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Parathyroid carcinoma presenting with type 1 renal tubular acidosis

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Abstract

Primary hyperparathyroidism usually caused by parathyroid adenoma, parathyroid hyperplasia or rarely parathyroid carcinoma (PTC). PTC accounts for 0.4% to 5.2% of all reported cases of hyperparathyroidism, which is approximately 0.2% to 0.5% of malignant endocrine tumors overall. Renal tubular acidosis has been reported to be associated with primary hyperparathyroidism. We report a case of parathyroid carcinoma diagnosed with only using the minor criteria who had concurrent renal tubular acidosis. This case highlights the importance of keeping the suspicion of PTC in mind when clinically hypercalcemic patients presented with abnormally high serum calcium levels.

Key words: Hypercalcemia, primary hyperparathyroidism, parathyroid carcinoma, type 1 renal tubular acidosis.

Case presentation

Sixty-one year old previously unevaluated male patient from Samantthurai presented with lethargy, constipation, vomiting and loss of weight for 6 months. He became wheel chair bound for last two weeks. There was no history of cold intolerance, fever, cough or altered bowel habits. He was neither a smoker nor an alcoholic. He was dehydrated and pale and did not have a neck lump. Rest of the examination was normal.

His serum ionized calcium was 3.68 mmol/l, 24hour urinary calcium level 204mg. Microcytic hypochromic anemia in blood picture favored anemia in chronic disorder. Metabolic acidosis, high urinary pH (>5.3) favored diagnosis of type 1 renal tubular acidosis (blood PH 7.302, HCO38.8 mmol/l, urinary PH 7.36 with normal anion gap). Subsequent work up included an ultrasound scan of the neck that showed possible left lower parathyroid adenoma and sestemibi scan demonstrated increased up take of left lower parathyroid gland. He had normal study of contrast enhanced computer tomogram of brain, chest and abdomen and upper and lower gastrointestinal endoscopy examination. He underwent left hemi thyroidectomy. Specimen showed 1cm^3 mass on left lower pole of thyroid gland. Histology revealed parathyroid carcinoma with readily identifiable mitotic figure, vascular and capsular invasion in the Hematoxylin and Eosine (figure 1,2,3). Post-operative fasting calcium level was reduced to 8.5 mg/dl.

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Table 1. Laboratory investigation results of the patient

Test	Results	Normal Range
White cell count	6.9	4-11x10^9
Hemoglobin	7.6	11 – 14 g/dl
Platlet count	237000	150-450,000
Serum Na	140	133-145 meq/l
Serum K	2.7	3.4-5 meq/l
Calcium	14.8	8.3 – 10.5 mg/dl
Phosphate	1.6	2.5-4.9 mg/dl
TSH	1.18	0.4-4.0 uIU/ml
Serum PTH	46.32	1.5—7.2 pmol/l
Fasting blood sugar	5.5	3.8 – 5.6 mmol
S.Creatinine	0.7	0.7 – 1.3 mg/dl

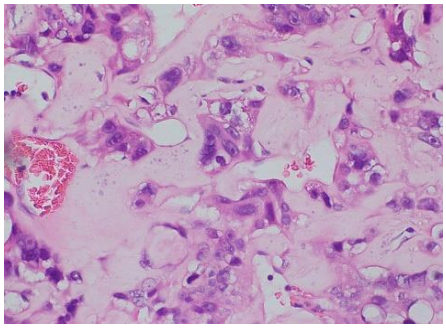

Figure 1: Parathyroid carcinoma showing vascular invasion

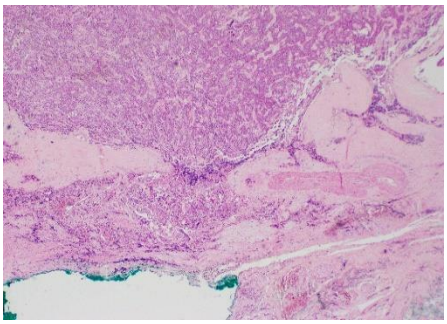

Figure 2: Parathyroid carcinoma showing capsular invasion

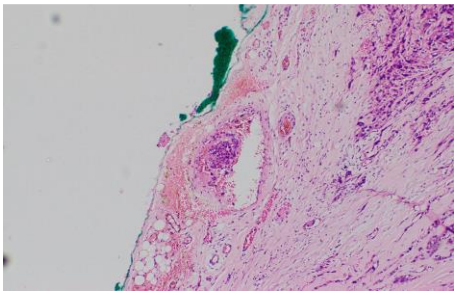

Figure 3: Parathyroid carcinoma showing mitotic figures

Discussion

Primary hyperparathyroidism usually caused by parathyroid adenoma, parathyroid hyperplasia or rarely parathyroid carcinoma (PTC) (1). PTC accounts for 0.4% to 5.2% of all reported cases of hyperparathyroidism, which is approximately 0.2% to 0.5% of malignant endocrine tumors overall (2). The suspicion of malignancy should be in the setting of high calcium level more than 14mg/dl and high parathyroid hormone (PTH) level (>five times of upper normal). Diagnosis PTC was difficult due to no single histopathological feature is pathognomonic for PTC (3,4). There are absolute criteria (table 2) and features associated with malignancy. In the absence of the absolute criteria, at least two, preferably three or more features associated with malignancy should be there (5). The diagnosis was made upon the three minor criteria; mitotic figures, vascular and capsular invasion.

Hypokalemia, metabolic acidosis, high urinary pH (>5.3) favored diagnosis of type 1 renal tubular acidosis (6). Renal tubular acidosis has been reported to be associated with primary hyperparathyroidism. Renal tubular dysfunction due to significant hypercalciuria appears to be one of the proposed mechanisms. We came to final diagnosis as parathyroid carcinoma associated with type 1 renal tubular acidosis and hypercalcemia. Normal parathyroid function was ensured indirectly with repeated serum calcium levels after the surgery where we did not have facilities for serum PTH level measurements in frequently.

Conclusion

This case report highlights the importance of considering parathyroid carcinoma in patients with severe symptomatic hypercalcemia even in the absence of tell tailed features of parathyroid malignancy. In our case all imaging studies favored to parathyroid adenoma. In severe hypercalcemia, histology confirmation should be considered.

Table 2. Absolute criteria and features associated with malignancy

Absolute criteria	Features associated with malignancy.
1. Invasion into surrounding tissue (Thyroid. Esophagus, nerve, soft tissues)	1. Capsular invasion
2. Histological feature presented with distant metastasis	2. Vascular invasion 3. Readily identifiable mitotic figure(>5/10HPF) 4. Broad intraturmoral fibrosis band splitting the parenchyma and separating expansile nodules 5. Coagulative tumor necrosis 6. Diffuse sheet- like monotonous small cells with high nuclear/cytoplasmic ratio 7. Diffuse cellular atypia 8. Macronucleoli present in many tumor cell

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