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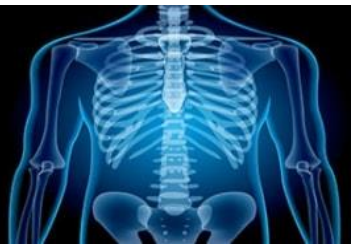

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An unusual presentation of hypercalcemia-imaging and interventions: A case report

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Abstract
Background: Multisystem manifestations of hypercalcemia include stones, painful bones, psychic groans, and abdominal moans. All manifestations in a single patient are extremely rare. Hypercalcemia is often diagnosed incidentally when a high calcium level is detected in blood samples.
Case illustration: Here we present a very rare case of hypercalcemia which presented as supra-refractory status epilepticus and other manifestations in the later phase. A young man's journey through neurological, pancreatic, and renal manifestations of hypercalcemia, the role of diagnostic imaging in revealing parathyroid adenoma, and interventional radiology in providing the definitive cure and management of complications are explained here.
Conclusion: Careful history and physical examination focusing on the clinical features of hypercalcemia, possible causative diseases, and medication is important. The laboratory workup and correct interpretation help the physician narrow down the differentials. Increased screening of calcium levels and the wide availability of reliable assays for intact parathyroid hormone (PTH) levels have led to more frequent and earlier diagnoses of primary hyperparathyroidism. The role of radiology in hypercalcemia is not limited to diagnosis, but it involves managing complications in each phase and in providing a definitive cure.

Keywords: Hypercalcemia, parathyroid hormone, parathyroid adenoma

Introduction

Case Report: 46-year-old Asian male with systemic hypertension on a single antihypertensive was brought to the emergency department with acute mental confusion and recurrent seizures. In view of the status of epileptics and low GCS score, elective intubation was done. The patient underwent CT brain which ruled out hemorrhage. MRI showed features of posterior reversible encephalopathy syndrome (PRES).

Blood test results were suggesting the possibility of primary hyperparathyroidism (Table 1). Bedside ultrasound of the neck (Figure: 1) showed a well-defined hypoechoic lesion inferior to the lower pole of the left lobe of the thyroid, raising the possibility of parathyroid adenoma. It was confirmed with TC-99^{m} SESTAMIBI scan (Figure: 1). Considering the hemodynamic instability and poor GCS score a minimally invasive ultrasound-guided percutaneous microwave ablation of the parathyroid adenoma was done. Successful ablation was performed in 1 minute and 30 seconds using the single-shot moving technique. Post-procedure blood results were really satisfying. There was no further episode of seizures.

A few days later the patient developed intermittent fever spikes and a rapid rise in serum lipase and amylase. Bedside ultrasound of the abdomen revealed bulky and heterogenous pancreas with peripancreatic fluid. Contrast-enhanced CT abdomen showed features of acute severe necrotizing pancreatitis. Thrombus in the superior mesenteric vein, left branch of the portal vein, and bilateral mild pleural effusion constituted a modified CT severity index of 10 (Figure: 2). An obstructive right distal ureteric calculus and a few tiny right renal calculi were present. The patient underwent laser lithotripsy and a DJ stenting for the same. Pancreatitis was initially managed conservatively, later when fever spikes were persisting and repeat imaging showed an increase in peripancreatic collection, a pigtail drain was inserted.

Bilateral iliofemoral venous filling defects were detected incidentally in CECT and Doppler confirmed deep venous thrombosis (Figure: 3). An IVC filter was placed to prevent proximal migration of thrombus and pulmonary embolism.

At the end of 75 days, decannulation of the tracheostomy tube was done and the patient started tolerating oral feeds. Relevant clinical, laboratory, and imaging findings and management of the patient during these 75 days are summarised in Table 1.

