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Case Report

Non-surgical Management of Isolated Aortic Arch Thrombus

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Abstract

Arterial embolization is a severe condition and leading cause of disability in the United States. Primary aortic thrombosis in the absence of obvious underlying arterial disease is a rare cause of arterial embolic disease. Advancements in imaging, have augmented our ability to detect vascular pathology. Despite this, there remains a paucity of literature describing the optimal management of primary isolated aortic thrombosis. We present the case of a large isolated thoracic aorta thrombus in a middle-aged male with no identifiable gross aortic atherosclerotic or aneurysmal changes, intracardiac abnormalities, hypercoagulability, or malignancy. We discuss surgical versus nonsurgical management.

Keywords: Primary Aortic Thrombosis; CT Angiography (CTA); Computed Tomography (CT)

Introduction

Primary aortic thrombosis in the absence of obvious underlying arterial disease is a rare cause of arterial embolic disease [1]. Due to its low prevalence, an established approach to management remains unclear. Management options include nonsurgical and surgical interventions. We present the unusual case of a middle-aged male found to have an isolated thrombus within the aortic arch and descending aorta. Consent was obtained from the patient for publication of this case report.

Case Presentation

A 48-year-old male presented with 2 days of abdominal pain, nausea, and emesis. His past medical history was significant for smoking, hypertension, diabetes, hyperlipidemia, coronary artery disease, peripheral arterial disease with stenting of the right iliac artery with no follow up, and a T12 burst fracture after a motor vehicle accident two years prior. He had no history of hypercoagulability, but family history was remarkable for an unspecified hypercoagulable disorder in his sister. Computed tomography (CT) abdomen/pelvis revealed bilateral wedge-shaped perfusion defects of both kidneys. Heparin was initiated, and he was transferred to our facility for further management. CT angiography (CTA) of the chest demonstrated a non-calcified filling defect within the proximal descending aorta extending into the left common carotid artery and left subclavian artery (Figure 1). Heparinization was continued in addition to adding aspirin and a high-intensity statin. Transesophageal echocardiogram (TEE) showed no evidence of intracardiac thrombus or vegetation. No aneurysmal or atherosclerotic changes were appreciated within the aortic arch. Laboratory investigation for hypercoagulability was unremarkable. Additionally, there was no evidence of obvious underlying malignancy.

Figure 1: CT angiogram of the chest at time of presentation showing the aortic arch thrombus extending into the left subclavian artery (A) and the left common carotid artery (B).

Given the location of the thrombus we felt an endovascular approach would carry high risk for embolization. Accordingly, he was offered the option of hybrid approach versus open thoracic surgery versus conservative therapy. He elected to pursue the conservative pathway. On hospital day 1, after starting heparin therapy, he developed right lower extremity acute limb ischemia requiring surgical intervention. He remained inpatient for 5 additional days with no further embolic incidence. Repeat CTA chest demonstrated slight regression of thrombus burden in the left common carotid artery. Patient insisted to be released on day 5. He was sent on Eliquis, Aspirin and statin. He was seen back with repeated images at 1 month, 4 months and 17 months (Figure 2,3) with his last chest CTA showing ~98% resolution of the filling defect in the aorta and complete resolution of the filling defect in the left common carotid and left subclavian artery (Figure 3).

Figure 2: CT angiogram of the chest at 4 months follow up showing complete resolution of the left subclavian artery (A) and left common carotid artery (B) thrombus with significant regression of the aortic arch thrombus (A).

Figure 3: CT angiogram of the chest at 17 months follow up showing complete resolution of the left subclavian artery (A) and left common carotid artery (B) thrombus with ~ 98% resolution of the filling defect (red arrow) in the aorta (C).

Discussion

Acute arterial thrombosis is a leading cause of death in the developed world [2]. A common etiology of arterial thrombosis is atrial fibrillation [3]. When the aorta is the site of thrombosis it is typically seen under conditions of hypercoagulability, atherosclerotic vascular changes, aneurysmal changes of the aorta, or intimal tear following trauma [4-6]. Primary aortic thrombosis is rare and typically occurs in the descending aorta, however, there are reports of thrombogenesis within the aortic arch [7,8]. These arterial thrombi can dislodge and cause severe vascular catastrophe in distal structures.

