Penetrating Atheromatous Ulcer As A Precursor Of Aortic Dissection: A Case Report

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Case report

Keywords: Atheromatous ulcer, penetrating, aortic dissection

Posted Date: August 5th, 2020

DOI: https://doi.org/10.21203/rs.3.rs-47460/v1

Abstract

Background: Acute aortic syndromes include a range of life-threatening conditions with the most familiar entity being aortic dissection. However, variants of aortic dissection also include intimal tear without hematoma, aortic intramural hematoma, and lastly penetrating aortic ulcer (PAU), which will be the focus of this case report. Most PAUs are located in the descending thoracic aorta (85-95%), but they can also occur in the ascending aorta or arch as in the current case.

Case Presentation: We report a case of a 77 year old male who presented with chief complaint of intermittent right-handed weakness associated with no numbness or mental status changes. Patient was admitted for stroke workup with unrevealing findings on CT (computed tomography) for acute abnormalities or any hemodynamically significant stenosis on carotid ultrasound. CT angiogram of head/neck revealed a penetrating aortic ulcer of the lateral aspect of the mid to distal ascending aorta. Patient was then transferred for further evaluation to a center of higher level care for further management.

Conclusions: Patient was evaluated for surgical repair of penetrating ascending aortic ulcer. Patient underwent serial imaging throughout hospital course which showed grossly similar findings to prior examination and thus no surgical intervention was needed at that time. Patient was recommended to have follow up CT scan in one month to monitor progression of aortic ulcer, however patient lost to follow-up thereafter. Penetrating aortic ulcers are rarely located in the ascending aorta and are considered precursors of life-threatening aortic dissections.

Background

Acute aortic syndromes include a range of life-threatening conditions with the most familiar entity being aortic dissection. However, variants of aortic dissection also include intimal tear without hematoma, aortic intramural hematoma, and lastly penetrating aortic ulcer, which will be the focus of this case report. Penetrating aortic ulcers (PAU) occur when the ulceration of an aortic atherosclerotic lesion penetrates the internal elastic lamina into the media in varying amounts, over which there may or not be an overlying thrombus. PAUs may be associated with hematoma within the media and may progress to perforation or aortic dissection, and are known to be the initiating lesion in <5% of all aortic dissections. Most PAUs are located in the descending thoracic aorta (85-95%), but they can also occur in the ascending aorta or arch as in the current case.

Case Presentation

Acute aortic syndromes include a range of life-threatening conditions with the most well-known being aortic dissection. These syndromes most commonly cause a disruption in the layers of the aortic all, most often initiated by an intimal injury of sorts. The major classifications of aortic dissection begin with intimal tears without a hematoma, penetrating aortic ulcers, aortic intramural hematomas, and periaortic

hematomas which all become equally as important when considering severity of illness. Penetrating aortic ulcers occur when the ulceration of an aortic atherosclerotic lesion penetrates the internal elastic lamina into the media in varying degrees, over which there may or may not be an overlying thrombus[1]. PAUs may be associated with a hematoma within the media and may progress to perforation or aortic dissection, and are known to be the initiating lesion in <5% of all aortic dissections ever since they were first described[2]. Most penetrating aortic ulcers are located in the descending thoracic aorta (85-95%)[3], and are rarely located in the ascending aorta or arch.

The general presentation of PAU is similar to that of the aforementioned aortic dissection etiologies, however the radiographic evidence is somewhat unique. Today, PAU is best diagnosed by contrast-enhanced CT scanning demonstrating an outpouching ulcer crater, intimal calcification, and localized intramural hemorrhage in conjunction with severe atherosclerotic disease. PAU tends to have a predilection in older gentleman >70 years of age with significant cardiovascular comorbidities including hypertension, tobacco abuse, coronary artery disease, chronic obstructive lung disease, and renal insufficiency[4]. This is of particular interest as the patient being discussed in this case report illustrates someone who falls into multiple categories including age, cardiovascular comorbidities given his history of paroxysmal atrial fibrillation, previous abdominal aortic aneurysm repair, hyperlipidemia, peripheral vascular disease, and previous tobacco abuse likely resulting in compromised pulmonary function.

