Inflammation and infection

A rare case – Escalation of care for high risk emphysematous pyelonephritis in rural Australia

Stuart Jackson a,b,*, Doruk Seyfi a, Keval Pandya a, Than-Htike Oo a, James Collett a, Balasubramaniam Indrajit a

a Dubbo Base Hospital, NSW, Australia
b University of Sydney, NSW, Australia

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ABSTRACT

Emphysematous Pyelonephritis (EP) is a rare necrotizing renal parenchymal infection characterised by gas within the kidney parenchyma. Management with emergency nephrectomy has transitioned to a graded medical, radiological intervention and surgical approach. We present a rare case of high-risk emphysematous pyelonephritis, outlining key high risk factors and demonstrating staggered care escalation within a rural Australian referral hospital.

Introduction

Emphysematous Pyelonephritis (EP) is a rare necrotizing renal parenchymal infection characterized by gas within the kidney parenchyma. Less than 1000 cases have been reported globally since 1966, with the majority occurring in Asia.1 There is only one Australian case reported prior in review of literature.2

Previously with documented mortality of 50–70%, recent studies have reported an EP mortality rate of 20%.3 Traditional emergency nephrectomy has transitioned to a graded medical, percutaneous and surgical approach based on evidence comprised of retrospective case series.

Despite progress in EP population outcomes, a subset of patients will ultimately necessitate emergency nephrectomy. We present a rare high-risk case of emphysematous pyelonephritis, demonstrating staggered care escalation within a rural Australian referral hospital.

Case presentation

A 51-year-old indigenous woman presented to a remote health service with back pain radiating to the left flank and symptoms of dehydration. Her past medical history included insulin dependent type two diabetes mellitus, medically managed ischaemic heart disease, hypertension, and chronic lower back pain. She was treated with intramuscular anti-inflammatories and discharged with a diagnosis of an acute exacerbation of lower back pain.

Five days later, the patient represented with vomiting, flank pain and confusion, and was transferred to a regional health service. On arrival, the patient’s blood pressure was 76/53 mmHg. She was tachypnoeic at 55bpm with impaired consciousness and oliguria, consistent with sepsis. Biochemistry revealed a non-ketotic high anion gap metabolic acidosis (pH 7.15, lactate 10.1mmol/L), hyperglycaemia (blood glucose 23.5mmol/L) and an acute kidney injury with a serum creatinine of 348 μmol/L. Urinalysis demonstrated leukocytes, haemolysed blood, and protein (300–500), with no nitrites. Inflammatory markers were elevated with a white cell count (WCC) of 14x10⁹ and a CRP of 156mg/L. She was hyponatraemic (126mmol/L) and thrombocytopaenic (57x10⁹) in the context of septic shock.

After receiving broad-spectrum antibiotics (piperacillin-tazobactam and gentamicin) and resuscitation, a non-contrast computed tomography (CT) scan (Fig. 1) of the abdomen and pelvis demonstrated marked oedema of the left kidney with hydronephrosis, and extensive loculated and mottled areas of gas encompassing the majority of renal parenchyma. This was consistent with emphysematous pyelonephritis (EP). There was trace perinephric gas but no fluid levels or drainable collection. A simple cyst of the superior pole was evident, with no gas internally.

The patient was admitted to the intensive care unit for vasopressor support, antibiotics, strict fluid balance with indwelling catheter, continuous veno-venous haemodialysis (CVVHD) and insulin infusion. Following urine and blood cultures for Klebsiella Pneumoniae, antibiotic therapy was narrowed (ceftriaxone and metronidazole). After 48-h of
medical management and no clinical progression, an 8Fr nephrostomy tube was placed under CT guidance to help facilitate source control and decompress the urinary system. Despite a further 72-h trial of non-operative management, nephrostomy drainage and organ support with ability to cease CVVHD after 3 days, the patient failed to show significant improvement. This was clinically evident in persisting confusion, high grade fevers, tachycardia, dependence on vasopressors, and parenteral analgesia infusion for pain. With new anaemia (Hb 82g/L), persisting inflammatory rise (WCC 13.6x10^9) and thrombocytopaenia (114x10^9), and recurring deterioration in renal function (Cr 142μmol/L) from post CVVHD nadir (71μmol/L), repeat imaging was undertaken. This demonstrated no parenchymal improvement. Considering high risk state, lack of drainable collections and worsening clinical status with ongoing refractory end organ dysfunction, the decision was made to undertake open nephrectomy rather than repeat attempts at percutaneous drainage, which had failed. Fibrous adhesions encountered intraoperatively necessitated a subcapsular approach (Fig. 2).

Post-operatively, the patient recovered well, and intravenous antibiotics were transitioned to oral after 4 days. She was subsequently discharged with creatinine of 106 μmol/L.

Discussion

EP is a severe necrotizing infection of the renal parenchyma rarely encountered by clinicians. It is particularly relevant in rural and remote locations where suitable escalation of care may necessitate urgent transfer or patient retrieval.

There are several risk factors associated with poor outcomes and higher mortality for patients with EP. These include: shock, altered mental status, renal failure, hyponatraemia, thrombocytopaenia, need for dialysis, and a high EP CT grade, or >50% renal parenchymal destruction on CT. This case illustrates a patient with such risk factors, yet who survived and had a good outcome because of rapid and appropriate escalation of treatment options.
Recent systematic reviews of case series have advocated for a staggered approach of medical management, drainage and nephrectomy – with nephrectomy only in severe cases where less invasive therapies have been unsuccessful. This approach has been associated with higher survival and preservation of renal function; however, this data is based on retrospective studies susceptible to selection bias and without clearly defined severity classifications.

Given EP’s rarity, the evidence base for a clear management algorithm is lacking. Clinicians should note that despite image-based grading systems, there are no studies that propose a clear management algorithm based on specific grades of severity or risk factor, nor outline time frames for necessary treatment escalation. There is no ‘one size, fits all’ approach. We feel our method of escalated intervention over 72-h periods was appropriate in the case of this high-risk EP patient, though encourage clinicians to be judicious – accelerated or lengthened time frames may be suitable with differing clinical response. Attention to signs of poor infection clearance in the form of repeat positive cultures, refractory end organ failure, sepsis and/or biomarkers (CRP, WCC) should be used primarily to guide clinical care.

Consideration must be made for early multidisciplinary consultation, and we advocate for patient transfer to centres with appropriate radiological and surgical intervention services. There is poor consensus as to the preferred treatments for patients with poor infection control by percutaneous drainage, and repeat attempts at drainage may be reasonable in carefully selected patients, those unfit for anaesthesia, or with bilateral disease.

**Conclusion**

EP is a rare, necrotising infection, which is associated with high mortality. An individualised, multi-disciplinary, staggered approach to treatment is appropriate. Due to the lack of high-quality evidence underpinning management, clinicians must be vigilant in their approach and consider rapid escalation of therapy if there is evidence of non-progressive non-operative management.

**Credit author statement**

- Jackson, Stuart MD, MS – conception and design, drafting and revision
- Seyfi, Doruk MBBS – drafting and revision of important intellectual content.
- Panda, Keval MBBS, FRACP – revision of important intellectual content.
- Oo, Than-Htike – analysis and interpretation of data for the work.
- Collett, James MBBS, FRACP – supervision and revision of important intellectual content.
- Indrajit, Balasubramaniam FRACS (Urol) – supervision and revision of important intellectual content.

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