venous end of the stent. In our case, the covered stent extended up to the hepatic vein ostia, thusthe junction of the covered and uncovered at the portal venous end was the vulnerable point.

TIPS stent fracture is very rare and the presence of TIPS stent angulation and use of overlapping covered and uncovered stents increases the risk of stent fracture with the most common site being the junction of covered and uncovered stents. The use of a single piece stent like the VIATORR stent-graft (Gore, Flagstaff, AR, USA) is thus ideal to decrease the risk of stent fracture rather than using two overlapping stents. Our case is unique compared to the other case reports because it represents the first case of TIPS stent fracture at the portal venous end of the stent with no displacement of the fractured fragment. Our patient was symptomatic and responded to stent revision with a covered stent graft across the fractured segment.

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Vitamin D Toxicity Causing Acute Pancreatitis with Acute Kidney Injury: A COVID-19 Era Mishap?

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Acute pancreatitis (AP) is an inflammatory condition of the pancreas defined by the revised Atlanta Criteria. The cause of pancreatitis can be easily identified in 75-85% of patients, most commonly being gall stone pancreatitis and alcohol induced pancreatitis. Hypercalcemia leading to pancreatitis is uncommon (~4%) and is seen more often with underlying hyperparathyroidism. It has been shown in experimental studies that increased calcium within the pancreatic acinar cells could accelerate conversion of trypsinogen to trypsin leading to acinar cell damage, auto digestion and subsequent pancreatitis.

Here, we present a case of severe AP, with acute kidney injury (AKI), induced by hypercalcemia due to Vitamin D toxicity.

Case Report

A 48 year old gentleman, with no comorbidities and no history of substance abuse, presented to the emergency room with non-radiating, severe periumblical abdominal pain that was associated with non-bilious vomiting since 5 days. He also complained of deep aching calf pains.

He was admitted with these complaints to his nearest health center, where, initial evaluation revealed hemoconcentration (Hemoglobin – 15.4 g/dl, raised haematocrit, Total leukocyte count – 11,500/mm^3, platelet count – 5,55,000/mm^3, significantly elevated serum

amylase (1651U/L[25-115U/L]) and lipase (2667U/L [73-393U/L]), with deranged creatinine (4.4 mg/dl) (however, with no obvious history of decreased urine output) and normal liver function tests. A diagnosis of acute pancreatitis (AP) was made and patient was treated with analgesics, hydration and bowel rest.

On admission to our hospital, after 3 days of onset of symptoms, further evaluation for the etiology of AP revealed elevated triglycerides (304 mg/dl), severe hypercalcemia (corrected calcium - 14.5 mg/dl) and hyperphosphatemia (5.1 mg/dl). We followed this up with estimation of serum PTH, which was low (2.5 pg/ml), and 25-hydroxy-Vitamin D, which was was >100 ng/ml, consistent with vitamin D toxicity.

Our patient had history of COVID 19 pneumonia 1.5 months prior to onset of this episode of AP and was on home isolation and oral medication for the same.

On taking careful drug history, the patient had apparently self-medicated himself with 60,000 units of Vitamin D3 capsules (available as 10,000 and 60,000 units in India), 6 capsules a day for around 20 days during COVID 19 illness (in view of a then low serum vitamin D3 level of 22 ng/ml).

To rule out presence of gall stones, transabdominal ultrasonography was performed which showed a well-distended gall bladder with sludge without any acoustic shadowing, and normal gall bladder wall thickness. The common bile duct (CBD) appeared normal. The echotexture of head and body of pancreas appeared altered with ill-defined peripancreatic collections. Since there was no evidence of gall stones, gall bladder wall and CBD appeared normal, and initial liver functions tests were normal, we opted not to get an endoscopic ultrasonography.

He was treated with aggressive hydration with isotonic saline at a rate of 200 to 300 ml/hour to maintain his urine output at 100 to 150 mL/hour - regardless his serum calcium level continued to be high, so salmon calcitonin 4 IU/kg was adminstered every 12th hourly. Although hypocalcemic response was noted with salmon calcitonin therapy over a period of 48 hours, tachyphylaxis developed, which compelled us to use Denosumab 60 mg (**Figure 1**) subcutaneously. Bisphosphonates were avoided due to severe renal impairment. Serum calcium levels normalised gradually over period of a few days (**Figure 2**).

Gradually, his pain subsided and he was started on oral soft diet. Meanwhile, his serum creatinine levels started to normalise.

A contrast CT imaging done after the AKI recovered, showed local complications in the form of an ill-defined acute necrotic collection (ANC), extending from the head, distal body and tail of pancreas into left

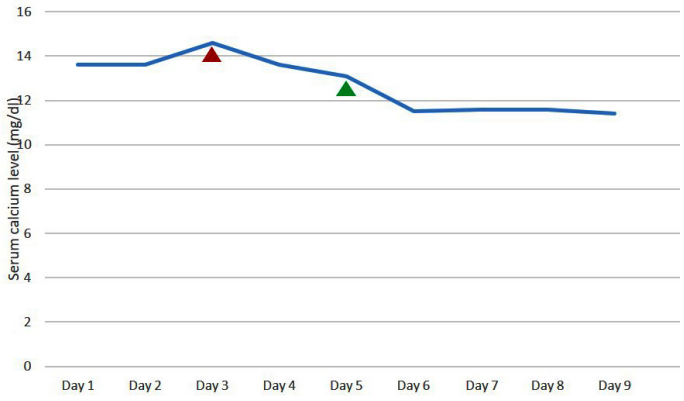

Figure 1: Progressive levels of sereum calcium. The arrow heads depict the calcium levels during therapy at which Calcitonin (red arrow head) and denosumab (green arrow head) were added.

