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CASE REPORT

A RARE CASE OF PARATHYROID ADENOMA WITH CONCURRENT VERTEBRAL BROWN TUMOUR AND ACUTE NECROTIZING PANCREATITIS- A CASE REPORT

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Abstract

Primary hyperparathyroidism is a disorder of one or more parathyroid glands characterized by increased production of parathyroid hormone. Fatigue, muscle weakness, bone pains, osteoporosis, bone fractures and kidney stones are common presenting features of primary hyperparathyroidism. Here we present a middle-aged female with primary hyperparathyroidism due to adenoma of parathyroid gland who presented with pathological fracture of femur, renal calculi, acute necrotizing pancreatitis and vertebral brown tumor.

Keywords: Primary hyperparathyroidism, parathyroid adenoma, pathological fracture, acute necrotizing pancreatitis, brown tumor, case report

1 | INTRODUCTION

Primary hyperparathyroidism is characterized by increased production of parathyroid hormone by the parathyroid gland , which may be due to adenoma of parathyroid gland in 80-85% of cases, hyperplasia of parathyroid gland in 15-20% cases and parathyroid carcinomas in only less than 0.5% (). Females over the age of 50 years usually have this disorder, the incidence being 21/1000 (). Most patients of primary hyperparathyroidism are asymptomatic and hypercalcemia is found in routine laboratory examination. Other cases may present with fatigue, muscle weakness, bone pains, osteoporosis and bone fractures, kidney stones, dyspepsia, constipation and altered sensorium. Some less common presentations include pancreatitis in less than 10% (), and brown tumors in less than 5% cases ().

CASE SUMMARY

A 35 years old woman presented with a history of painful swelling of right lower limb for 3 months. She had a fracture right femur 9 months ago which

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was conservatively managed by the orthopedic department. She also complained of generalized body aches, lethargy, anorexia, nausea, vomiting, constipation, decreased sleep and appetite. She complained of constant back ache as well.

Examination:

Examination revealed a middle aged lady, slightly distressed, was fully conscious and well-oriented. Pulse was 86/min, BP 110/70 mmHg, respiratory rate 20/min, temperature 98.6° F. Chest, cardiovascular and abdominal examinations were unremarkable. On central nervous system examination, plantars were bilaterally up-going and the power of right and left lower limb was 0/5 and 1/5 respectively. Initial diagnosis of osteomyelitis, metabolic bone disease and multiple myeloma was made.

Laboratory Investigations:

Initial investigations showed hemoglobin 7.4 g/dL (normal= 12-15.5 g/dL), total leucocyte count $4.1x10^9$ /L (normal= 4-$11x10^9$/L), platelets $301x10^9$/L (normal= 150-$450x10^9$/L), hematocrit 23.1% (normal= 34.5-44.5%), serum bilirubin 0.7 mg/dL (normal upto 1.0 mg/dL), serum alanine aminotransferase 13 U/L (normal upto 43 U/L), serum alkaline phosphatase 2529 U/L (normal upto 300 U/L), serum lipase 1526 U/L (normal= 23-65 U/L). Renal function tests were normal. Total serum calcium was 13 mg/dL (normal= 9-11 mg/dL), ionized serum calcium was 1.78 mmol/L (normal= 1.13-1.32 mmol/L), vitamin D3 11 ng/ml (deficiency <10 ng/ml, insufficiency= 10-30 ng/ml, sufficiency= 30-100), serum parathyroid hormone 1592.1 pg/ml (normal= 15-68 pg/ml). Bence Jones proteins were negative and serum protein electrophoresis was also normal.

Other Investigations:

Ultrasonogram of abdomen showed 6mm single calculus in right kidney with mild hydronephrosis. CT scan of abdomen was done, the findings of which were suggestive of acute necrotizing pancreatitis with modified CT severity index of 7-8 with peripancreatic fluid collection, left sided pleural effusion, bilateral nephrolithiasis with mild hydronephrosis, multiple lytic lesions diffusely involving the skeleton with pathological fracture of bilateral femori and left sacral ala and subcentimeter mesenteric lymphadenopathy. A soft tissue homogeneously enhancing mass was also seen at level of T11 vertebral body, destroying the T11 pedicle, anteriorly extending intraspinally compressing the spinal canal and spinal cord at this level. MRI of the thoracolumbar spine showed a diffuse mass lesion involving the spinous processes of T11-T12 thoracic vertebrae with extension and involvement of the pedicle of T10 vertebra. Lesion was quite well-defined and did not involve adjacent soft tissues.