Table 1: Relevant clinical, laboratory, and imaging findings and management of the patient during these 75 days

DAY	CLINICAL DIAGNOIS	LABS	IMAGING	MANAGEMENT/INTERVENTION
1-2	STATUS EPILEPTICUS, ALCOHOL ABUSE BP-180/80 E1V2M2	**AST/ALT-1116/480** **S.CA-15.7mg/dl(8.6-10.2)** **ION.CA-2.2 mmol/l(1.1-1.3)** **S.PHOS-1.1 mg/dl(2.7-4.5)** **PTH-348pg/ml(15-65)** **AMYLASE-812** **LIPASE-2357** **S.CREAT-1.2** **TC-15400 cells/cumm**	**CT HEAD**-POST ICTAL CHANGES. NO HEMORRHAGE **MRI BRAIN**-SUGGESTIVE OF PRES	INTUBATED ANTIEPILEPTICS ANTIBIOTICS THIAMINE IV NORMAL SALINE INJ.CALCITONIN CA FREE HEMODIALYSIS
3-8	HYPERCALCEMIC SEIZURES, SUPRAREFACTORY STATUS EPLILEPTICUS HYPERPARATHYROIDISM AKI, GCS-2T	S.CA-9.9 ION. CA-1.5 PTH-462	**USG NECK**-SINGLE PARATHYROID ADENOMA **SESTAMIBI SCAN**-CONFIRMS PARATHYROID ADENOMA	MINIMALLY INVASIVE USG GUIDED **MICROWAVE ABLATION OF PARATHYROID ADENOMA**(MOVING SHOT TECHNIQUE(1MIN 30SEC)
9-12	POST ABLATION POST TRACHEOSTOMY NO FURTHER SEIZURES GCS-8T	POST ABLATION PTH-66 S.CA-9.9 ION. CA-1.4 HB-8.1→6.8 RISING- ALT/AST	**USG ABDOMEN**- S/O PANCREATITIS. **CECTABDOMEN**-ACUTE SEVERE NECROTIZING PANCREATITIS WITH COMBINED PANCREATIC AND PERIPANCREATIC NECROSIS. MCTSI-10. OBSTRUCTIVE RIGHT DISTAL URETERIC CALCULUS	CONSERVATIVE MANGEMENT OF PANCREATITIS. **RIGHT URS LASER LITHOTRIPSY+DJ STENTING**
13-27	POST ABLATION POST TRACHEOSTOMY ACUTE NECROTIZING PANCREATITIS PERSISTING FEVER SPIKES, ABDOMINAL SEPSIS.	HB-7.3 TC-10200 CRP-92.7 S. CREAT-1.2	**REPEAT CECT ABDOMEN**- LARGE PERIPANCREATIC COLLECTION, B/L ILIAC VEIN THROMBOSIS **B/L LOWERLIMB VENOUS DOPPLER**-LEFT ILEOFEMORAL VENOUS THROMBOSIS	**PIGTAIL CATHETER INSERTION AND DRAINAGE** **INFRA RENAL IVC FILTER PLACEMENT**
28-70	PARATHYROID ADENOMA HYPERCALCEMIC CRISIS POST ABLATION ACUTE NECROTIZING PANCREATITIS. SEPSIS ILEOFEMORAL VENOUS THROMBOSIS POST TRACHEOSTOMY	TC-12000 HB-7.9 BACTERIAL CULTURE AND SENSITIVITY (PUS)-MODERATE GROWTH OF BURKHOLDERIA CEPACIA	NIL	SYMPTOMATIC AND SUPPORTIVE MEASURES
70-75	PARATHYROID ADENOMA HYPERCALCEMIC CRISIS POST ABLATION ACUTE NECROTIZING PANCREATITIS. SEPSIS ILEOFEMORAL VENOUS THROMBOSIS POST TRACHEOSTOMY	**TC-10400** **PTH-31.5** **S.CA-9.2** **ION.CA-1.37** **S.PHOS-4.4** **S.CREAT-0.6**	NIL	**DECANULATION OF TRACHEOSTOMY TUBE** **TOLERATING ORAL FEEDS** **NO FURTHER SEIZURE/ FEVER SPIKES** **HEMODYNAMICALLY STABLE**

