Case Report

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Multiple Left Ventricular Apical Thrombi in Mild Acute Pancreatitis Despite Preserved Ventricular Function: A Case Report

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ABSTRACT

Left ventricular thrombus is most commonly associated with acute myocardial infarction, significant regional wall motion abnormalities, ventricular aneurysm, or severe left ventricular systolic dysfunction. Its occurrence in a patient with preserved ventricular function and no significant obstructive coronary artery disease is unusual. We report the case of a middle-aged patient who presented with mild acute pancreatitis and a mature pancreatic pseudocyst measuring 6.8×6.9×6.0 cm. During contrast-enhanced abdominal CT performed for evaluation of the pancreatic pathology, an incidental left ventricular apical filling defect was identified. Subsequent transthoracic echocardiography and cardiac magnetic resonance imaging confirmed multiple left ventricular apical thrombi, the largest measuring approximately 1.7 cm, despite a preserved left ventricular ejection fraction of 65% and absence of overt regional wall motion abnormality.

Coronary angiography demonstrated non-obstructive coronary artery disease, with a 40% lesion in the left circumflex artery. A thrombophilia evaluation was non-contributory. In view of the known systemic prothrombotic effects of acute pancreatitis, pancreatic inflammation was considered a potential contributor to thrombus formation, although a direct causal relationship could not be established.

The patient was treated with apixaban 5 mg twice daily following multidisciplinary cardiology and surgical assessment. Elective intervention for the pancreatic pseudocyst was deferred because of the competing thromboembolic and procedural bleeding risks. At 6-week follow-up, repeat echocardiography demonstrated partial regression of the thrombus without clinical evidence of systemic embolization.

This case highlights an unusual association between acute pancreatitis and left ventricular thrombus formation in the absence of the conventional major cardiac substrates. It also emphasizes the importance of careful review of cardiac structures visible on abdominal cross-sectional imaging and the need for multidisciplinary decision-making when an intracardiac thrombus is identified in a patient requiring elective surgery.

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Introduction

Left ventricular thrombus (LVT) is a potentially serious condition because of its association with systemic embolization, stroke, and other adverse cardiovascular outcomes. It is classically encountered in patients with acute myocardial infarction, particularly when associated with apical akinesia or aneurysm, as well as in patients with significant left ventricular systolic dysfunction. The pathogenesis of LVT can be understood using Virchow's triad: blood stasis, endocardial injury, and hypercoagulability [1]. Current American Heart Association guidance recognizes the importance of identifying both the underlying cardiac substrate and the individual patient's thromboembolic and bleeding risks when managing LVT.

Acute pancreatitis is increasingly recognized as a systemic inflammatory disease with local and extra-pancreatic complications. Coagulation abnormalities and thrombotic complications, particularly splanchnic venous thrombosis, are well described. Acute pancreatitis has also been shown to be associated with a hypercoagulable state, with evidence of increased coagulation activity, platelet activation, and tissue-factor expression.

Intracardiac thrombosis, however, is extremely uncommon. A previously published case described intraventricular thrombus as a rare complication of acute pancreatitis, supporting the biological plausibility of this association. The present case is distinctive because multiple left ventricular apical thrombi were identified in a patient with mild acute pancreatitis, preserved left ventricular systolic function, and no significant obstructive coronary artery disease [2, 3]. The thrombi were discovered incidentally during abdominal cross-sectional imaging. The case also presented a management dilemma because the patient had a mature pancreatic pseudocyst for which intervention was being considered.

Case presentation

A middle-aged patient presented to the emergency department with severe, constant epigastric abdominal pain radiating to the back, associated with nausea and non-bilious vomiting. Clinical assessment with elevated pancreatic enzyme levels established the diagnosis of acute pancreatitis, and the patient was admitted under the general surgery department for conservative management.

The medical history was notable for newly diagnosed type 2 diabetes mellitus and systemic arterial hypertension. There was no previous personal or family history of thromboembolic disease, premature coronary artery disease, or cardiac arrhythmia.

On physical examination, the patient had epigastric tenderness without clinical features of an acute abdomen. There were no clinical signs of peripheral systemic embolization.

