Cholesterolosis as a cause of acute pancreatitis

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ABSTRACT

Acute pancreatitis is an inflammatory condition of the pancreas manifesting with abdominal pain and elevated serum levels of pancreatic enzymes. Gallstones and chronic alcohol use are the most commonly described causes. A less studied cause is cholesterolosis, gallbladder polyps that cause mechanical obstruction of the sphincter of Oddi. Here, we present the case of a 55-year-old woman who presented with acute pancreatitis and was found to have cholesterol polyps in her gallbladder with no evidence of gallstones. The patient underwent cholecystectomy with complete resolution of her symptoms.

KEYWORDS Acute pancreatitis; biliary sludge; cholesterolosis; gallstone pancreatitis

The two most common causes of acute pancreatitis, accounting for more than two-thirds of cases, are gallstones and chronic alcohol use.¹ A less studied etiological factor is acute pancreatitis due to gallbladder polyps. These are outgrowths of the gallbladder mucosal wall and are often detected incidentally. Gallbladder polyps can either be benign, such as hyperplasia and lipid deposits (cholesterolosis), or malignant, which is rare.² These polyps can slough off from the mucous membranes and result in mechanical obstruction at the sphincter of Oddi. We report a case of acute pancreatitis that we believe was due to cholesterolosis.

CASE STUDY

A 55-year-old woman with a history of idiopathic thrombocytopenic purpura for 3 years was admitted for acute pancreatitis. While vacationing in France, she developed a sharp, severe midepigastria pain. She took pantoprazole for 36 hours without improvement. She then presented to a hospital in Paris where she was diagnosed with pancreatitis given a lipase of 4000 U/L, elevated aminotransferases, and hyperbilirubinemia. A computed tomography (CT) scan of her abdomen showed evidence of gallbladder polyps but no necrosis or gallstones. After receiving intravenous fluids, she returned to the United States and presented to our hospital.

She endorsed about one drink of alcohol every 3 months and had one glass of wine daily for 3 days before her attack. She had a similar episode of midepigastria pain a year earlier

but was diagnosed with gastritis given a normal lipase and an unremarkable CT scan.

Her laboratory values on transfer to our hospital were notable for a lipase of 161 U/L, alanine transaminase of 335 U/L, aspartate transaminase of 214 U/L, and total bilirubin of 1.7 mg/dL. No tests were ordered for autoimmune pancreatitis given our low clinical suspicion. A repeat CT scan of the abdomen revealed findings consistent with acute pancreatitis. There was moderate intrahepatic and extrahepatic biliary ductal dilation suggestive of biliary obstruction but no visible intraductal filling defect. The gallbladder was moderately distended with a small amount of pericholecystic fluid. Notably, there were two enhancing polypoid lesions arising from the gallbladder mucosa, measuring 9 mm and 6 mm (*Figure 1*), and no evidence of gallstones. She was treated with intravenous fluids and pain control.

A right upper quadrant ultrasound and magnetic resonance cholangiopancreatography were obtained for further evaluation of the gallbladder and biliary duct. On ultrasound, the gallbladder was noted to have multiple polyps measuring up to 7 mm. There was biliary sludge without sonographic evidence of cholecystitis. The common bile duct was enlarged at 7 mm. The magnetic resonance cholangiopancreatography was consistent with CT and ultrasound findings, confirming the presence of biliary ductal dilation without choledocholithiasis.

The consulting surgery team elected to proceed with laparoscopic cholecystectomy for obstructive pancreatitis. Her perioperative course was unremarkable. The final pathologic diagnosis was benign biliary-type polyp with chronic cholecystitis.

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Figure 1. CT of the abdomen showing evidence of (a) a 9-mm and (b) a 6-mm gallbladder polyp on the mucosal surface.

The gallbladder measured $7.5 \times 2.9 \times 2.0$ cm, the walls averaged 0.3 cm thick, and the cystic duct was unobstructed. The mucosa was notable for two loosely adherent 0.5-cm and 0.6-cm maximum dimension green excrescences and no gallstones. Her postcholecystectomy laboratory values markedly improved, with an alanine transaminase of 131 U/L, aspartate transaminase of 43 U/L, and total bilirubin of 0.6 mg/dL. The patient was discharged home a day later in good condition. She followed up with her primary care physician multiple times and was pain free without any recurrence of pancreatitis 15 months after surgery.

DISCUSSION

Cholesterosis is a benign condition that is usually discovered incidentally, either during cholecystectomy or on ultrasonography. These cholesterol polyps are common, with a prevalence ranging from 9% to 26%. In fact, cholesterol polyps are commonly found coexisting with gallstones. Given this association, there is reported evidence that the risk factors and the pathology of cholesterosis are similar to those of gallstone disease.³ However, there are few studies that have investigated the relationship between gallbladder polyps and acute pancreatitis. These studies have reported conflicting results, suggesting that the question of whether gallbladder polyps can cause acute pancreatitis is still unknown.

One of the earliest studies suggesting gallbladder polyps as a cause for acute pancreatitis described a patient with recurrent attacks of pancreatitis in whom cholecystogram was normal.⁴ The patient underwent a cholecystectomy and the operation revealed cholesterol polyps in the mucosa of the gallbladder and a widened (10-mm) common bile duct. The authors postulated that the cholesterol polyps may have sloughed off, causing a widening of the common bile duct and leading to the development of pancreatitis.^{4,5} This hypothesis was supported by Paricio et al in a retrospective study involving a cohort of 3797 cases of cholecystectomy, 55 of which were not

associated with cholelithiasis; 27 of those patients had presented with recurrent attacks of acute pancreatitis.⁶ Their findings led them to postulate a mechanism involving temporary impaction of cholesterosis polyps at the sphincter of Oddi, causing increased pressures in the pancreatic duct.⁵⁻⁷

Other studies have suggested that cholesterosis may simply be a marker for gallstones that may have passed undetected, as is the case in microlithiasis.⁸ Dairi et al performed a retrospective study of a cholecystectomy database of 6868 patients⁹ and did not find a direct correlation between cholesterosis and pancreatitis among patients without gallstones. They found no patients with pancreatitis and cholesterosis, and the lipase levels of patients with cholesterosis were significantly lower, suggesting that cholesterol polyps may actually be protective against pancreatitis.⁹

We hypothesize that cholesterol polyps in our patient acted like gallstones and caused temporary obstruction of the pancreatic duct leading to pancreatitis. Clinical suspicion for other causes of pancreatitis such as alcohol use disorder or autoimmune pancreatitis was very low. However, we cannot exclude microlithiasis and biliary sludge as possible causes. The 15-month negative medical follow-up in this patient reassures us of our proposed etiology for her pancreatitis.

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