOLEDRONIC ACID INFUSION IN A PATIENT WITH SLEEVE GASTRECTOMY

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Zoledronic acid is commonly used to treat hypercalcaemia and osteoporosis, as well as to prevent skeletal complications from haematological and solid organ malignancies. We report the case of a 38 year old female who presented with severe hypocalcaemia following the administration of zoledronic acid. Her significant background history included vitamin D deficiency and sleeve gastrectomy 3 years ago. She had prolonged hypocalcaemia requiring IV calcium replacement for 2 months. Her prolonged hypocalcaemia was attributed to her vitamin D deficiency at the time of zoledronic acid infusion as well as her history of bariatric surgery. This case emphasizes the importance of ensuring vitamin D levels are replete prior to zoledronic acid infusion and ensuring the calcium levels are checked frequently in patients with a history of bariatric surgery obtaining zoledronic acid.

RENEAL CLINICIAN SELF-REPORTED SHARED DECISION MAKING COMPETENCY AND HEALTH SYSTEM OUTCOMES BEFORE AND AFTER A MEDICAL COMMUNICATION COURSE (IVALIDATE)

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Background: Shared decision-making is important for people with chronic medical disorders. Advance care planning (ACP) improves end-of-life outcomes for renal patients and is cost effective.

Aim: Understand the impact of clinicians undertaking a medical communication course on their self-reported confidence of shared-decision making and ACP and impacts on health system outcomes.

Methods: We provided a commercial medical three-session experimental small-group communication program (iValidate) to renal clinicians at SWSLHD. Clinicians undertook surveys self-assessing their competency and the program at four time-points—immediately after, 1 week, 2 and 6 months after the program. The impact on patient care was assessed by measuring palliative care referrals, number and route of admissions, ACPs, medical emergency team (MET) reviews and intensive care unit (ICU) admissions in a prevalent dialysis population for 6 months before and after the program. Analysis was undertaken with SPSS.

Results: Of the 24 participants that registered 8 were nursing/allied health and 16 medical. All advanced trainees and 72% of senior medical staff attended. Participants reported an improvement in confidence in initiating ACP conversations, eliciting patient goals, shared decision making and initiating difficult decisions (all p < 0.01). After course participation a reduction in referrals to palliative care service (adjusted odds ratio) aOR 0.2(95%CI 0.05–0.74, p = 0.016) and ICU admissions aOR 0.43(95% CI 0.2–0.9) was observed. There was no difference in documentation of ACPs, admission number or route, or number of METs.

Conclusions: A medical communication course improved self-reported confidence in undertaking difficult decisions and engaging in shared decision making in renal clinicians. This was associated with a reduction in referrals to the palliative care service and number of ICU admissions, suggesting increased clinician engagement in end of life decision-making.

NO EFFECT OF DESMOPRESSIN ADMINISTRATION BEFORE KIDNEY BIOPSY ON THE RISK OF MAJOR POST-BIOPSY BLEEDING

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Background: The most important complication of kidney biopsy is bleeding, and it is unclear whether desmopressin is effective in preventing it.

Aim: This study was conducted to compare post-biopsy bleeding with or without desmopressin prescription prior to percutaneous kidney biopsy.

Methods: In this single-centered, retrospective, and observational study, 3018 adult patients who underwent kidney biopsy between January 1, 2003 and March 31, 2019 at our institute were recruited. Of these, 776 patients received desmopressin. To compare the differences in major bleeding events between patients administered and not administered with desmopressin, propensity score matching was performed.

Results: Before propensity score (PS) matching, it was observed that patients in the desmopressin group were significantly older (p < 0.001) and had a higher blood pressure (p < 0.001), higher serum creatinine (p < 0.001), lower haemoglobin levels (p < 0.001), and lower platelet counts (p = 0.001) than those in the no-desmopressin group. Furthermore, the incidence of renal artery embolization was not significantly different between the two groups (p = 0.077); however, blood transfusions occurred significantly more frequently in the desmopressin group (p < 0.001). A comparison of the two groups after PS matching did not reveal any differences in the incidence of renal artery embolization (p = 0.341), blood transfusion (p = 0.579), and total major bleeding events (p = 0.442). Furthermore, there was no difference in the incidence of perinephric hematoma on computed tomography or ultrasound (p = 0.120).

Conclusions: We do not recommend desmopressin administration before kidney biopsy.

DEVELOPMENT OF A NOVEL POINT OF CARE DEVICE FOR URINARY ALBUMIN AND CREATININE MEASUREMENT

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Aim: To develop a fluorescent based and colorimetric based portable medical device for point-of-care (POC) testing for urine albumin: creatinine ratio (ACR).

Background: Fluorescence and colorimetric monitoring are optical based measurement techniques, which have been used to detect and evaluate the presence of multiple chemicals in biological fluids. They have potential as POC medical devices due to small sample volume requirement and high sensitivity and specificity. Currently, an important part of chronic kidney disease (CKD) detection and monitoring is by measuring urinary ACR, but samples are usually tested in laboratories. There is a need for devices which can provide accurate ACR results at the point of care with low cost.

Methods: We have developed and optimized novel aggregation induced emission biosensors specified for albumin detection in urine. This has been combined with a low-cost colorimetric based device that has been manufactured through 3D printing technology for urine creatinine measurement through the Jaffe creatinine method.

Results: The above POC fluorescent device was used to detect albumin levels and the commercial Jaffe creatinine kit with a developed POC colorimetric device was used to detect creatinine in urine samples from 56 patients with CKD. The ACR results from the devices were compared with the results from the laboratory results. The Bland–Altman plot and intraclass correlation coefficient (R = 0.92,
ACUTE COVID-19 WITH ASSOCIATED RENAL INFARCTION

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Background: Acute COVID-19 infection is known to result in a hypercoagulable state with an increased risk of both arterial and venous thrombosis. Pulmonary embolism is the most reported thrombotic complication of COVID-19, with thrombosis at other sites occurring less commonly. Here we report a case of renal infarction as a complication of mild COVID-19 infection.

Case Report: A 41-year-old Caucasian male with no significant medical history presented with acute flank pain in the setting of mild COVID-19 infection. A contrast computed tomography (CT) scan showed an area of hypoattenuation in the left interpolar region, suggestive of infarction with a differential diagnosis of renal abscess. No others risk factors for thrombosis were identified, and a prothrombotic screen (Factor V Leiden, Prothrombin G20210A, Anti-thrombin III, Protein C and S concentrations and Antiphospholipid antibodies) was negative. CT angiography did not reveal any vascular abnormalities. Urine culture was positive for Escherichia coli, and he was treated with a course of intravenous and oral antibiotics and anticoagulated with oral apixaban. Follow up CT imaging showed evolutionary changes consistent with the diagnosis of renal infarction.

Conclusion: Renal infarction, though rare, can occur in isolation as a complication of acute COVID-19 in the absence of other thrombotic risk factors. Further studies are needed to provide guidance on the long-term management of this condition, especially regarding the duration and effectiveness of anticoagulation.

OUTCOMES OF PATIENTS WITH SEVERE AND CRITICAL COVID-19 INFECTION MANAGED WITH HEMOPERFUSION: A RETROSPECTIVE COHORT STUDY

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Aim: To determine the outcomes of patients with severe or critical COVID-19 infection who underwent hemoperfusion.

Background: As an emerging disease, various COVID-19 therapies have been proposed. During the pandemic, countries worldwide have utilized hemoperfusion for cytokine storm syndrome and have reported beneficial effects.

Methods: A single-center retrospective cohort was done by reviewing the electronic medical records of adult patients with severe or critical COVID-19. Comparative analyses were conducted using Chi-Square Test or Fisher’s Exact Test. Association between hemoperfusion and outcomes was analysed using univariate linear regression and binary logistic regression.

Results: A total of 100 patients underwent hemoperfusion for severe or critical COVID-19 infection. The patients in the hemoperfusion group showed significant improvement in IL6 levels than in control (28% vs 10%, p = 0.001). Hemoperfusion was not associated with improvement in length of hospitalization, length of intensive care unit stay, mortality, duration on a mechanical ventilator, oxygenation requirements, and other inflammatory markers: ferritin, C-reactive protein, lactate dehydrogenase, and D-Dimer (p > 0.05). The timing of hemoperfusion in terms of the day of illness had no significant effect on the outcomes (p > 0.05). The likelihood of secondary infection is 3.35 times less likely among those who had ≥3 hemoperfusion sessions compared to those with ≥4 sessions. The prevalence of bleeding was significantly higher (r² = 12.14, p = 0.003) among patients who had ≥4 hemoperfusion sessions (31.58%).

CONCLUSION: Hemoperfusion has been shown to decrease IL6 levels in patients with critical or severe COVID-19. It did not exhibit significant improvement in clinical outcomes and other inflammatory markers.

Hemoperfusion of four or more sessions has been linked to increasing the risk of secondary infection and bleeding complications.

INDOLENT MANTLE CELL LYMPHOMA OF RENAL SIGNIFICANCE: A CASE REPORT

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Background: Mantle cell lymphoma (MCL) is an aggressive but rare form of B cell lymphoma. Most extra-nodal disease involves the bone marrow, skin or gastrointestinal tract. Renal involvement is rare and occurs in the context of extensive aggressive or relapsed disease associated with systemic symptoms.

Case Report: A 62-year-old male was referred with stage 3 acute kidney injury (AKI) (serum creatinine 506 μmol/L) on routine three-monthly blood testing, in the absence of urinary symptoms, hypovolaemia, recent medication changes or systemic symptoms. He had underlying low-risk indolent MCL diagnosed from a lymph node biopsy 2 years earlier and was being managed with surveillance only. Notably, previous serial surveillance scans and a staging bone marrow biopsy performed just 1 month prior to the AKI episode showed ongoing indolent disease with stable cellularity.
We report potentially the first case of seemingly indolent MCL infiltration. This prompted initiation of chemotherapy with resultant remission and partial recovery of renal function, with a serum creatinine stable at 150-200umol/L on last follow up (2 years later).

**Conclusion:** We report potentially the first case of seemingly indolent MCL presenting with extensive tumour infiltration of the kidney and associated AKI in an otherwise systemically well patient. The discordance between indolence of cellularity and the aggressive renal infiltration underscores the importance of pursuing a high index of suspicion for renal involvement in patients with new renal impairment and indolent haematological malignancies, as timely diagnosis and treatment significantly improves patient outcomes.

## THE DILEMMA OF HYPOADRENALISM IN NEPHROLOGY: A CASE SERIES

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**Background:** Adrenal insufficiency is an uncommon disorder in patients with chronic kidney disease. It presents with non-specific symptoms that can be attributed to renal failure itself. Furthermore, significant blood pressure changes attributed to dialysis may hinder diagnosis. We highlight this diagnostic uncertainty in a case series of three patients with hypoadrenalism from a single centre.

**Case Series:** We present three cases from a large single centre of hypoadrenalism. All cases were in relatively younger women. All patients symptomatically and hemodynamically improved with steroid replacement therapy.

**Case 1:** Fifty-four-year-old woman on haemodialysis following bilateral nephrectomies. She experienced post-dialysis hypotension that did not improve despite increase in ideal body weight and intravenous antibiotics. Laboratory tests revealed low serum cortisol level. Histopathology revealed that an adrenalectomy was inadvertently performed at the time of right nephrectomy.

**Case 2:** Twenty-four-year-old woman with Joubert’s syndrome and juvenile nephronophthisis experienced recurrent episodes of shock secondary to peritoneal dialysis related peritonitis on a background of long-term corticosteroid therapy because of renal transplantation with graft loss. Significant hypotension prompted investigation for concomitant hypoadrenalism, which was confirmed by low serum cortisol level during a short synacthen test.

**Case 3:** A 27-year-old woman with juvenile nephronophthisis required vasopressor support for significant hypotension following months of diarrhoea, nausea, and light-headedness. She had previously required long-term prednisone as immunosuppressive therapy following renal transplantation. Unsuccessful crystalloid resuscitation and antibiotic therapy prompted a short synacthen test which demonstrated an inadequate cortisol response.

**Conclusions:** Adrenal insufficiency is under recognized in patients with renal disease who present with unexplained persistent hypotension, as symptoms are commonly attributed to the haemodynamic effect of dialysis. Steroid replacement improves symptoms and haemodynamic parameters.

