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PARATHYROID ADENOMA: A CASE REPORT

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ABSTRACT: Parathyroid adenoma, generally presenting with hypercalcemia and high parathyroid hormone, is a rare cause of primary hyperparathyroidism. We present the case of a 48-year old woman who presented with persistent vomiting for 3 months along with mild epigastric pain and retrosternal burning associated with heaviness after meals, bloatedness and indigestion of food. She had generalized body aches and easy fatigability, lethargy and generalized weakness. She reported difficulty in going to sleep along with repeated awakenings during the night, low-mood and feeling anxious throughout the day along with inability to concentrate. On work up, she had hypercalcemia and was subsequently diagnosed with Parathyroid adenoma causing Primary Hyperparathyroidism.

Keywords: Hypercalcemia, Parathyroid Adenoma, Primary Hyperparathyroidism.

I. INTRODUCTION

The parathyroid glands are primarily responsible for maintaining plasma calcium level by enhancing calcium and phosphate release from bone matrix, enhancing renal calcium reabsorption, and stimulating renal 1,25-dihydroxyvitamin D production to subsequently increase intestinal calcium absorption.[1] Elevated parathyroid hormone production thus leads to raised serum calcium concentrations. Hyperparathyroidism is categorised as primary, secondary or tertiary hyperparathyroidism depending on the etiology. Parathyroid adenoma, generally presenting with hypercalcemia and high parathyroid hormone, is a rare cause of primary hyperparathyroidism seen in about 1:1000 in the US being twice more frequent in women as compared to men.[2] Majority of hyperparathyroid patients are clinically asymptomatic. Common presentations of hyperparathyroidism include features of hypercalcemia, osteoporosis and bone fragility. Calcium deposition in renal parenchyma can lead to

recurrent stone formation and subsequently renal dysfunction.[3] Management of parathyroid adenoma is aimed at complete surgical resection and patients generally recover without recurrence or long-term complications.

II. CASE REPORT

We present the case of a 48-year old woman admitted through the out-patient department because of vomiting, epigastric pain and bodyaches. She was in her usual state of health 3 months ago when she developed complaints of anorexia and nausea along with persistent vomiting, multiple times a day, 5-10 minutes after ingestion of meals. The vomitus was yellowish in colour, 1-1.5 cup per episode and contained ingested food materials. Along with this, she had complaints of mild epigastric pain and retrosternal burning associated with heaviness after meals, bloatedness and indigestion of food. It was non-radiating, dull in character and had no specific aggravating or relieving factors. She complained of relative constipation, passing hard stools every 4-5 days after taking laxatives. There was history of weight loss in the last 3 months, from 72 kg to 48 kg. She had generalized body aches without joint pain, swelling or stiffness. She complained of easy fatigability, lethargy and generalized weakness especially when getting up from squatting position. There was history of polyuria but no complaint of lumbar pain, frothing of urine or blood in urine. She reported difficulty in going to sleep along with repeated awakenings during the night, low-mood and feeling anxious throughout the day along with inability to concentrate. There was no history of dysphagia, odynophagia, shortness of breath, cough, sputum, fever, fits, palpitations, chest pain or tightedness, head ache, blurring of vision, memory loss, bleeding tendency, fractures, lumps or bumps.

She had been diagnosed with type-II diabetes mellitus 3 months ago and was taking oral antihyperglycemics regularly. She was married with one child and menopausal for 2 years. She had no known allergies and did not smoke or use illicit drugs. There was no family history of diabetes, hypertension, heart disease, tuberculosis malignancy or any autoimmune disorder. On examination the patient appeared anxious but had normal vitals signs. Movements of wrist, elbow, ankle and knee joints caused mild discomfort but no tenderness or swelling were noted. She had difficulty standing up from squatting but neurological examination was normal. There was no palpable mass in the neck. The remainder of her physical examination was unremarkable. Initial blood workup of the patient is shown in Table I which demonstrates hypercalcemia. Urinalysis revealed clear, yellow urine, with a specific gravity of 1.010, a pH of 6.0, albumin trace positive and negative results for occult blood, leukocyte esterase, nitrite, glucose, ketones, bilirubin, and urobilinogen. Abdominal ultrasound revealed bilateral renal parenchymal changes with normal renal size and a uterine fibroid; rest of the scan was unremarkable. Chest X-Ray (PA view) was unremarkable. CT scan (plain) of the brain was also unremarkable. Viral markers (HBsAg/Anti-HCV) were negative.