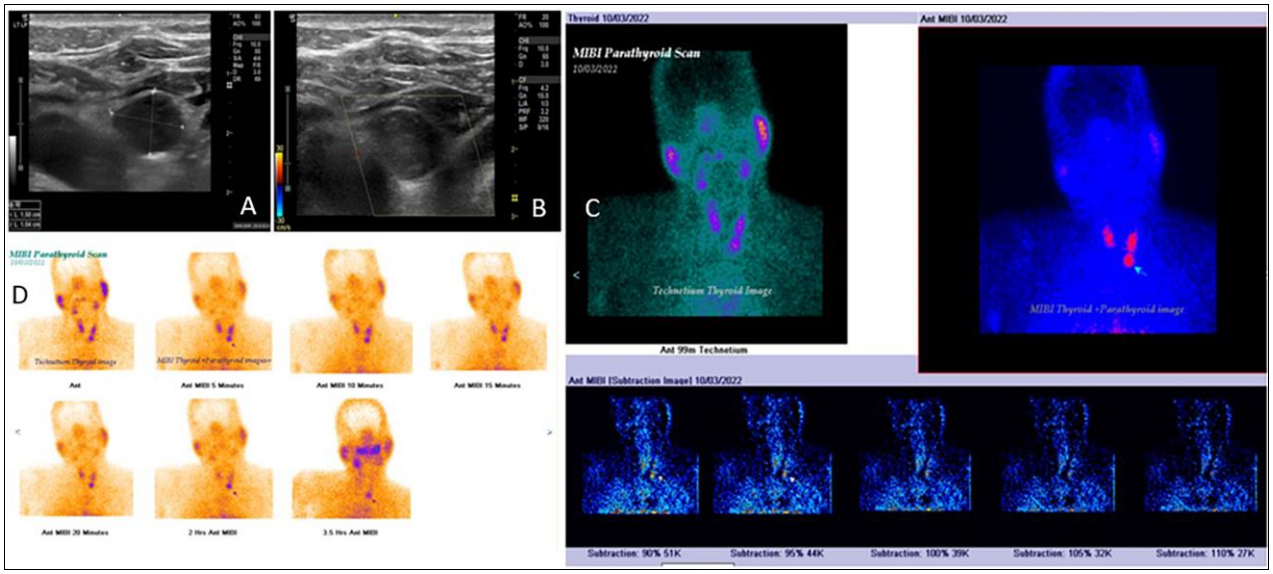

Fig 1: A, B: USG of the neck showed a well-defined solitary hypoechoic area inferior to the lower pole of the left lobe of the thyroid suggesting the possibility of parathyroid adenoma. C, D: SESTAMIBI scan shows, focal nodular tracer uptake inferior to the lower pole of the left lobe of the thyroid with retention of the tracer in the delayed images. Parathyroid adenoma involving the lower group of the left parathyroid gland was confirmed. No ectopic glands were detected