## TREATMENT INTENSITY OF RENAL INPATIENTS DURING THEIR TERMINAL ADMISSION, A CLINICAL AUDIT

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**Aim:** To identify the intensity of interventions and treatment renal patients experience during their final hospital admission.

**Background:** Renal patients, particularly those on dialysis, are known to have high morbidity and mortality risks, and their illness trajectory often leads to death in hospital.

**Methods:** Utilizing electronic medical records and Rapid Response Team (RRT) data, we performed an audit of the terminal admissions of renal adult inpatients who died in hospital between January 2020 to September 2021 and their final 6 months of life. We collected demographic data and treatment information regarding their final 6 months as well as escalations of care during the terminal hospital admission prior to death, including time in intensive care, RRT data and number of invasive interventions such as surgery.

**Results:** We identified 90 patients who died in hospital during the specified period. The majority were on haemodialysis (51/90, 56.7%) and had over 3 comorbidities (56.7%). Of those not on dialysis at presentation, 32% (8/25) were initiated on dialysis. During the final admission, 52.2% (47/90) had a RRT call, 38.9% (35/90) an intensive care stay and 54.4% (49/90) underwent an invasive intervention. In the preceding 6 months, 44.4% (40/90) had had 2 or more hospital admissions and 23.3% (21/90) attended the emergency department 4 or more times. Only 10% had an advanced care directive prior to the final admission.

**Conclusion:** Our audit highlights the significant treatment and intervention burdens renal patients can experience towards the end of life. Being able to identify patients with terminal trajectories may facilitate advanced care planning and avoid recurrent hospital presentations and unnecessary interventions.
TWO-YEAR EFFICACY AND SAFETY OF RAVULIZUMAB IN ADULTS AND CHILDREN WITH ATYPICAL HEMOLYTIC UREMIC SYNDROME: ANALYSIS OF TWO PHASE 3 STUDIES

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Aim: To investigate the effect of fibroblast growth factor 23 (FGF23) on myoblast proliferation and differentiation.

Background: FGF23 is a bone-derived hormone whose signalling depends upon several factors, including FGF receptors (FGFRs), α-klotho coreceptor, and cell types. FGF23 levels are elevated in chronic kidney disease patients, and are linked to left ventricular hypertrophy and mortality risk. However, effects on skeletal muscle remain unclear.

Methods: Human skeletal muscle myoblasts were cultured in growth medium (SkGM™-2 Medium, Lonza) at 37°C in a controlled humidified 5% CO2 atmosphere. To induce differentiation into myotubes, upon 70%-80% confluence growth medium was switched to differentiation medium (DMEM) supplemented with 2% horse serum and 1% penicillin–streptomycin. The expression of FGFRs and key regulatory myogenic genes (MyoD, MyoG and MTSN) were measured using quantitative RT-PCR. For proliferation assays, undifferentiated human myoblasts were treated with vehicle, FGF23 (100 ng/mL), FGF2 (100 ng/mL) as positive control, uraemic toxin (indoxyl sulfate 1 mM and p-cresyl sulfate 0.2 mM) as negative control for 48 and 72 hr. Proliferation was measured in 96-well plates using the CellTiter-Glo® (Promega) and BrdU (Roche) assays.

Results: FGFR1, FGFR4 and α-klotho (in decreasing order) were expressed in human myoblasts and myotubes. FGF23 stimulated myoblast proliferation after 48 and 72 hr treatment, similar to FGF2, while uraemic toxins had opposite effects. At 48 hr of differentiation, compared to control, the expression of MyoD decreased significantly in cells treated with FGF23, FGFR2 and with similar trend in the uraemic toxin group. MyoG was significantly downregulated in the FGF2-treated group, with similar trends in FGF23 and uraemic toxin-treated groups. MSTN was significantly downregulated in all three groups.
Conclusions: FGF23 promotes myoblast proliferation but appears to inhibit myogenic differentiation.

RELATIONSHIP BETWEEN SARCOPENIA AND FRAILTY IN CHRONIC KIDNEY DISEASE: A CROSS-SECTIONAL STUDY

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Aim: To determine the relationship between sarcopenia and frailty in patients with chronic kidney disease (CKD).

Background: Sarcopenia is defined as loss of muscle mass, strength and/or performance while frailty is characterized by a reduction in functional reserve, with an increased risk of developing adverse outcomes upon exposure to stressors. Both sarcopenia and frailty are strongly associated with all-cause mortality. However, no study has assessed the coexistence of sarcopenia and frailty in CKD patients.

Methods: A single institution cross-sectional study was performed. Sarcopenia and frailty were assessed according to the standard diagnostic criteria. Sensitivity and specificity using sarcopenia as a predictor of frailty were calculated. Logistic regression was performed to assess the relationship between sarcopenia and frailty.

Results: Twenty-one healthy controls (mean age 42.0 ± 13.1 years), 26 CKD patients with estimated glomerular filtration rate between 15 and 90 ml/min/1.73 m² (mean age 68.9 ± 12.9 years) and 45 dialysis-dependent patients (mean age 67.4 ± 17.0 years) were included. None of the healthy control subjects were sarcopenic though one (4.7%) was frail. Four (15.4%) CKD patients had sarcopenia and 4 (15.4%) were frail; while 25 (55.6%) patients who were dialysis-dependent had sarcopenia and 18 (40.0%) were frail. The prevalence of frailty among those with sarcopenia was 58.6%; whereas the prevalence of sarcopenia among frail people was 73.9%. Sarcopenia showed a moderate sensitivity (73.9%) but a higher specificity (82.6%) for the diagnosis of frailty, with a negative predictive value of 90.5%.

Using multivariate logistic regression (adjusted for age, gender, diabetes and CKD), sarcopenia was significantly associated with frailty (OR = 5.88, 95% CI = 1.41, 31.5).

Conclusions: The absence of sarcopenia might be useful to exclude frailty but has limited utility as a biomarker of frailty.

MOLECULAR ALTERATIONS IN SKELETAL MUSCLE IN CHRONIC KIDNEY DISEASE: A SYSTEMATIC REVIEW

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Aim: The aim of this systematic review is to summarize the current evidence on molecular changes in the skeletal muscle of humans and rodents with chronic kidney disease (CKD) and to assess the strength of such evidence.

Background: Loss of skeletal muscle mass is prevalent among CKD patients and is associated with significant morbidity and mortality. The underlying molecular pathogenesis has yet to be fully elucidated.

Methods: The PubMed and EMBASE databases were searched using three main themes: messenger ribonucleic acid/protein/microRNA expression, skeletal muscle and CKD. This study was conducted in accordance with the PRISMA standards.

Results: Ninety-eight studies were included in the systematic review, comprising 26 prospective human clinical studies, 4 human and rodent studies, and 68 rodent-only studies (32 mouse and 36 rat). The sample sizes of human studies were predominantly small (73% of studies had ≤40 participants). Qualitative polymerase chain reaction (qPCR) was the most commonly used method for gene expression. None of the studies fulfilled the Minimum Information for Publication of qPCR Experiments (MIQE) criteria for quality of gene analysis. Most studies investigated only a few genes or a specific signalling pathway. The identified differentially expressed genes and proteins belonged to eight major pathways, including apoptosis, autophagy, inflammation, insulin/insulin-like growth factor 1 signalling, lipid metabolism, mitochondrial function, muscle cell growth and differentiation, and protein degradation, similar to other chronic disease states.

Conclusions: The current evidence regarding molecular alterations in the skeletal muscle in CKD is largely derived from small, heterogeneous studies. Nevertheless, markedly similar modifications in the major biological pathways between CKD and other chronic diseases supports shared deleterious molecular mechanisms producing muscle atrophy, irrespective of the underlying chronic disease.

GENERAL NEPHROLOGY—PREGNANCY ASSOCIATED

PARENTHOOD IN PEOPLE WITH KIDNEY FAILURE: EVOLUTION AND EVALUATION OF THE PARENTHOOD DATA COLLECTION OF THE ANZDATA REGISTRY

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Aim: This study aimed to further evaluate the parenthood event data reported to the Australia and New Zealand Dialysis and Transplant Registry (ANZDATA) over time, to determine whether expansion of the parenthood survey influenced reporting completeness and data quality.
Background: ANZDATA has collected parenthood event data since its inception, advancing our understanding of outcomes of pregnancies for this high-risk parent group. The data collection process was formalized in 2001 with a specific parenthood survey and subsequently expanded in 2017. We evaluated the parenthood event data to determine whether expansion of the parenthood survey influenced reporting completeness and data quality.

Methods: ANZDATA is a prospective, national registry collecting data on all dialysis and transplant patients in Australia and New Zealand annually. Descriptive statistics (absolute numbers and percentages) were used to quantify the completeness of data and compare parenthood surveys over time.

Results: The key finding was that the core data items were recorded with 100%, or near to, completeness across all data collection tools. Completeness of data reporting did not substantially change from 2001 to 2017 for items that were recorded in both surveys. The majority of additional items in the 2017 Parenthood survey, focused on dialysis intensity, graft outcomes, and medical complications during pregnancy, had less than 70% completeness of data.

Conclusion: This study was the first to analyse the data completeness of the ANZDATA parenthood survey. Our findings underpin the robustness of the data collection, but also highlight the need for more in-depth review of the data items and education of data collectors to support completion.

PREGNANCY OUTCOME IN DIALYSIS PATIENTS AT A LARGE MATERNITY CENTRE

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Background: Women with chronic kidney disease (CKD) are at increased risk of complications during pregnancy including requiring dialysis. Current guideline recommended intensive dialysis up to six times per week of dialysis and dialysis frequency increased with increasing gestational age for all patients. All patients achieved a pre-dialysis urea level of less than 12 mmol/L throughout their pregnancy. Only one patient was not dialysis dependent following pregnancy with an eGFR of 15 ml/min/1.73 m².

Conclusions: This study highlights the importance of frequent dialysis targeting a urea below 12.5 mmol/L to achieve successful pregnancies in women with advanced CKD. It also demonstrates how pregnancy in women with advanced CKD accelerates the commencement of maintenance dialysis.

GENETICS

WHOLE GENOME ANALYSIS OF THREE CHILDREN WITH FSGS AND POST-TRANSPLANT RECURRENCE

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Background: 20% of Childhood Nephrotic Syndrome is resistant to steroid therapy (SRNS) and of these, 50% will develop progressive chronic kidney disease leading to end stage within 5 years. In 20% of SRNS patients, monogenic causes (dominant and recessive) have been identified primarily in genes expressed in the podocyte. In the remaining cohort, aetiology is presumed immune-mediated with putative circulating factor(s) causing nephrotic syndrome. This theory is exemplified by risk of recurrence in gene-negative disease of >40%. Suggested circulating factors/pathways include both Th2 and ILC2 (Type 2 Innate Lymphoid Cells) pathways due to the association with allergy and Interleukin-4/Interleukin-13.

Method: We performed Whole Genome Sequencing (WGS) on three unrelated children with post-transplant recurrent SRNS where diagnostic gene panels had been uninformative. We re-interrogated the WGS using the 83 genes in the PanelApp knowledge base associated with proteinuria and then also using 17 genes associated with ILCs (Innate Lymphoid Cells).

Results: No disease-causing variants were identified in the genes associated with monogenic causes of nephrotic syndrome. About 183 rare variants were identified in 4 genes associated with ILC function: ST6GALNAC3, CDON, ATP8A2, and SLC27A6. However, no exonic or splice site variants were shared between the three unrelated children.

Conclusions: Aetiology of recurrent disease post-transplant in childhood SRNS remains poorly understood. Risk alleles or monogenic causes within the immune system could offer an explanation. This preliminary study did not reveal strong candidate variants within ILC genes but points to a new research focus that requires study.
WHOLE GENOME SEQUENCING REVEALS A NOVEL NPHP18 DELETION IN TWO COUSINS WITH CILIOPATHY

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Background: Nephronophthisis (NPHP) is typically an autosomal recessive disease of progressive interstitial kidney fibrosis and renal scarring. Over 20 genes have been identified as causes of NPHP with many involved in ciliary function. CEP83/NPHP18 was originally identified in seven families of children presenting with end-stage kidney failure between 1 and 4 years of age with associations including developmental delay, retardation, and hepatic fibrosis.

