ejpmr, 2016,3(5), 442-445

SJIF Impact Factor 3.628

EUROPEAN JOURNAL OF PHARMACEUTICAL AND MEDICAL RESEARCH

www.ejpmr.com

Case Report

ISSN 2394-3211

EJPMR

PRIMARY HYPERPARATHYROIDISM: CASE REPORT OF ADVANCED DISEASE

[1]Dr. Mahesh Brungi*, [2]Dr. Prathap B., [3]Dr. Abdul Gafoor P. M., [4]Dr. Nazneen Abdul Kader, [5]Dr. Ajith T. Abraham

[1]Junior resident, Orthopedic department, Baby Memorial Hospital, Calicut, Kerala India.
[2]Junior resident, Department of General medicine, Kurnool medical college, Andhra Pradesh India.
[3]Junior resident, Orthopedic department, Baby Memorial Hospital, Calicut, Kerala India.
[4]Junior resident, Department of Pathology, Calicut medical college, Calicut, Kerala India.
[5]Junior resident, Orthopedic department, Baby Memorial Hospital, Calicut, Kerala India.

***Corresponding Author: Dr. Mahesh Brungi**

Junior resident, Orthopedic department, Baby Memorial Hospital, Calicut, Kerala India.

Article Received on 03/03/2016 **Article Revised on 24/03/2016** **Article Accepted on 13/04/2016**

ABSTRACT

Primary hyperparathyroidism is a condition characterized by hyper function of one or more parathyroid glands leading to uncontrolled secretion of parathyroid hormone. The most common cause of primary hyperparathyroidism is parathyroid adenoma, followed by hyperplasia and carcinoma in 1 to 2% of cases. Patients most commonly presents with symptoms of hypercalcemia which occurs due to increased resorption of calcium from bones, increased absorption of calcium in bowels and decreased urinary elimination of calcium. Clinically patient presents with multiple bone pain, loss of appetite, nausea, vomiting, loss of weight, constipation, depression and symptoms of nephrolithiasis. Diagnosis is mainly based on the clinical presentation and serum levels of calcium, phosphorus, alkaline phosphatase, albumin and parathormone levels, ultrasonography of neck with FNAC studies and sestamibi scan for parathyroid gland. Here we are reporting a case of a middle aged woman with primary hyperparathyroidism who presented to us with advanced disease. After thorough clinical, laboratory and radiological evaluation, primary hyperparathyroidism due to adenoma in the left parathyroid lobe was diagnosed for which she underwent focused parathyroidectomy. Intra op frozen section confirmed the adenoma. After symptomatic and supportive therapy, she was completely normal and resumed all her daily activities.

KEYWORDS: Primary Hyperparathyroidism, Parathyroid adenoma, Brown tumour, Sestamibi scan.

INTRODUCTION

In primary hyperparathyroidism, hyper function of one or more parathyroid glands leads to uncontrolled secretion of parathyroid hormone. Excessive secretion of PTH due to hypocalcaemia results in secondary hyperparathyroidism. The most common cause of primary hyperparathyroidism is parathyroid adenoma followed by hyperplasia, and carcinoma in 1 to 2% of cases.[1]

Primary hyperparathyroidism occur in 1/1000 individuals in general population with a female:male ratio of 3:1.[1,2] Patients most commonly presents with symptoms of hypercalcemia which occurs due to increased resorption of calcium from bones, increased absorption of calcium in bowels and decreased urinary elimination of calcium. Patients with primary hyperparathyroidism have calciuria with an increased tendency for urolithiasis.

They also have polyuria because of osmotic diuresis which leads to dehydration and loss of weight. Reabsorption of phosphate in the kidneys is reduced which leads to hypophosphatemia and hyperphosphaturia.[1-3]

Clinically patient present with multiple bone pain, loss of appetite, nausea, vomiting, loss of weight, constipation, depression and symptoms of nephrolithiasis.[4,5,6] Clinical presentation depends on the level of parathormone and hypercalcemia. Diagnosis is mainly based on the clinical presesntation, serum level of calcium, phosphorus, alkaline phosphatase, albumin and parathormone levels, ultrasonography of neck with FNAC studies and ^{99m}Tc-sestamibi scan for parathyroid gland.

Here we are reporting a case of a middle aged woman with primary hyperparathyroidism who presented to us with advanced disease.