Contrast-enhanced CT and MRCP demonstrated a large, mature, well-defined, and encapsulated intrapancreatic pseudocyst involving the pancreatic head and body. The lesion measured 6.8×6.9×6.0 cm and produced extrinsic compression of the portal vein, with prominent superior mesenteric venous tributary collaterals. The splenic vein remained patent, and there was no evidence of pseudoaneurysm formation. During review of the contrast-enhanced abdominal CT, a hypodense filling defect was incidentally identified in the left ventricular apex (Figure 1).

Transthoracic echocardiography demonstrated preserved left ventricular systolic function with an ejection fraction of 65%. Concentric left ventricular hypertrophy and trivial mitral regurgitation were noted. Multiple distinct apical thrombi were visualized, with the largest measuring approximately 1.7 cm in maximal dimension. There was no reported major regional wall motion abnormality. No right heart strain or evidence of pulmonary embolism was identified.

Cardiac magnetic resonance imaging was performed for further characterization of the intracardiac lesions, which demonstrated an apical thrombus measuring 15×13 mm. Focal apical myocardial thinning was present. Non-ischemic late gadolinium enhancement was noted in the apical and mid-ventricular regions. Importantly, there was no overt regional wall motion abnormality. The absence of enhancement within the intracardiac mass and the subsequent reduction in thrombus size on anticoagulation supported the diagnosis of thrombus rather than an intracardiac tumor.

Coronary angiography was performed to evaluate for occult ischemic myocardial injury as a potential substrate for LVT, and non-obstructive coronary artery disease, with a 40% lesion in the left circumflex artery, was noted. There was no significant obstructive coronary lesion that could explain a major infarction-related apical thrombus.

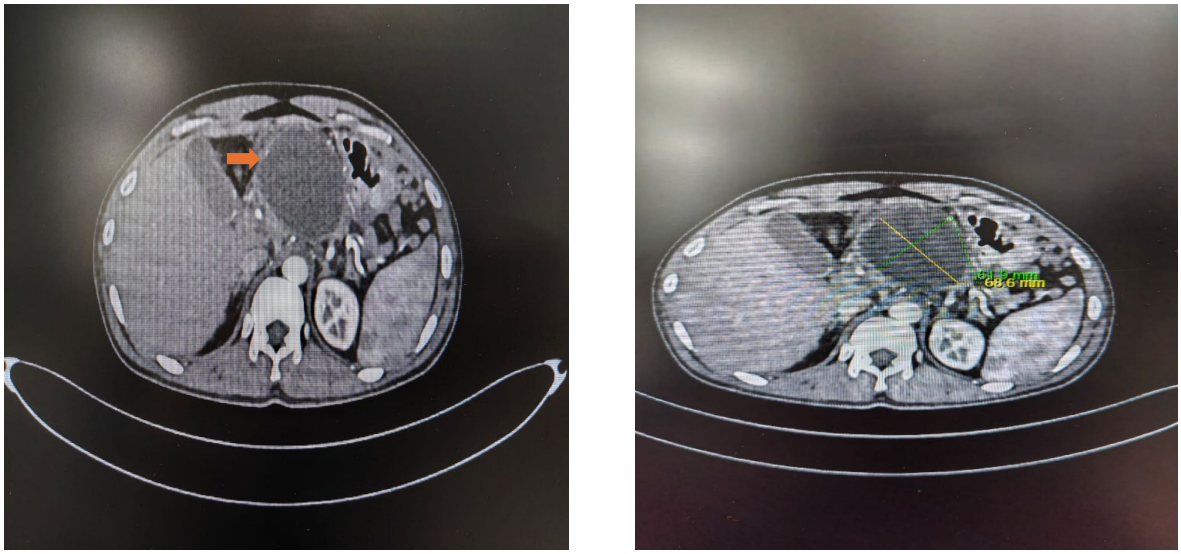


Fig. 1. Computed tomography images showing a pancreatic pseudocyst.

A prothrombotic evaluation was performed. Protein C, protein S, antithrombin III, lipoprotein(a), and serum homocysteine levels were within normal ranges.