Case Report: The proband presented in end-stage renal failure before 2 years of age. He also had significant developmental delay. The pedigree is highly consanguineous from the Middle East. Targeted panel exome in 2014 was non-informative. Trio whole genome sequencing (WGS) (Garvan Institute) analysed using SEQR (the Center for Mendelian Genomics at the Broad Institute) revealed a novel homozygous 3 bp deletion in NPHP18, Nucleotide 12:94702642 2050–2052 del GAA; p. Glu684del. His first cousin, presented with a similar picture at less than 2 years of age. Both children received dialysis and subsequent kidney transplant, which has been complicated by vascular rejection in the proband and loss of his transplant requiring a second kidney transplant. Both also have cerebellar vermis hypoplasia and liver involvement in the proband.

Conclusions: Trio WGS was able to identify a novel pathogenic mutation in NPHP18, which broadens known variants and the clinical phenotype of known NPHP genes. This provided diagnostic certainty for the family and allows for potential future family planning.

OSTEOMALACIA FOLLOWING ANTI-RESORPTIVE THERAPY FOR OSTEITIS FIBROSA IN CKD-MBD

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Background: Chronic kidney disease—mineral and bone disorder is a well-recognized complication of advanced chronic kidney disease (CKD). This manifests as a spectrum of disease, with osteitis fibrosa being a consequence of high-turnover bone disease, and osteomalacia as a result of low-turnover bone disease. We report a case of biopsy-proven osteomalacia following denosumab administration in a patient with known osteitis fibrosa.
Case Report: A 43 year old male with end stage kidney disease on maintenance intermittent haemodialysis was referred for investigation following multiple atraumatic fractures. Background history was significant for Fabry’s disease and tertiary hyperparathyroidism without parathyroidectomy.

Initial investigations demonstrated corrected calcium of 2.24 mmol/L, phosphate 1.25 mmol/L, parathyroid hormone 86.7 pmol/L, 1.25 OH-vitamin D 110 pmol/L, bone-specific alkaline phosphatase 53.7 ug/L, procollagen type 1 n-terminal peptide 2219ug/L and c-terminal collagen type 1 teleopeptide >6000 ng/L.

Skeletal scintigraphy demonstrated exquisite uptake throughout the axial skeleton and peripheral joints, with patchy sclerosis throughout the pelvis and end plate sclerosis of the lumbar spine. Double tetracycline labelled bone biopsy was performed 5 weeks after 60 mg denosumab was administered for fracture prevention. Bone biopsy was compared with bone histopathology from both hips from previous fracture repairs. Earlier histopathology demonstrated increased osteoblastic and osteoclastic activity with peritabracular fibrosis consistent with osteitis fibrosa. Following denosumab, biopsy showed complete absence of osteoclastic activity, with markedly thickened osteoid and decreased mineral bone consistent with severe osteomalacia.

Conclusion: We present a case of iatrogenic osteomalacia following the administration of denosumab in a patient with atraumatic fracture history and osteitis fibrosa. The use of antiresorptives in patients with osteoporosis and advanced CKD should be considered carefully, as it may induce pathologic low-turnover bone disease without reducing fracture risk.

A SINGLE CENTRE STUDY REVIEWING PHOSPHATE BINDER PRESCRIPTION AND MINERAL METABOLITES IN MORNING AND AFTERNOON HAEMODIALYSIS SHIFTS

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Background: Hyperphosphataemia has been associated with increased mortality and cardiovascular events in CKD. Complicating phosphate management are the following issues: (1) the accuracy of serum phosphate given diurnal phosphate variability has been observed in the general population and haemodialysis patients, with unclear clinical impact upon phosphate binder prescription. (2) the impact of pill burden, maintaining a phosphate binder prescription without high exogenous calcium administration.

Aim: To review our current management of hyperphosphataemia in patients on haemodialysis; analysing time of measurement of mineral metabolites and impact on phosphate binder prescription.

Methods: Retrospective cohort study of patients on satellite haemodialysis at Western Health between December 2021 to March 2022. We collated baseline demographics, mineral metabolites and phosphate binder details on patients in either morning or afternoon sessions of haemodialysis. Exclusion criteria involved patients being managed by a private nephrologist due to insufficient data, or if on twilight dialysis.

Results: We reviewed 162 patients with 105 patients eligible for assessment. Mean age was 65 ± 13 years, 69% male, 46% with known ischaemic heart disease, 73% diabetic. 88% of patients were prescribed phosphate binders. These included calcium carbonate (45%), lanthanum carbonate (32%), sevelamer (35%), aluminium (1%), sucroferric oxyhydroxide (3%), with median six phosphate binder units (IQR3-8). Mean serum phosphate was 1.90 ± 0.67 mmol/L, with 33% of patients within normal range. Neither mean serum phosphate or phosphate binder prescription were different between morning and afternoon shifts.

Conclusion: Phosphate management comprises a high volume of pill burden with exogenous calcium administration comprising 45% of binders prescribed at our institution. Phosphate variability between timing of phosphate measurements did not result in a clinically significant difference in our cohort of patients.

HYPERPROLACTINAEMIA IN A HAEMODIALYSIS PATIENT AND FRACTURE EVALUATION BEYOND CKD-MBD: A CASE REPORT

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Background: Hyperprolactinaemia is common in patients with end stage renal disease, occurring in about 37–70%, with levels not affected by haemodialysis. However, significant elevation of prolactin should raise suspicion of pituitary abnormalities, which may impact on metabolic bone health.

Case Report: A 30-year-old man with severe tertiary hyperparathyroidism, presents with an atraumatic subacute right neck of femur fracture (NOF) after haemodialysis. Previous fractures include atraumatic contralateral subcapital NOF, multilevel vertebral crush fractures, and bilateral rib fractures. Initial investigations demonstrated a serum corrected calcium of 2.40 mmol/L, phosphate of 1.18 mmol/L and an alkaline phosphatase of 2740 units/L (reference value 30–110 units/L). 25-dihydroxy Vitamin D level was 30 nmol/L, and a parathyroid hormone level was unrecordably high at >212.0 pmol/L. He was managed on 120 mg of cinacalcet prior with intermittent non-compliance. A secondary osteoporosis screen was performed, which
revealed hypogonadotropic hypogonadism caused by hyperprolactinaemia of 5719 mIU/L and 4412 mIU/L pre- and post-dialysis, with undetectable macroprolactin. A pituitary MRI revealed a 4.6 × 5 mm pituitary microadenoma with no chiasmal compression. He commenced cabergoline 0.5 mg weekly. Subtotal parathyroidectomy was performed. To minimize hungry bone syndrome, he received calcitriol 0.75 mcg three times a day for 3 days prior, 2 mg of intravenous zolendronic acid, and 100 000 IU of intramuscular cholecalciferol. Nonetheless, he had protracted hypocalcaemia requiring 6 days of intravenous calcium infusion. He was discharged with calcitriol and calcium supplementation, and cabergoline weekly.

**Conclusion:** Profound hypogonadism should prompt further investigations of the pituitary-gonadal axis which can be suppressed by prolactin-producing pituitary adenomas in patients with end stage renal failure. Hyperprolactinaemia can potentially compound already deleterious effects on bone health.

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**OUTCOMES WITH INCREMENTAL HAEMODIALYSIS: A SINGLE CENTRE COHORT STUDY**

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**Aim:** To describe outcomes of patients being treated with Incremental Haemodialysis (IHD) at Austin Health.

**Background:** IHD involves adjusting the haemodialysis (HD) prescription according to measured residual kidney function. IHD is not widely practiced in Australia and the outcomes are not well characterized.

**Methods:** A retrospective and prospective audit was conducted from May 2018 to December 2021 comparing IHD to standard haemodialysis (SHD) or peritoneal dialysis (PD). Clinical criteria for IHD included measured residual urea clearance of greater than 2 ml/min, and capacity to comply with regular timed urine collections and adjustments to HD prescription.

**Results:** The audit included 95 patients with 31, 32 and 32 in the IHD, SHD and PD groups respectively. The median ages were 69 (IHD), 77 (SHD) and 68 (PD) years and the most common cause of kidney failure was diabetes. There were no significant differences in gender, ethnicity, cardiovascular disease, cancer and smoking status between groups. Patient survival at 3-years was IHD 93%, SHD 54%, PD 86% (NS, p = 0.066, log-rank). Median technique survival for IHD, defined as remaining on less or equal to 10.5 hours of HD per week was 8 months, lower than both SHD (31 months) and PD (19 months) (p = 0.0007, log-rank). Patients doing IHD received a median 9 h dialysis per week, compared to 12 h in the SHD group (p < 0.0001). However, mean total urea clearance (dialysis plus renal), measured as weekly Std Kt/V, was greater in the IHD group (3.4) than the SHD (2.4) and PD (2.3) groups (p < 0.0001).

**Conclusion:** In selected patients, IHD enabled less dialysis for a median of 8-months, with reassuring safety outcomes in terms of patient survival.

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**HAEMODIALYSIS—OTHER**

**CASE REPORT: RHINORBITAL MUCORMYCOSIS AND DESFERRIXAMINE—REVISITING AN OLD FOE IN PATIENTS RECEIVING DIALYSIS**

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**Background:** Mucormycosis is a highly virulent and rare invasive fungal infection seen in immunocompromised patients, diabetics, and patients with iron overload or on maintenance dialysis. Mucorales thrive in iron-rich environments and specific use of intravenous (IV) desferrioxamine markedly increases risk of infection and mortality. Mucormycosis is now rarely seen in dialysis patients since introduction of oral iron chelators (deferasirox), widespread ESA use reducing iron overload, and less prevalent aluminium toxicity.

**Case Report:** A 66-year-old female on dialysis for 12 years due to ADPKD with previous transfusion-dependent aplastic anaemia was receiving long-term IV desferrioxamine due to deferasirox intolerance for chronic iron overload. She presented with sudden-onset of unilateral facial pain and headache. Examination and investigations were unremarkable aside from left-sided cheek numbness and a C-reactive protein of 135. Broad-spectrum IV antibiotics were commenced, but over 24 h she developed a complex opthalmoplegia, propotis, diplopia, and eventual full visual loss. Repeat CT scan was unremarkable aside from enlargement of the left medial and inferior rectus muscles. A presumptive diagnosis of orbital mucormycosis was made. IV liposomal amphotericin B was commenced and she underwent debridement and orbital exenteration. Histopathology confirmed mucorales. Despite further debridement and IV antifungal therapy, an MRI 3 days post-exenteration demonstrated multifocal cerebral ischemia from angioinvasive rhinobitoral mucormycosis. Haemodialysis was withdrawn due to poor prognosis and she died 11 days after initial presentation.

**Conclusion:** Although Mucormycosis is rare, clinicians should carry a high index of suspicion in at-risk patient groups due the absence of clinical signs at presentation, and rapid progression to an often fatal outcome despite aggressive treatment. Importantly, desferrioxamine use and iron overload confer a markedly increased risk to dialysis patients.

**COVID-19 INCIDENCE IN HAEMODIALYSIS UNIT IN TULUS AYU HOSPITAL; A RURAL SETTING**

**DEDHY PRIMADONA MULIA1,2,3,4, RICO IRAWAN2, IDA TRIKANDIANI3, FIRDA ARIYANTI2, SUGIHARTONO SUGIHARTONO3, FAHRIZAL FAHRIZAL4, AMRIZAL AMRIZAL2**
PATIENT AND UNIT FACTORS ASSOCIATED WITH INPATIENT DIALYSIS INITIATION—A SINGLE UNIT RETROSPECTIVE COHORT STUDY

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Aim: To examine patient and unit factors resulting in unplanned dialysis starts, to determine factors that could be altered to improve patient outcomes.

Background: Commencing dialysis as an inpatient has been demonstrated to have negative impacts on patients both psychologically and physically. The available literature suggests that unplanned dialysis starts occur in 40%-60% of patients, and that increased age, increased comorbidity burden and cardiovascular disease are the most common associations with unplanned dialysis initiation.

Methods: We undertook a retrospective cohort analysis of all new dialysis starts in our unit from January 1 2017 to December 31st 2019. We used our inpatient database compared with ANZDATA registry information to examine the differences between patients who commenced dialysis as inpatients vs outpatients.

Results: 310 new patients commenced dialysis between 2017 and 2020. 171 (58%) of new start dialysis patients commenced dialysis as inpatients. Median length of stay was 8 days. 111 (64%) inpatients were Male. One-hundred and twenty-eight (76%) of inpatients commenced dialysis with a tunnelled or temporary line compared with 30 (21%) of outpatient starts. Twenty-seven (70%) of ATSI patients started as inpatients. Fluid overload was the most common reason for admission at 58(33.3%). Diabetes and IHD were commonly associated with inpatient starts. We also compared SEIFA indices of patients to show socio-economic factors that impact unplanned starts. Median SEIFA index for inpatients was 932 compared with 956 for outpatients.

