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Case Report

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An Ascending Aortic Thrombus

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Abstract

An ascending aortic thrombus is a rare finding, but it is a widely known cause of systemic embolization. Our case presents a 50-year-old man who presented with acute mesenteric ischemia. This was the result of a peripheral embolism originating from the ascending aortic thrombus. There was no pre-existing clotting abnormality. Conservative treatment with apixaban was initially tried since the patient was deemed not to be a suitable surgical candidate. However, the patient did not adhere to treatment. He later presented to the emergency department with severe abdominal pain and was found to have numerous embolic lesions to various body organs comorbid to his acute mesenteric ischemia. Only a few cases of intra-aortic thrombus without any coagulation abnormality are described in the literature. Occasionally, they present as acute mesenteric ischemia. We write about this case to emphasize its existence and highlight the serious morbidity and mortality associated with systemic embolization as a result of an intra-aortic thrombus.

Keywords: Ascending aortic thrombus; mesenteric ischemia.

Introduction

There is a paucity of cases where an ascending thoracic aorta thrombus leads to systemic embolization. There are a few cases that have been reported in the literature of an ascending aortic thrombus leading to cerebral infarction [1], acute limb ischemia [2], and splenic infarction [3]. We present, however, a case of an aortic atheroma leading to celiac and superior mesenteric artery thromboses, as well as infarction of the spleen,gallbladder, kidneys, and pancreas.

Case Presentation

A 49-year-old male with a past medical history of six cerebrovascular accidents, hypertension, hyperlipidemia, and a history of a thrombus or atheromatous plaque in the ascending thoracic aorta presents to the ED complaining of severe abdominal pain qualified as 10/10 in severity. Associated symptoms included nausea and vomiting. Vital signs upon arrival were as follows: heart rate 91 bpm, oxygen saturation 100%, blood pressure 162/81 mmHg, respiratory rate, and temperature 97.4 F.

Workups showed white blood cell count 15,800/mm^3, nucleated red blood cell count 0.0, neutrophils 10,800/mm3, lymphocytes 3,100/mm^3, monocytes 1,500/mm^3, eosinophils 300/mm^3, basophils 100/mm^3, and immature granulocytes 90/mm^3. red blood cell count 4.90 x 106/mm^3, hemoglobin 14.8 g/dL, hematocrit 44.8%, mean corpuscular volume 91 µm^3, red blood cell distribution width 15%, platelet 2.92 x 10^5/mm^3. Sodium 143 mEq/L,

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potassium 3.4 mEq/L, chloride 106 mEq/L, bicarbonate 20 mEq/L, anion gap 17 mEq/L, glucose 208 mg/dL, blood urea nitrogen 10 mg/dL, creatinine 1.21 mmol/L, estimated glomerular filtration rate by creatinine 73 mL/min, calcium 9.1 mEq/L, lactic acid was elevated at 4.3 mmol/L, and serum troponin was slightly elevated at 0.09 ng/L. The CTA of the abdomen (Figures 1 and 2) revealed findings that were suspicious for thrombi within the superior mesenteric artery (SMA), the superior mesenteric vein, and the left renal vein, as well as hypoperfusion of the spleen, the small bowel, and the right and left kidneys.

Go to July 18 imaging for the SMA thrombosis. Sagittal cut image 71 CT abd/pelvis venous phase. Intaluminal filling defect axial image 34 and 35

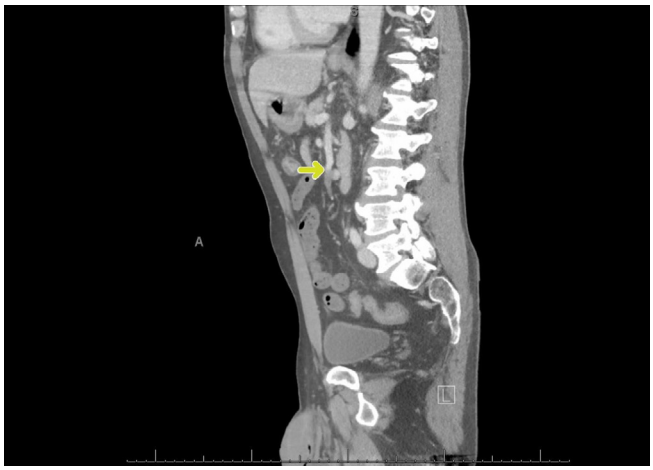

Figure 1: CT abdomen (sagittal view) showing a thrombus in the superior mesenteric artery(arrow)