CASE REPORT

43 year old female who was apparently normal in the past was referred to our institution with h/o multiple pathological fractures, loss of weight, generalized weakness, nausea, vomiting, psychological disturbances, and menstrual irregularity. Complaints began one year

back when she had episodes of edema of the feet and muscle weakness for which she underwent native treatment.

Later she developed difficulty in walking following which she sustained an intertrochanteric fracture of the right femur (Fig. 1) while getting down from the bed (without any traumatic event). She was treated surgically with a proximal femoral nail for right femur (Fig. 2) from a local hospital.

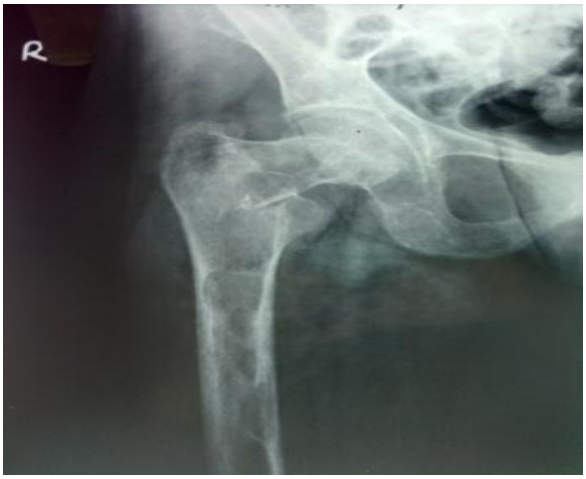

Fig1.intertrochanteric fracture (RT)hip With multiple lytic lesions in proximal femur

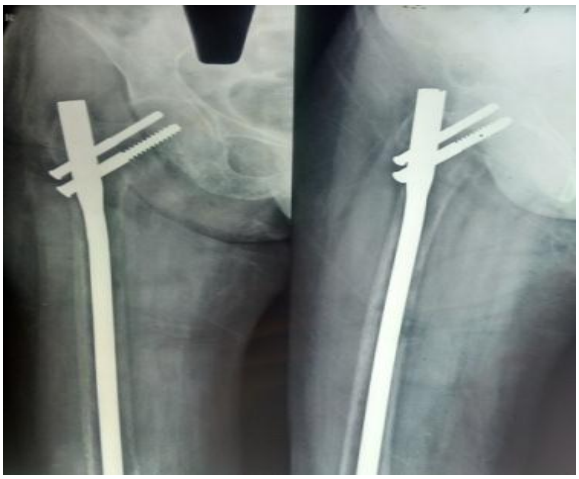

Fig2. PFN right hip

She was hospitalized for 12 days, and was mobilized with a walker after two months. While mobilization she developed severe pain in both forearms, for which radiographs were done which revealed multiple lytic lesions with pathological fractures (Fig. 3). She was treated with rush nail for both forearms (Fig. 4).

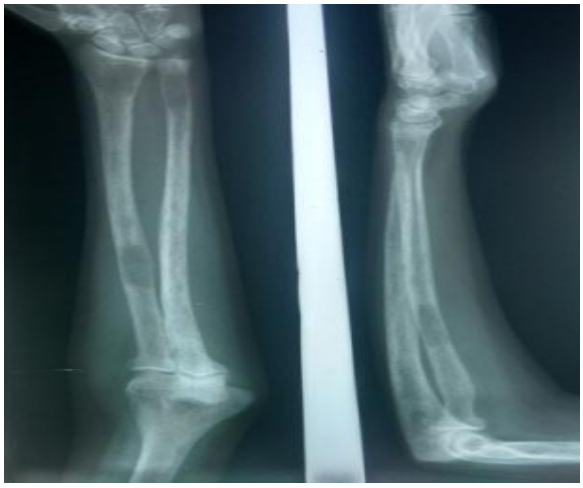

Fig 3. Multiple lytic lesions in both bone forearm with pathological fracture

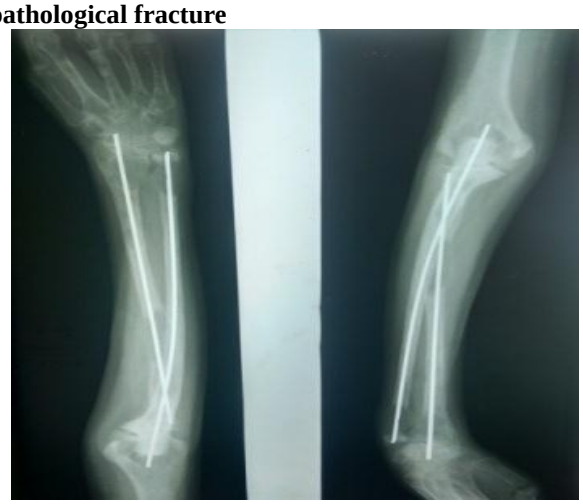

Fig.4. Rush nailing both bone forearm

Three months later she sustained an intertrochanteric fracture of the left hip (Fig. 5) and diaphyseal fracture of left tibia spontaneously without any trauma and she was treated with femoral nail for left hip (Fig. 6) and intra medullary nail for left tibia.