Bone biopsy and bone scan were done which were suggestive of osteomyelitis of right femoral shaft and fracture on same side, with multiple focal lesions in rest of the skeleton. Ultrasonogram of neck gave suspicion of parathyroid adenoma in right lobe, 20x15 mm in size. Parathyroid MIBI scan was done, the findings of which were consistent with parathyroid adenoma of right inferior parathyroid gland. Biopsy of the spinal cord mass was taken, which showed a fibromuscular mass, diagnosed as brown tumour, after clinical correlation.

Diagnosis and treatment:

A final diagnosis of parathyroid adenoma, complicated by pathological fracture of femur, deep venous thrombosis (DVT), renal calculi, acute necrotizing pancreatitis and vertebral brown tumour was made. During her stay in medical ward, she was treated for DVT and pancreatitis. She was then referred to surgery department for right inferior parathyroidectomy.

2 | DISCUSSION

Primary hyperparathyroidism has various manifestations which reflect the combined effects of increased parathyroid hormone secretion and hypercalcemia. Our case discusses a 35 year old female patient of primary hyperparathyroidism, presenting with advanced skeletal disease, renal stones, acute necrotizing pancreatitis and vertebral brown tumour. This is a rare presentation as acute pancreatitis and brown tumour have not been reported till date to occur concomitantly in a single case.

Primary hyperparathyroidism is due most often to a parathyroid adenoma secreting parathyroid hormone. Elevated PTH levels cause bone resorption, the formation of polyostotic lesions and a reduction in bone mineral density, predisposing to pathological fractures. Advanced skeletal disease is the most common presentation at a young age (). The final stage of this disease is osteitis fibrosa cystica. Osteitis fibrosa cystica is the end stage manifestation of primary hyperparathyroidism. Histologically, the lesions are characterized by the presence of osteoclasts associated with areas of irregular bone resorption, which is replaced by fibrous vascularised tissue and giant cells. Accumulation of red blood cells and their pigments give a hue to the lesions, hence the name "brown tumour". The first case of brown tumour was reported in 1953 as a fronto-ethmoidal brown tumour (). It usually occurs in the long bones, pelvic girdle, clavicle, ribs, and the mandible. Our case had a vertebral brown tumour in the thoracic region which resulted in paraparesis of lower limbs. Vertebral brown tumours are a rare entity. According to a study, 30 cases of vertebral brown tumours have been reported so far of which 56.7% were in the thoracic region (). According to another study, thoracic brown tumours were found in 59%, followed by lumbar in 27%, cervical 9% and sacral 4.5% (). Our case also had concurrent acute necrotizing pancreatisis. Acute pancreatitis due to hypercalcemia secondary to primary hyperparathyroidism is a known medical entity but it is very uncommon. According to a study, the incidenceof pancreatitis in primary hyperparathyroidism was 8% (). Mixter et al. reported 62 cases of pancreatitis in patients of primary hyperparathyroidism (). A study reported 28 folds increase in the risk of developing pancreatitis in patients of primary hyperparathyroidism as compared to patients without parathyroid disease. Also mean calcium levels were higher in patients having pancreatitis and primary hyperparathyroidism as compared to those having no involvement of pancreas. Thus, it suggested a causal relationship between the two entities and correlated it with higher calcium levels (). A case was reported in 2010 in which acute necrotizing pancreatitis was the first manifestation of primary hyperparathyroidism (). Other cases have also been reported in which pancreatitis was associated with primary hyperparathyroidism and it was suggested that it can serve as a diagnostic clue for primary hyperparathyroidism (). Renal stones have also been linked to primary hyperparathyroidism and according to a study, there was fourfold increase in the prevalence of renal stones in patients of primary hyperparathyroidism compared with subjects not affected by the disorder (). A study found a similar prevalence of kidney stones in normocalcemic and hypercalcemic primary hyperparathyroidism (). Another study found no significant association between 24-hour urinary calcium and renal stone formation although the average value was higher in stone formers compared to non-stone formers. It concluded biochemical factors to be unreliable in the prediction of renal stones in primary hyperparathyroidism (). Also notable in our case is the vitamin D insufficiency, which leads to more severe bone disease and the effects of primary hyperparathyroidism on biochemical, densitometric, and histomorphometric indices are more pronounced (). Another study found the disease to be more severe in patients with concomitant primary hyperparathyroidism and vitamin D deficiency ().

