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Acute Pancreatitis After Percutaneous Mechanical Thrombectomy of a Thrombosed Arteriovenous Fistula

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Background: In the United States, approximately 275,000 cases of acute pancreatitis are diagnosed annually, making acute pancreatitis the leading gastrointestinal cause of hospitalization. The most common etiologies are gallstones and alcohol use, but many cases have inconspicuous etiologies.

Case Report: A 69-year-old female with a history of end-stage renal disease on hemodialysis presented with acute epigastric abdominal pain hours after undergoing an uncomplicated percutaneous mechanical thrombectomy of a stenosed arteriovenous fistula. Laboratory and imaging findings were consistent with acute pancreatitis without evidence of gallstones. Her pancreatitis was attributed to inflammation caused by hemolysis that occurred during the procedure she underwent earlier that same day.

Conclusion: During thrombectomy, intravascular hemolysis releases free hemoglobin and heme products. These substances can generate an inflammatory response through the production of acute phase reactants and free oxygen radicals. The microvasculature of the pancreas is susceptible to this inflammatory cascade, and in some patients, the result is acute pancreatitis. The mechanism through which the pancreas is susceptible to inflammation associated with hemolysis is poorly understood.

Keywords: *Abdominal pain, hemolysis, inflammation, kidney failure–chronic, pancreatitis, thrombectomy*

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INTRODUCTION

In the United States, approximately 275,000 hospitalizations for acute pancreatitis occur annually, making acute pancreatitis the leading gastrointestinal cause of hospitalization.[1] The prevalence of this diagnosis has been on the rise in the United States, from 172,508 cases in 1990 to 264,954 cases in 2019.[2] The most common etiologies are gallstones and alcohol use. However, other commonly recognized causes include medication-related adverse effects, elevated triglyceride levels, trauma, and autoimmune disorders.[1] Acute pancreatitis is characterized by abdominal pain, elevated lipase or amylase levels, and findings of pancreatic inflammation on imaging.[1] Treatment may vary by etiology but routinely consists of intravenous hydration, bowel rest, and pain management. While most cases of acute pancreatitis can be readily explained, a portion of patients have inconspicuous etiologies.

We present the case of an elderly female who underwent successful percutaneous mechanical thrombectomy and within hours of the procedure presented with the signs and symptoms of pancreatic inflammation.

CASE REPORT

A 69-year-old female with a medical history of end-stage renal disease on hemodialysis and congestive heart failure presented with acute-onset epigastric abdominal pain, nausea, vomiting, and diarrhea. Earlier the same day, she had undergone a successful fistulogram with percutaneous mechanical thrombectomy of a stenosed arteriovenous fistula of her left upper extremity. No complications were documented, and the patient remained hemodynamically stable throughout and after the procedure.

Physical examination showed a stable but ill-appearing female with considerable epigastric abdominal pain and distention. She denied any current or previous alcohol use and, per medical record review, had been reporting alcohol abstinence for much of her life. Her history was negative for cholelithiasis or recent trauma. Initial laboratory workup was notable for leukocytosis with a white blood cell count of 13.84 K/μL (reference range, 3.90-12.70 K/μL), lipase of 1,453 U/L (reference range, 4-60 U/L), and bilirubin elevated to 1.2 mg/dL (reference range, 0.1-1.0 mg/dL), with baseline near 0.5 mg/dL. During the patient's first 24 hours of admission, her hemoglobin decreased from 12.8 g/dL to 10.8 g/dL (reference range, 12.0-16.0 g/dL) without any signs of bleeding. Triglyceride levels were within normal limits. Haptoglobin, lactate dehydrogenase, and reticulocyte count were not collected. Computed tomography angiography of the abdomen and pelvis showed diffuse peripancreatic fat stranding. No evidence of gallstones, pancreatic fluid collection, or abdominal arterial thrombosis or stenosis was seen. Based on her symptoms, laboratory findings, and imaging

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results, the patient was diagnosed with and treated for acute pancreatitis. Her medication list was reviewed for common causes of medication-induced pancreatitis. Torsemide was held but was not considered a likely culprit as the patient had been stable on the medication for 4 years.

Her symptoms improved after 24 hours of continuous intravenous hydration with lactated Ringer solution, 24 hours of bowel rest followed by gradual diet advancement during the next 24 hours, and management of pain with hydrocodone-acetaminophen 5-325 mg every 6 hours as needed. She was discharged after 3 days without any additional complications. Laboratory values at discharge were white blood cell count of 10.56 K/μL, lipase of 28 U/L, and bilirubin of 0.4 mg/dL.

A home health evaluation was conducted 2 days after discharge. The patient's symptoms remained resolved, and no acute concerns were noted.

DISCUSSION

Percutaneous mechanical thrombectomy is a rare cause of a common presentation, with most literature about acute pancreatitis following percutaneous mechanical thrombectomy limited to case series.[3] Consequently, the frequency of acute pancreatitis following percutaneous mechanical thrombectomy is unknown.

In a 1991 retrospective analysis, Druml et al estimated that after massive intravascular hemolysis secondary to various causes—including autoimmune hemolysis and microangiopathic hemolytic anemia—acute pancreatitis develops in approximately 20% of patients.[4] The Druml et al analysis did not include any patients who had undergone thrombectomy, further highlighting the rarity and need for additional understanding of pancreatic inflammation after thrombectomy. The mechanism through which intravascular hemolysis leads to pancreatitis is not well understood, but hemolysis and the subsequent production of heme products are well documented to result in a proinflammatory state.[5]

Independent of the cause, intravascular hemolysis releases free hemoglobin and heme products. Heme, a recognized damage-associated molecular pattern, acts as a chemoattractant for various immune cells, activates the complement system, and is associated with the production of reactive oxygen species.[5] Additionally, this inflammatory state can activate the coagulation cascade, leading to formation of microthrombi.[6] When the innate clearing mechanisms of damage-associated molecular patterns are overwhelmed, the microvasculature of the pancreas is susceptible to these insults, leading to the development of acute pancreatitis. Risk factors believed to contribute to the development of pancreatic inflammation after mechanical thrombectomy include renal dysfunction, large thrombus size, and involvement of the central vasculature.[3]

Promptly recognizing postthrombectomy pancreatitis can spare patients extensive workups, and diagnosis only requires a physical examination, a serum lipase level, and pancreatic imaging. Knowing that symptoms of pancreatitis after mechanical thrombectomy typically develop within 24 hours of the procedure can aid in rapid diagnosis. Management involves the routine practices of intravenous fluids, bowel rest, and pain management.

CONCLUSION

Acute pancreatitis after percutaneous mechanical thrombectomy is rare but can occur secondary to intravascular hemolysis and the release of free heme products, a recognized proinflammatory damage-associated molecular pattern. While no single diagnostic tool confirms this diagnosis, clinical history, serum lipase, and pancreatic imaging, combined with exclusion of other causes, are sufficient for workup. Symptom onset within 24 hours of mechanical thrombectomy, history of end-stage renal disease, and laboratory evidence of hemolysis should raise suspicion for this process. Early recognition of this etiology can prevent unnecessary procedures and potential patient harm.

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