We report a case of a 77 year old male with significant PMH for paroxysmal atrial fibrillation (on Xarelto and amiodarone), abdominal aortic aneurysm (AAA) status post repair in 2012, right iliac artery aneurysm status post repair in 2018), chronic obstructive pulmonary disease (COPD), hyperlipidemia (HLD), peripheral vascular disease (PVD), and hypertension (HTN) who presented with a chief complaint of intermittent right-handed weakness. Patient had unrevealing findings on CT head for acute abnormalities or any hemodynamically significant stenosis on carotid US (Figure 1). Echo revealed severe concentric left ventricular hypertrophy (LVH) and ejection fraction (EF) of 55-60% with moderate diastolic dysfunction (Figures 2 and 3). CT angiogram of head/neck revealed an irregular mural hypoattentuation of the lateral aspect of the mid to distal ascending aorta and to which contrast extended (Figure 4). CT angiogram of the chest with and without contrast was then performed which demonstrated contrast extension into the anterior lateral aspect of the ascending thoracic aorta which likely represented a penetrating atheromatous ulcer measuring 0.9x0.4x1.3cm with no definite dissection flap identified (Figure 5). At this point, decision was made to transfer patient to a center of higher level care for further management.

Upon transferring, the patient was evaluated for surgical repair of PAU. Surgery was initially planned for ascending aortic replacement via minimally invasive sternotomy, however given >6% risk of major adverse cardiac events (MACE), significant smoking history, previous AAA rupture repair, and increased risk for surgical complications, more detailed imaging was obtained via CT-TAVR with gated technique to rule out change in size of PAU necessitating surgery. This study redemonstrated a focal PAU in the anterolateral ascending thoracic aorta unchanged in size. Left heart catheterization showed non-obstructive coronary artery disease (CAD) with aneurysmal right coronary artery (RCA). Patient was then

managed medically and started on dual antiplatelet therapy and high-dose statin with optimization of blood pressure control. Patient was discharged with recommendation to obtain serial CT scans. The first scan 4 weeks later and a second scan three months later both showed unchanged focal outpouching of ascending aorta concerning for penetrating ulcer. Patient will continue to obtain serial CT scans to monitor progression of ulcer and to determine if a change in size necessitates surgery.

Discussion And Conclusions

Penetrating aortic ulcers account for 2-7% of acute aortic syndromes, and thus it is crucial to recognize the predisposition of an aortic dissection upon a patient's presentation. Atheromatous ulcers develop in patients with advanced atherosclerosis. Initially, the lesions are confined to the intimal layer however they can penetrate through elastic intima and into the media. Once a penetrating atheromatous ulcer (PAU) is formed, the ulcer may remain quiescent or progress to acute processes like dissection or aneurysm formation. PAU's also tend to have a scarcity of symptoms which is in contrast to the classic symptoms associated with dissections including severe back or chest pain or aortic regurgitation[5]. Given this patient's risk factors and history of abdominal aortic aneurysm, right iliac artery aneurysm, hypertension, and hyperlipidemia, the threshold for further workup became even lower for this patient. The risk of aortic rupture is significantly higher in patients with PAU, and medical management often is ineffective in ascending aortic pathology and surgery is often indicated. Thus, PAUs are considered precursors of life-threatening aortic dissections, and are rarely located in the ascending aorta.

Life expectancy of patients affected by PAU has not been noted to exceed ten years after diagnosis, which reinstates the severity of disease in these patients[6, 7]. Patients have also been noted to have a concurrent abdominal aortic aneurysm in up to 60% of patients with abdominal PAU[8, 9]. Stanson et. al and Cooke et al. have both illustrated that PAU requires aggressive surgical treatment of symptomatic patients since conservative medical therapy had often times led to recurrence or worsening of symptoms and propensity to rupture[10,11]. An early study by the Yale group found that 40% of patients who were primarily treated conservatively needed emergency interventions for rupture[12]. The Stanford group again demonstrated that progression with PAU occurred in 48% of cases, and a strictly conservative regimen led to 10% mortality within a mean of 9.3 days[3]. These findings were accepted as the mainstay of treatment unless the risk associated with a patient's comorbidities outweighed the benefit of surgical intervention. These conclusions support the recommendation for surgery. However, in some cases, studies have also shown that many PAU have been managed non-operatively. Hussain et. al argues that a confined intramural hematoma is not exposed to the turbulent aortic flow that one might find in a double lumen and thus potentially more likely to remain stable[13]. Kazerooni et. al describes patients with typical symptoms that had abnormal imaging including chest radiograph showing an enlarged thoracic aorta, and CT scans showing intramural hematoma (n = 16), focal ulcer (n = 15), displaced intimal calcification (n = 13), pleural and/or extrapleural fluid (n = 7), mediastinal fluid (n = 4), and a thick or enhancing aortic wall (n = 6). The study demonstrated that by comparing films and treating patients surgically (n=7) and conservatively (n=9), eight of the nine patients treated conservatively were asymptomatic after