Conclusions: Our study is a unique look at patient and unit factors on unplanned dialysis starts. Careful identification of risks will help us highlight areas our unit can target to reduce our rate of inpatient dialysis starts. This study has resulted in the creation of the dialysis transition group in our unit.

MANAGEMENT OF HAEMODIALYSIS PATIENTS UNDERGOING RADIOACTIVE IODINE THERAPY FOR THYROID CANCER

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Background: Radioactive iodine (RAI) is an established treatment for thyroid carcinoma following thyroidectomy. I-131 is predominantly excreted renally hence its clearance is reduced in patients with end-stage kidney disease (ESKD). There are, however, no established protocols for dosing in this population aimed at balancing treatment efficacy with red marrow toxicity. Studies in the dialysis population have modelled I-131 distribution in thyroid tissue and bone marrow, and its clearance from the body; however limited evidence exists to guide dosing, dialysis timing, or duration of isolation following the initial RAI dose. There is therefore a need to understand the real-world kinetics of I-131 in relation to haemodialysis, in order to optimize treatment efficacy whilst minimizing radiation hazard to the patient and dialysis staff.

Case Report: We report two haemodialysis patients undergoing RAI therapy for papillary thyroid carcinoma. A protocol was created by a multidisciplinary team comprising of medical physicists, nuclear medicine physicians, nephrologists and dialysis nursing staff. Modifications to haemodialysis included pre-treatment training in self-cannulation, implementation of haemodialysis in an unplumbed lead-lined room,
and safe isolation and disposal of haemodialysis equipment. I-131 dosing was individualized accounting for patient weight, residual renal function, as well as capacity to safely suspend dialysis.

**Conclusions:** Our protocol introduced the use of measuring I-131 levels in blood to monitor for red marrow toxicity, whilst one-meter dose-rate measurements were used to determine safety for release. Our report additionally provided a comparison between an anephric (bilateral nephrectomy) patient and a haemodialysis patient with residual renal function, characterizing the clearance of I-131 solely by haemodialysis. Our protocol may be useful in informing future RAI therapy in haemodialysis patients.

**HAEMODIALYSIS—VASCULAR ACCESS**

**AN UNUSUAL MISPLACEMENT OF A FEMORAL VASCULAR CATHETER IN A RENAL TRANSPLANT RECIPIENT**

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**Background:** Femoral vascular catheter misplacements are rare events more commonly reported in the paediatric population with potentially catastrophic sequelae. Left sided femoral catheters have been misplaced in the iliolumbar vessels, lumbar vessels and abdominal cavity. To our knowledge, this is the first reported case of a femoral vascular catheter tip positioned in the pelvis of a kidney transplant.

**Case Report:** A 62 year old female with a right iliac fossa renal transplant from 2007 was admitted with severe acute kidney injury and coronavirus disease 2019 (COVID-19) pneumonitis. A right sided femoral vascular catheter was inserted for continuous venovenous haemofiltration (CVVHDF) on admission in the intensive care unit under ultrasound guidance using the seldinger technique. No resistance was encountered at insertion and there was no difficulty aspirating or flushing the catheter. The catheter tip position was not confirmed radiologically. CVVHDF was commenced and patient appeared to receive adequate haemodialysis clinically and biochemically.

A computer tomography of the abdomen performed a week after catheter insertion revealed the catheter tip to be within the superior pole parenchymal vein of the kidney transplant. This was associated with a small renal cortical defect representing a renal infarct. The catheter was promptly removed and replaced. The patient remained dialysis dependent and subsequently passed away from severe COVID-19 pneumonitis.

**Conclusion:** Clinicians should be aware of this unusual complication in patients with femoral vascular catheters inserted ipsilateral to a kidney transplant. Ongoing vigilance of catheter malposition and catheter migration or displacement should be maintained. However, this may be challenging in an intubated and sedated patient with a denervated kidney transplant.

**A PROTOCOLISED APPROACH TO THE MANAGEMENT OF THROMBOSED ARTERIO-VENOUS FISTULAS AND GRAFTS**

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**Aim:** To assess the effectiveness of an implemented international guidelines based protocol for the management of haemodialysis patients presenting with thrombosed arterio-venous fistulas and grafts.

**Background:** Arteriovenous fistulas and grafts (AVF/Gs) are important forms of vascular access for patients requiring haemodialysis, but are prone to patency complications such as stenosis and thrombosis. This may adversely impact dialysis quality and patient morbidity and mortality.

**Methods:** A before and after retrospective observational cohort study was conducted of all incident haemodialysis patients presenting with thrombosed/stenosed AVF/Gs to Liverpool Hospital. Patients presenting 12 months before (2020) and 12 months after (2021) the protocol was implemented were included. Patients were assessed against key performance indicators (KPI) set as per guideline recommendations. Data were analysed with SPSS, and there was no blinding; all parties were engaged with change in practice prior to implementation.

**Results:** There were a total of 40 incidents of blocked access in 2020, and 59 in 2021. Comparison of data pre and post implementation of the protocol showed no difference in time to percutaneous intervention within 24 h (32% vs. 45%, p = 0.3), percentage of AVF/Gs with no attempt at de-clotting (38% vs. 31%, p = 0.7) and length of stay (3 days [IQR 2–10.5] vs. 3 days [IQR 1–8], p = 0.3). Initially there was a difference in the rate of vascaht insertion (55% vs. 23%, p = 0.02), but not after adjustment of group differences (p = 0.6).

**Conclusion:** The implemented protocol did not result in improvements in outcomes for haemodialysis patients presenting with blocked AVF/Gs. Further review of current practices, plan-do-study-act cycles and a quality-audit-reporting-system are needed by relevant stakeholders.

**INTERVENTIONAL NEPHROLOGY**

**THE LAST LINE: A CASE OF A TRANSHEPATIC CATHETER FOR HAEMODIALYSIS**

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Background: The use of transhepatic and translumbar catheters have been described for patients with no other conventional haemodialysis access options. We have previously employed the latter. Here we describe the first case of a haemodialysis patient in our unit who required a transhepatic line.

Case report: A 51 year old morbidly obese haemodialysis patient with MRSA bacteraemia secondary to an infected right thigh PTFE loop graft in the context of a history of access difficulties had the infected graft ultimately explanted, albeit complicated by a septic shower. A transhepatic (in preference over translumbar) line was inserted 2 days later by interventional radiology with a 31 cm 14.5 French Bard tunnelled haemodialysis catheter via the right hepatic vein with the tip positioned within the right atrium. Post procedurally she complained of pain, associated with a drop in haemoglobin by 13 g/L and an intra-hepatic haematoma (44 × 26 × 28 mm) demonstrated on a CT of her abdomen. She dialysed successfully through the transhepatic line on multiple occasions prior to her death 3 weeks after insertion.

Conclusion: Previously studies have demonstrated a high mortality in patients with hepatic lines which is more likely due to pre-existing patient factors, and a mean catheter-day survival that ranged from 27 to 141.2. Complications secondary to the insertions were low, and the main causes for catheter failure was due to thrombosis. In patients with limited access options or congenital inferior vena cava abnormalities the transhepatic approach remains a viable alternative for dialysis access as was demonstrated in this patient.

SETTING UP NATIVE KIDNEY BIOPSY: EXPERIENCE FROM REGIONAL AUSTRALIA

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Aim: To share the experience of setting up kidney biopsy program in a regional centre.

Background: Kidney biopsy remains a gold standard investigation when it comes to establishing a histopathological diagnosis in a large category of kidney diseases. Historically, patients from Mildura who required native kidney biopsy had to travel >500 kilometres to Melbourne for the procedure.

Methods: This prospective study describes the steps of setting up successful kidney biopsy program and clinical parameters including post-biopsy complications.

Results: The process of setting up included: (a) Planning—proposal of the procedure and credentialing of the Nephrologist, (b) Creating protocol and policy suitable for local health service in collaboration with all stakeholders involved, (c) Approval by the policy review committee, (d) Implementation and initiation of the procedure, (e) Regular in service education of the nursing staff involved in patient care, and (f) auditing the service provided. Since April 2021 till date, 12 native and one transplant kidney biopsy were performed. Bard max core disposable kidney biopsy guns were used for sampling. All biopsy samples were performed under real-time ultrasound guidance with sonographers’ assistance. Eight of the biopsies were performed in the inpatient setting. Patients were admitted for investigations under general medical team and referred to nephrology for further investigation when a renal pathology was suspected. Two of the inpatients were for the management of anticoagulants and one resided >100 km away from the health service. One patient had haematuria, which improved following rest. One had hypotension not related to internal haemorrhage and one had hypertension which improved with anti-hypertensives post procedure.

Conclusion: With good planning and policies, kidney biopsy can be safely performed in regional Australian setting.

LUMBAR ARTERY HAEMORRHAGE AND PSEUDOANEURYSM: A RARE COMPLICATION FOLLOWING RENAL BIOPSY

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Background: Lumbar artery puncture and haemorrhage following renal biopsy is an exceedingly rare complication of clinical significance. Only a few cases have been reported in the literature to date.

Case Report: We report a case of a 62-year-old man who underwent an ultrasound-guided percutaneous renal biopsy for suspected drug-induced tubulointerstitial nephritis. He initially presented in a hyperosmolar hyperglycaemic state precipitated by lower limb osteomyelitis. He was treated with a one-week course of high dose penicillin-based antimicrobial therapy and developed dialysis-dependent renal failure with a serum creatinine elevation from 150μmol/L to 833μmol/L. A standard 16-G Bard automated core biopsy gun was utilized under direct visualization with a curvilinear ultrasound probe. The left kidney was biopsied with two cores obtained after four passes. There were no immediate complications post biopsy.

The following day, the patient reported mild flank tenderness with a subsequent haemoglobin drop from 81 to 64 g/L. No superficial bruising or haematuria was noted. CT renal tract revealed a large left retroperitoneal haematoma approximately 10 × 5.5 cm axially and 24 cm
cruently from the perirenal space down to the left inguinal ring with associated displacement of the left kidney. Angiography studies identified arterial enhancement at left L2-3 level, representing a lumbar artery pseudoaneurysm and fistula between the lumbar artery and paired veins.

Successful embolization of the fistula was performed by interventional radiology via catheterization through the lumbar artery. This was achieved using 0.5 cc of 1:1 glue and lipiodol.

**Conclusions:** Extra-renal arterial injury is a rare but significant complication of renal biopsy. Angiographic imaging and embolization techniques can be effectively applied in management of significant bleeding.

**NUTRITION**

**UTILIZATION OF DIET CODES IN INPATIENTS WITH KIDNEY DISEASE ADMITTED TO VICTORIAN HOSPITALS**

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**Aim:** To compare the utilization of diet codes used for nutrition provision of inpatients with kidney disease in Victorian hospitals.

**Background:** Patients with kidney disease admitted to hospital are frequently prescribed a therapeutic diet code to align nutrition provision with their clinical needs. These include renal, low sodium, low potassium and low phosphate diets. Renal diets have traditionally been restrictive in nature with an inability to target individual micronutrient requirements.

**Methods:** Renal dietitians working within Victorian hospitals participated in a survey distributed via the Dietitian’s Australia Victorian Renal Interest Group. Quantitative data collected included number and types of diet codes in hospital food service systems for inpatients with kidney disease. Qualitative data included compliance specification measures used by each health service and their clinical application. Results across different hospitals were compared.

**Results:** Surveys were distributed to 15 health services with nine responding. Of these, 33% offered all four diet options; 77% of services offered a renal diet, 77% low sodium, 66% low potassium and 44% low phosphate diets. Reported compliance specifications found six services utilizing pre-existing guidelines with three references identified. Two services modified these compliances. One service did not use a guideline and two services used modified versions of non-renal diet codes. Comments on diet code clinical application included renal diets being too restrictive for patients and guidelines not updated for current clinical evidence.

**Conclusions:** Use of diet codes to manage nutrition provision of inpatients with kidney disease is inconsistent across Victorian Hospitals. Development of guidelines to support the clinical application of standards would assist in providing consistency of nutrient provision and allow for individualisation of micronutrient targets as recommended by current clinical evidence.

