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CASE REPORT

Acute Pancreatitis Following Percutaneous Mechanical Thrombectomy of Left Common Iliac Vein

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Abstract

While parental or oral anticoagulation remains a mainstay of therapy for thrombosis, in sporadic clinical situations, percutaneous mechanical thrombectomy is favored. Percutaneous mechanical thrombectomy is a well-tolerated subtype of catheter-directed intervention resulting in thrombus breakdown and removal. This procedure combines endovascular mechanical thrombectomy in combination with pharmacologic thrombolysis allowing for a significant reduction in procedure time. Similar to other catheter-based procedures, common complications include hemorrhage, dissection, pseudoaneurysm, and perforations. Acute pancreatitis, in contrast, is a rare complication of percutaneous mechanical thrombectomy with only limited cases reported and is hypothesized to occur secondary to release of heme byproducts. Here, we present a case of acute pancreatitis following outpatient percutaneous mechanical thrombectomy of the left common iliac vein that ultimately required hospitalization, intensive care unit (ICU) admission, and standard medical management for pancreatitis.

Keywords: Pancreatitis, Mechanical Thrombectomy

1. Introduction

Percutaneous mechanical thrombectomy, a subtype of catheter-directed thrombectomy, was found to be an effective and well-tolerated option for management of vascular thrombi of arterial, venous, or embolic origin. Recent studies have shown the superiority of percutaneous mechanical thrombectomy for management of deep venous thrombus (DVT) as compared to standard care with anticoagulation, reducing the likelihood of post-thrombotic syndrome, as well as in cases where traditional anticoagulation has failed to achieve clot burden reduction or symptomatic improvement.[1] Percutaneous mechanical thrombectomy differs from traditional catheter-directed thrombolysis in that percutaneous mechanical thrombectomy couples a shorter duration (in general no more than 45 min) of tissue plasminogen activator (tPA) with mechanical thrombectomy, whereas traditional catheter directed thrombolysis involves a longer duration of tPA infusion and only balloon maceration of residual thrombus.[1] Since the introduction of percutaneous mechanical thrombectomy as an adjunctive treatment option under the umbrella of catheter-directed thrombolysis for management of DVT, patient outcomes have improved significantly with lower morbidity, mortality, hospital length of

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Table 1. Laboratory values from the day of presentation and day 6 of admission.

	Na (135–145 mmol/L)	K (3.5–5 mmol/L)	CO2 (22–33 mmol/L)	BUN (8–20 mg/dL)	Cr (0.6–1.5 mg/dL)	Hb (g/dL)	Hct (%)
On admission	136	4.9	19	34	1.8	15.4	44
After episode (6 days after presentation)	136	3.5	24	31	1.97	10.1	29.6

stay, and complications, particularly when compared to traditional mechanical thrombectomy methods.[2] While complications remain sporadic and primarily involve procedure-related adverse events - hemorrhage, pseudoaneurysm formation, perforation, and dissection[3] – the marked hemolysis may result in heme byproduct release resulting in an inflammatory response with secondary organ dysfunction. Here, we present a rare case of post-percutaneous mechanical thrombectomy acute pancreatitis.

2. Case presentation

A 74-year-old female with chronic venous insufficiency status post bilateral common iliac vein stent placement and unprovoked occlusive deep venous thrombosis (DVT) was incidentally found to have a left common iliac vein in-stent non-occlusive thrombus on surveillance imaging. Her history was notable for a prior episode of left iliofemoral occlusive DVT that was managed with IVC filter insertion followed by Zelante catheter-based thrombectomy, catheter-directed TPA-mediated thrombolysis, and percutaneous suction thrombectomy, with subsequent anticoagulation with Xarelto and IVC removal. She occasionally drank alcohol and no prior history of pancreatitis in her or her family members. Following her recurrent diagnosis, she was resumed on Xarelto and underwent scheduled mechanical thrombolysis via Zelante catheter-based thrombectomy and left common iliac vein balloon venoplasty with Denali IVC filter insertion for PE prevention. The patient tolerated the procedure without immediate complication and was discharged home. Unfortunately, over the course of the evening she developed acute onset upper abdominal discomfort that radiated to her back. Her symptoms were associated with severe nausea, multiple episodes of bloody non-bilious vomiting and dark cola-colored urine for which she presented to our facility.

