

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.

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

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

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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.

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PRIMARY HYPERPARATHYROIDISM DURING PREGNANCY: A CASE REPORT

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

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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.