In situ aortic thrombosis secondary to intra-abdominal abscess

Thomas D. Willson¹, Vijaya Rao², Francis J. Podbielski³, Matthew J. Blecha¹,³

¹ St. Joseph Hospital, Chicago, IL, U.S.A.
² Loyola University Medical Center, Maywood, IL, U.S.A.
³ University of Chicago Hospital, Chicago, IL, U.S.A.

Summary

Background: Abdominal aortic mural thrombus is uncommon in the absence of aneurysm or atherosclerosis.

Case Report: We report the case of a 46-year-old man who presented to our institution with perforated appendicitis for which he initially declined surgery. Four days after admission he ultimately consented to appendectomy and abdominal washout. Follow-up imaging to evaluate for intra-abdominal abscess revealed mural thrombus of the infra-renal abdominal aorta extending into the left iliac artery. This thrombus was not present on the admission CT scan. The patient had no clinical signs of limb ischemia. Conservative treatment with therapeutic anticoagulation resulted in resolution of the thrombus.

Conclusions: While portal, mesenteric, and major retroperitoneal venous thrombosis are well associated with major intra-abdominal infection and inflammatory bowel disease, aorto-iliac arterial thrombus formation in the absence of associated aneurysm, atherosclerosis or embolic source is exceedingly rare. We are unaware of other reports of in-situ aorto-iliac arterial thrombus formation secondary to perforated appendicitis.

key words: aorta • thrombosis • abdominal abscess

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Author’s address: Matthew J. Blecha, Department of Surgery, Saint Joseph Hospital, 2900 N Lake Shore Drive, Chicago, IL 60657, U.S.A., e-mail: matthew.blecha@yahoo.com
BACKGROUND

We report the case of asymptomatic in-situ aorto-iliac arterial thrombus formation in a patient with recent perforated appendicitis. The true prevalence of asymptomatic aortic mural thrombi is not known [1]. Most reported cases involve either atherosclerotic plaque or aneurysmal vessels. Primary mural thrombus is uncommon [2]. While generalized hypercoagulable states and vasculitis have been proposed as possible culprits, immunologic and genetic testing frequently fails to identify a specific cause [3–5]. While portal, mesenteric, and major retroperitoneal venous thrombosis are well associated with major intra-abdominal infection and inflammatory bowel disease [6,7], aorto-iliac arterial thrombus formation in the absence of associated aneurysm, atherosclerotic plaque, or proximal embolic source is exceedingly rare. We are unaware of other reports of in-situ aorto-iliac arterial thrombus formation secondary to perforated appendicitis.

CASE REPORT

A 46-year old otherwise healthy man was evaluated for one week of progressive abdominal pain without nausea, vomiting, diarrhea, fever, or chills. He also endorsed anorexia. Abdominal computed tomography revealed perforated appendicitis with abscess formation. The patient was placed on appropriate antibiotics and offered surgical management, which he declined at that time. Eventually he chose to proceed with surgery and underwent open appendectomy with abdominal washout. A postoperative CT scan was ultimately performed due to ongoing leukocytosis and abdominal pain after the washout. This revealed an intra-loop abscess as well as new clot in the distal aorta extending to the iliac arteries (Figure 1AB). This thrombus was not present on the patient’s admission CT scan.

The patient related no limb pain and there was no exam evidence of distal embolization. Arterial Doppler exam revealed normal bilateral triphasic arterial Doppler waveforms and ABI (Figure 2). Platelet count was 453,000. On detailed questioning, the patient had no prior history of limb swelling, claudication, or thromboembolism. There was no family history of hypercoagulability or bleeding diathesis. He did have a current, fifty pack-year smoking history. Transesophageal echocardiography demonstrated normal cardiac function and no evidence of atrial thrombus, valvular abnormalities, or vegetation. There were minimal vascular calcifications on computed tomography. Blood cultures were negative. In-situ thrombus formation rather than embolization was diagnosed given: the normal TEE; and, the location of thrombus burden being both at the anterior aortic wall well above the bifurcation as well as in the left common iliac artery.

The patient’s abscess resolved with repeat laparotomy and antibiotic treatment. He was placed on therapeutic anticoagulation and eventually transitioned to warfarin. At three month follow up, the thrombus had completely resolved (Figure 3). Anticoagulation was discontinued and the patient has had no evidence of recurrent thrombus or embolism at one-year. Hypercoagulable work was negative one month after discontinuing warfarin.

DISCUSSION

We are unaware of other reports of in-situ aorto-iliac arterial thrombus formation secondary to perforated appendicitis. We hypothesize that this patient developed retroperitoneal inflammation secondary to the intra-peritoneal infectious process with rare secondary intra-arterial thrombus development. While the actual incidence of aortic mural thrombus is unknown, Machleider demonstrated its presence in 0.45% of 10,671 patients in an autopsy study [4]. The true incidence is suspected to be much higher when factors such as post-mortem thrombosis and perimortem embolization are taken into account [5]. In Gagliardi’s series, aortic mural thrombus accounted for 3.8% of surgically treated nonaneurysmal aortoiliac lesions and 5% of peripheral arterial emboli [1].

The pathogenesis of nonocclusive aortic mural thrombus is not clear; surgical specimens reveal a wide spectrum of disease from micro- and macroscopically normal aortic walls to those with a ‘shaggy,’ atheromatous intima [5]. Both generalized hypercoagulability and vasculitis have been suggested.
as potential causes of this phenomenon. Indeed, hypercoagulable conditions may be present in up to 10% of vascular surgery patients [7]. In this population, however, hematology workup only occasionally identifies a specific syndromic cause [2,3,5]. A number of common factors have been identified in these patients. A substantial smoking history is common to many patients [9]. Oral contraceptive pills, which are associated with deep venous thrombosis, have also been associated with aortic mural thrombosis [9]. Recent heparinization, post-operative or post-catheterization state, and cardiac disease history have also been noted in association with mural thrombus [10]. Chronic inflammatory diseases including Crohn’s disease and rheumatoid arthritis have been associated with the condition, lending credence to systemic vasculitis as a potential culprit [2,11]. Interestingly, acute inflammatory conditions including Crohn’s flares and pancreatitis have also been seen in these patients [2]. The acute appendicitis seen in this case fits that category.

The most definitive treatment for aortic mural thrombus would target the underlying condition, if one exists. Until that is clarified, however, a number of options for management remain. There is little disagreement in the literature that anticoagulation is indicated, at least in the short term, for patients without an identifiable hypercoagulable condition and in the longer term for those who do. No arterial intervention was performed given the lack of symptoms and normal resting arterial hemodynamics in this patient.

Certainly in symptomatic cases of embolism, embolectomy or percutaneous mechanical thrombectomy is necessary for limb or organ salvage. A number of authors have, however, treated both symptomatic and asymptomatic patients with anticoagulation alone with good results [2,8,12–14]. Embolizing or hemodynamically significant aortic thrombus lesions should be treated with either open surgical approaches ranging from simple thrombectomy or thrombendarterectomy to bypass of the affected aortic segment [1,2,5,10]. Endovascular therapy has also been described with covered stent treatment for the embolizing source [6,15].

CONCLUSIONS

We suggest that asymptomatic individuals can be tried on a course of conservative therapy while patients with signs of limb ischemia require revascularization, consideration of removal of source thrombus, and postoperative anticoagulation. All patients presenting with aortic mural thrombus not without associated aneurysm or atherosclerotic plaque should undergo a hypercoagulable workup.

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