appropriate control of blood pressure[14]. However, it is still important to determine the threshold of an aortic ulcer being present and the verge of it dissecting, which becomes solely surgical when a patient is hemodynamically unstable.

The risk stratification of the presented patient required serial imaging to quantify the rate of progression, if any, as well as determining the etiology and method of resolution of the patient's right hand weakness. Risk factors for progression are thought to be of various groupings, including simply symptom onset, as well as depth and diameter of PAU. Ganaha et al. identified both the maximum PAU diameter and maximum PAU depth (21.1±8.0mm and 13.7± 4.2mm), as well as rapid increase of aortic diameter and hemodynamic instability as indications for immediate surgery[3]. Given our patient's risk of MACE and comorbidities, it was extremely integral to weigh out advantages and disadvantages. Patient's resolution of symptoms did not necessarily imply lesion stability, and thus it was integral for the patient to have follow up imaging to assess for worsening disease as described above. While the patient was admitted in the intensive care unit (ICU), serial imaging did not show progression of PAU and thus the recommended interval of follow up imaging was prolonged upon discharge from the hospital. The most recent interval for imaging has been prolonged to one year, with most recent CT angiogram of the chest in March 2020 again showing a focal outpouching in ascending thoracic aorta. Patient will return for follow up imaging in March 2021.

If this patient did not have the comorbidities they did, surgical intervention would have prevailed as the primary treatment of choice. Thoracic endovascular aortic repair (TEVAR) is especially effective as an intervention modality. This procedure usually involves a stent delivered through a sheath introduced into the common femoral artery or common iliac artery into the abdominal aorta and further into the level of the thoracic aorta. Brinster et al. showed that this approach in 21 patients all presenting with uncontrolled pain associated with PAU or radiologic evidence of impending rupture, or both. Sixteen of these patients had an acute presentation with symptoms being present for less than fourteen days[15]. All patients treated with endoluminal graft placement had 100% relief of their symptoms with no operative mortalities. This is especially notable since many patients who have previously been managed conservatively often can have disease progression. Conservative management involves adequate blood pressure control with normalization and left ventricular ejection fraction (LVEF) reduction as they are the main determinants of dissection extension and rupture. Beta blockers have been shown to decrease mortality from 67% to 95%[16], and for this reason are usually the agents of choice when initiating blood pressure management or titrating medications.

The patient presented in this case report was one of the 5-15% of penetrating aortic ulcers located in the ascending aorta. This is a rare finding, and management remained non-conservative after close following.

Abbreviations

Penetrating aortic ulcer (PAU)

Abdominal aortic aneurysm (AAA)

Chronic obstructive pulmonary disease (COPD)

Hyperlipidemia (HLD)

Peripheral vascular disease (PVD)

Hypertension (HTN)

Left ventricular hypertrophy (LVH)

Ejection fraction (EF)

Major adverse cardiac events (MACE)

Coronary artery disease (CAD)

Right coronary artery (RCA)

Intensive care unit (ICU)

Thoracic endovascular aortic repair (TEVAR)

Left ventricular ejection fraction (LVEF)

Declarations

Ethics approval and consent to participate: Not applicable.

Consent for publication: Informed consent was obtained from the patient/daughter for publication of this case report and accompanying images.

Availability of data and materials: Not applicable.

Competing interest: The authors declare that they have no competing interests.

Funding: The authors declare that they were not provided any funding for this paper.

