KANSAS JOURNAL of MEDICINE

Acute Pancreatitis Secondary to Hypertriglyceridemia Presenting as Sepsis Without Abdominal Pain

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Received March 5, 2023; Accepted for publication April 25, 2023; Published online June 20, 2023
https://doi.org/10.17161/kjm.vol16.19634

INTRODUCTION

Acute pancreatitis is a common emergency room diagnosis that often can lead to inpatient admission. In the U.S., acute pancreatitis accounts for 7% of yearly emergency department (ED) visits and 200,000 hospital admissions annually with an average hospital stay of 6.1 days.[1] While it often can be seen as a benign disease, it boasts a mortality rate of 10-30%.

Diagnosis is made by establishing two of three main criteria that are composed of both subjective and objective findings: epigastric abdominal pain, elevated serum amylase/lipase greater than three times the upper limit of normal, and imaging consistent with the diagnosis.[1] It is possible to see lipase elevation in the setting of acute renal failure, small bowel obstruction/perforation, acute cholecystitis, and diabetic ketoacidosis; however, the diagnosis of acute pancreatitis is considered most often in the context of acute abdominal pain and estimated to be present in up to 90% of cases.

The following case is an example of acute pancreatitis secondary to hypertriglyceridemia without abdominal pain in a patient presenting with sepsis who was subsequently found to be in diabetic ketoacidosis.

CASE REPORT

A 32-year-old male patient with a past medical history of newly diagnosed diabetes mellitus, recently diagnosed hyperlipidemia, hypertension, mood disorders, and methamphetamine use in remission presented to the ED with palpitations and constant right-sided chest wall pain for two days. He denied any abdominal pain and his physical exam showed no abdominal tenderness, distension, guarding, or rigidity. The patient did not appear to have any triggering medications that could have precipitated the event.

Initial cardiac workup showed an electrocardiogram with normal sinus rhythm, troponin < 0.01 ng/ml, and an unremarkable chest x-ray. The patient was tachycardic and tachypneic with a white blood cell count of 16.4 x 103/uL and normal lactic acid. Additional labs obtained in the ED showed blood glucose 356 mg/dl, anion gap 24 mEq/L, bicarbonate 7 mEq/L, pH 7.17, 2+ ketones on urinalysis, and hemoglobin A1c greater than 17.6%. His calculated body mass index was 23.5 kg/m^2.

The patient was diagnosed with diabetic ketoacidosis (DKA) and was started on DKA protocol with insulin drip and intravenous fluids. A lipid panel was obtained due to a history of hyperlipidemia and showed triglycerides 2172 mg/dL, cholesterol 351 mg/dL, and HDL 14 mg/dL. Subsequently, a lipase test was found to be elevated at 472 U/L. Computed tomography (CT) without contrast of the abdomen revealed diffuse peri-pancreatic edema compatible with acute pancreatitis without a well-defined fluid collection. On nil-per-os status and an insulin drip, triglycerides decreased to < 500 mg/dL over the course of two days at which point the insulin was discontinued. The patient was started on fenofibrate and a statin prior to discharge.

DISCUSSION

Acute pancreatitis is a disease where prompt diagnosis and treatment are essential to avoid poor outcomes. While the quality and radiation of abdominal pain can vary, it is important to note how frequently this physical exam finding is used as a basis for diagnosis. This means that a lack of abdominal pain inadvertently may preclude some physicians from considering further acute pancreatitis workup with labs and imaging. When considering a diagnosis of acute pancreatitis, it is important to evaluate the entire clinical picture rather than anchoring to the presence of abdominal pain. Although there is a prevalence of abdominal pain at presentation, the level of pain poorly correlates with disease severity or specific etiology of pancreatitis due to the subjective nature of pain perception.[1]

Nair et al.[2] showed that DKA may mask coexisting acute pancreatitis occurring in at least 10-15% of cases. Based on that study, the severity index based on CT findings has been shown to better correlate with outcome. Out of the eleven patients included in the study, two did not have abdominal pain on admission, like our case. Additionally, an estimated 10% of patients presenting with chronic pancreatitis reported no abdominal pain.[3] Pancreatic nociception primarily is mediated by chemo-sensitive mechanisms stimulated by inflammatory mediators and mechanosensitive mechanisms on parenchymal blood vessels that are stimulated by ischemia, stretching, and necrosis.[4] This cascade of pain stimulus begins with the release of pro-inflammatory mediators and continues down primary, secondary, and tertiary sensory neurons in the central nervous system. Eventually, neurotransmitters are released at both the dorsal horn of the spinal cord and primary nerve endings on the pancreas where they act as inflammatory mediators.

Poorly or uncontrolled diabetes can have unfortunate consequences that affect multiple organ systems.[4-5] This commonly manifests in the gastrointestinal system as gastroparesis, esophageal dysmotility, and intestinal dysmotility. The mechanism of autonomic dysregulation and visceral neuropathy in a diabetic is multifactorial; however, neuronal damage and a decrease in sensory neuropeptides are thought to contribute heavily to decreased organ nociception. It has been proposed that insulin lowers the serum triglycerides (TG) levels by increasing the enzymatic activity of lipoprotein lipase which metabolizes chylomicrons and VLDLs into the free fatty acids and glycerol ultimately decreasing the serum TG levels.

In our patient, possible explanations for his lack of abdominal pain in the acute setting includes poorly controlled diabetes causing pancreatic nociceptive dysfunction that masked acute pancreatitis or undiagnosed painless chronic pancreatitis masked by diabetic nociceptive dysfunction that was potentiated by chronic inflammatory neuronal destruction. In the setting of acute pancreatitis, indirect laboratory findings combined with pertinent medical history are important when evaluating a patient who is presenting with atypical physical exam findings such

as a lack of abdominal pain. Unfortunately, there is a paucity of literature highlighting the importance of seeing beyond abdominal pain as an initial indicator of acute pancreatitis.

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Keywords: abdominal pain, diabetes mellitus, hypertriglyceridemia, insulin, pancreatitis