**GLOBAL RENAL INTERNET COURSE FOR DIETITIANS (GRID COURSE) SUPPORTS ADVANCED NUTRITION TRAINING TO IMPROVE OUTCOMES OF PATIENTS WITH CHRONIC KIDNEY DISEASE**

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**Aim:** To develop and implement advanced nutrition training for clinical dietitians globally.

**Background:** Chronic kidney disease (CKD) is a public health problem worldwide. Nutrition is integral to medical management for both prevention to treatment of all stages of CKD including transplantation. To improve outcomes for renal patients globally, standardized advance training for dietitians is vital to increase their knowledge and understanding, especially in lower-to-middle-income countries (LMIC) where access to training is limited.

**Methods:** The GRID committee was formed following a meeting in Wurzburg, Germany in 2014. It was led by an expert physician in nephrology nutrition who collaborated with specialist renal dietitians from the United States, Australia and Malaysia. Consultations were also sought with relevant stakeholders to develop the course structure and curriculum.

**Results:** This state-of-the-art course comprised three modules developed for dietitians of all levels of expertise in renal nutrition, from gaining new skills to enhancing sophistication of practice. Module I, which is the fundamentals of renal nutrition and metabolism; and Module II, adapting the Nutrition Care Process (NCP) of dietetic practice were launched in 2020 and 2021, respectively. Module III, which focuses on implementation skills with practical case studies, is currently underway for expected launching by the end of 2022. The three modules are delivered by 70 international faculty experts for 80 webinars, addressing regionocultural nutrition issues as well. The National Kidney Foundation (NKF) of the United States provides administrative and technical support to deliver the course on-line. To date, participants from more than 21 countries have enrolled in this course.

**Conclusions:** The GRID course providing global access facilitates capacity building for dietitians in specialist renal care.

**HEALTH LITERACY IS ASSOCIATED WITH NUTRITION KNOWLEDGE IN CAREGIVERS OF PATIENTS WITH CHRONIC KIDNEY DISEASE**

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¹—

ASSESSMENT OF DIETARY MACRONUTRIENT AND MICRONUTRIENT INTAKE AMONG HAEMODIALYSIS PATIENTS IN SRI LANKA

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Background: Malnutrition is very common among haemodialysis (HD) patients.

Aim: We aimed to assess the macronutrient and micronutrient intake and determined the association between nutritional intake and socio-economic status among HD patients.

Methods: This is a descriptive cross-sectional study which is conducted among 305 HD patients at National Hospital, Kandy, Sri Lanka. Interviewer administered, semi-structured questionnaire was used to collect data from the patients. Nutritional data was collected using 24-h dietary recall method. Statistical analysis was performed via SPSS version 20. Ethical approval was obtained from Ethics Review Committee, Faculty of Allied Health Sciences, University of Peradeniya.

Results: The mean age of the study participants was 50.45 ± 13.063 years and majority were males (69.2%; n = 211). Only 1.6% (n = 5) of the patient fulfilled the recommended calorie intake per day (>30 Kcal/day). Majority of the patients had more than 60% of the calorie intake from carbohydrates (n = 232; 76.1%). Overall, energy, protein and fat intakes in 98.4%, 96.1% and 91.8% of the participants were less than the recommended values. All the micronutrient intake amounts were in less than the recommended level. Significant differences found between micronutrients based on the monthly income. Such as, dietary PUFA (p = 0.021), cholesterol (p = 0.002), vitamin E (p = 0.018), vitamin B2 (p = 0.000), vitamin B6 (p = 0.008), folic acid (p = 0.004), sodium (p = 0.000), potassium (p = 0.010), calcium (p = 0.001), magnesium (p = 0.038), phosphorus (p = 0.000), iron (p = 0.042) and zinc (p = 0.003).

Conclusions: There is a wide gap between recommended nutrient intake and the actual nutrient intake of this study patients. Nutritional status and other clinical measures were influenced by patients’ age and monthly income. Regular optimal nutritional counselling and monitoring is very important to improve their outcomes. Introduction of socio-economical dietary guidelines can be warranted.

RELATIONSHIP PROTEIN ENERGY-WASTING AND PSOAS MUSCLE INDEX IN CHRONIC KIDNEY DISEASE PATIENTS

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Background: PEW, sarcopenia, and frailty are commonly seen in advancing CKD and have been linked with adverse outcomes in various studies. Psoas Muscle index (PMI) has been shown to be effective in predicting sarcopenia in CKD patients. This study was conducted to see the relationship between PEW and PMI.

Methods: This was a cross-sectional study and included diagnosed cases of CKD who were attending Nephrology outpatient care. The protein-energy wasting (PEW) was diagnosed by the criteria proposed by The International Society of Renal Nutrition and Metabolism (ISRNM). Psoas Muscle Index (PMI) was calculated on a CT scan.

Results: A total of 90 patients were included. The mean age of the study population was 51.81 ± 14.61 yrs., 68.9% were male, mean BMI was 19.85 ± 3.20 Kg/m2. Male had significantly more PMI compared to females (4.512 ± 1.929 vs. 3.229 ± 1.231, p = <0.001). Age had strong relationship with PMI (r = −0.375, 95% CI −0.539 to −0.180,
USE OF A VERY LOW CALORIE DIET FOR WEIGHT LOSS IN A PATIENT WITH ACUTE KIDNEY INJURY—A PATIENT CENTRED-APPROACH TO WEIGHT LOSS DURING A PROTRACTED HOSPITAL ADMISSION

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Background: Very low calorie diets (VLCD) have been used safely for weight loss in chronic kidney disease, despite previous concerns about further damage to kidney function through increased protein catabolic load, diuresis, and risk of electrolyte derangements. However, no data is available to guide the use VLCDs in acute kidney injury (AKI). This case study reports on successful VLCD use in the acute setting in a patient with AKI and class III obesity with comorbid complications.

Case Report: A 45-year woman (271 kg; BMI 86 kg/m²) attended hospital following a fall. Developing complications attributed to obesity and preventing discharge including immobility, deconditioning, fluctuating renal function and AKI, fluid overload, oxygen desaturations and hypoxia. A VLCD was initiated for rapid weight loss despite the manufacturer-stated contraindication for AKI. VLCD adjustments were made to account for the patient’s higher requirements, fluid and salt restrictions. Daily medical and twice weekly dietetic reviews were made to ensure safety of the VLCD program. Electrolyte levels were monitored daily until AKI resolved at VLCD week 5 and thereafter with high regularity as the patient remained on diuretics. No adverse side effects regarding electrolytes, fluid, or kidney function were observed. The VLCD program was continued for 13 weeks of admission, with various adjustments undertaken in consultation with the patient to address weight plateaus and promote adherence. The patient was discharged home after 6 months of hospitalization, having attained a 76 kg weight loss (BMI 62 kg/m²).

Conclusions: VLCD may be safe for use in patients with AKI during hospitalization under close medical supervision. Seizing the opportunity of protracted hospital admission to address obesity can improve overall patient outcomes.

EXPLORATION OF RENAL OUTPATIENT REFERRAL PRIORITIZATION IN VICTORIAN HOSPITALS

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Aim: To explore the clinical prioritization of dietetics referrals for outpatients in Victorian Hospital Nephrology services.

Background: The KDOQI Clinical Practice Guideline for Nutrition recommends medical nutrition therapy across all stages of kidney disease and post transplantation to optimize nutrition status. Nutrition requirements are varied depending on the stage of kidney disease, and presence of other co-morbidities. Access to dietetic services is inconsistent and clear criteria for clinical prioritization is lacking. Effective triaging of referrals ensures timely access to nutrition care for patients most at risk.

Methods: A benchmarking survey was distributed via email to renal dietitians working in Victorian public health services. Participants ranked 11 dietetic referral indications based on their local renal nutrition ambulatory services. Options included a priority rating of 1–3 (1 being the highest clinical prioritization and 3 the lowest) and ‘unlikely to be seen’. Qualitative data relating to the triage process were also collected.

Results: The response rate to the survey was 53% (n = 8). Malnutrition and hyperkalaemia were most frequently rated as priority 1 (75%), with the lowest priority rating given to pre-transplant education (25% as Priority 3 and 75% as unlikely to be seen). Other referral indicators for therapeutic diet educations were ranked across all domains with low agreement between health services. Subjective data noted that a patient’s overall clinical presentation informs referral prioritization (50%) and resource limitations are a barrier for access to nutrition service (50%).

Conclusions: Dietetic prioritization of nutrition needs for patients with kidney disease in ambulatory care was broadly consistent across services. A comprehensive evaluation of service delivery including barriers and facilitators to access to care may better inform future service innovations.

PAEDIATRICS

ALPHA LIPOIC ACID THERAPY IS EFFECTIVE IN A PERSONALIZED MOUSE MODEL OF CYSTINURIA

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Aim: To evaluate the effect of alpha-lipoic acid (α-LA) in a personalized model of type II cystinuria.

Background: Cystinuria is an inheritable genetic disorder characterized by the accumulation of cystine in the urine. Cystinuria accounts for 1%-2% of all adult kidney stones and 3%-10% in children. Type II cystinuria is caused by mutations in the SLC7A9 gene that is primarily expressed in the proximal tubules. To test the effectiveness of therapeutics in cystinuria, cystine transporter gene deficient (−/−) mice have been developed. However, these models do not represent the clinical situation, where point mutations cause cystinuria. As cystinuria is a rare disease, running clinical trials is challenging. Therefore, creating personalized models of clinically relevant point mutations may afford individualized approaches. α-LA has been shown to be an effective treatment in an experimental gene-deficient mouse model of type I cystinuria and has entered clinical trials. We sought to test α-LA in a personalized model of type II cystinuria.

Methods: We have identified a pathogenic mutation in a severe early-type II cystinuria. α clinical trials. We sought to test α-LA dietary supplementation in reducing cystine stone formation using computed tomographic (μ-CT) imaging.

Results: 100% of male Slc7a9G105R mutant mice developed cystine stones by 9-weeks. α-LA treatment significantly reduced stone number, volume, and weight with 80% of α-LA-treated mice presenting with no stones.

Conclusions: Our findings prove that the development of translational personalized models of monogenic kidney stone diseases are feasible, and that α-LA is effective in preventing type II cystinuria. The crucial next step is to take α-LA into broader clinical trials, including in paediatrics.

BASELINE CHARACTERISTICS OF PARTICIPANTS IN THE NAVKIDS2 TRIAL: A PATIENT NAVIGATOR PROGRAM IN CHILDREN WITH CHRONIC KIDNEY DISEASE

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Background and Aim: Children with chronic kidney disease (CKD) require multidisciplinary care to meet their complex healthcare needs. Patient navigators are trained non-medical personnel who assist patients and caregivers to overcome barriers to accessing health services through care coordination. This trial aims to determine the effectiveness of a patient navigator program in children with CKD.

Methods: The NAVKIDS2 trial is a multi-center, waitlisted, randomized controlled trial of patient navigators in children with CKD.
Conclusion:
The NAVKIDS2 trial is designed to evaluate the effectiveness of tacrolimus IPV in pediatric KTR patients. The trial completed recruitment in October 2021 with expected completion of follow-up by October 2022. There were 162 patients enrolled with 80 and 82 patients randomized to the immediate intervention and waitlisted groups, respectively. Fifty-eight (36%) participants were from regional/remote areas, with a median (IQR) age of 9.5 (5.0, 13.0) years, 46% were of European Australian ethnicity and 65% were male. A total of 109 children (67%) had CKD stages 1–5, 42 (26%) were transplant recipients and 11 (7%) were receiving dialysis.

**Conclusion:** The NAVKIDS2 trial is designed to evaluate the effectiveness of patient navigation in children with CKD from families experiencing socioeconomic disadvantage.

**TACROLIMUS INTRA-PATIENT VARIABILITY AND ITS EFFECT ON ACUTE REJECTION AND DEVELOPMENT OF DONOR SPECIFIC ANTIBODIES IN PAEDIATRIC KIDNEY TRANSPLANT RECIPIENTS**

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**Background:** Adequate immunosuppression is essential for long-term graft outcomes after kidney transplantation (KT). High intra-patient variability (IPV) in tacrolimus trough levels (TTL) is associated with acute rejection, graft loss, and de novo donor-specific antibody (dnDSA) development in adult KT recipients (KTR).

**Aim:** Our study aimed to determine the effect of tacrolimus IPV on acute rejection and dnDSA development in pediatric kidney transplant recipients (KTR).