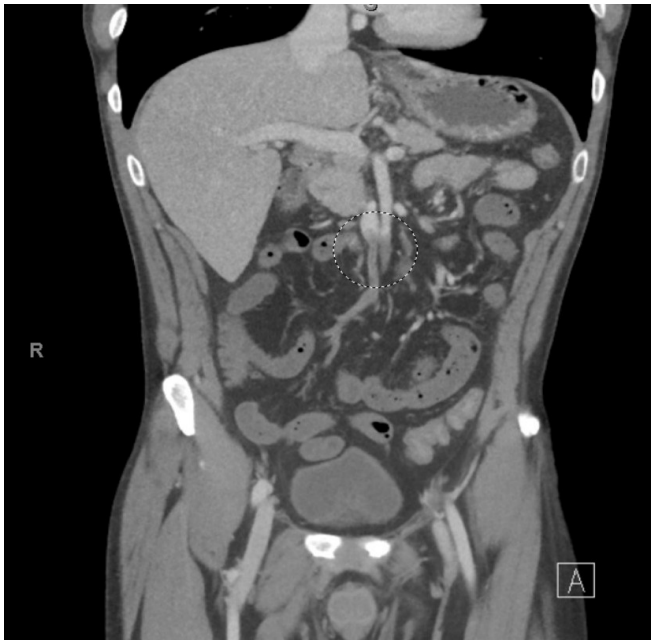

Figure 2: CT imaging (coronal view) of a thrombus in the celiac artery(circled)

On the physical exam, the patient was alert and cooperative, but was in distress. On examination of his abdomen, he was noted to be tender on palpation but did not exhibit any rebound tenderness or guarding, and there was no appreciable splenomegaly or hepatomegaly.

Heart sounds were normal, with no murmurs, rubs, or gallops present. The patient's lung exam was unremarkable. Pulses in his lower and upper extremities were palpable,2+.

The patient was moved to the intensive care unit because of his elevated lactate, and signs of bowel ischemia on physical examination and on imaging. He received analgesics, was started on a heparin 25,000units/24hours, and then the interventional radiology department and general surgery department were consulted for further management.

On Day 1 of admission, a superior mesenteric artery (SMA) arteriogram was done, a 5 cm unifuse catheter was placed in the mid-SMA, and tissue plasminogen activator (TPA) was injected into it. Below, we have images of the SMA pre-TPA (Figure 3) and post-TPA (Figure 4).

After the catheter thrombolysis, the patient continued to complain of abdominal pain, and there was tenderness to percussion as well as other peritoneal signs. Also, there was a noticeable reperfusion to the right colon and ileum, but the proximal jejunum remained unperfused; hence, a decision was made to pursue an exploratory laparotomy. During the exploratory laparotomy, procedural findings included small bowel infarction and a SMA thrombus. The cecum and 225 cm of the small bowel (the distal jejunum, the entirety of the ileum) was resected, and the surgical team did an open embolectomy of the superior mesenteric artery.