3 | CONCLUSION

Primary hyperparathyroidism has several manifestations with fatigue, muscle weakness, bone pains, pathological fractures, renal stones, dyspepsia being more common. It is commonly seen in women over 50 years of age. We present a case of a 35 year old female patient with pathological fracture of femur complicated by deep venous thrombosis, renal stones with hydronephrosis, vertebral brown tumour resulting in neurological deficit and acute necrotizing pancreatitis making it a rare presentation of parathyroid adenoma.

WHAT IS NEW?

Primary hyperparathyroidism has various manifestations which reflect the combined effects of increased parathyroid hormone secretion and hypercalcemia. Other cases may present with fatigue, muscle weakness, bone pains, osteoporosis and bone fractures, kidney stones, dyspepsia, constipation and altered sensorium. Some less common presentations include

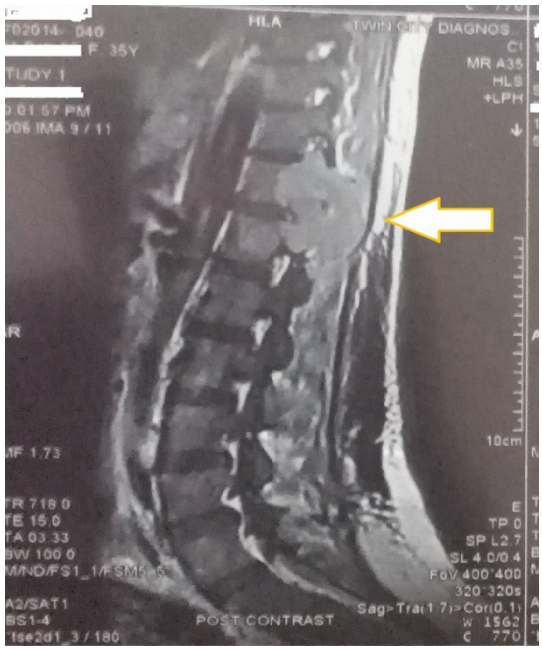

FIGURE 1: MRI of the thoracolumbar spine showing a diffuse mass lesion involving the spinous processes of T11-T12 thoracic vertebrae with extension and involvement of the pedicle of T10 vertebra.

pancreatitis in less than 10%, and brown tumors in less than 5% cases. In this article, the authors present a case of parathyroid adenoma with concurrent vertebral brown tumor and acute necrotizing pancreatitis which has not been published in literature so far.

4 | DECLARATIONS

Ethical approval:

Ethical approval is not required at our institution to publish an anonymous case report.

Consent of patient:

Consent was taken from the patient for publication.

Conflict of interest:

The authors declare that there is no conflict of interest regarding the publication of this case report.

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TABLE 1: Summary of the case

1	**Patient (gender, age)**	Female, 35 years old
2	**Final Diagnosis**	Parathyroid adenoma complicated by pathological fracture of femur, deep venous thrombosis (DVT), renal calculi, acute necrotizing pancreatitis and vertebral brown tumour.
3	**Symptoms**	Painful right lower limb swelling, body aches, lethargy, anorexia, nausea, vomiting, constipation, decreased sleep and appetite, constant backache
4	**Medications**	Medications for treating DVT
5	**Clinical Procedure**	Referred for right inferior parathyroidectomy
6	**Specialty**	medicine

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