nODULES on ultrasound. She underwent prophylactic thyroidectomy. Neither the patient, nor his siblings, have any progeny. The patient screened negative for primary hyperparathyroidism and pheochromocytoma. Calcitonin (739, normal <=14.3 pg/mL) and CEA levels (31.7, 0.3-5.0 ng/mL) were elevated. Thyroid ultrasound (US) showed two solid hypoechoic nodules with lobulated margins and internal coarse calcifications in the right and two in the left thyroid lobe; 1.5 cm and 1.2 cm in maximum diameter, and 1.2 cm and 3 mm in maximum diameter, respectively. Based on elevated calcitonin and CEA levels, known RET mutation and evidence for thyroid nodules, we recommended a total thyroidectomy and central neck dissection. Pathology revealed multifocal, bilateral medullary carcinoma (largest focus of 1.5 cm), with 4/4 lymph nodes positive for metastasis. This was classified as mpT1bN1aM0 (Stage III). Patient was started on levothyroxine with plans to repeat calcitonin and CEA levels and neck ultrasound, 3 months following surgery. CT chest, abdomen and pelvis did not reveal any distant metastasis. Conclusion: Inherited MTCs are rare. Early diagnoses by screening of at-risk family members in MEN2 kindreds is important because MTC can be life-threatening and can be cured and prevented by early thyroidectomy. While our suspicion for FMTC in this patient and his siblings is high, FMTC is now considered a variant of MEN2A and ongoing screening for pheochromocytoma and primary hyperparathyroidism is recommended.

Reproductive Endocrinology
TRANSGENDER MEDICINE AND RESEARCH

Prevalence of Polycythaemia with Different Formulations of Testosterone Therapy in Transmasculine Individuals

Brendan J. Nolan, MBBS, FRACP,1 Shalem Y. Leemaqz, PhD2, Olivia Ooi, MD1, Pauline Candill, BM, FRACGP3, Nicholas Silverstein, MFamMed, FRACGP, FACRM4, Peter Locke,3 Mathis Grossmann, MD, PhD, FRACP, Jeffrey D. Zajac, MBBS, FRACP, PhD2, Ada S. Cheung, MBBS, FRACP, PhD2.

1University of Melbourne (Austin Health), Melbourne, Australia,
2University of Adelaide (Robinson Research Unit), Adelaide, Australia,
3Equinox Gender Diverse Clinic, Melbourne, Australia.

SUN-046

Background: Masculinising hormone therapy with testosterone is used to align an individual's physical characteristics with their gender identity. Testosterone therapy is typically administered via intramuscular or transdermal routes and polycythaemia is the most common adverse event.

Aims: To compare the risk of polycythaemia with different formulations of testosterone therapy in transmasculine individuals.

Methods: A retrospective cross-sectional analysis was undertaken of transmasculine individuals at a primary and secondary care clinic in Melbourne, Australia. 180 individuals who were on testosterone therapy for >6 months were included. Groups included those receiving (1) intramuscular testosterone undecanoate (n=125), (2) intramuscular testosterone enantate (n=31), or (3) transdermal testosterone (n=24). Outcome was prevalence of polycythaemia (defined as haematocrit >0.5).

Results: Mean age was 28.4 (8.8) years with a median duration of testosterone therapy 37.7 (24.2) months. 27% were smokers. There was no difference between groups in serum total testosterone concentration measured. Whilst there was no difference between groups in haematocrit, there was a higher proportion of patients with polycythaemia in those who were on intramuscular testosterone enantate (23.3%) than on transdermal testosterone (0%), p=0.040. There was no statistically significant difference in polycythaemia between intramuscular testosterone undecanoate (15%) and transdermal, p=0.066 nor between intramuscular testosterone enantate and undecanoate, p=0.275.

Conclusions: One in four individuals treated with intramuscular testosterone enantate and one in six treated with testosterone undecanoate had polycythaemia. No individual treated with transdermal testosterone had polycythaemia. This highlights the importance of regular monitoring of haematocrit in transmasculine individuals treated with testosterone and findings may inform treatment choices.