On presentation she was hemodynamically stable. Physical examination was notable for a soft and non-distended abdomen with normoactive bowel sounds, but diffuse tenderness to palpation, most pronounced in right upper quadrant, without peritoneal signs. Initial laboratory analysis revealed an elevated white blood cell count (18.9 k/uL), blood urea nitrogen (34 mg/dL), creatinine (1.80 mg/dL), high-sensitivity Troponin (11,711 ng/L), lactic acid (5.6 mmol/L), and lipase (1538 units/L) (Tables 1 and 2). Lipid panel was obtained for rule out hypertriglyceridemia for a possible cause of pancreatitis, that was within normal (Table 3). Urinalysis showed dark yellow urine positive for blood, bilirubin, and urobilinogen with only 5–10 RBC/hpf. Initial electrocardiogram revealed no ischemic changes. Computed tomography (CT) gastrointestinal bleed protocol showed fatty infiltration of the pancreatic head with concerns for necrotizing pancreatitis with no presence of cholelithiasis noted (Fig. 1). She was admitted to the ICU and Ceftriaxone, Metoclopramide, Zofran, morphine, and fluids were initiated. Heparin drip was also started given the associated troponin elevation and concern for non-ST elevation myocardial infarction (NSTEMI).

Her hospital course initially complicated by hematemesis and melena with a marked hemoglobin decline (15.4 g/dL to 7.0 g/dL), requiring transfusion of two units of packed red blood cells and heparin discontinuation. With continued conservative management, her abdominal pain and vomiting gradually resolved, but she remained unable to tolerate oral intake, necessitating fluoroscopy-guided nasojejunal tube (NJ) tube placement and tube feedings. No evidence of obstruction was appreciated on CT or abdominal radiographs. She underwent esophagogastroduodenoscopy that demonstrated a medium-sized hiatal hernia with a single 8-mm clean-based ulcer within the hiatal hernia. Over the course of the next five days she

Table 2. Liver panel values from the day of presentation and day 6 of admission.

	Total Bilirubin (0.1–1.0 mg/dL)	Direct bilirubin (0.-0.4 mg/dL)	ALP (35–115 IU/L)	AST (0–45 IU/L)	ALT (–50 IU/L)
On admission	2.5	0.49	86	116	31
After episode (6 days after presentation)	0.8	0.38	257	111	131

Table 3. Lipid Panel on admission.

	Total Cholesterol (mg/dL)	HDL (mg/dL)	LDL (mg/dL)	Triglycerides (mg/dL)
On admission	219	55	140	118

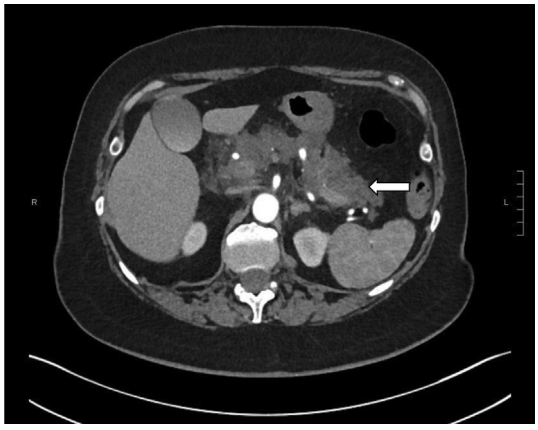

*Fig. 1. **Computed Tomography of the abdomen and pelvis.** CT on presentation demonstrated the presence of distorted pancreatic architecture with fatty infiltration along the pancreatic head, concerning for pancreatitis with associated pancreatic necrosis.*

demonstrated gradual symptomatic improvement, was challenged with Heparin without complication, and transitioned to Xarelto on discharge.

3. Discussion

Deep vein thrombosis (DVT) is a major cause of morbidity with numerous complications including venous insufficiency, post-thrombotic syndrome, and pulmonary embolism. Management had classically focused on the use of unfractionated heparin or low molecular weight heparin followed by vitamin K antagonists or direct oral anticoagulants for a minimum of three months.[4] Endovascular modalities including catheter-directed thrombolysis and percutaneous mechanical thrombectomy were developed to achieve accelerated thrombolysis with less bleeding risk. Catheter-directed thrombolysis was shown to be superior to anticoagulation alone in terms of higher rates of thrombolysis and lower rates of recurrence and post-thrombotic syndrome.[5]