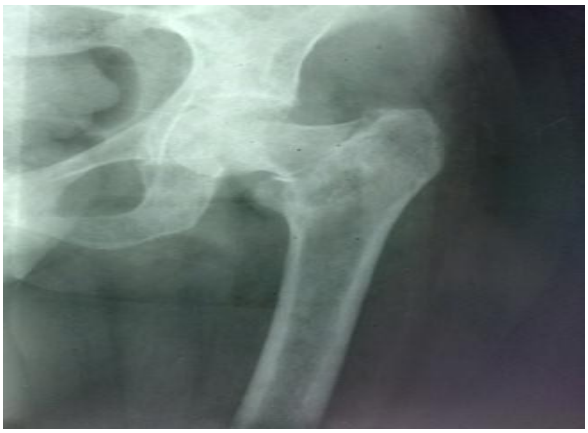

Fig.5. intertrochanteric fracture (RT)hip With multiple lytic lesions in proximal femur

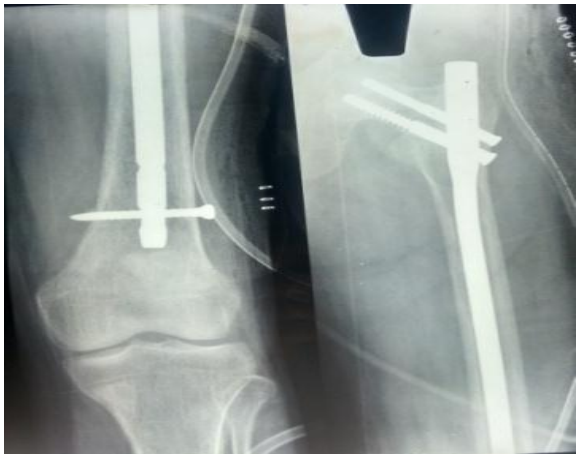

Fig6. PFN left femur

All the surgical procedures were done by a surgeon elsewhere who missed to evaluate the patient to identify her basic pathology. During this year she lost 18 kg of her weight and had developed recurrent episodes of nausea, vomiting, muscle weakness, psychiatric disturbances and menstrual irregularities.

She was referred to our institute for further evaluation and management. At the time of admission serum calcium, ALP, PTH and serum albumin were done and calcium (13.8 mg/dl) parathyroid hormone (1924.7 pg/ml) and alkaline phosphatase (673 U/L) were found to be elevated. On examination there was a palpable swelling on the left side of the neck. USG neck showed an enlarged left sided inferior pole of thyroid gland (33X15.4X24.1 mm). Parathyroid scintigraphy (MIBI) scan was done which showed increased uptake in the left inferior parathyroid gland. Skull x-ray (Fig. 7) showed pepper pot appearance, and absence of lamina dura, and x ray of the hand (Fig. 8) showed sub periosteal resorption of middle phalanx on radial side.