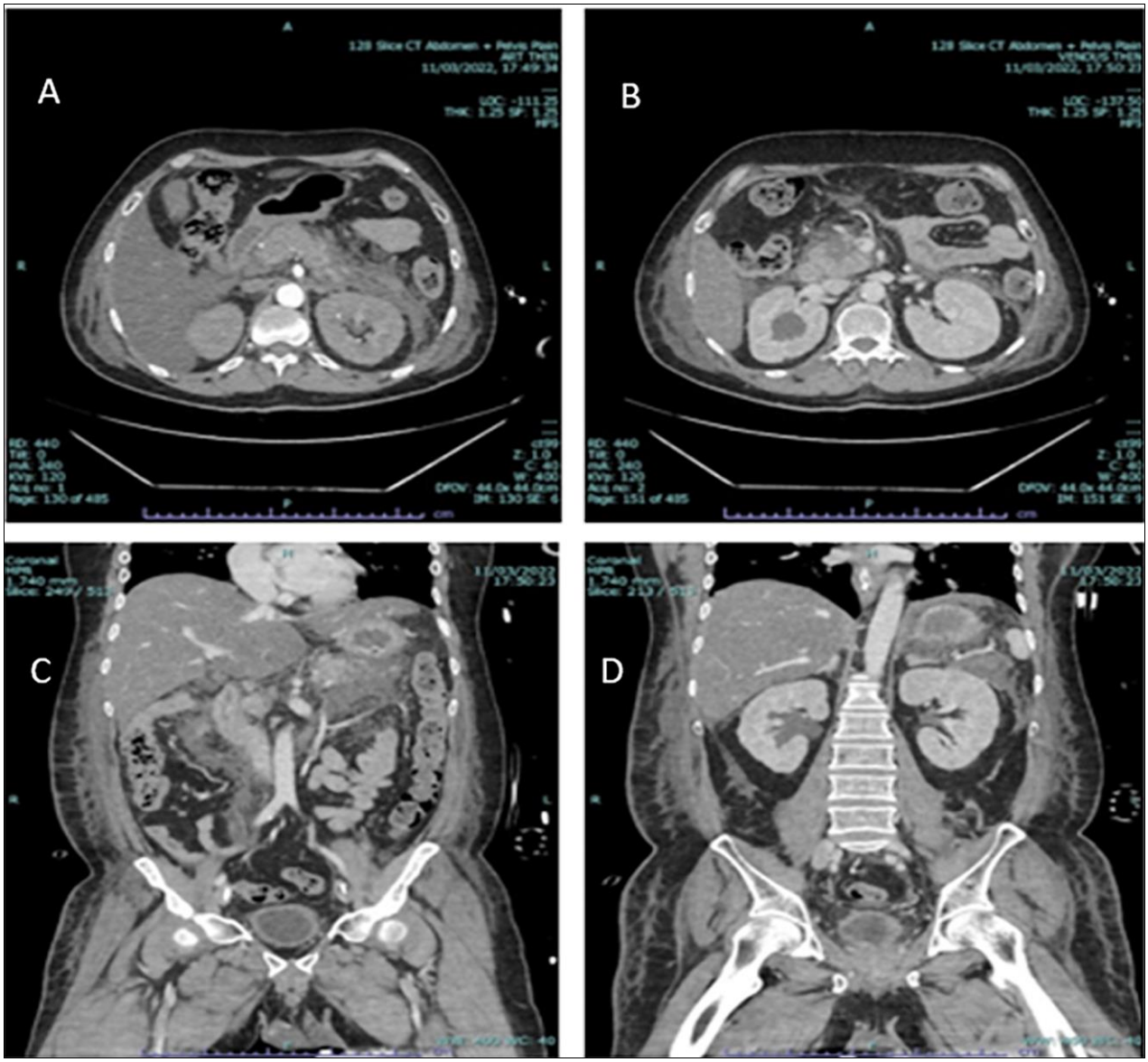

Fig 2: A, B: CECT axial and C, D (coronal) sections: Bulky and heterogenous pancreas with combined pancreatic, and peripancreatic necrosis. Right mild hydronephrosis is present