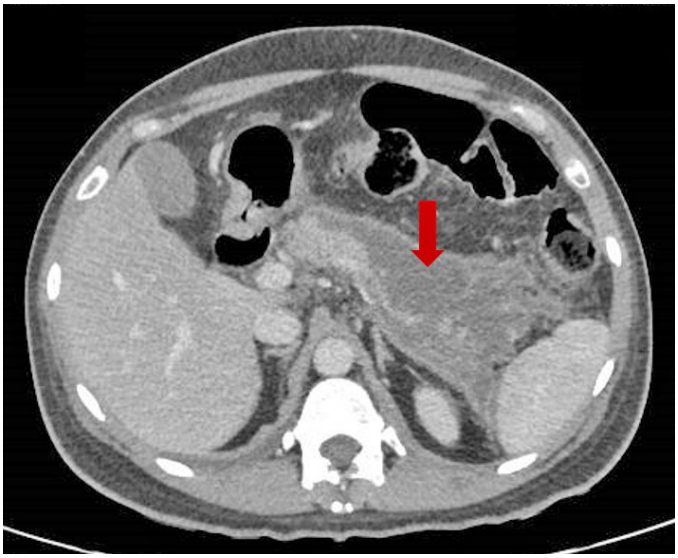

Figure 2: The arrow points to the acute necrotic extending from the head, distal body and tail of pancreas into left anterior pararenal space.

and right anterior pararenal space, measuring around 5.2 x 11.3 x 4.7 cm.

Thereafter, he was planned for conservative management with respect to the ANC and he was discharged after complete symptomatic relief and normalisation of serum creatinine and calcium. He was advised for close follow-up.

Discussion

Vitamin D plays a pivotal role in calcium homeostasis and bone health. Its major role is in intestinal absorption of calcium and phosphorous and formation of bone mineral matrix. Even though reference values in different laboratories may vary, usually serum 25 (OH) D levels less than 30 nmol/L is considered to be deficient and levels of 50 nmol/L or more are sufficient for most people[1]. 30-50 nmol/l is considered as insufficiency. Levels of greater than 125 nmol/L are potentially associated with toxic effects[1]. The recommended dietary allowance (RDA) for Vitamin D in adults is 400-1000IU/day[2].

Vitamin D also plays an immunomodulatory role in the human body. Amidst the current COVID 19 pandemic, it has found a place in various treatment protocols across the world. Mahdavi *et al*.[3] reported that vitamin D is a negative renin-angiotensin system (RAS) modulator and has a potential protective role against acute lung injury/ARDS. A randomized, placebo controlled study by Rastogi *et al*[4] showed significant fibrinogen decrease and SARS-CoV-2 RNA negativity with high-dose cholecalciferol supplementation. However, more studies are warranted to evaluate its actual benefit. Supplementation has been widely done in documented deficiency, but self-medication, and overdosing as seen in this case can lead to unprecedented complications – emphasising the need for monitoring Vitamin D levels while on therapy.

The recommended pharmacological dose is oral administration of 50,000 units weekly for 6-8 weeks followed by maintenance dose of 800 IU/day from food and supplements once plasma levels are attained. Toxicity is rare and can occur if intake is > 4000 IU/day[2]. Toxicity is usually manifest with symptoms due to hypercalcemia and can present with neuropsychiatric and neuromuscular manifestations such as difficulty in concentration, confusion, muscle cramps, muscle twitching, apathy, drowsiness, depression, psychosis and in extreme cases, a stupor and coma; gastrointestinal symptoms such as recurrent vomiting, abdominal pain, polydipsia, anorexia, constipation, peptic ulcers, and pancreatitis; cardiovascular manifestations of such as hypertension, shortened QT interval, ST segment elevation, and bradyarrhythmia with first-degree heart block on the electrocardiogram; renal symptoms due to hypercalciuria leading to polyuria, polydipsia, dehydration, nephrocalcinosis, and renal failure[5]. In our case, patient developed severe acute pancreatitis and AKI. He had no neurological or cardiac complications. A similar case of Vitamin D toxicity leading to acute pancreatitis and AKI was reported by Singh *et al*[2].

To conclude, vitamin D over dosage must be considered in the setting of pancreatitis presenting in the background of hypercalcemia and PTH suppression with high 25-hydroxy-Vitamin D levels. This is especially so in endemic areas, and given the COVID 19 scenario and widespread practise of self-treatment. Our case is one such example of a much avoidable severe complication.

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