**Methods:** This retrospective cohort study evaluated pediatric KTR patients at The Children’s Hospital at Westmead. We collected TTL 1–5 years post KT and calculated coefficient of variance (CV = standard deviation/mean × 100). We examined its association with acute rejection and dnDSA development using Cox-proportional hazard models.

**Results:** Thirty pediatric KTR (16 males; 54%) with a mean age of 6.0 (s.d.) (4.0) years were included. Of 28 patients, acute graft rejection occurred in 6 patients (21%). The mean tacrolimus CV was 43%(18.6%) compared to 30%(8.1%) in patients who did not experience acute rejection. Univariate cox-proportional hazards model found a 1% increase in tacrolimus IPV resulted in a 7% increased risk of acute rejection (HR: 1.07, 95% CI: 1.02–1.14). dnDSA were detected in 13 (48%) of 27 patients. The mean tacrolimus CV was 36%(16%) compared to 28%(8.3%) in patients who did not develop dnDSA. Univariate cox-proportional hazards models found a 1% increase in tacrolimus IPV resulted in a 3.0% increased risk of dnDSA development (HR: 1.03, 95% CI: 0.99–1.07).

**Conclusion:** Acute rejection in pediatric KTR is associated with increased levels of tacrolimus IPV. An association between development of dnDSA and high tacrolimus IPV was not demonstrated in this study.

**UNUSUAL CAUSES OF ANASARCA AND RESPONSE TO USUAL IMMUNOSUPPRESSIVE MEDICATIONS**

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**Background:** Immunosuppression treatment plays no role in genetic and developmental kidney diseases. Reporting three different non-immune causes of anasarca and proteinuric kidney diseases and their symptomatic improvement & remission with medications.

**Cases:** Presenting three cases, all girls, P1, P2, and P3, aged 7, 2 and 1.5 years, weight 50th, 90th, 50-75th centiles and height 75th, 90th, and 50–75th centiles respectively, referred for generalized swelling and proteinuria. They all had serum albumin <2 gm/dL and normal kidney functions. P1 & P3 had 6 weeks, 2 mg/Kg of corticosteroids course was prior. P1 also had partial bilateral elbow extension difficulty since birth but doing all routine work. The family denied genetic and biopsy testing initially. With a clinical diagnosis of steroid-resistant NS, tacrolimus started and remained in remission for 2 years. Her proteinuria reappeared after stopping tacrolimus. Histopathology done showed FSGS, while genetics revealed LMX1B c.668G > A on exon 4 heterozygous AD mutation. P2 had gross haematuria too. She responded well to initial corticosteroid therapy. But intermittent proteinuria and episodes of haematuria episodes persisted for the next 3 years. Biopsy suggested features of Alport syndrome. Clinical exome analysis reports COLA 4AS c.2678-42A > G heterozygous X-linked dominant heterozygous. Both parents were negative for the mutation. Ear and eye examinations were normal. P3 was referred for biopsy but deferred as ultrasound suggested crossed fused ectopic kidney. DMSA nuclear imaging showed no scaring. During the period of waiting for USG report, she was started on tacrolimus but continued later as she achieved remission. Twice tried tapering and stopping tacrolimus but anasarca reappeared so required to restart therapy again.

**Conclusion:** More data and studies are required to understand non-immune causes of proteinuria.
IS RESIDUAL URINE OUTPUT ASSOCIATED WITH THE BETTER PERITONEAL DIALYSIS OUTCOMES IN A SINGLE CENTRE?

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Background: Urine volume (UV) has been shown to be positively correlated with improved outcomes and a reduction in the relative risk of death in patients on peritoneal dialysis (PD). For the 2013–2018 period, St Vincent’s Hospital Melbourne (SVHM) had superior patient and PD technique survival compared to the national average on the ANZDATA Centre Report.

Aim: To provide insight into the SVHM PD patient cohort, with a focus on residual urine output as a potential explanation for the difference in outcome.

Methods: A retrospective analysis was conducted on PD patients at SVHM who commenced PD between January 2013 and September 2020. Patient characteristics and the UV recorded on PD adequacy tests at <12, 12–24 and >24 months after commencing PD were analysed. Peritonitis rates were also examined.

Results: 214 patients were included in the demographic analysis. The average age at commencement was 66 ± 14.5 years. The mean urea, calcium, phosphate, and haemoglobin were 27.5 ± 11.5 mmol/L, 2.32 ± 0.21 mmol/L, 1.82 ± 0.52 mmol/L, and 107 ± 14 g/L, respectively. About 186 patients had a PD adequacy test recorded. The average 24-h UV was 1192 ± 665 ml at <12 months after commencing PD, 887 ± 592 ml and 964 ± 638 ml, at 12–24 months and >24 months, respectively. UV was similar between patients who had an episode of peritonitis and those who did not.

Conclusions: With demographic and biochemical data of the SVHM cohort being comparable to the national average, it is possible that high residual UV of our PD cohort may be associated with the better outcomes. We should not underestimate the appeal of using UV in ESRD as a simple, measurable, comparable metric to help guide the commencement of PD if better outcomes can be achieved.

PLASMID ACQUIRED ANTIMICROBIAL RESISTANCE IN STAPHYLOCOCCUS EPIDERMIDIS IN A PERITONITIS CASE

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Aim: To sequence the genomes of three longitudinal S. epidermidis isolates (C099, C100 and C101) obtained from a patient with peritonitis.

Background: Coagulase-negative staphylococci such as S. epidermidis are common colonizers on human skin, however as opportunistic pathogens they can cause serious infections. S. epidermidis is resistant to a range of antibiotics such as methicillin and tetracycline and most antimicrobial resistance (AMR) genes are plasmid encoded. Plasmids can facilitate the rapid spread of AMR between bacteria and we have observed this in S. epidermidis isolates obtained from a treatment resistant peritonitis case.

Method: C099 was isolated from the patient on day 1 of infection, C100 was obtained on day 6 and C101 was obtained on day 15. DNA was extracted using lysostaphin lysis of cells followed by the use of the Monarch® Genomic DNA purification kit. Extracted DNA was checked for quality and sequenced using the Illumina NextSeq and Oxford Nanopore MinION. Replicons were confirmed using gel electrophoresis. Genomes were assembled using long nanopore sequence reads using Flye. Final assemblies were polished with both long reads using Racon and with short reads (Illumina) using Pilon. The broth micro-dilution (BMD) method was performed using Sensititre™ Custom plates according to manufacturer’s instructions (Thermo Scientific™).

Results: A total of five plasmids were identified in C099 and six in C100 and C101. Sequencing confirmed the additional plasmid in C100 and C101 contained the ermC gene. Both C100 and C101 were resistant to erythromycin using the BMD method.

Conclusions: The rapid acquisition of erythromycin resistance highlights the issue of increasing AMR in bacteria. Sequencing can be used for surveillance and to further study the emergence of AMR in peritonitis cases.

POINT OF CARE PERITONEAL DIALYSIS: INITIAL USABILITY ASSESSMENTS

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Aim: To assess the usability of a novel affordable dialysis system that produces peritoneal dialysis (PD) fluid at the point-of-care.

Background: The Ellen Medical Devices Point-Of-Care (EM-POC) system can be used by patients and care-givers to produce PD fluid from potable water at the point-of-care. Early identification of potential use errors is important within the medical device design process. IEC 62366-1 is a medical device human factors engineering process standard used to establish the safety of
medical devices with respect to usability. Formative evaluations are completed iteratively, early and often throughout an optimal design process, with feedback used to improve device design. We present results from early formative evaluations which have informed the ongoing design and development of the EM-POC system.

**Methods:** Formative human factors evaluations compliant to IEC 62366–1 were completed with an experienced PD patient, PD nurses and internal company personnel:

1. Participants assessed usability of a prototype device when used to complete PD bag fills with sterile water produced by the EM-POC system at home (n = 4).
2. Participants completed tasks and provided opinions regarding aspects of design and usability through online evaluations using images of the prototype and software simulation of the user interface (n = 3).

**Results:** The most common potential harm identified was concern over contamination risk leading to peritonitis. Resulting design adjustments have included:

1. Tactile and auditory feedback during connection of the PD bag
2. Improved geometry and visibility of the connection port
3. A single step connection process
4. Prohibited distilling if the connection is incomplete or incorrect

**Conclusions:** Early formative evaluations have successfully identified potential use errors enabling design refinement and improvement prior to further clinical evaluation.

**TRANSPLANTATION**

**SYSTEMATIC REVIEW AND META-ANALYSIS ON THE EFFECT OF BLOOD TRANSFUSIONS ON KIDNEY TRANSPLANT OUTCOMES: SHINING NEW LIGHT ON AN AGE-OLD DEBATE**

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**Aim:** Determine the effects that blood transfusions have on kidney transplant outcomes.

**Background:** The use of transfusions in kidney transplant medicine has long been a point of contention. While they are a solution to the anaemia plaguing this patient population, they can also induce a sensitized state, increasing the risk for poorer graft outcomes. Recently, however, with the advent of tacrolimus and with transfusions primarily being given in the post-transplant period only when they are clinically indicated, transfusions may not pose the same risk. No meta-analyses have been published including these newer studies. Therefore, we believed it prudent to undertake a systematic review and meta-analysis exploring the effect transfusions have on transplant outcomes.

**Methods:** A systematic search was conducted using MEDLINE, Embase, Cochrane library and Clinicaltrials.gov. Including only English articles published between 1/1/2000 and 01/03/2022, we found nine studies that met our inclusion criteria. A meta-analysis was thereby carried out comparing delayed graft function, the development of de novo DSA, and antibody mediated rejection.

**Results:** Low-level evidence exists for no difference or an increase in DGF and AMR for participants who received at least one blood transfusion. Low-level evidence exists for an increase in AMR for participants who received at least one blood transfusion. No study reported any improvement with blood transfusion usage.

**Conclusion:** The transfusion effect appears not to exist in the 21st century even in patients receiving postoperative blood transfusions. However, while transfusions are avoided wherever possible, transfusions in kidney transplant recipients do not appear to be associated with any worse outcomes compared to their non-transfused counterparts. While more research is required, clinicians may be able to administer blood transfusions to these patients with a clear conscience.

**TWO-YEAR FOLLOW UP OF CKD-MBD POST-TRANSPLANTATION: A SINGLE CENTRE STUDY**

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**Background:** Chronic kidney disease mineral and bone disorder (CKD-MBD) can persist post-transplantation and is associated with increased fracture risk. CKD-MBD and fracture screening post-transplantation is variable and specific management remains uncertain.

**Aim:** To assess the prevalence of CKD-MBD post-transplantation and evaluate our practice pertaining to fracture risk stratification and treatment.

**Methods:** We conducted a retrospective study of patients (n = 88) who underwent kidney transplantation at Monash Health in 2018 with a 24-month follow-up. Demographic, biochemical and dual-energy X-ray absorptiometry (DXA) results were obtained from electronic medical records.

**Results:** Baseline eGFR was 7 ml/min (SE ± 0.34), increasing to 52 ml/min (SE ± 2.00) and 55 ml/min (SE ± 2.23) at 12 and 24 months respectively. Baseline mean parathyroid hormone (PTH) level was 45.2 pmol/L (SE ± 3.26), improving to 14.4 pmol/L (SE ± 1.60) and 14.2 pmol/L (SE ± 1.49) at 12 and 24 months. Mean alkaline phosphatase (ALP) improved from 116 IU/L (SE ± 6.41) to 112 IU/L (SE ± 4.58) and 95 IU/L (SE ± 3.77) at 12 and 24 months respectively.

At 24 months, 34 patients (38%) had persistent secondary hyperparathyroidism (SHPT) post-transplantation. Fifty-two patients (59%) received a DXA and of those, 40 patients (77%) had osteoporosis at the femoral neck. However only 7 patients (13%) received anti-fracture therapy. Management of SHPT was variable: 17 patients received cholecalciferol, whilst 2 patients received cinacalcet and 3 patients received calcitriol.
Conclusions: SHPT persists in a significant proportion of patients. 59% of patients had a fracture risk assessment and of those 77% had osteoporosis. Specific anti-fracture therapies were only utilized in 13% of patients. Better protocols for fracture risk stratification and management are needed.

RAPIDLY PROGRESSIVE CALCIPHYLAXIS IN A RENAL TRANSPLANT PATIENT

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Background: Calciphylaxis can be categorized into uremic, classically seen in patients with end stage kidney disease (ESKD) and non-uremic, which is calciphylaxis in patients with normal renal function or early stages of kidney disease. The latter is associated with a 1-year mortality between 25% and 50%.

