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CLINICAL VIGNETTE

Influenza A as a cause of acute pancreatitis: A Case Report

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Our patient was a 55-year-old female in good health with a history of intermittent gastroesophageal reflux disease. She had no history of diabetes or hyperlipidemia. She was not taking any routine medication; however, she would occasionally use omeprazole or ranitidine as needed for her GERD. She had no known allergies and no prior surgeries. She did not smoke or drink alcohol. Her family history was positive for diabetes in her father, but no family history of hyperlipidemia, pancreatitis, or gallbladder disease. She had opted out of routine adult immunizations.

She was feeling well until she developed fever, cough, body aches, epigastric abdominal pain, nausea, and vomiting. This occurred during the winter, and she was felt likely to have influenza. Treatment was initiated with oseltamivir phosphate 75 mg twice a day for 5 days, and the laboratory test of her nasal specimen was positive for influenza A. Her fever and cough improved; however, her gastrointestinal symptoms continued and worsened.

She presented to the emergency room with nausea, vomiting, loose stools, and severe upper abdominal pain. She was unable to tolerate any oral intake and was admitted to the hospital. Her temperature was 99.3 degrees F, blood pressure 120/75, heart rate 70, and respirations 18/min. Her head and neck exam was unremarkable and lungs were clear bilaterally. Heart revealed a regular rhythm – no murmurs, rubs, or gallops. There was moderate tenderness in the epigastrium but no rebound or rigidity. Her initial lipase measurement was 110 U/L (<63 UL), which subsequently rose to 653. The amylase, electrolytes, calcium, creatinine, and liver function tests were normal. Glucose was 123 mg/dL (70-99 mg/dL), white blood cell count was 3,700/uL (range 4,000-11,000/uL), and hemoglobin was 13.2 g/dL (11.6-15.4 g/dL), unchanged from baseline. Urine culture, stool testing for the Helicobacter pylori antigen, and Clostirium difficile PCR were negative. Her chest x-ray and abdominal ultrasound were normal with normal gallbladder and no gallstones. CT of her abdomen and pelvis had no significant findings and no evidence of pancreatitis with a normal gallbladder, no pancreatic edema, or pseudocyst.

The patient was treated conservatively with intravenous fluids, tramadol, ondansetron, and pantoprazole, and her symptoms improved. Her diet was gradually advanced, and she was discharged 6 days later on oral medication. Her nausea and vomiting resolved, and the abdominal pain had significantly lessened. Her lipase level dropped to 152.

There are about 210,000 hospital admissions for acute pancreatitis every year in the United States.[1] The two major causes of acute pancreatitis are alcohol use and gallstone disease, which account for over 75% of cases. Other causes of pancreatitis include hypercalcemia, hypertriglyceridemia, traumatic injury, complication of medical procedures, pregnancy, or are idiopathic.[2]

Acute pancreatitis can also be due to infectious agents. Bacteria, fungi, and parasites have all been reported as causes of acute pancreatitis. Viral etiologies are responsible for <1% of cases. Viral causes include HIV, mumps, Hepatitis E, Hepatitis A, coxsackie virus, cytomegalo virus, varicella zoster, herpes simplex, and influenza A.[2-5]

The diagnosis of acute pancreatitis must include 2 out of 3 of the following criteria: abdominal pain, elevation of serum amylase and/or lipase to 3 times the upper limit of the normal range, and an imaging study, usually CT or MRI, which reveals signs of pancreatitis.[2,4-6] The abdominal pain occurs mainly in the epigastrium, radiates to the back in 50% of cases, and may be associated with nausea and vomiting. The lipase level may remain elevated longer than the amylase. The severity of pancreatitis does not correlate with the height of amylase or lipase elevation.[1,7]

Acute pancreatitis is classified as mild or severe. "Mild acute pancreatitis was defined as pancreatitis associated with minimal organ dysfunction and an uneventful recovery. Severe pancreatitis was defined as pancreatitis associated with organ failure and/or local complications (necrosis, abscess, or pseudocyst)."[7] Approximately 80% of cases are considered to be mild. The mortality rate of acute mild pancreatitis is low, <1%. Treatment of mild acute pancreatitis is mainly supportive care with IV hydration, antiemetics, and analgesia. Oral intake is usually possible within a few days.[1,7]

The risk of recurrent acute pancreatitis is increased by continued alcohol use and untreated biliary tract disease. Cholecystectomy for gallstone disease is recommended when the pancreatitis has resolved and the patient is stable.[8]

The cases reported of flu-related acute pancreatitis have been from influenza A. One case was initially attributed to influenza B, but this turned out to be a lab error.[2] Some cases of subclinical flu related acute pancreatitis may occur; however, there are no data.[4] We did not find reports of recurrent acute pancreatitis related to influenza.

Conclusion

Acute pancreatitis may be caused by influenza A. Influenza symptoms include fever, cough, myalgias, headache, and gastrointestinal symptoms.[3] During the winter months, for a patient presenting with suggestive symptoms and epigastric pain radiating through to the back, acute influenza related pancreatitis should be considered. For this diagnosis, the patient must meet 2 out of 3 of these criteria: abdominal pain; elevation of serum amylase and/or lipase 3 times normal; and an imaging study, usually CT or MRI, which shows signs of pancreatitis. Patients may require hospitalization. For mild acute pancreatitis, treatment is mainly supportive care, and hospital stays are usually only a few days.

Our patient presented in wintertime with characteristic influenza symptoms and abdominal pain. She had not received the flu vaccine earlier in the season. She met 2 of the 3 criteria required for the diagnosis of acute pancreatitis. She had mild acute pancreatitis because there was no evidence of organ failure. Her white blood cell count was low, suggestive of a viral syndrome. Her nasal specimen was positive for influenza A. She improved with supportive care in the hospital and went home after 6 days. She has not had further episodes of pancreatitis.

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