Table I: Initial blood work up of the patient

Investigation	Patient's Value	Reference Range
Hemoglobin (G/dl)	11.0	12.0-16.0
TLC (per mm^3)	8,800	4,000-11,000
Platelets (per mm^3)	226,000	150,000-450,000
Hematocrit (%)	34	35-46
MCV (fL)	81.4	77-91
MCH (pg)	26.7	26-32
MCHC (G/dl)	32.8	32-36
PT (seconds)	15	13
APTT (seconds)	36	34
Blood Urea (mg/dl)	35	10-50
Serum Creatinine (mg/dl)	1.0	0.6-1.4
Albumin (G/dl)	4.1	3.5-5.0
Globulin (G/dl)	3.3	1.8-3.2
Serum Bilirubin (mg/dl)	0.6	Upto 1.0
AST (U/L)	27	Less than 35
ALT (U/L)	17	Less Than35
Alkaline Phosphatase (U/L)	202	30-120
Sodium (mmol/L)	144	133-150
Potassium (mmol/L)	3.8	3.5-5.0
Calcium (mg/dl)	18.6	8.4-10.2
Phosphate (mg/dl)	3.3	3.0-7.0
Bicarbonate (mmol/L)	24.9	22.0-29.9
Chloride (mmol/L)	105.9	97.0-110.0
Uric Acid (mg/dl)	6.5	2.5-7.0
CRP (mg/dl)	65.20	Upto 6.0

Blood work up to look for cause of hypercalcemia and Endocrinology profile to rule out complex hereditary syndromes such as multiple endocrine neoplasia (type I and IIA), familial isolated hyperparathyroidism, familial hypocalciuric hypercalcemia are shown in Table II which demonstrates primary hyperparathyroidism. Neck Ultrasound followed by a Sestamibi radioactive-isotope scan showed a 34x20x19mm sized well-circumscribed oval heterogenous hypoechoeic mass, present posterolateral to the left lobe of the thyroid lying outside the thyroid capsule suggestive of parathyroid adenoma. The patient was diagnosed with Parathyroid adenoma causing primary hyperparathyroidism and hypercalcemia. Treatment with intravenous anti-emetics, intravenous proton-pump inhibitors, vigorous intravenous fluids, intravenous furosemide, subcutaneous calcitonin, intravenous bisphosphonates was initiated and subsequently consultation for surgical resection of parathyroid adenoma was planned.

Table II: Blood work up for hypercalcemia and Endocrinology profile

Investigation	Patient's Value	Reference Range
Intact PTH (pg/ml)	990	11-67
Repeat S/Calcium (mg/dl)	20.1	8.4-10.2
Repeat S/Creatinine (mg/dl)	1.5	0.6-1.4
Urinary Calcium (mg/dl)	18.88	6.8-21.3
Urinary Creatinine (mg/dl)	14.23	28-217
25-hydroxyvitamin D (ng/ml)	12.74	40-100
Intact PTH (pg/ml)	990	11-67
TSH (iIU/ml)	0.526	0.35-4.95
T4 (ug/dl)	6.65	4.9-11.7
Free T3 (pg/ml)	2.10	1.71-3.71
ACTH (pg/ml)	17.4	9-52
Serum Cortisol (ug/dl)	27.76	6.2-19.4
FSH (mIU/ml)	30.100	21.7-153
LH (mIU/ml)	13.800	11.3-39.8
Prolactin (ng/ml)	8.52	1.90-25
Urinary VMA (mg/24 hours)	4	2-7

III. DISCUSSION

Primary hyperparathyroidism is not an uncommon disease having a notable female predominance characterized by hypercalcemia and elevated parathyroid hormone levels. Causes of isolated primary hyperparathyroidism include parathyroid hyperplasia, parathyroid adenoma and parathyroid carcinoma. However parathyroid tumours may not be clinically visible or palpable. The presentation of these patients is generally with clinical features of hypercalcemia which are very diverse.[4] The classic pentad of hypercalcemia includes renal stones, painful bones, psychic moans, abdominal groans and fatigue overtones.[5] Early hypercalcemia is often not diagnosed especially in developing countries like Pakistan due to limited monetary and laboratory resources. Common long-term complications of untreated hypercalcemia include low bone mineral density leading osteopenia and osteoporosis, and subsequently increased bone fragility with increased fracture risk.[4] In addition to high levels of serum calcium and parathyroid hormone, other laboratory findings of primary hyperparathyroidism include hypophosphatemia and increased urinary calcium which indicate altered calcium homeostasis.[5] The most frequently used technique to localize parathyroid adenoma is to combine neck ultrasound and ^{99m}Tc-sestamibi scintigraphy (MIBI) scan. Limitation of using neck ultrasound alone is that it may not reveal the exact extent of adenoma and miss out on ectopic lesions in the mediastinum.[6]

Surgical exploration of neck with resection of pathological parathyroid glands is the definitive treatment of parathyroid adenoma and patients generally recover without recurrence or long-term complications.[7] Primary Hyperparathyroidism rarely may exist as part of complex hereditary syndromes such as multiple endocrine neoplasia (type 1 and 2A), familial isolated hyperparathyroidism, familial hypocalciuric hypercalcemia and adequate assessment on history, examination and investigations to rule out these should be done.[8] In conclusion, primary hyperparathyroidism and parathyroid adenoma need to be considered when patients present with longstanding non-specific features of hypercalcemia including bodyaches, lethargy and abdominal pains as in our patient to avoid delay in diagnosis and prevent complications. Parathyroid adenoma shows excellent outcome after complete surgical resection.

Consent: Informed consent was taken from the patient.

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