Case Report: We present a 63-year-old female of Caucasian background with the primary disease of familial glomerulonephritis. She had haemodialysis for 10 years prior to transplantation in 1990 (31 years ago). She had a baseline creatinine between 150 and 250 μmol/L and her transplant was managed with tacrolimus, azathioprine and prednisolone. Of note she had previously had a total parathyroidectomy prior to transplantation. She presented with multiple necrotic, extremely painful indurated lesions over shins, calves and buttocks, with some areas of eschar. This coincided with delirium in the context of hypercalcaemia (corrected calcium of 3.26 mmol/L). Renal function deteriorated with a creatinine peak to 300 μmol/L. She had normal phosphate (0.84 mmol/L) and PTH (0.9 pmol/L). Biopsy of the lesions returned with small arterioles with coarse calcification within their walls, in keeping with calciphylaxis. Warfarin (for stroke prevention in the setting of atrial fibrillation) and calcium supplement was ceased and dialysis commenced with supplemental sodium thiosulphate. Despite maximal treatment, the necrotic lesions continued to progress in size and subcutaneous nodules increased in number leading to a bed bound state. She was transitioned to comfort care within 3 weeks post re-commencement of dialysis to optimize biochemistry.

Conclusion: Calciphylaxis is associated with a high mortality rate, including patients with renal transplantation.

LEFLUNOMIDE USE IN KIDNEY TRANSPLANT RECIPIENTS WITH BK VIRUS INFECTION

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Aim: BK virus infection is an increasingly common clinical challenge encountered in kidney transplantation. The current universally accepted management approach is reduction of immunosuppression. We present our local experience in managing BK infection with leflunomide, a pyrimidine synthase inhibitor, when initial reduction of immunosuppression is unsuccessful.

Methods: This study includes kidney transplant recipients managed through the Kidney Health Services at the Royal Brisbane and Women’s Hospital as of September 2021. All patients with detectable serum BK virus DNA were included. BK titres at the time of diagnosis and transplant kidney biopsy results were collected. Clinical and pharmacy dispensary records were reviewed to identify patients treated with leflunomide. Outcomes were collected for leflunomide treated patients including suppression of BK titres to below detectable levels, persistent reduction in titres, or graft loss.

Results: Of the 241 kidney transplant recipients reviewed; 32 patients (13%) had a positive serum BK result. BK titre at diagnosis ranged from 13 to 160 000 copies/mL. Transplant kidney biopsy was performed on 12 patients (38%) with BK viraemia with six renal biopsies (19%) demonstrating concurrent BK nephropathy. Eleven BK-positive patients (34%) received leflunomide of which four (36%) had BK titres persistently reduced to ≤350 copies/mL. Five leflunomide treated patients (45%) achieved BK titres below undetectable levels and graft loss from BK nephropathy occurred in two leflunomide treated patients (18%).

Conclusion: Leflunomide may promote the suppression of BK viraemia in kidney transplant recipients with BK virus infection unresponsive to initial reduction of immunosuppression.

EARLY TRANSPLANT RENAL ARTERY STENOSIS: COULD DONOR ARTERIES BE TO BLAME?

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Background: The incidence of transplant renal artery stenosis (TRAS) is between 1% and 23%. Whilst TRAS can occur anytime, it most commonly occurs between 3 months and 2 years post-transplantation. We present a case series of three patients with early TRAS and explore the impact of anatomical quality of donor and recipient vessels.

Cases: Between 2017 and 2021, three renal allograft recipients aged between 49 and 55 experienced hemodynamically significant TRAS. The intraoperative findings reported that the arterial vessels of all three recipients were of good calibre with no macroscopic atheroma and all patients had a satisfactory end-to-side anastomosis. All three donors allografts had a single renal artery, but in one case a small calibre renal artery was noted. All donor arteries were noted to have macroscopic atheroma during the operation. The initial post-operative course was complicated by delayed graft function in two patients due to acute tubular necrosis. All three patients presented with persistent hypertension and graft dysfunction within 2–3 months of their transplantation and TRAS was suspected. They proceeded to have a renal Doppler ultrasound which revealed raised peak systolic velocity (300–
519 cm/sec) in the proximal renal artery. All cases proceeded to have balloon angioplasty with improvement in blood pressure and graft function. One patient required stent placement for recurrence of stenosis 6 months later.

**Conclusions:** TRAS should be suspected in patients with persistent hypertension post-transplantation as it is potentially treatable condition. Atheromatous disease in the donor vessels appears to be a risk factor. Doppler ultrasonography can be used to detect most hemodynamically significant stenoses. Angioplasty is usually first line for patients with significant stenoses, but medical management or surgical intervention can be considered.

**COMPENSATORY RENAL HYPERTROPHY IN RENAL TRANSPLANT RECIPIENTS**

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**Aim:** To evaluate compensatory renal hypertrophy (CRH) of the allograft kidney in renal transplant recipients and relationship to estimated glomerular filtration rate (eGFR).

**Background:** CRH is observed in patients nephrectomised for renal malignancy and living donors following nephrectomy. The extent of transplant kidney hypertrophy and influence on graft function is not well studied. Understanding the factors associated with CRH in the transplanted kidney could shed light on the mechanisms driving CRH.

**Methods:** A single centre retrospective study was undertaken of renal transplant recipients cared for by the Flinders Medical Centre Renal Unit from January 2007 to June 2020. Donor and recipient details, renal allograft length from serial transplant ultrasounds at months 0, 1, 3, 6 and 12 were collected. The primary outcome was occurrence and magnitude of CRH; secondary outcome was association of CRH with eGFR.

**Results:** 191 renal transplant recipients were studied, 61.3% male (n = 117) and 38.7% female (n = 74). One hundred and eleven of 169 patients (65.7%) demonstrated CRH defined as any increase in maximal longitudinal diameter on serial ultrasounds. Twenty-two patients were excluded due to absence of serial ultrasounds. The mean increase in kidney size over the first 12 months was 0.54 cm. Eighty-five of 148 (57.4%) patients with renal ultrasound within a month post-transplant demonstrated enlargement. Nine patients (5.3%) had no significant change in transplant size and 49 patients (29%) had a decreased size. Univariate analysis demonstrated that eGFR increased by 3.3 ml/min/1.73 m2 (95% CI 1.4–5.1, p < 0.001) with every 1 cm increase in kidney length.

**Conclusion:** CRH is common in renal transplant recipients and associated with enhanced eGFR. Further studies will determine factors that influence the occurrence of CRH and improved transplant function.

**PERI-OPERATIVE COVID-19 INFECTION FOLLOWING DECEASED DONOR KIDNEY TRANSPLANTATION: A CASE REPORT**

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**Background:** COVID-19 infection is associated with higher disease burden and mortality among kidney transplant recipients. In the immediate post-transplant period, there is limited evidence to guide changing immunosuppression to treat incidental COVID-19 infection. We present a patient who tested positive for SARS-CoV-2 within 24 h following kidney transplantation.

**Case Report:** Forty-two-year-old gentleman with end stage kidney disease secondary to IgA nephropathy on peritoneal dialysis presented for kidney transplantation via donation after circulatory death (DCD) pathway. He had received 3 doses of the BNT162b2 Pfizer vaccine. Pre-operative rapid antigen test (RAT) and polymerase chain reaction (PCR) test were negative for COVID-19. Serologic testing was negative for nucleocapsid protein IgG and showed high SARS-CoV-2 spike protein IgG titre (>8000 AU/mL). He received rabbit anti-thymocyte globulin (rATG) induction and underwent successful transplantation. The patient tested positive for COVID-19 (RAT and PCR) on day +1 as part of close contact screening after his wife tested positive. He developed mild respiratory symptoms and was treated with molnupiravir and sotrovimab. To reduce risk of severe illness, third dose of rATG was withheld and mycophenolate dose was reduced. He was also treated with ceftriaxone and fluconazole for Klebsiella and Candida from donor’s urine culture. He was weaned off nasal oxygen by day +5 and discharged on day +7. Maintenance immunosuppression includes prednisolone 20 mg daily, mycophenolate mofetil 1 g b.d and tacrolimus targeted to trough of 5–8 ng/mL. The patient remains well with no residual signs of COVID-19 infection and good allograft function.

**Conclusions:** Our report illustrates the first case of peri-operative COVID-19 following kidney transplantation in Western Australia. Vaccination, antiviral, and monoclonal antibody treatment alongside immunosuppression reduction were able to prevent severe complications.

**DIARRHOEA, HYPERKALAEMIC ACIDOSIS AND GRAFT DYSFUNCTION: CASE REPORT OF ADRENAL FAILURE IN A TRANSPLANT RECIPIENT**

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**Background:** Surgical procedures for primary or recurrent cancer in the renal bed may involve adrenalectomy. Adrenal insufficiency may
be partial or delayed in patients already on steroids. This can cause diagnostic uncertainty, especially in other settings known to similarly result in hypotension and renal dysfunction.

**Case Report:** A 67-year-old Caucasian male required dialysis after sequential native radical nephrectomies in 2017 (left adrenal was spared at surgery) and 2004 for renal cell carcinoma. Two years after a kidney transplant in 2020, he developed recurrent tumour in the left renal bed. He subsequently underwent an uncomplicated excision and was discharged home. Three weeks later, he presented with severe diarrhoea, hypotension and acute kidney injury (creatinine 226 μmol/L). In hospital, his diarrhoea resolved; blood pressure and renal function partially recovered with intravenous hydration. Prednisolone was increased to 20 mg daily. He presented a week after discharge with constellations of non-specific signs and symptoms, a potassium of 7.4 mEq/L and a persistently high creatinine (140-160 μmol/L). He had a hyperchloremic non-anion-gap acidosis (pH 7.25, pCO2 47.5, HCO3 20.5, Cl 113). Serum ACTH was elevated just above the normal range, serum aldosterone was undetectable (<50 pmol/L), and renin was elevated (104 μU/L). Biochemical parameters stabilized and kidney function returned to normal after adding fludrocortisone 100mcg daily. Two weeks later, he had a normal potassium, normal blood pressure and a creatinine of 99 μmol/L.

**Conclusions:** Hypoadrenalism has protean manifestations but can be masked in kidney transplant recipient already receiving steroids. Diarrhoea at presentation can suggest alternate causes for hypotension and renal failure, but failure of metabolic parameters to improve despite haemodynamic stability should prompt the consideration of less common aetiology.

**PRE-TRANSPLANT HUMAN LEUKOCYTE ANTIGEN (HLA) MATCHING, RENAL RESISTIVE INDEX (RI), AND DELAYED GRAFT FUNCTION (DGF) IN KIDNEY TRANSPLANTATION**

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**Aim:** To determine the difference in pre-transplant Human Leukocyte Antigen (HLA) matching and renal resistive index (RI) between patients with delayed graft function (DGF) and without DGF.

**Background:** Delayed graft function (DGF) is a major complication of kidney transplantation. It causes high rates of morbidity and determines poor graft functional outcomes. Pre-transplant HLA matching and RI are important early predictive factors for the development of DGF in kidney transplantation.

**Methods:** We analysed pre-transplant HLA matching and RI in 24 patients who underwent kidney transplantation, then we determined the difference in HLA-matching and renal resistive index between patients who developed DGF and non-DGF.

**Results:** There was no significant difference of number HLA matching (6.5 vs. 8.2; MD –1.7 95% CI MD –6.1 to 2.71; p = 0.433) between patients with DGF and without DGF. However, there was a significant difference of renal resistive index (0.75 vs. 0.62; MD 0.13 95% CI MD 0.06–0.2; p = 0.001) between patients with DGF than without DGF.

**Keywords:** Delayed graft function, renal resistive index, Human Leukocyte antigen.

**PRELIMINARY FEASIBILITY ANALYSES FOR IMMUNOSUPPRESSION MEDICATIONS IN KIDNEY TRANSPLANTS (IMET) STUDY**

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**Aim:** To determine the feasibility of assessing the effect of different immunosuppression regimens on kidney graft failure secondary to glomerular disease (GD) recurrence using registry data.

**Background:** Current registry studies investigating the effect of immunosuppression regimens on graft outcomes have primarily used discharge medications to categorize treatment groups which does not account for medication changes.