Bone and Mineral Metabolism
BONE AND MINERAL CASE REPORTS II

Isolated Extrapulmonary Sarcoidosis Presenting as Symptomatic Hypercalcemia

Janya Swami, MD.

Carle Foundation Hospital, Urbana, IL, USA.

MON-340

Introduction: Sarcoidosis is a granulomatous disease of unknown etiology often involving multiple organ systems. Sarcoidosis most frequently affects the lungs, but in up to 30% of cases, can present with extrapulmonary manifestations. Less than 10% of patients with sarcoidosis present with disease at extrapulmonary sites. 10%-20% patients with sarcoidosis present with hypercalcemia. Hypercalcemia in sarcoidosis is secondary to increased intestinal calcium absorption due to increased levels of 1,25-dihydroxy Vitamin D. Clinical Case: 71-year-old Caucasian male presenting to his primary care physician with 3-week duration of fatigue, anorexia, mild confusion and unintentional weight loss was found to have moderate hypercalcemia and acute kidney injury. He was admitted the hospital for evaluation and management. Labs at admission revealed albumin-corrected calcium of 13.5 mg/dL (normal 8.5-11.0 mg/dL), creatinine of 1.78 mg/dL (normal 0.7-1.3 mg/dL) and alkaline phosphatase of 173 U/L (normal 45-117 U/L). Workup noted low PTH (3, normal 14-72 pg/mL), normal 25-hydroxy Vitamin D (50.7, normal 30-100 ng/dL), normal PTHrP and normal serum electrophoresis and immunofixation indicating non-PTH dependent hypercalcemia. 1,25-dihydroxy Vitamin D (125, normal 18-64 pg/mL) and ACE levels (159 U/L, normal 16-85 U/L) were elevated. Patient denied being on any Vitamin D supplements. A CT chest, abdomen and pelvis was done to look for occult malignancy and hepatosplenomegaly was noted with only mild compressive atelectasis of lungs. Abdominal ultrasound confirmed hepatomegaly with lobulated outer contour consistent with cirrhosis. Acute hepatitis, infectious and autoimmune workup was negative. Patient was discharged with mild improvement in calcium and mental status with hydration.
Due to high suspicion for granulomatous disease, a liver biopsy was done. Liver biopsy confirmed granulomatous hepatitis with stage 2 of 4 fibrosis with numerous foci of non-caseating granulomatous inflammation. With negative acid-fast staining, no fungal organisms, absence of foreign material, normal eosinophil counts and low clinical suspicion for Crohn’s disease, a diagnosis of abdominal sarcoidosis was made. The patient was started on 10 mg prednisone daily and within one week, his albumin-corrected calcium levels improved to 10.4 mg/dL with significant improvement in appetite and mental status. Conclusion: While isolated extrapulmonary sarcoidosis is rare, it is an important cause of hypercalcemia due to elevated 1,25-dihydroxyvitamin D levels. Management of hypercalcemia secondary to sarcoidosis often consists of initiating glucocorticoids which act mainly by inhibition of 1,25-dihydroxy vitamin D synthesis in addition to inhibiting calcium absorption and osteoclast activity.

Bone and Mineral Metabolism

BONE AND MINERAL CASE REPORTS II

Osteitis Fibrosa Cystica and Pathological Fractures: The Classic but Neglected Skeletal Manifestation of Primary Hyperparathyroidism

Sirinart Sirinvarawong, MD, Nontouch Singsumpun, MD, Ekasame Vanitcharoenkul, MD, Aasis Unnanuntana, MD.
Mahidol University Faculty of Medicine Siriraj Hospital, Bangkok, Thailand.

MON-347

Background: Osteitis fibrosa cystica is one of the classic manifestations of primary hyperparathyroidism (PHPT), yet it has become increasingly rare due to early detection of PHPT.

