CONCLUSION
Bodily pain, a component of physical health and hypovitaminosis D had negative impact on bone-turnover. Overall, majority of participants had SF-36 health survey scores that trended towards good physical and mental health signaling satisfactory QOL despite being largely affected by comorbidities associated with transfusion-dependent thalassemia.

PP-46

A CASE OF SEVERE PROXIMAL MYOPATHY IN A PATIENT WITH ATYPICAL PARATHYROID ADENOMA

https://doi.org/10.15605/jafes.036.S72

Shireen Lui Siow Leng, Alfieyanto Syaripuddin, Serena Khoo Sert Kim
Endocrinology Unit, Department of Medicine, Queen Elizabeth II Hospital, Kota Kinabalu, Sabah, Malaysia

INTRODUCTION
Parathyroid-induced myopathy is a rare neuromuscular manifestation of primary hyperparathyroidism leading to progressive proximal muscle weakness, pain and atrophy. Severity of the weakness varies in relation to the duration and degree of hyperparathyroidism.

RESULTS
We describe a 30-year-old male who presented with progressive debilitating muscle weakness, severe muscle wasting and recurrent muscle spasms over 1 year. He also experienced bone pain, anorexia and weight loss. He had a symmetrical proximal myopathy and muscle wasting of both upper and lower limbs with MRC grade 3/5 on shoulder abduction, adduction, hip extension and flexion. Corrected calcium 4.08 mmol/L (2.10-2.55), phosphate 1.22 mmol/L (0.72-1.52), iPTH >3000.0 pg/ml (15.0-68.3) were suggestive of primary hyperparathyroidism. Parathyroid ultrasound and SESTAMIBI scan localised a hyperfunctioning left superior parathyroid adenoma. His 25 OH-Vitamin D was 39.7 nmol/L suggestive of Vitamin D insufficiency. An elevated alkaline phosphatase at 1807 U/L (40-150), skeletal survey with cortical thinning and generalise low bone density along with bilateral nephrocalcinosis and nephrolithiasis reflected skeletal and renal involvement, common complications of primary hyperparathyroidism. However, an elevated creatine kinase (CK) of 861 U/L (30-200) despite a normal nerve conduction study and electromyography was indicative of a rare myopathic involvement. He underwent successful parathyroidectomy following treatment with hyperhydration, intravenous pamidronate and denosumab. There was resolution of severe muscular spasms, improvement in muscle strength, weight gain and normalisation of his CK, calcium, PTH and vitamin D levels. The histopathological examination confirmed an atypical parathyroid adenoma.

CONCLUSION
Severe proximal myopathy is a rare complication of primary hyperparathyroidism. Cases of atypical parathyroid adenoma, a rare intermediate neoplasm of uncertain malignant potential may present with a more severe clinical and biochemical profile. Prompt diagnosis and parathyroidectomy can prevent complications and improve clinical outcomes.

PP-47

PRIMARY HYPERPARATHYROIDISM DURING PREGNANCY: A CASE REPORT

https://doi.org/10.15605/jafes.036.S73

Loh YD, Nor Akidah M, Wan Mohd Hafez WH, Masliza Hanuni MA
Endocrinology Unit, Medical Department, Hospital Sultanah Nur Zahirah, Terengganu, Malaysia

INTRODUCTION
Primary hyperparathyroidism (PHPT) is a bone and mineral metabolism disorder caused by autonomous secretion of parathyroid hormone (PTH). PHPT is rare in pregnancy with a quoted incidence of 1%. PHPT during pregnancy is challenging to diagnose and difficult to manage. This is due to limited diagnostic and therapeutic options available during pregnancy and the lack of clinical guidelines. PHPT poses serious maternal and foetal complications such as hyperemesis gravidarum, hypercalcaemic crises in the mother, preterm delivery or miscarriage, and neonatal hypocalcaemia. The definitive treatment for PHPT in pregnancy is parathyroidectomy.

We report a case of PHPT diagnosed and managed during pregnancy.

RESULTS
A 35-year-old female who was 27 weeks pregnant, G3P2, presented with prolonged nausea and vomiting up to her second trimester of pregnancy. Blood results showed serum corrected calcium of 3.17 mmol/L (reference range 2.20-2.65), serum phosphate level of 0.56 mmol/L (reference range 0.81-1.45), alkaline phosphatase of 601U/L (reference range 30-120), intact PTH of 346 pmol/L (reference range 14.9-56.9) and normal renal function. Her calcium clearance to creatinine clearance ratio was 0.016. Ultrasound of the neck showed an enlarged left superior parathyroid gland. She was admitted to the ward for intravenous rehydration with forced diuresis. After 1 week trial of outpatient oral rehydration, repeated serum corrected calcium was 2.77 mmol/L. After multi-disciplinary discussion and family conference, a decision was reached to perform parathyroidectomy. Following left superior parathyroidectomy, her serum calcium returned to normal, and symptoms of nausea as well as vomiting has resolved.