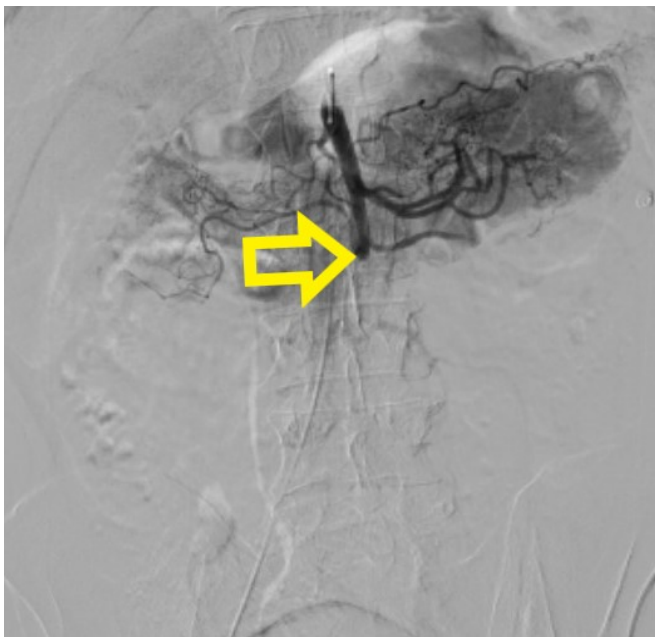

Figure 3: Selective superior mesenteric arteriogram–Digital Subtraction angiography. The arrow depicts the location the superior mesenteric thrombus.

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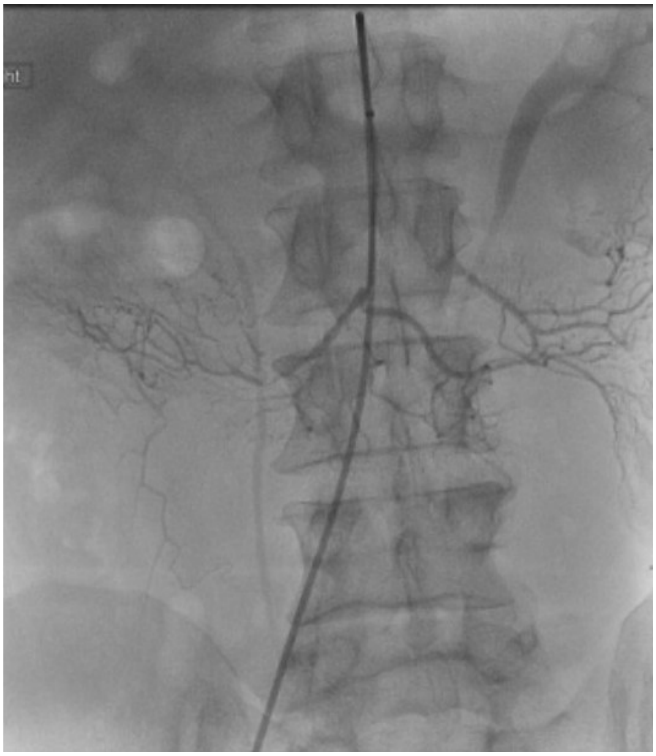

Figure 4: Digital subtraction angiography of SMA post catheter directed thrombolysis showing a patent superior mesenteric artery

A recheck exploratory laparotomy was done on day 2 of admission. The findings included two segments of ischemic jejunum that were identified and resected (approximately 64 cm total). A side-to-side, end-to-end small bowel to small bowel (jejunal-jejunal) anastomosis was created, and another side-to-side, end-to-end small bowel to ascending colon anastomosis was created. In addition, a gangrenous gallbladder was identified and removed.

The abdomen was closed after the second exploratory laparotomy, and the patient was restarted on a heparin infusion and moved to the ICU for close management.

On Day 3 of admission, the most significant events included markedly elevated transaminase levels (AST of 15,239 U/L and ALT of 4,183 U/L), the patient being oliguric, serum creatinine levels trending up from 1.2 mmol/L on admission to 2.87 mmol/L, and the patient being hypotensive and needing vasopressors. Due to the findings listed above, the patient was started on continuous renal replacement therapy. A repeat CTA of the abdomen did show hypoenhancement of the majority of the spleen and wedge-shaped areas of hypoenhancement involving the lower poles of the bilateral kidneys. A transthoracic echocardiogram was done, and it showed a left ventricular ejection fraction of 65% with no left ventricular thrombus and no valvular abnormalities.