**Methods:** All patients who received a kidney transplant for glomerular disease between 1985–2020 were extracted from the Australian and New Zealand Dialysis and Transplant (ANZDATA) registry. Immunosuppression regimens were assessed at discharge, 1 year and 5 years post-transplant. Power calculations were completed using the sample size and outcome rates in the dataset to determine relative risk between the groups at a significance level of 0.05.

**Results:** 3615 kidney transplants were performed for GD. 1 year, 2 year and 5 year graft failure rates were 6.1%, 8.2% and 13.5%, respectively. In total, 1075 (29.7%) grafts failed (death-censored) with 122 (3.4%) due to disease recurrence, 579 (16.0%) due to chronic rejection, 724 (20.0%) due to death with functioning graft, 95 (2.6%) due to acute rejection, and 10 (0.3%) due to BK virus nephropathy. Immunosuppression medications changed significantly at 1 year and 5 years compared to discharge. Assuming a combined sample size of 3200, an observed rate of graft failure due to disease recurrence of 3.4%, and a significance cut off of 0.05; a log-rank test would be able to detect a relative risk between the groups of 0.50 with a power 0.90.
Conclusions: Even with conservative estimates for sample size, it is feasible to assess the effect of different immunosuppression regimens on graft failure due to disease recurrence in people with GD.

CLINICAL PROFILE AND OUTCOME OF TUBERCULOSIS IN KIDNEY TRANSPLANT RECIPIENTS: A CASE SERIES IN DR. SARDJITO GENERAL HOSPITAL, INDONESIA

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Background: Tuberculosis (TB) is one of the most severe bacterial infections after kidney transplantation and is the leading health problem in developing tropical countries. Anti-tuberculosis treatment (ATT) has side effects and frequent drug interactions with immunosuppressant drugs.

Case Report: We present two cases of active TB in kidney transplant recipients with different clinical presentations and outcomes. Case 1: A 42-year-old man with end-stage kidney disease (ESKD) caused by hypertension had comorbid heart failure, an unrelated living donor, and a history of acute rejection 2 weeks post-transplantation. He was diagnosed with disseminated TB one-year post-transplantation. The three-month post-ATT found that tacrolimus level decreased drastically, but stable allograft function was achieved. However, allograft kidney failure occurred in the fourth-month post-ATT, and the patient underwent haemodialysis. Case 2: A 31-year-old female with ESKD caused by hypertension had comorbid tertiary hyperthyroidism, an unrelated living donor, and a history of delayed graft function. She was diagnosed with pulmonary TB at one-year post-transplantation. The three-month post-ATT found that tacrolimus level decreased drastically, but stable allograft kidney function was found until completion of ATT.

Conclusions: The diagnosis and management of TB after kidney transplantation are challenging, especially with atypical symptoms and variable clinical outcomes. Significant interactions between ATT and tacrolimus can occur at the beginning of 3 months of ATT, so drug adjustments with frequent and close monitoring are needed. TB prophylaxis in kidney transplant recipients is essential within 1 year after transplantation, especially in endemic countries.

Keywords: Kidney Transplantation, Tuberculosis, Allograft Kidney Function.

IS CREATININE AND TACROLIMUS MEASUREMENT THROUGH VAMS MICROSAMPLING THE FUTURE FOR ADULT KIDNEY TRANSPLANT PATIENTS?

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Background: Kidney transplant patients require repeated and frequent venepunctures in the management of their allograft to measure both immunosuppressant drug concentrations and estimate kidney function. Microsampling methods that use a finger-prick draw of capillary blood, like volumetric absorptive microsamplers (VAMS), have the potential to dramatically reduce the pain, inconvenience, and volume of blood loss associated with venepuncture. This study aimed to clinically validate VAMS for simultaneous measurement of tacrolimus and creatinine compared to gold standard venous blood samples based on venepuncture in adult kidney transplant patients.

Methods: Blood samples for measurement of tacrolimus and creatinine concentrations were simultaneously collected using Mitra® volumetric absorptive microsamplers (VAMS) and venepuncture in 40 adult kidney transplant recipients, immediately prior and 2-h after tacrolimus dosing. Analyte concentrations were measured via high-performance liquid chromatography. Samples taken via the two collection methods were compared using Passing-Bablok regression and bias was assessed using Bland–Altman analysis. The predictive performance of analyte concentrations from VAMS measurement compared to venepuncture was also assessed through estimation of the median prediction error (MPE) and median absolute percentage prediction error (MAPE).

Results: There was a systematic difference between tacrolimus VAMS and venepuncture with a Passing-Bablok regression slope of 1.08 (1.03–1.13). Tacrolimus values were adjusted for this difference, with corrected values showing elimination of the systematic difference. Creatinine measured with VAMS showed a systematic difference with a slope of 0.65(0.6–0.7) and no systematic difference when adjustment was applied. Tacrolimus (corrected) and creatinine (corrected) microsampling values met MPE and MAPE predefined acceptability limits of <15%.

Discussion: VAMS collected in a controlled environment reliably measured tacrolimus and creatinine. This represents an enormous opportunity for more frequent and less invasive sampling for patients.

NOCARDIA CAUSING INTRAMUSCULAR ABSCESS IN A RENAL TRANSPLANT RECIPIENT

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Background: Nocardia is a gram-positive bacterium capable of causing both local and systemic opportunistic infection in immunocompromised patients. The most common clinical presentation is pulmonary infection. Presentation with an intramuscular abscess without disseminated nocardiosis is rare.

Case Report: A 67-year-old male with polycystic kidney disease received a renal transplant 3 years prior complicated by cytomegalovirus viraemia and persistent neutropaenia with subsequent cessation of mycophenolate. His immunosuppressants at the time of presentation included tacrolimus and prednisolone. He presented with swelling of the right bicep muscle in the vicinity of an intravenous cannula inserted at the time of left radiocephalic fistula ligation. Ultrasound confirmed a 10×20×20 mm intramuscular fluid collection which was managed conservatively and presumed to be a haematoma from intravenous cannulation and in the setting of anticoagulation for a pulmonary embolism. He represented two-weeks later with increasing pain and swelling without constitutional symptoms. Computer tomography (CT) was suggestive of an intramuscular abscess. Surgical incision and drainage was performed with intraoperative samples showing gram-positive bacilli resembling Nocardia spp., treated initially with meropenem and linezolid. CT of the brain, chest, abdomen and pelvis showed no other focus of infection. The Nocardia was identified as N. pseudobrasiliensis which was resistant to carbapenems. Therapy was changed to trimethoprim/sulfamethoxazole (SXT) and ciprofloxacin however SXT was not tolerated. He was subsequently treated with 6-months of ciprofloxacin and azithromycin with a good clinical response and no recurrence to date.

Conclusions: Nocardia requires consideration in the differential diagnosis of atypical infections in the immunocompromised kidney transplant recipient. The antimicrobial susceptibilities of Nocardia are variable depending on species with carbapenem resistance recently described in N. pseudobrasiliensis and this should be considered when initiating antimicrobial therapy.

RENAL TRANSPLANT FAILURE FOLLOWED BY WEANING IMMUNOSUPPRESSION: INCIDENCE OF ALLOGRAFT NEPHRECTOMY AND IMPACT ON PATIENT OUTCOME

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Aim: To review and compare mortality and retransplantation outcomes of patients with transplant failure in our centre who underwent allograft nephrectomy against those who did not. Immunosuppression cessation patterns post graft failure was also examined in detail.

Background: There is a lack of clinical consensus in the management of a failed renal allograft with respect to weaning immunosuppression and decision of nephrectomy, the latter of which is associated with risks of surgery and increased anti-HLA sensitisation. The optimal pathway for failed transplantation requires further study.

Methods: A retrospective analysis was performed on 48 patients who returned to dialysis following kidney transplantation failure in the years 2010–2020. Of these, 27 (56.3%) underwent a transplant nephrectomy and 21 (43.8%) weaned immunosuppression without requiring nephrectomy. Patients’ clinical data, transplant characteristics, immunosuppression management, mortality and retransplantation outcomes were collected and compared between the two groups.

Results: The allograft nephrectomy group had a trend towards lower mortality compared to those who did not undergo nephrectomy (11.1% vs. 33.3%, RR 0.33, p = 0.060). There was a significant difference in the number of patients relisted or worked up for retransplantation favouring the allograft nephrectomy group (70.4% vs. 38.1%, RR 1.85, p = 0.025). Of those relisted, a smaller proportion of those in the nephrectomy group proceeded to retransplantation (52.6% vs. 62.5%, p = 0.64). 85.2% of the nephrectomy group and 71.4% of the non-nephrectomy group ceased immunosuppression within the first year.

Conclusions: Despite concerns regarding the risks of allograft nephrectomy, the nephrectomy group demonstrated a trend towards improved mortality outcomes and no significant difference in rates of retransplantation. This suggests that nephrectomy may be a valuable treatment option for patients with transplant failure.

AN INTEGRATED SKIN CANCER EDUCATION PROGRAM IN RENAL TRANSPLANT RECIPIENTS

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Aims: To determine the effect of an integrated skin cancer education program on skin cancer awareness and sun-protective behaviours in renal transplant recipients (RTRs).

Background: Sun-protective strategies focusing on skin cancer awareness are needed in immunosuppressed patients at risk of skin cancers.

Methods: A pilot prospective cohort study in Central Queensland was undertaken among adult RTRs, who completed survey questionnaires.
Results: Twenty-five patients participated in the study. All participants completed questionnaires at pre-education, 3-month post-education and 92% (n = 23) at 6-month post-education. The mean age of all participants was 59 years (standard deviation, SD 11.4 years) and 64% were male. All participants were Caucasians and 72% had a history of skin cancer. There was a significant increase in SCSK scores from baseline at 3-months (19.7 [SD 3.1] vs. 21.8 [SD 1.5], p = 0.004) and 6-months post-intervention (19.3 vs. 20.9, p = 0.004). The frequency of outdoor activities of participants did not change significantly pre- and post-education, p = 0.549. Compared to baseline, the mean total number of sun-protective methods used changed significantly at 3-months (5.1 [SD 2.3] vs. 5.4 [SD 2.1], p = 0.043) and 6-months post-intervention (5.1 [SD 2.3] vs 6.0 [SD 1.9], p = 0.038). Compared to baseline, more RTRs stayed in the shade (p = 0.031) and wore sunglasses (p = 0.031) at 6 months. Interventional education enhanced regular self-skin examination rate (p = 0.002) but it did not significantly change the frequency of full skin checks by general practitioners (p = 0.219).

Conclusions: An integrated skin cancer education program improved knowledge of skin cancer and skin health knowledge and self-skin examination. However, it was not completely associated with an improvement in compliance with sun-protective practices.

NEPHROLITHIASIS IN THE TRANSPLANT RECIPIENT: A CASE OF EARLY RENAL FAILURE

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Background: Nephrolithiasis in the transplant recipient has an incidence of approximately 1%, however remains an uncommon but important cause of renal failure post transplantation.

Case Report: We report a case of oliguric renal failure secondary to nephrolithiasis without imaging evidence of hydronephrosis in 57-year-old female 7 months post renal transplantation. Past history of renal failure was secondary to presumed reflux nephropathy and a previous episode of obstruction secondary to a renal calculus. Her sister also had reflux nephropathy. She underwent deceased donor renal transplantation with stable baseline graft function (serum creatinine 104 μmol/l, eGFR 52 ml/min/1.73 m²) and 7 months post-transplant presented with oliguric renal failure. Imaging demonstrated a large stag-horn calculus without features of hydronephrosis. Calcification of the ureter was first noted following stent removal 4 months prior to admission and serial imaging showed increasing calcification. The patient was awaiting a urology opinion.

Renal biopsy excluded rejection and demonstrated acute tubular injury with calcium oxalate deposition without other features of oxalate nephropathy. Due to concern for obstruction a nephrostomy was placed with improvement in transplant function. Stone analysis demonstrated composition of calcium phosphate (80%) and magnesium ammonium phosphate struvite (20%), and she was found to have cornebacterium urealyticum infection contributing to stone formation. Percutaneous nephrolithotomy was unsuccessful. Novel treatment with a citric acid and micodacyn super oxidized solution was used to attempt stone dissolution with some improvement.

Conclusions: This case highlights an unusual cause of acute renal failure in the early post-transplant period with a staghorn calculus. The role of the ureteric stent as a ‘nidus’, and exclusion of genetic and metabolic causes of stone formation is required. Timely urology opinion in cases of nephrolithiasis post-transplant is essential.