The identification of an apical intracardiac mass in a patient with preserved left ventricular systolic function prompted consideration of several differential diagnoses. LVT was considered the most likely diagnosis based on the apical location, morphology, cardiac MRI characteristics, and subsequent partial regression during anticoagulation.

Primary cardiac tumors, including myxoma and fibroma, were considered. These were considered less likely because of the apical location, multiplicity of the lesions, and lack of enhancement characteristics expected of a vascularized intracardiac mass.

These conditions were considered because of the myocardial late gadolinium enhancement. However, the reported peripheral eosinophil count was normal, and there was no evidence of restrictive hemodynamics or other characteristic structural findings. Apical hypertrophic cardiomyopathy or Takotsubo-related apical abnormality was suspected because apical hypertrophy was present, but there was no characteristic apical ballooning or severe contractile abnormality on cine CMR.

Overall, the combined clinical, echocardiographic, and CMR findings favored multiple left ventricular apical thrombi.

The mature pancreatic pseudocyst represented a potential indication for elective intervention. However, immediate invasive treatment was considered inappropriate because of the newly identified intracardiac thrombi and the potential risk of systemic thromboembolism.

Conversely, initiation of therapeutic anticoagulation would increase the potential bleeding risk associated with an invasive pseudocyst procedure. After multidisciplinary discussion, elective intervention for the pseudocyst was therefore deferred.

Oral apixaban 5 mg twice daily was initiated for therapeutic anticoagulation. Treatment for the newly diagnosed hypertension and type 2 diabetes mellitus was also initiated, with planned cardiology follow-up. The choice of a direct oral anticoagulant was made following individualized cardiology assessment. Contemporary observational and meta-analytic evidence suggests that DOACs may have efficacy comparable with vitamin K antagonists for LVT, although the evidence remains heterogeneous and largely observational, and DOAC use in this setting has historically been off-label. The patient was discharged in a clinically stable condition.

At 6-week outpatient follow-up, the patient remained hemodynamically stable. The abdominal pain had completely resolved, and there were no ongoing gastrointestinal symptoms or clinical features of systemic embolization. Repeat transthoracic echocardiography demonstrated partial regression of the left ventricular thrombus burden, although residual apical thrombus remained. Apixaban was therefore continued. Drainage or other elective

intervention for the mature pancreatic pseudocyst remained deferred pending further resolution or stabilization of the intracardiac thrombus and repeat cardiology assessment.

Discussion

This case describes multiple left ventricular apical thrombi occurring in a patient with mild acute pancreatitis despite preserved left ventricular systolic function. The principal clinical challenge was that the patient lacked the usual major substrate for LVT. The ejection fraction was 65%, there was no overt regional wall motion abnormality, and coronary angiography demonstrated only a 40% left circumflex lesion. Thus, a large infarction with apical akinesia was not identified. The finding therefore raises the possibility that systemic prothrombotic mechanisms associated with acute pancreatitis contributed to thrombus formation [4].

Thrombotic complications of acute pancreatitis are well recognized, particularly involving the splanchnic venous circulation. Systematic reviews have reported clinically important rates of splanchnic venous thrombosis in acute pancreatitis [5, 6].

The pathophysiology is complex. Pancreatic inflammation can activate coagulation pathways, promote endothelial dysfunction, and alter the balance between procoagulant and anticoagulant mechanisms. Experimental and translational data also suggest increased platelet activation and tissue-factor activity during acute pancreatitis. Importantly, a previously reported case described intraventricular thrombus as an extremely rare complication of acute pancreatitis.

Our case adds to this limited literature by describing multiple left ventricular apical thrombi [7] in the setting of mild acute pancreatitis with preserved systolic function and without significant obstructive coronary artery disease.

However, the association should not be interpreted as proof of causation. The patient had newly diagnosed diabetes mellitus and hypertension, concentric LV hypertrophy, and subtle myocardial abnormalities on CMR. These factors may have contributed to the thrombotic substrate.

Virchow's triad in this patient: The case can be considered in the context of Virchow's triad.

Hypercoagulability: Acute pancreatitis may produce systemic activation of coagulation and a transient prothrombotic state.

Endothelial or myocardial injury: The CMR findings of focal apical myocardial thinning and late gadolinium enhancement suggest that subtle myocardial abnormalities may have been present, although their precise significance cannot be established.