There is a paucity of literature regarding primary aortic thrombosis in the healthy aorta. In 1958, Gaylis described the primary aortic thrombus which he defined as thrombosis in an aorta with no identifiable gross underlying pathology [1]. Advancements in imaging technology has allowed for easier identification of arterial thromboembolic disease, as well as better insight into their exact etiology. Despite this, there is a lack of consensus in literature regarding the etiology and management of primary aortic thrombus. This may be in part the multifactorial and heterogenous nature of this condition [9].

A meta-analysis performed by Fayad., *et al*. on primary aortic mural thrombosis in the "normal aorta", included 200 patients from 98 articles and found the mean age to be 50 years [10]. Several of the papers with similar presentations of primary aortic thrombosis reported a mean or median age in the fourth decade of life, with ages ranged from 27 to 69 years [11-15]. This is an interesting finding given that age is an important risk predictor for arterial

thrombosis [16]. A retrospective study looking at 30 autopsied cases of aortic thrombosis in healthy aortas found the mean age to be 37 years, with the majority of thrombosis occurring in the third to fourth decades of life [9]. There is a wide spectrum of risk factors and comorbid conditions amongst individuals with primary aortic thrombosis. Fayad., *et al.* demonstrated the most common comorbidities to be hypertension (22%), insulin-dependent diabetes (11%), and dyslipidemia (11%). Hypercoagulability, malignancy, inflammatory bowel disease, and underlying hematologic disorders were present in 25%, 10%, 3% and 3% of patients, respectively. Additionally, 35% of patients had a history of smoking [10].

Treatment options are grossly divided into primary surgical intervention and nonsurgical management with anticoagulation. In the meta-analysis by Fayad., *et al*. their analysis favored surgical management. Their results demonstrated a significant increase in persistence or recurrence of aortic thrombosis, recurrence of peripheral arterial embolization, and major limb loss in the group treated primarily with anticoagulation when compared to those treated primarily with surgery. Non-significant reductions in mortality and complication rates were seen in the surgery group when compared with conservation management [10]. In a more recent study, 13 patients with identified thrombosis in a "healthy" aorta were compared based on outcomes of management approach. Six patients were treated with anticoagulation and had overall lower morbidities and reduced length of hospitalization compared to patients treated with primary surgical intervention [15]. Perhaps an agreed upon approach to management does not yet exist, not only due to the rarity of this disease, but also due to the variability and complexity of this disorder, and management should be tailored to each individual's presentation.

In the case we present, the patient's initial diagnostic evaluation revealed no clear etiology of thrombosis. Notably, he was involved in a motor vehicle accident 2 years prior to this admission, which could have produced an intimal tear serving as a lead point for thrombosis that was undetected. However, the prolonged time gap and anatomic location of the thrombus make this possibility less likely. Additionally, his cardiovascular risk factors support underlying atherosclerotic disease (ASO). Although his CTA did not show clear evidence of macro thoracic aorta ASO changes, there is always the possibility of non-identifiable microscopic atherosclerotic lesions contributing to thrombus formation in the aorta.

Our approach to management began with immediate anticoagulation combined with antiplatelet and statin therapy. However, he developed sudden onset acute limb ischemia requiring surgical intervention within one day of initiation anticoagulation therapy. This complication could be explained by the effects of heparin on thrombus stability/dissolution. He was kept inhouse for 5 days after initiation of anticoagulation to capture such complication. However, in the long run the anticoagulation in addition to antiplatelet and statin did help with thrombus regression and was successful to prevent its progression or further embolization incidence.

Conclusion

Isolated primary aortic thrombus in an otherwise healthy aorta is a rare condition. Due to its limited prevalence, an optimized approach to management is unclear. Although surgical management can be the preferred approach in suitable anatomy, recent case reports have found early detection and initiation of anticoagulation without surgical intervention to be effective in selective cases. In house monitoring for thrombus destabilization and distal embolization after initiation of anticoagulation is advised. However, the length of inhouse observation is not clear and further studies are needed.

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