Catheter-directed thrombolysis delivers thrombolytic agents via transluminal catheters allowing for reduced doses of thrombolytic agents and consequentially a reduction in bleeding complications compared to systemic thrombolysis, though still markedly elevated compared to systemic anticoagulation, hampering its widespread use.[6] Percutaneous mechanical thrombectomy is a specific subtype of catheter-directed thrombolysis in which thrombectomy devices are passed to the site of DVT and blood clots are removed by different mechanical means. It can also be used as an adjunctive device to CDT or pharmacomechanical thrombectomy. When these two methods are used in combination, the dosage of thrombolytic agents can be lowered further and the duration of procedure can be shortened.[7] Percutaneous mechanical thrombectomy provides the greatest benefits for young and functionally active patients with acute presentation of extensive proximal DVT (<14 days, or presence of phlegmasia cerulea dolens).[4] While complications of percutaneous mechanical thrombectomy such as distal embolization of thrombus fragments, vessel perforation, vessel dissection and bleeding - are similar to other catheter based interventions,[8] the force of the localized saline jets lead to clot-based and blood-based erythrocyte trauma. As a result of these local forces, hemolysis, with a concomitant decline in hemoglobin and development of indirect hyperbilirubinemia and hemoglobinuria, have been noted post-procedure. Furthermore, this catheter-directed thrombolysis induced hemolysis has been linked to acute renal insufficiency and pancreatitis.[2,3,9,10,11,12]

Prior reports have highlighted that hemolysis is a rare yet definitive cause of pancreatitis.[13–18] While hemolysis results in the release of free heme groups, pathophysiologic effects of this release are minimized secondary to the presence of heme-scavenging proteins and heme-degrading enzymes such as hemopexin and heme oxygenase, respectively.[19] Unfortunately massive hemolytic events overwhelm normal physiology and allow for heme escape with subsequent oxidative damage, inflammatory cell recruitment, vascular dysfunction, and cytokine cascades.[19] Indeed, the pathophysiology of hemolysis-induced pancreatitis is hypothesized to be secondary to free heme group mediated inflammation.[20,21] These moieties are responsible for inflammatory cascade activation, leukocyte recruitment, and Interleukin-8 induced neutrophil activation.[18] Free heme molecules have been shown to increase pancreatic tissue levels of tumor necrosis factor α (TNF- α), platelet-activating factor and α-2 macroglobulin leading to acute pancreatitis.[18,20–22] Free heme has also been shown to increase pancreatic vascular permeability through the up-regulation of the adhesion molecules ICAM-1 and P-selectin, facilitating not only leukocyte migration but further contributing to the developing inflammatory cascade.[21,22]

In cases of catheter-directed thrombolysis, and more specifically, percutaneous mechanical

thrombectomy, the risk of developing free-heme induced pancreatitis has been associated with numerous factors − thrombus size, blood flow within the target vessel, procedure duration, baseline renal function.[2,23] The patient presented in this case was noted to have only a limited procedural time, however, was noted to have a larger thrombus and significant blood flow within the target vessel, which when combined with the patients underlying chronic kidney disease, likely contributed to acute pancreatitis following her procedure. Renal dysfunction limits heme clearance, thus allowing for increased free heme concentrations in the blood and resultant inflammatory cascade activation. Typically, symptoms of percutaneous mechanical thrombectomy-induced pancreatitis commence within 24 h of the procedure but may present as last as four days post-procedure.[23,24] Given the temporal relationship between the percutaneous mechanical thrombectomy and the development of acute pancreatitis, coupled with a negative, albeit limited workup for other causes of pancreatitis, our patient was given a diagnosis of percutaneous mechanical thrombectomy induced pancreatitis secondary to hemolysis. Prevention strategies including procedural time limitations, perioperative rehydration, and urine alkalization may prevent massive hemolysis and subsequent complications.

4. Conclusion

Pancreatitis secondary to percutaneous mechanical thrombolysis is a relatively rare phenomenon dependent not only on predisposing patient factors (renal function), but also upon target factors (clot size, clot location, local blood flow) and technician dependent factors (catheter saline jets). Further studies evaluating these factors may help in determining the risk of post-procedure pancreatitis as a function of hemolysis and free heme release. Furthermore, studies targeting prophylaxis whether by hydration, urinary alkalization, or nutritional supplementation to facilitate free radical scavenging would be beneficial to mitigate the effects of free heme. Physicians should be aware of post-percutaneous mechanical thrombectomy pancreatitis and evaluate lipase and amylase levels in the context of unexplained abdominal pain post-procedure, acknowledging that such presentations may be delayed by over 96 h.

Specific author contributions

Ashik Pokharel, Xinhang Tu, Haider A. Naqvi: Manuscript writing, proofreading, figures and review of data.

Christopher J. Haas & Rafi Raza: Overall supervision and proofreading.

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Conflict of interest

None.

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