Stasis: Although there was no major global LV dysfunction, local alterations in ventricular geometry associated with concentric hypertrophy and apical structural abnormalities could potentially have contributed to local thrombus formation.

Thus, the combination of systemic inflammation and individual cardiovascular risk factors may have provided a sufficient environment for thrombosis despite preserved global systolic function.

The thrombi were initially identified incidentally on abdominal contrast-enhanced CT. This highlights the importance of careful inspection of the cardiac structures included in upper abdominal cross-sectional imaging.

Transthoracic echocardiography established the presence of multiple apical thrombi and demonstrated preserved ventricular function. CMR then provided further tissue characterization and helped differentiate thrombus from intracardiac tumor.

The AHA scientific statement on LVT also recognizes CMR as an important imaging modality when echocardiographic findings are uncertain or when further tissue characterization is required.

Therapeutic anticoagulation was selected because of the potential embolic risk associated with the intracardiac thrombi.

The use of apixaban in this case should be interpreted as an individualized treatment decision rather than evidence that DOACs are universally superior to vitamin K antagonists [8]. Contemporary meta-analyses have generally found comparable thrombus resolution between DOACs and vitamin K antagonists, although differences in bleeding and mortality have been reported, and the evidence base remains limited by observational data and small randomized studies. In the present case, partial thrombus regression was observed at 6 weeks without clinical evidence of systemic embolization [9].

The pancreatic pseudocyst created an additional

management challenge. Although elective drainage could potentially be considered for a mature pseudocyst depending on symptoms, complications, and anatomical considerations, intervention in a patient with an untreated intracardiac thrombus may carry significant thromboembolic risk. Conversely, therapeutic anticoagulation increases the potential bleeding risk associated with invasive procedures.

The decision to defer elective pseudocyst intervention therefore represented a balance between two competing risks and was made following multidisciplinary assessment.

Limitations

This is a single case and cannot establish a causal relationship between acute pancreatitis and left ventricular thrombus formation. The patient also had newly diagnosed diabetes mellitus and hypertension, and CMR demonstrated subtle myocardial abnormalities. Therefore, pancreatitis should be regarded as a plausible contributing prothrombotic trigger rather than a proven sole cause. Longer-term imaging follow-up would also be required to establish complete thrombus resolution and determine the appropriate duration of anticoagulation.

Conclusion

Multiple left ventricular thrombi can rarely occur in the setting of acute pancreatitis even when left ventricular systolic function is preserved and significant obstructive coronary artery disease is absent. In this patient, acute pancreatitis was considered a potential contributor to a systemic prothrombotic state, although causality could not be established.

Careful inspection of cardiac structures visible on routine abdominal CT facilitated early detection and prompted definitive cardiac imaging. CMR was valuable in characterizing the intracardiac lesions and excluding alternative diagnoses. When an intracardiac thrombus is discovered in a patient with a concurrent surgical condition, management should be individualized through multidisciplinary assessment of thromboembolic, anticoagulation, and procedural bleeding risks.

Learning points

- **Acute pancreatitis may be associated with a systemic prothrombotic state** and can rarely be accompanied by thrombotic complications outside the commonly recognised splanchnic circulation.

- **Left ventricular thrombus can occur despite preserved systolic function**, particularly when other structural, metabolic or inflammatory risk factors coexist.

- **Careful review of cardiac structures on abdominal CT can reveal clinically important incidental findings** and may alter subsequent management.

- **Cardiac MRI is valuable for characterising an intracardiac mass** and distinguishing thrombus from other cardiac lesions.

- **The association between pancreatitis and left ventricular thrombus should be described as plausible rather than definitively causal** in an individual case.

- **The presence of an intracardiac thrombus can substantially alter the timing of elective surgery**, necessitating multidisciplinary assessment of thromboembolic and bleeding risks.

Ethical Considerations

Compliance with ethical guidelines

There were no ethical considerations to be considered in this article.

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Conflict of Interests

The authors have no conflict of interest to declare.

Contributors

All authors contributed to patient care, manuscript preparation and drafting, critical revision of the manuscript and approval of the final version.

Patient consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images.

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