

## Pancreatic Panniculitis Sans Pancreatitis in a Patient with Diabetic Ketoacidosis

### Dear Editor,

A 23-year-old Malay woman with no past medical illness was admitted for a 4-day history of epigastric pain, vomiting and drowsiness. Investigations showed a high anion-gap metabolic acidosis with elevated serum ketones and hyperglycaemia, consistent with diabetic ketoacidosis (DKA). Anti-glutamic acid decarboxylase autoantibodies were positive, consistent with type 1 diabetes mellitus.

In the first week of admission, the patient had hyperamylasaemia (670 units/L, upper limit 100 units/L) and hyperlipasaemia (>400 units/L, upper limit 40 units/L) accompanying her epigastric pain, fulfilling the diagnosis of acute pancreatitis.<sup>1</sup> The gastroenterology consultant who reviewed the patient, however, requested for contrast-enhanced computed tomography (CT) scan of the abdomen to confirm the diagnosis as he felt the patient's DKA could have accounted for the elevated serum amylase and lipase levels, with similar epigastric pain. Two contrast-enhanced CT scans performed 6 days apart were negative for acute pancreatitis. The patient's hyperamylasaemia and hyperlipasaemia, together with epigastric tenderness, was attributed to DKA by the reviewing gastroenterologist.

Two weeks into admission, dermatology consult was sought for a 4-day history of painful lesions over the patient's legs, associated with bilateral ankles arthralgia. On examination, multiple discrete tender erythematous nodules were present over her shins and dorsal feet (Figs. 1a and 1b). Septic workup was unremarkable. Anti-streptolysin O titre was <200 IU/mL. Serum alpha-1 antitrypsin level was not performed. Clinical differentials considered included pancreatic panniculitis, erythema nodosum, erythema induratum, other causes of panniculitis, and cutaneous polyarteritis nodosa. A 6 mm punch biopsy was performed over one of the patient's right leg nodules. Histology showed lobular panniculitis with fat necrosis and neutrophils. Bluish saponified fat, basophilic deposits of calcium and ghost cells were present. No vasculitis was seen (Fig. 2). This was consistent with pancreatic panniculitis. No tissue was sent for direct immunofluorescence.

The patient was treated with indomethacin 25 mg thrice a day for symptomatic relief of her painful leg nodules. On outpatient review 2 weeks later, the patient was well. Nodules over her legs were less painful with flattening

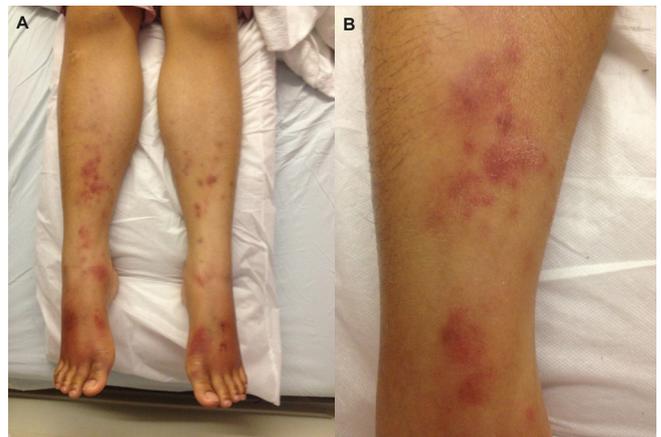


Fig. 1. A) Multiple erythematous nodules are seen over the patient's shins and dorsal feet. B) A close-up view of the erythematous nodules over the patient's right shin.

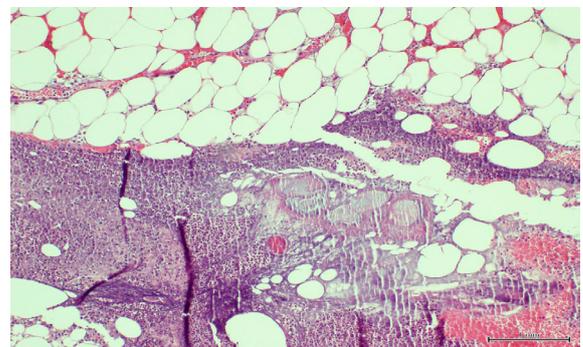


Fig. 2. Lobular panniculitis with fat necrosis and neutrophils. Bluish saponified fat, basophilic deposits of calcium and ghost cells seen. No vasculitis is seen (haematoxylin and eosin, original magnification x 200).

and post-inflammatory hyperpigmentation changes were seen.

The diagnosis of acute pancreatitis requires 2 of the following 3 features: 1) abdominal pain consistent with acute pancreatitis (acute onset of a persistent, severe, epigastric pain often radiating to back); 2) serum lipase or amylase activity at least 3 times greater than the upper limit of normal; and 3) characteristic findings of acute pancreatitis on contrast-enhanced CT and less commonly magnetic resonance imaging (MRI) or transabdominal ultrasonography.<sup>1</sup>

Pancreatic panniculitis is rare, affecting 0.3% to 3% of all patients with pancreatic disease, ranging from pancreatitis to pancreatic carcinoma.<sup>2</sup> It tends to affect the distal lower limbs, presenting with tender erythematous subcutaneous nodules that may spontaneously ulcerate with thick oily discharge.<sup>2</sup> Other possible sites of involvement include breasts, buttocks, thighs, and abdomen. Pancreatic lipase and amylase have been postulated to cause subcutaneous fat necrosis via enzymatic destruction of fat, supported by findings of these enzymes in lesional biopsies.<sup>3</sup>

Hyperamylasaemia and hyperlipasaemia are known to occur with DKA (16% to 25% of cases).<sup>3</sup> Three times the upper limit of normal for these enzymes is thought to be suggestive of pancreatic involvement since acute pancreatitis can precipitate or coexist with DKA.<sup>4</sup> However, hyperamylasemia and hyperlipasemia have been reported without pancreatitis findings on clinical examination or CT scan (gold standard for confirmation).<sup>4,5</sup> The source of these elevated enzymes in DKA patients without acute pancreatitis remains uncertain. Possibilities include subtle injury to pancreatic acinar cells, dysmetabolic state with release of salivary gland amylase or its accumulation from suboptimal urinary excretion, and release of lipolytic enzymes from other non-pancreatic sources (e.g. tongue, oesophagus, stomach, small bowel, liver).<sup>4,5</sup>

Although our patient presented with epigastric pain (not uncommon with DKA) with elevated serum amylase and lipase levels, contrast-enhanced CT scans of the abdomen did not reveal any pancreatitis. Pancreatic panniculitis lesions can precede the development of pancreatic disease in 40% of cases by 1 to 7 months.<sup>2</sup> However, in our patient, we postulate that her lesions were likely secondary to hyperlipasaemia associated with DKA and not due to underlying pancreatic disease per se. Patient's epigastric tenderness resolved shortly after her second CT scan. Her serum lipase level, which was persistently >400 units/L, only downtrended 1 week after resolution of her epigastric tenderness. Ten months on, there was no development of pancreatitis or other pancreatic disease clinically.

We report, to the best of our knowledge, the first case of pancreatic panniculitis sans pancreatitis in a newly diagnosed type 1 diabetes mellitus patient presenting with diabetic ketoacidosis.

#### REFERENCES

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