CONCLUSION

Clinicians should have a high index of suspicion for PHPT in pregnancy and manage the condition with a multidisciplinary team (consisting of endocrinologist, endocrine surgeon, obstetrician, paediatrician and anaesthesiologist) due to its potential serious maternal and foetal adverse outcomes if left untreated.

PP-48

A CHALLENGING CASE OF PARATHYROID CARCINOMA WITH SEVERE HYPERCALCEMIA

https://doi.org/10.15605/jafes.036.S74

Wan Mohd Hafez Wh and Masliza Hanuni Ma
Endocrinology Unit, Medical Department, Hospital Sultanah Nur Zahirah, Terengganu, Malaysia

INTRODUCTION

Parathyroid carcinoma is a rare condition and it accounts for < 1% of cases of sporadic primary hyperparathyroidism (PHPT). Parathyroid carcinoma is commonly associated with severe and refractory hypercalcemia. We described a patient with parathyroid carcinoma who presented with multiple pathological fractures.

RESULTS

A young female presented with closed fractures of the right proximal humerus and bilateral femoral necks after a trivial fall. Her initial serum calcium was 3.18 mmol/L, serum phosphate was 0.56 mmol/L. The diagnosis of primary hyperparathyroidism was confirmed with high serum parathyroid hormone (iPTH) level of 1187.2 pg/mL (14.9-56.9). Neck ultrasound showed a right parathyroid lobulated lesion measuring 1.6 x 2.3 x 3.3 cm. The challenge in management was the refractory severe hypercalcemia despite standard treatment of hydration, bisphosphonate and calcitonin. Her serum calcium still ranged between 3.6-4.5 mmol/L despite the above therapy. She developed ECG changes typical for hypercalcemia (short QT interval). The surgery cannot proceed due to severe hypercalcemia. Hence, the decision for hemodialysis was made. She underwent 2 sessions of hemodialysis with low calcium dialysate and proceeded with emergency right inferior parathyroidectomy and right hemithyroidectomy. Postoperatively, she was put on calcium gluconate infusion for a few days on top of oral calcium supplement and oral vitamin D, in anticipation of hungry bone syndrome. Histopathological examination confirmed a right parathyroid carcinoma with infiltration to adjacent thyroid tissue. She was discharged well with oral calcium and oral vitamin D supplement. As for her multiple pathological fractures, they were treated conservatively.

CONCLUSION

Hypercalcemia in parathyroid carcinoma is very challenging to manage because it tends to be severe and refractory. Hemodialysis for treatment of severe hypercalcemia was shown to be effective for reduction of hypercalcemia while the patient prepare for parathyroidectomy.

PP-49

CURING HYPERTENSION: SUCCESFUL ADRENALECTOMY FOR PRIMARY ALDOSTERONISM USING CONTRALATERAL SUPPRESSION INDEX

https://doi.org/10.15605/jafes.036.S75

Md S Md Amin, L Ibrahim, S R Vethakkan
Endocrine Division, Medical Department, University Malaya Medical Centre, Malaysia

INTRODUCTION

The diagnosis of Primary Aldosteronism can be challenging especially when it comes to localising an adenoma in patients who fail adrenal venous sampling. Even in expert hands, the right adrenal vein has been known to be difficult to cannulate during AVS. We present a case of a hypertensive woman with metabolic alkalosis and hypokalemia who was evaluated for primary hyperaldosteronism.

RESULTS

A 57-year-old Chinese female with a history of mini-gastric bypass with resultant resolved DM, attended clinic for poorly-controlled hypertension on 2 anti- hypertensives. She also had persistent hypokalemia (2.9-3.5 mmol/l) with metabolic alkalosis. She denied paroxysms or abnormal weight gain suggestive of phaeochromocytoma or Cushing's syndrome. Physical examination was unremarkable. Her aldosterone-renin-ratio (ARR) was raised (>91) with a serum aldosterone of 747 pmol/L and renin of <1.8mU/L. A saline suppression test showed a non-suppressed aldosterone (767pmol/L). A CT-adrenal 3-phase confirmed a subcentimeter benign right adrenal gland nodule with an absolute washout of 92%, suggestive of Conn's adenoma. 24-hour urine catecholamines/metanephrines and 24-hour urinary cortisol were negative. She underwent adrenal-vein-sampling (AVS) with cosyntropin stimulation to localise the source of excess aldosterone secretion. Three series of cortisol samples demonstrated selectivity indices between 0.9-1 for the right and 6.4-24.4 for the left adrenal veins, reflecting failed right adrenal vein cannulation. Contralateral suppression index (CSI) was <1 for all 3 samples from the left adrenal vein. She was referred to the surgeons for right retroperitoneal adrenalectomy. Histopathology confirmed a 1 x 1 cm adrenal cortical adenoma. She was able to cease all anti-hypertensives and potassium supplementation immediately after the surgery.