CR-GE-31

THE MANY FACES OF PARATHYROID CARCINOMA: A CASE SERIES

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INTRODUCTION
Parathyroid carcinoma is rare, affecting less than 1% of patients with primary hyperparathyroidism (PHPT). Parathyroid carcinoma with concomitant papillary thyroid carcinoma is also rare, and the etiology is not fully defined.

CASE
We present 3 patients with parathyroid carcinoma and discuss their presenting symptoms, characteristics, and treatment. Two women and 1 man (2 Filipinos and 1 Marshallese) ages 53-68 years old had parathyroid carcinoma. Two had primary hyperparathyroidism, one had non-functioning type of parathyroid carcinoma. Two had concomitant bone or renal disease. All three had concurrent thyroid disease- 2 had papillary thyroid microcarcinoma, one had colloid goiter. Because each one had different indications for neck surgery, different surgical techniques were done. Parathyroid cancer sizes ranged from 2.0-5.2 cm, with capsular and vascular invasion in all three. One case had double parathyroid carcinomas. On follow-up, the patient with the largest tumor size had tumor recurrence within 1 year from surgery.

CONCLUSION
This case series, to the best of our knowledge, includes the first reported case of synchronous parathyroid carcinoma and papillary thyroid carcinoma in a Marshallese patient, as well as the 7th case of double parathyroid carcinomas. Parathyroid carcinoma is a rare condition, and coupled with its highly variable presentation, as seen in the 3 cases, presents clinicians with a difficulty in arriving at a diagnosis. Histopathology remains the gold standard in diagnosis and is a key in guiding management. Since coexisting thyroid and parathyroid disease may occur, thyroid pathology should be evaluated in the presence of PHPT.

KEY WORDS
cancer, hyperparathyroidism, parathyroidectomy, microcarcinoma

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THE PERILOUS PROTON PUMP INHIBITOR

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INTRODUCTION
Proton pump inhibitors (PPIs) are the mainstays of therapy for all gastric acid related diseases. PPIs have been associated with various adverse effects, including hypomagnesemia. The postulated mechanism of PPI-related hypomagnesemia involves inhibition of intestinal magnesium absorption via transient receptor potential melastin (TRPM) 6 and 7 cation channels. PPI-induced hypomagnesaemia (PPIH) has become a well recognized phenomenon since it was first reported in 2006. In this article, we report 2 cases of PPIH referred to endocrine unit for severe hypocalemia related to PPIH.

CASE
Patient 1 was newly diagnosed with pulmonary tuberculosis (PTB). She was started on pantoprazole due to vomiting after taking anti-TB medications. She presented a week later with supraventricular tachycardia. Blood investigations showed multiple electrolyte abnormalities, including hypomagnesaemia, hypocalcaemia, and hypokalemia. Her serum electrolytes failed to return to normal despite multiple corrections given. After pantoprazole was discontinued, the serum levels of magnesium, potassium and calcium started to respond to corrections given and returned to normal. Patient 2 was diagnosed with peptic ulcer disease and started on pantoprazole. 4 months later, she presented with lethargy and bilateral hand numbness. Blood investigations revealed severe hypocalcaemia and hypomagnesaemia. Her magnesium and calcium levels slowly returned to normal after the pantoprazole was discontinued.

CONCLUSION
Known risks of long-term PPIs administration must be considered in clinical practice and judicious use of PPIs is important to avoid potentially fatal complications.

KEY WORDS
proton pump inhibitor, hypocalcemia, hypomagnesemia