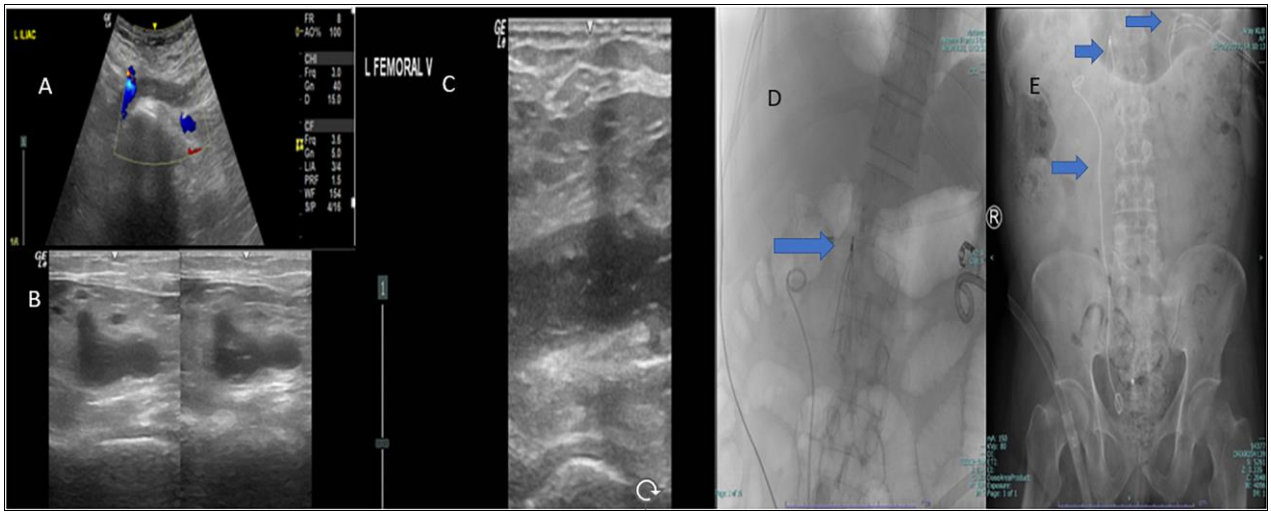

Fig 3: A, B, C: Venous Doppler showed distended iliofemoral veins with echogenic filling contents and absent Doppler flow suggestive of thrombosis. D: Denali IVC filter (arrow) was placed at infra-renal IVC by right trans jugular approach. E: Radiograph of KUB shows IVC filter, right DJ stent, and left pigtail drainage tube in situ (arrows pointing)

Discussion

Hyperparathyroidism is characterized by anorexia, nausea, vomiting, thirst, and polyuria, with associated muscular weakness, confusion, and drowsiness which may progress to lethargy and coma (1). Calcium homeostasis is maintained by the actions of three primary hormones- parathyroid hormone (PTH) 11, 25-dihydroxy vitaminD-3 (Vitamin D3), and calcitonin that regulate calcium transport in the gut, kidneys, and bone (2). Neuronal membrane excitability changes, hypertensive encephalopathy, and vasoconstriction induced by hypercalcemia have been hypothesized to be the aetiologies of seizures and posterior reversible encephalopathy syndrome (PRE S) (3). Calcium binds to tropomyosin and allows for the interaction of myosin and actin in the sarcomere, leading to muscle contraction. Vasospasm of cerebral vessels is the cause of vasogenic edema in the subcortical white matter presenting as PRES.

Primary hyperparathyroidism (PHPT) is rarely associated with the development of pancreatitis (5). Plausible mechanisms include calcium-phosphate deposition in the pancreatic ducts, calcium-dependent conversion of trypsinogen to trypsin, increased permeability of pancreatic duct due to hypercalcemia, and an apparent direct toxic effect of PTH on the pancreas (6). Microwave ablation (MWA), radiofrequency ablation, and laser ablation are advanced treatment techniques in patients who do not meet surgery criteria or decline surgical resection (7). Ablation is performed using the 'moving-shot' technique using Power 20–30 W for each microwave application (8). Skeletal involvement in hyperparathyroidism is a result of bone resorption (9). The commonly used non-invasive imaging techniques include sonography, scintigraphy, CT, and MRI. Multiple endocrine neoplasia (MEN) type 1, 2A,4, and hyperparathyroidism-jaw tumor syndrome are caused by known germline genetic mutations (10).

Teaching points

- The primary purpose of imaging in HPT is to accurately localize the diseased gland and identify patients who are suitable for minimally invasive surgery.
- Sonography and ^{99}mTc-sestamibi scintigraphy are the dominant imaging techniques for the preoperative localization of parathyroid adenomas.
- Contrast-enhanced CT (4D) and magnetic resonance imaging (MRI) can also effectively locate parathyroid adenomas.

MCQ

1. Among the following metabolic derangements, choose the right one suggestive of primary hyperparathyroidism

a) High PTH, Low Calcium
b) High PTHrP, Low PTH
c) High 1,25 dihydroxy vitamin D, High PTH
d) High ALP, High PTH

2. Which is the confirmatory imaging modality in the diagnosis of parathyroid adenoma.

a) Ultrasound
b) ^{99}mTc-sestamibi scintigraphy
c) 4D CT
d) MRI

3. If skeletal survey was performed for this case, which would have been the first radiograph taken and earliest finding

a) Pelvis, Whiskering of ischial tuberosities
b) Chest, Erosion of Lateral end of clavicles
c) Hand, Subperiosteal resorption along the radial aspect of proximal and middle phalanges of the 2nd and 3rd fingers
d) Skull, Salt and pepper sign**.**

Key: 1-a, 2-b, 3-c

Conclusion

Rare clinical manifestations of hypercalcemia can mislead the diagnosis. A failure in early diagnosis in cases of malignancy-related hypercalcemia can even result in loss of lives. Rare presentations like PRES, pancreatitis, and vague intraoral symptoms are challenging to diagnose early. Knowing the standard diagnostic algorithm, and appropriate medical and surgical interventions can save patients with atypical manifestations of hypercalcemia.

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Author's Contribution
Not available

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