HYPERCALCAEMIA MASQUERADING AS FATIGUE IN ELDERLY

Singh Anup[1,*], Annesh Bhattacharya[2]

[1]Assistant Professor, [2]Ex-Junior Resident, Department of Medicine, Institute of Medical Sciences, Banaras Hindu University, Varanasi, Uttar Pradesh, India, Pin-221005

***Corresponding Author:**
E-mail: Dranupbhu@gmail.com

ABSTRACT:

The clinical spectrum of hypercalcaemia ranges from being asymptomatic to life threatening situations. Primary hyperparathyroidism is one of cause of hypercalcemia caused by excessive secretion of parathyroid hormone from one or more parathyroid glands. It results from an abnormally high level of serum calcium and an increased level of parathyroid hormone. We present a case of hypercalcaemia presenting as fatigue in an elderly female. So high level of suspicion is needed in evaluating elderly people.

CASE:

A 65 year old postmenopausal female presented with the complaints of generalised fatigue for 1 month and loss of appetite. There was no history of fever, burning micturition, constipation, altered behaviour, pain at any site, altered bladder and bowel symptoms, loss of weight.

General examination revealed only a moderately elevated Blood Pressure of 150/90 mmHg and mild pallor. Rest of the systemic examination was within normal limits.

The patient was subsequently subjected to investigation, which revealed normocytic normochromic anemia with haemoglobin of 9.8gm%, with normal total leucocyte count and platelet count. Serum creatinine of patient was 1.29mg/dl, with serum total Protein of 8.3g/dl, Albumin of 4.5g/dl, Alkaline Phosphatase of 373 U/l, and elevated serum Calcium (15.5 mg/dl), elevated Ionic Calcium (2.25 mmol/l) with a low Phosphate (2 mg/dl). Thyroid profile was within normal limits. Chest X-Ray and ECG was within normal limits, while X-Ray of the pelvis revealed features of bilateral sacroilitis. Ultrasonography of Abdomen showed presence of bilateral nephrolithiasis, with no evidence of any other pathology. This was further confirmed on CT scan of abdomen, which additionally revealed presence of multiple lytic lesions in bilateral iliac bones, right femoral head and left femoral neck. With this a provisional diagnosis of an unknown primary malignancy with multiple skeletal metastasis was made, and further investigations was planned accordingly.

To rule out multiple myeloma, serum electrophoresis and urinary Bence-Jones protein excretion was performed which were inconclusive. 24 hour Urinary Calcium excretion was 168 mg and Phosphate excretion was 930 mg. Serum Parathyroid (PTH) was markedly elevated (2101 pg/ml , Normal 11-60 pg/ml). Skeletal survey showed resorption of distal phalanges of 1st, 3rd and 4th digits of left hand. Skull X-ray showed a typical mottled salt-Pepper appearance. Ultrasonography of the neck detected only an isolated left-sided thyroid adenoma, but failed to detect any parathyroid lesion. Hence patient was resorted to 123-I Metaiodobenzylguanidine scintigraphy(MIBG) scan, which showed increased uptake of radioactive tracer in left lower parathyroid gland (figure 1). Hence the diagnosis of hyperparathyroidism due to single parathyroid adenoma was made.

The patient was symptomatically treated by hydration with intravenous fluids and given diuretics for treating hypercalcaemia. She was also given a single injection of Zoledronic Acid (5mg) intravenously. Blood pressure was controlled with S-Amlodipine 5mg, once daily dosing. Serum calcium returned to normal and subsequently the patient was referred to higher centre for removal of Parathyroid Adenoma.

Table 1: Clinical presentations of hypercalcemia

Renal	Nephrolithiasis, Nephrocalcinosis, chronic kidney disease, Nephrogenic diabetes insipidus Polyuria, nocturia, prerenal acute kidney injury
Gastrointestinal	Abdominal pain, nausea, Constipation, Anorexia, weight loss, Pancreatitis
Skeletal	Bone pain, Osteoporosis
Neurological	Impaired cognition Fatigue, Weakness ,Decreased level of consciousness
Cardiovascular	Hypertension ,Bradycardia ,Shortened QT interval, Arrhythmia

