

Calcification at the Level of 12th Dorsal and 1st Lumbar Vertebrae

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Received: 03 January 2025; Accepted: 17 February 2025

A 46-year-old man presented with pain in his left lower limb for the past 2 months. He was taking herbal medications for diabetes, which he had for the past 3 years. He had a history of alcohol dependence for the last 12 years, but recently he was able to overcome his addiction with the help of counseling. He had no history of recurrent abdominal pain and had no complaints of steatorrhea. Clinical examination revealed his body mass index (BMI) was 18 kg/m², pulse 110 beats per minute, blood pressure 124/78 mm Hg, and absence of icterus. The blood tests showed hemoglobin 11.5

gm/dL, blood sugar (fasting) 440 mg/dL (reference range 60–110), and glycated hemoglobin (HbA1c) 9.5% (good control <6.5), serum calcium 8.9 mg/dL (reference value 8.5–10.5) and vitamin D₃ 16 ng/mL (reference range 20–50), serum creatinine 1.2 mg/dL (reference value 0.7–1.4), alanine transaminase 35 U/L (reference range 5–45). For pain in the lower limb, a radiograph of the lumbosacral vertebral spine (Fig. 1) was taken; however, numerous dense calcifications were noted across the 12th dorsal and 1st lumbar vertebrae, more so on the right side. Unenhanced abdominal computed tomography (CT) scans (Figs 2A and B) were obtained. The patient received an oral dosage of 500 mg of metformin twice a day, along with 10 units of human regular insulin delivered *via* subcutaneous injection in the morning and 8 units in the evening. His glucose levels were monitored at regular intervals. In a week's time, the patient's fasting blood glucose level decreased to 110 mg/dL. He was also prescribed calcium supplementation alongside weekly oral administration of vitamin D₃ (60,000 IU). Additionally, oral pregabalin at a dosage of 75 mg was administered twice a day.

Calcifications across dorsolumbar vertebrae (Fig. 1) identified on radiograph may indicate a variety of conditions, such as pancreatic calcification, splenic artery calcification, cholelithiasis in the common

bile duct, and contrast retention within a duodenal diverticulum. The calcification of splenic artery atherosclerosis and pancreatitis resemble each other closely; nevertheless, splenic artery calcification has a characteristic tram-track appearance.¹ Abdominal CT scans (Figs 2A and B) resolved the ambiguities, showing diffuse parenchymal calcifications within the pancreas; they were profusely present in the head of the pancreas, while they were sparse in the body and the tail, indicating chronic pancreatitis. In this particular case, the predominant etiology of chronic pancreatitis is chronic alcohol consumption. The patient's low BMI, vitamin D deficiency and borderline serum calcium levels indicate possible nutritional deficiency. Intense pain in the lower limb could be a sign of diabetic neuropathy, which may worsen with uncontrolled blood sugar levels. A symptom indicating exocrine insufficiency, steatorrhea, may not be detected until >90% of the pancreatic parenchyma is lost.² Although abdominal pain is a predominant symptom of chronic pancreatitis, a subset of patients, approximately 10%, may experience a painless course.³ Endocrine insufficiency, brought on by a loss of insulin and islet cell mass, manifests as diabetes called pancreatic diabetes or type 3c diabetes. This kind of diabetes differs from type 1 and type 2 diabetes as there is loss of counter-regulatory hormones, besides pancreatic exocrine insufficiency. Metformin alone will suffice in management of type 3c diabetes, when HbA1c is <8.0%, or else insulin has to be added to metformin with frequent checks for blood due to significant fluctuations in blood glucose levels. In individuals with

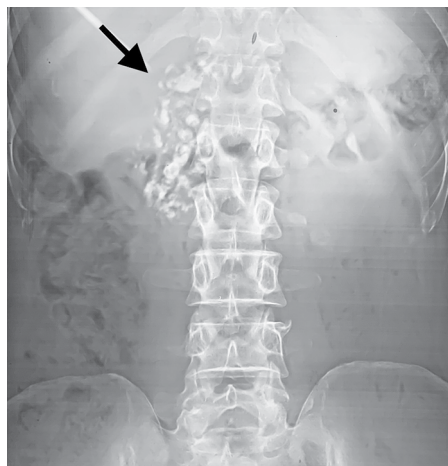
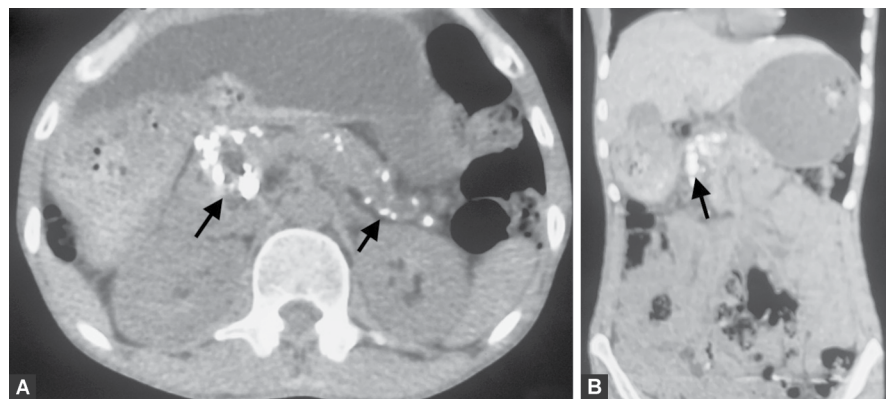


Fig. 1: An abdominal radiograph reveals calcifications across D12 and L1 vertebrae (black arrow)



Figs 2A and B: Black arrows show numerous pancreatic calcifications over the head and a few in the body and tail in axial and coronal views of a noncontrast CT scan, respectively

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How to cite this article: Singh RK. Calcification at the Level of 12th Dorsal and 1st Lumbar Vertebrae. J Assoc Physicians India 2025;73(9):101–102.

chronic alcoholic pancreatitis, the lifetime prevalence of diabetes mellitus is 80% and the corresponding insulin demand is 50%.⁴ Despite the fact that Ewald and Bretzel⁵ developed diagnostic guidelines for type 3c diabetes mellitus, this is not widely recognized.

Recognition of type 3c diabetes is crucial for tailoring the treatment approach, as there is a decline in pancreatic function (both endocrine and exocrine) and the loss of counter-regulatory

hormones, leading to hyperglycemia with wide swings in blood glucose levels.

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