CLINICAL VIGNETTE

Drug-Induced Pancreatitis

Nisha Viswanathan, MD

Case Presentation

A 52-year old female presented to the hospital with new onset of sharp abdominal pain. Her symptoms began two days prior to admission with mild abdominal "aching" that gradually worsened to a sharper pain over the next two days. The pain was worst in her left upper quadrant and radiated slightly to her mid-epigastric region and was associated with nausea, vomiting and decreased appetite. She denied any aggravating or alleviating factors and reported no improvement with over-the-counter pain medications. She denied constipation, diarrhea, recent weight loss or night sweats. She also was free of chest pain, dyspnea or urinary symptoms. Two weeks ago, she started citalopram 20 mg daily for depression and also takes levothyroxine chronically. There was no recent alcohol use, abdominal procedures, trauma or scorpion stings. She is current in vaccinations, including childhood MMR.

The patient had no other significant past medical history except hypothyroidism and depression. She had prior cholecystectomy years before and no other surgical history. She denied alcohol, tobacco or drug use and any other new medications. Family history was negative for gastrointestinal diseases, malignancies, or autoimmune disorders.

On admission, the patient was diaphoretic and uncomfortable. She was afebrile and tachycardic to 100 with normal BP, RR and oxygen saturation. Abdominal exam was remarkable for increased tenderness to palpation in the left upper quadrant with some guarding, but no rebound tenderness. Other than dry mucous membranes, the remainder of her exam was unremarkable.

Labs on admission included elevated WBCs to 12.5 with neutrophilic predominance, mildly elevated AST and ALT of 34 and 30. Lipase was markedly elevated at 3000 with amylase elevated to 400. Triglycerides and calcium were within normal limits. ANA returned negative as well as no detectable Ethanol. CT Abdomen revealed pancreatic inflammation and stranding without pseudocysts or masses.

The patient was treated with fluids, pain control, and maintained on NPO status for two days. Citalopram was held on admission. Her symptoms resolved and she was discharged home after three days in the hospital without abdominal pain. Because of the timing, Citalopram was discontinued on discharge.

Discussion

Drug-induced pancreatitis (DIP) is considered a rare cause of pancreatitis with reports of around 0.1 to 2% in the literature.[1] Case reports of drug-induced pancreatitis are common and it is considered a diagnosis of exclusion. The majority of cases are mild which may contribute to underreporting. It is less common than other causes of pancreatitis: biliary, alcoholic, and hypertriglyceridemia.[2] The prevalence is equal across all age groups with similar rates in men and women.[3]

The mechanism of pancreatic injury in DIP is not well understood and various mechanisms have been proposed. Most postulate that different drugs have varying mechanisms of injury including direct pancreatic duct constriction, immunologic reactions, as well as accumulation of toxic metabolites.[4] In addition, some propose that certain drugs such as statins and oral contraceptives could lead to pancreatic necrosis from hypercoagulability.[5]

To establish diagnosis of true DIP versus an idiopathic or other etiology requires four criteria.[1] First, a clear diagnosis of pancreatitis. Second, clear exclusion of the most common causes of pancreatitis. Third an adequate time interval for symptoms to develop after drug initiation and improvement with drug withdrawal. This depends on drug half-life, as drugs such as valproic acid, pentamidine, and didanosine may not cause pancreatitis for months after initiation. Lastly, recurrence of DIP with reinitiation of the drug, which was not established.

Studies report scales to rank medications on their likelihood of causing drug-induced pancreatitis. In 2007, a four-point classification system was published, based on data from case reports on drugs associated with pancreatitis.[3] The I-IV classifications, categorized drugs with the greatest potential of causing DIP (Class I) versus those lower potential of causing DIP (Class IV). Class IV contained sertraline, a selective-serotonin release inhibitor in the same class as citalopram.

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