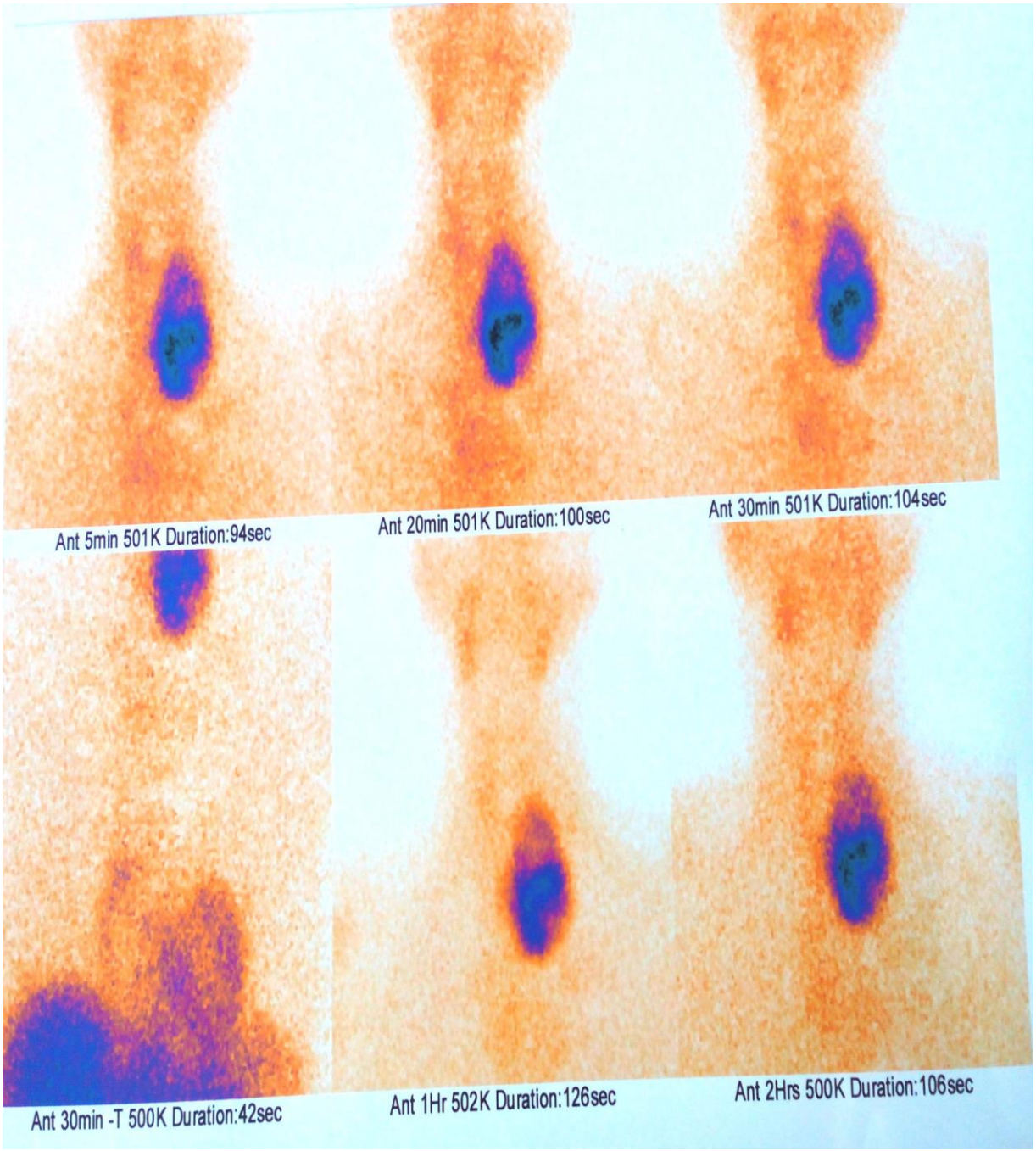

Fig. 1: Legend: Metaiodobenzylguanidine scintigraphy(MIBG) scan showing increased uptake of radioactive tracer in left lower parathyroid gland.

DISCUSSION

Clinical spectrum of hypercalcemia ranges from being asymptomatic in patients with mild chronic hypercalcemia to life threatening severe obtundation and coma(Table 1). The rate of rise of serum calcium concentration and the degree of hypercalcaemia, often determines symptoms and the urgency of therapy. The treatment should be guided accordingly [1-3].

Patients with asymptomatic or mildly symptomatic hypercalcemia (calcium <12 mg/dL [3 mmol/L]) do not require immediate treatment. Similarly, a serum calcium of 12 to 14 mg/dL (3 to 3.5 mmol/L) may be well-tolerated chronically, and may not require immediate treatment. However, an acute rise to these concentrations may cause marked changes in sensorium, which requires more aggressive measures. In addition, patients with a serum calcium concentration >14 mg/dL (3.5 mmol/L) require treatment, regardless of symptoms.

Treatment for hypercalcemia should be aimed both at lowering the serum calcium concentration and, if possible, treating the underlying disease. Effective treatments reduce serum calcium by inhibiting bone resorption, increasing urinary calcium excretion, or decreasing intestinal calcium absorption. The optimal choice varies with the cause and severity of hypercalcemia. In practice the most common etiologies of hypercalcemia are primary hyperparathyroidism and malignancy, two diseases that present quite differently.1 Isolated hyperparathyroidism generally causes mild, asymptomatic hypercalcemia that is detected on unrelated investigations. In contrast, malignancy may present with severe, symptomatic hypercalcemia along with symptoms due to the malignancy itself. Common malignancies include: multiple myeloma, lung cancer, breast cancer, and lymphoma. While primary hyperthyroidism and malignancy account for approximately 90% of hypercalcemia, there exist numerous other causes. Recently there has also been increased recognition of multifactorial hypercalcemia, including coexistence of primary hyperparathyroidism and malignancy[4]. Due to the broad differential and the potential for multifactorial etiologies, an organized approach to the underlying cause of hypercalcemia is essential. Diagnosing the underlying cause of hypercalcemia often requires days, therefore initial management of the hypercalcemia is uniformly empiric. In contrast, chronic management relies on both treatment of the underlying etiology, as well as etiology-specific treatment of hypercalcemia.

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