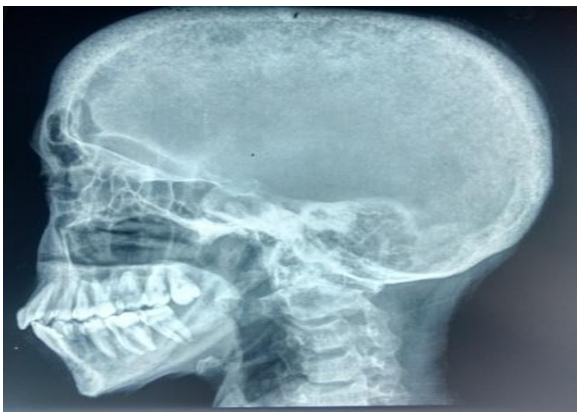

Fig7. Skull x ray showing pepper pot appearance absence of lamina dura

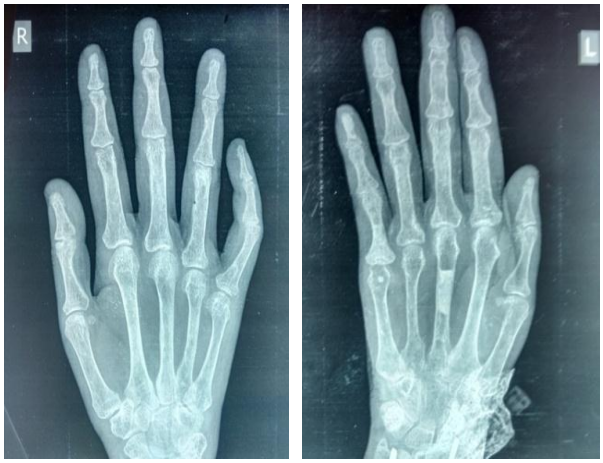

Fig8. Hand x ray showing sub periosteal resorption of and middle phalanx on radial side

Radiography of right whole femur (Fig. 9) showed multiple lytic lesions (brown tumors) and resorbed bone. Endocrine surgery consultation was done and she underwent focused parathyroidectomy. After 30 mins of surgery PTH levels dropped to 106pg/ml from 1749pg/ml. Frozen section was done intraoperatively and parathyroid adenoma was confirmed on histopathological examination. Post operatively serum ca and serum albumin levels were evaluated daily and calcium supplementation was given to prevent hungry bone syndrome.

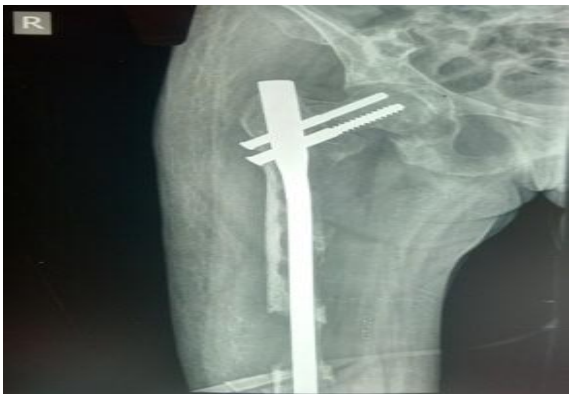

Fig9. X ray RT hip with femur showing multiple Lytic lesions (brown tumor)

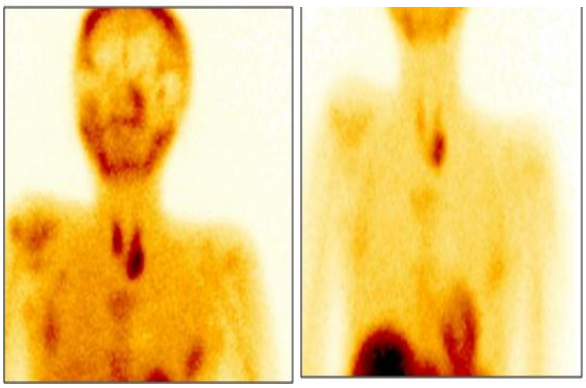

Fig10. Increased uptake of left parathyroid lobe in Parathyroid scintigraphy (MIBI) scan

DISCUSSION

Primary hyperparathyroidism (PHPT), is the third most common endocrinopathy, mainly affects middle aged women with 3:1 ratio.[1] It results from intrinsic hyper functioning of one or more of the parathyroid glands.[1] An individual usually have four glands which weights about 25-50 mg.[1] The most common cause of primary hyperparathyroidism is a solitary adenoma(75%-85%) followed by hyperplasia(10-20%), multiple adenomas (4%-5%) and carcinoma (1 to 2%).[1,2,3]

Majority (70%-85%) of patients with primary hyperparathyroidism are asymptomatic.[1,4,5,6] They are usually diagnosed when hypercalcemia is revealed in routine blood tests which triggers further investigation. Symptomatic patients may present with systemic features of nausea, vomiting, loss of weight, anorexia, dyspepsia, muscle weakness, anxiety, irritability, impaired concentration, psychosis, neurosis, depression, ataxia, coma, hypertension, arrhythmia, coronary artery disease, chondrocalcinosis, pseudogout, multiple kidney stones, constipation, multiple bone pains and pathological fractures.[1,4,5,6] Our case was an advanced disease with most of the above mentioned clinical features.