On Day 4 of hospitalization, the patient was off vasopressors. He was tolerating spontaneous breathing trials, and thus was extubated. His INR was 2.9, his platelet count had trended down from 1.68 x 10^5/mm^3 to 7.9x 10^4/mm^3, and his AST and ALT values had trended down to 665 U/L and 809 U/L, respectively.

The patient remained stable and on anticoagulation until day 8 of hospitalization, where a repeat CT (Figure 5) was done, which indicated concern for the interval development of a thrombus in the celiac trunk. There was improved contrast opacification of the superior mesenteric artery, superior mesenteric vein, splenic vein, and bilateral renal arteries, all of which appeared to be thrombosed on the prior exam. There was still concern for development and/or worsening of multifocal infarcts throughout the liver, spleen, kidneys, and pancreas.

Due to the above findings and the impending poor prognosis associated with the infarction of multiple organs and a short bowel, a palliative care consult was placed.

Discussion

This is a 67-year-old male with several factors (history of cerebrovascular disease, smoking, hypertension, and hyperlipidemia) that indicate a high risk for athero-emboli phenomena who was prescribed lifelong apixaban due to the finding of an ascending aortic thrombus.

Our case highlights the unexpected embolizations and gravity of outcome associated with an ascending aorta thrombus. Ascending aortic thrombus can be deadly; hence, we must pay close attention to treating them and following up on the size of the thrombus.The therapeutic cornerstone for ascending aortic thrombi management is lifelong anticoagulation therapy. However, certain factors determine the utilization of thrombectomy and endovascular stent placement over oral anticoagulation as first-line treatment. These factors include: -location of the thrombus; for example, there is a very high risk of stroke associated with a thrombus located in the ascending aorta, transverse aortic arch, or isthmic region; -recurrence of embolic events while on oral anticoagulation; and -the non-resolution of an aortic thrombus on subsequent imaging while on oral anticoagulation [4]. Our patient was deemed ineligible for surgical management of his ascending aortic thrombus, and he also failed to follow up with cardiothoracic surgery for repeat imaging. The above scenario emphasizes the importance of regular follow-up imaging in patients who have an ascending aortic thrombus.

Even in patients who underwent surgical management of an aortic thrombus, there have been cases in the literature of recurrence after surgical management [5]. Strict adherence to anticoagulation therapy is still paramount to the management of this condition, even after thrombectomy. In the case of our patient, he failed to adhere to medical therapy. With the life-threatening morbidity and mortality associated with this disease state, clinicians have a duty to educate their patients regarding the risk they are undertaking if they do not follow through or stop taking their oral anticoagulation.

The presence of an ascending aortic thrombus with several embolic events raises the question of the etiology of the ascending aortic thrombus. The sheer force and velocity of blood flow in the ascending aorta make the formation of an aortic thrombus very unlikely; but in cases like this, there is a pathological phenomenon causing this anomaly. Non-atherosclerotic aortic thrombi are usually associated with hypercoagulable disorders like antiphospholipid syndrome [6] and factor V leiden mutation [7]; protein S deficiency [8,9] and protein C deficiency, prothrombin mutation, and autoimmune diseases like systemic lupus erythematosus [10].

In conclusion, an ascending aortic thrombus is rare and should be considered one of the possible causes of systemic embolization. It carries a high risk of morbidity and mortality. Anticoagulation therapy and surgical management remain the cornerstones of its treatment.

Conclusion

An ascending aortic thrombus is a rare but uncommon cause of systemic embolization. It carries a high mortality risk and could result in acute myocardial infarctions, cerebral infarctions, splenic infarctions, acute mesenteric ischemia, and acute lower extremity arterial embolism. Its management requires lifelong anticoagulation therapy and close monitoring of the thrombus size and evolution. In some cases, surgery is the preferred method of management.

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