Clinical case: A 37-year-old woman was referred to our hospital for fixation of multiple fractures. Before transferring to our hospital, she was admitted to a local hospital due to right distal humerus and left tibial fractures without history of trauma. During the hospitalization, a new fracture at left femur had occurred. Her past medical history was significant for fractures of right shoulder, left elbow and right femur when she fell on the ground 5 years ago and treated by fixation. She had a history of kidney stones in the remote past. She denied family history of calcium or skeletal disorders.

Clinical examination revealed gross deformities at right elbow, left thigh and painful swelling of the left tibia. On HEENT examination, there were no palpable neck masses. Upon review of plain radiographs from outside hospital, we found not only fractures but diffuse osteopenia and brown tumors at multiple sites including shaft of right humerus, right proximal radius, left proximal femoral shaft and left tibia. Skull X-ray showed salt-and-pepper appearance. There were prominent subendplate densities at multiple lumbar spines (Rugger-jersey spine). These findings were consistent with osteitis fibrosa cystica which prompted further laboratory investigation for PHPT. The patient had a high corrected serum calcium level of 13.6 (8.6-10.0) mg/dL, low serum phosphate of 2.2 (2.5-4.5) mg/dL, serum creatinine of 1.16 (0.51-0.95) mg/dL, glomerular filtration rate (GFR) of 60.29 ml/min, high serum alkaline phosphatase of 1,482 (35-105) U/L. The serum parathyroid hormone (PTH) level was significantly high at 3,850 (15-65) pg/mL. Vitamin D level was low at 5.98 (≥30) ng/mL. The patient was diagnosed with PHPT. The left lower parathyroid adenoma was identified by ⁹⁹ᵐTc-Sestamibi scan with single photon emission computed tomography and neck ultrasound. Given severe skeletal manifestations and extremely elevated PTH level, urgent parathyroidectomy was performed. A 2.5-cm parathyroid adenoma was removed which was histologically confirmed by a pathologist. Postoperatively, she developed a hungry bone syndrome which resolved 10 months after.

Clinical lesson: In current practice, skeletal manifestation of PHPT is uncommon with a reported incidence of 1.4% in US. Osteitis fibrosa cystica is a radiographic feature of PHPT characterized by demineralized skeleton, salt-and-pepper appearance of the skull, subperiosteal resorption of the phalanges, bone cysts, and brown tumors. Although PHPT has evolved to asymptomatic disease in majority of patients, the classic skeletal involvement should not be overlooked, particularly in young patients who present with multiple pathological fractures.

Thyroid

THYROID DISORDERS CASE REPORTS II

A Grave Complication: Pulmonary Arterial Hypertension

Wajeeha Saeed Butt, MD¹, Vijay Srinivasan, MD².
¹JFK Medical Center, Atlantis, FL, USA, ²Aventura Hospital and Medical Center, Aventura, FL, USA.

SAT-459

There is currently emerging evidence that thyroid disease can have an impact on pulmonary pathologies but a direct causation, as opposed to correlation, is yet to be established. We present a unique case of newly diagnosed Graves’ disease with pulmonary sequelae. 69 year old non-smoking female with no known medical history presented to the hospital with acute onset of chest tightness, shortness of breath and palpitations. She had been experiencing night sweats and 10 lbs weight loss in the 3 weeks preceding hospitalization. She did not endorse any pertinent family history. On exam, she presented with tachycardia. The rest of the physical exam remained unchanged. Complete blood count and comprehensive metabolic panel did not reveal any abnormalities. Her TSH was suppressed below lab capability of measurement (<0.015). Free T4 was 5.97 ng/dL, and total T3 was 492 ng/dL. Subsequently obtained TPO antibody level was > 1000 IU/ml and TSI was 29.3 IU/L. EKG showed atrial fibrillation with RVR. Thyroid U/S showed that the thyroid gland was diffusely enlarged to the upper limit of normal but no nodules. CT chest showed diffuse ground glass opacities. She was treated with propranolol, and methimazole after resolution of acute symptoms. On outpatient follow up she continued to have mild shortness of breath on exertion. She had normal systolic and diastolic function of heart but an elevated PAP of 38mm Hg on a transthoracic echocardiogram, and her pulmonary function...