Primary Hyperparathyroidism Presenting with Acute Painful Paraparesis: A Case Report

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Background

Primary hyperparathyroidism (PHPT) is associated with many musculoskeletal complaints which can occasionally be the presenting symptoms [1,2]. We present an unusual case where acute paraparesis was not associated with spinal cord compression and resolved with the correction of the hypercalcemia. Therefore, this case demonstrates how it is imperative that clinicians always consider hypercalcemia in cases of acute painful paraparesis.

Methodology

Case Report

A 47-year-old male sheep farmer presented with acute onset paraplegia, self-reporting "difficulty getting up from sitting and squatting position". He had deep-seated bone pain in the lower limbs but no bowel or bladder incontinences. He had a medical history notable for Barrett’s esophagus, depression, obstructive sleep apnea and gout. On physical examination, vital signs were normal and neuromuscular examination revealed lower limb weakness with 3/5 power in the hip flexors and extensors, and 4/5 in the knee flexors and extensors (by MRC scale). There were intact deep tendon reflexes, plantare reflexes and sensation. Laboratory investigations revealed mild neutrophilia (9.4x10^9/microliter), acute kidney injury with urea of 21 mmol/L (3-8.0 mmol/L), creatinine of = 302 umol/L (60-110 umol/L) and eGFR of = 41 ml/min (>90ml/min). Corrected calcium was very high at 4.52 mmol/L (2.20-2.55 mmol/L) with phosphate of = 1.36 mmol/L (0.75-1.50), magnesium of = 0.79 mmol/L (0.7-1.50 mmol/L) and very high parathyroid hormone of = 169.3 pmol/L (1.2-5.8 pmol/L) and mildly low vitamin D at = 56 nmol/L (35-120 nmol/L). The diagnosis of primary hyperparathyroidism with resultant severe hypercalcemia was made. A Technetium (99mTc) sestamibi scan showed huge right lower parathyroid adenoma extending down to level of 4th the fourth thoracic vertebral. The patient was treated with intravenous fluid and calcitonin infusions. The bone pain, proximal myopathy and paraparesis resolved as the calcium level fell with appropriate treatment. He subsequently underwent a parathyroidectomy to remove a giant parathyroid adenoma weighing about 9.8g (Figure 1). The serum
calcium and renal function rapidly returned to normal by the third post-operative day.

Discussion

The classical bone manifestation of PHPT is osteitis fibrosa cystica (OFC). OFC is defined as the classical musculoskeletal manifesiatio of advance primary hyperthyroidism with bone resorption and appearance of "ground glass/salt and pepper "changes especially in the bones of the finger. But the observed musculoskeletal manifestations of this condition have evolved over time from symptomatic to subtle disease with the advances in the screening for hyperthyroidism and calcium measurement 

[2,3]. Many manifestations of PHPT are now recognized in orthopedic surgery, neurology and rheumatology practices. The common musculoskeletal presentations seen in PHPT include back pain, generalized bone pain, chest and/or rib pain, mandibular giant cell tumors, pathological fractures and Achilles tendon rupture [1,3,4]. The neurological manifestations include spinal cord compression and myopathy [1]. The acute painful paraparesis in our case was an acute neurological emergency. The painful proximal muscle weakness was a consequence of severe hypercalcemia rather than spinal cord compression. This has been reported in earlier case reports of PHPT.

Conclusion

The case presented highlights the need to consider hypercalcemia due to PHPT in cases of acute painful paraplegia when there is no other alternative cause identified such as spinal cord compression, paraneoplastic syndrome or hypercalcemia of malignancy.

References