In laboratory analysis, patients with PHPT exhibit hypercalcemia (normocalcemia in some cases) with normal to high levels of PTH. Even normal level of PTH is inappropriate, considering the coexistent hypercalcemia. Hypophosphatemia, decrease in 25-hydroxyvitamin D, and increase in 1, 25-dihydroxyvitamin D Levels will also be present.[5,6,7] Our case had hypercalcemia with high PTH levels. The differential diagnosis for patients with hypercalcemia associated with normal levels of PTH are sarcoidosis, Paget's disease, hypervitaminosis A or D, lithium or thiazide consumption.[6] For localizing the tumor imaging tests such as ultrasonography (US), computed axial tomography (CT), magnetic resonance imaging (MRI) and 99mTc-sestamibi scintigraphy are available. 99mTc-sestamibi scintigraphy has the highest sensitivity (nearly 90% in single adenomas, but only 45%-60% if hyperplasia, and 30%-73% if double adenoma are implicated). It has the ability to detect ectopic mediastinal parathyroid glands.[8]

Focused parathyroidectomy is the treatment option in patients with primary hyperparathyroidism. After removal of tumor, immediate drop in the level of PTH will be present introperatively. Histopathologically parathyroid adenoma is usually a well circumscribed, tan brown colour tumour of size less than 5 grams. Most often they are capsulated. Microscopically the tumour is composed of uniform, polygonal cells with clear cytoplasm, small centrally placed nuclei with no capsular invasion. A brown tumour microscopically shows increased osteoclastic activity predominantly in cortical bone. The surrounding marrow is replaced by fibrovascular tissue and giant cells. There is increased vascularity, hemorrhage and hemosiderin deposition which gives the characteristic brown colour. In severe cases there is peritrabecular fibrosis, increased osteoclastic activity and brown tumour which is called osteitis fibrosa cystic or Von Recklinghausens disease.

Post parathyroidectomy patients may develop hungry bone syndrome because of the rapid usage of serum calcium and phosphorus for remineralization of the resorbed bone.[9] Regular monitoring of serum calcium and phosphorus levels with uninterrupted vitamin D and calcium supplementation is important to prevent hungry bone syndrome.

CONCLUSION

Hypercalcemia, pathological fractures and multiple lytic lesions are manifestations of hyperparathyroidism and malignancies. Though hyperparathyroidism is a common entity encountered during clinical practice, high index of suspicion with meticulous clinical examination and detailed laboratory evaluation is required to diagnose the condition and prevent the complications.

REFERENCES

1. Fraser WD. Hyperparathyroidism. Lancet., 2009; 374(9684): 145-58.
2. Wermers RA, Khosla S, Atkinson EJ, Hodgson SF, O'Fallon WM, Melton LJ 3rd. The rise and fall of primary hyperparathyroidism: a population-based study in Rochester, Minnesota, 1965–1992. Ann Intern Med., 1997; 126: 433–40.
3. Catherine Cormier, Jean-Claude Souberbielle, André Kahan. Primary hyperparathyroidism and osteoporosis in 2004. Joint Bone Spine., 2004; 71(3):183-9.
4. Udelsman R, Pasieka JL, Sturgeon C, Young JE, Clark OH. Surgery for asymptomatic primary hyperparathyroidism: proceedings of the Third International Workshop. J Clin Endocrinol Metab., 2009; 94(2): 366-72.
5. Shoback D, Sellmeyer D, Bikle DD. Metabolic bone disease. In: Gardner DG, Shoback D. Greenspan's Basic & Clinical Endocrinology. 8th edition. McGraw-Hill companies., 2007; 281-345.
6. Blanchard C, Mirallié E, Mathonnet M. Sporadic primary hyperparathyroidism. J Visc Surg., 2010; 147(5): e285-95.
7. Mendoza V, Ramírez C, Espinoza AE, González GA, Peña JF, Ramírez ME, et al. Characteristics of ectopic parathyroid glands in 145 cases of primary hyperparathyroidism. Endocr Pract., 2010; 16(6): 977-81.
8. Jaskowiak N, Norton JA, Alexander HR, Doppman JL, Shawker T, Skarulis M, et al. A prospective trial evaluating a standard approach to reoperation for missed parathyroid adenoma. Ann Surg., 1996; 224(3): 308-20; discussion 320-2.
9. Smith D, Murray BF, McDermott E, O'Shea D, McKenna MJ, McKenna TJ. Hungry bones without hypocalcaemia following parathyroidectomy. J Bone Miner Metab., 2005; 23(6): 514-5.