Seven centimeter aortic mural thrombus in a 40-year-old female: Review and management

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CASE REPORT

A 40-year-old African-American female with a past medical history of diabetes mellitus type II, uterine fibroids, obesity and chronic anemia presented with right-sided mid-sternal chest pain associated with dyspnea for several weeks. She described her chest pain as feeling an object was stuck in her throat. She denied associated diaphoresis, weakness, or radiation. Her pain improved in the supine position and worsened with sitting upright. Her troponin was initially 0.22 ng/ml and trended to a peak of 0.64 ng/ml. Electrocardiography showed T wave inversions in leads III and V1, and flattening in V2–V5 and aVF. CBC showed a hemoglobin of 7 g/dL which dropped to 6.1 g/dL with MCV 59, platelets 358x10^9/L and 1.5% reticulocyte count. The patient denied hemoptysis, hematuria, hematochezia or melena. She reported a family history of anemia of unknown etiology, and stated that she had been taking iron supplementation since age 13, and frequently consumed ice chips. She reported regular menstrual cycles which had not been heavy or excessive. There was no history of smoking and her medications included metformin and glyburide. A computed tomography angiogram of the chest was negative for pulmonary embolus but resulted in an incidental finding of a large thrombus measuring 7x1 cm adherent to the anterior wall of the thoracic aorta with its distal portion positioned centrally within the lumen (Figure 1).

The patient was transfused two units of packed red blood cells which improved her anemia and dyspnea. She was given aspirin, a statin and a beta-blocker. Vascular surgery was consulted and recommended against surgical management, given that the patient...
was hemodynamically stable and without signs of embolization. She was subsequently started on a heparin drip. An echocardiogram revealed borderline normal left ventricular ejection fraction (LVEF).

A hypercoagulability panel (antithrombin III, protein C/S, anti-cardiolipin antibodies) was normal. The patient’s rheumatoid factor was negative and her ANA antibody titer of 1:40 was insignificant. Hemoglobin electrophoresis showed slightly decreased hemoglobin A2 and normal hemoglobin A and F. Hemoglobin A1c was 9.5% and a lipid panel was positive for hyperlipidemia.

The patient was discharged after three days with a statin, lovenox, and warfarin therapy. She had follow-up imaging one month after presentation which showed an interval decrease in thrombus size by approximately 1.5 cm. A repeat CT angiogram at eight months on warfarin therapy revealed near-complete resolution of her intraluminal thrombus (Figure 2). Her follow-up clinic visit three days post-discharge and at three, four, five, and seven months revealed no chest pain symptoms or signs of distal ischemic events.

**DISCUSSION**

Aortic wall thrombus is a rare cause of thromboembolism and is typically detected after presentation of distal pain, paresthesia, or weakness suggestive of ischemic disease [1–4]. In a literature review of 78 cases from 1981 to 2001, Bowdish et al. discovered 96% of patients with aortic thrombus presented with thromboembolic disease [1]. Fayad et al. completed a systematic review of 200 cases of aortic thrombus with a mean patient age of 50 years and noted high prevalence of embolic disease, with the majority of patients presenting with limb ischemia followed by visceral ischemia and stroke [2]. The embolization potential of an aortic thrombus is associated with its morphology. Those that are narrow-based and protruding into the lumen are associated with a 73% incidence of embolic events compared to a 12% incidence when lesions are broad-based and immobile [5]. The most common locations for aortic thrombus formation are the aortic arch and descending aorta [2, 5]. The patient in this case was a 40-year-old female presenting with chest pain and dyspnea and found to have an incidental 7 cm thrombus in the descending thoracic aorta without evidence of distal embolization. Although the specific morphology of the thrombus was not characterized, its size and distal position within the aortic lumen was believed to pose an increased risk of embolization, and would have been the likely outcome if the diagnosis was further delayed.

Risk factors for aortic thrombus formation include atherosclerosis, smoking, oral steroid use, primary aortic tumors, and hypercoagulable disease states [1, 4, 6, 7]. Given our patient’s young age it was less likely that her thrombus was due to atherosclerotic disease, as there was no overt evidence of aortic pathology, distal arterial disease, or claudication symptoms reported by the patient. Perler et al. in their case series noted that concomitant cigarette smoking and oral steroid use was a more likely etiology for aortic thrombus generation in premenopausal women than a diagnosis of premature arteriosclerosis [6]. Our patient, however, did not report steroid use or a smoking history, arguing against either of the described pathophysiology. Lastly, her hypercoagulopathy workup was negative and there was no evidence of malignancy.

Aortic thrombus with distal extremity emboli or cerebral infarct has been observed in young females with iron-deficiency anemia secondary to menorrhagia [8, 9]. Iron deficiency anemia may cause thrombocytosis or a possible hyperdynamic state that can lead to tears in the vascular endothelium predisposing it to thrombus formation [8, 9]. In our patient, a mild thrombocytosis was observed two years prior and was again noted at the end of her current hospital course, though such levels were unlikely to have contributed to clot formation.

Treatment strategies for aortic mural thrombus include thrombectomy, endovascular stenting, thrombolysis or anticoagulation, though currently there are no best practice guidelines. Retrospective reviews have shown thrombus resolution and prevention of additional embolic events when primary anticoagulation or thrombolysis is used [1, 3]. Turley et al. in their review of 13 cases, noted a lower complication rate for patients managed with anticoagulation and thrombolysis compared to surgical treatment (17% vs. 71%), including a lower median length of stay (9 days vs. 30 days) [3]. Given these findings and resulting clot resolution in both groups, they proposed medical management for initial thrombus management. Conversely, in their systematic review of 200 aortic mural thrombi cases, Fayad et al. suggested that primary surgical intervention is more beneficial given the lower rate of persistence and recurrence of thrombus compared to primary anticoagulation (5.7% vs. 26.4%; p <0.001) [2]. They also noted a higher complication rate in the anticoagulation group compared to the surgical group, although the difference was not significant.
As our patient did not have signs or symptoms of distal embolization and was hemodynamically stable, conservative management with anticoagulation was more appropriate than surgical intervention given the risk of surgical complications.

CONCLUSION

Aortic wall thrombus is a rare finding, usually presenting in the older population with underlying atherosclerotic disease or in younger patients with a hypercoagulable disease state. For the patient in this study, a hypercoagulable workup was negative. The underlying pathophysiology is unclear. Management options include anticoagulation, thrombolysis, and surgery. In our stable patient, we opted for warfarin therapy and avoided the risk of surgical complications. The patient remained asymptomatic over the follow-up period and imaging at eight months revealed clot resolution.

**Keywords:** Aortic thrombus, Descending thoracic aorta, Management

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**REFERENCES**


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**Author Contributions**

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**Guarantor**
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**Conflict of Interest**
Authors declare no conflict of interest.
SUGGESTED READING