Authors' contributions: PS contributed to project conception, acquisition and analysis of data, and preparing the manuscript by writing the draft. She also contributed to review and revisions. Patient contact, management of the patient while inpatient prior to transfer was handled by PS. EP contributed to the project by serving as project supervisor as the attending physician taking care of the patient. He also helped prepare the manuscript, specifically critical review, commentary and revisions, and final approval of the version to be published. Both authors, PS and EP, read and approved the final manuscript.

Acknowledgements: Not applicable.

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Figures

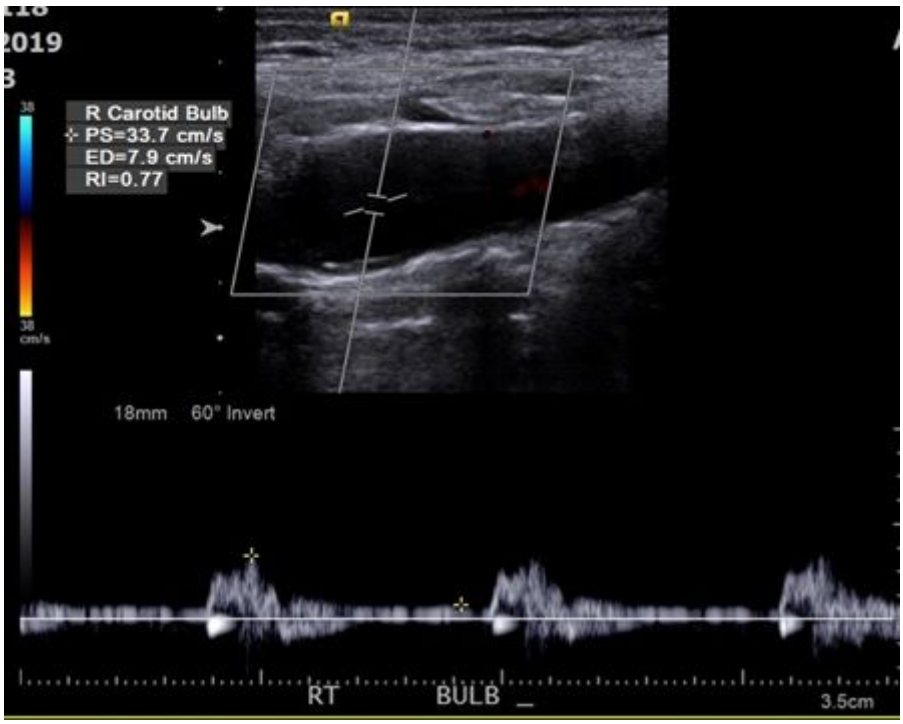

Figure 1

Patient had unrevealing findings on CT head for acute abnormalities or any hemodynamically significant stenosis on carotid US

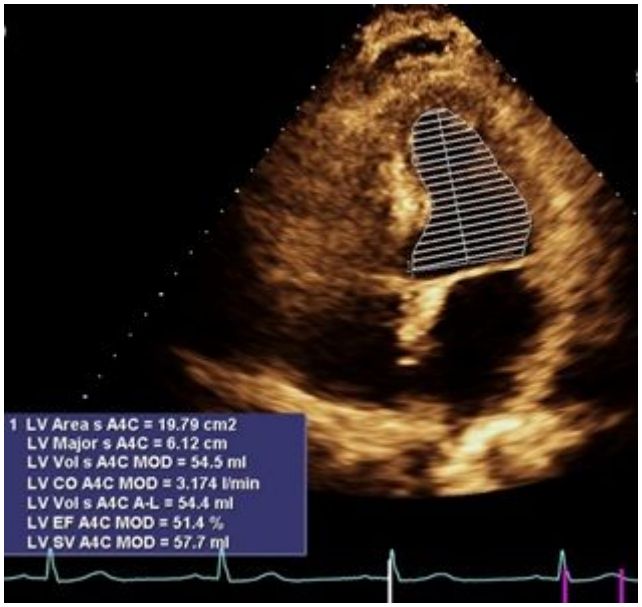

Figure 2

Echo revealed severe concentric left ventricular hypertrophy (LVH) and ejection fraction (EF) of 55-60% with moderate diastolic dysfunction

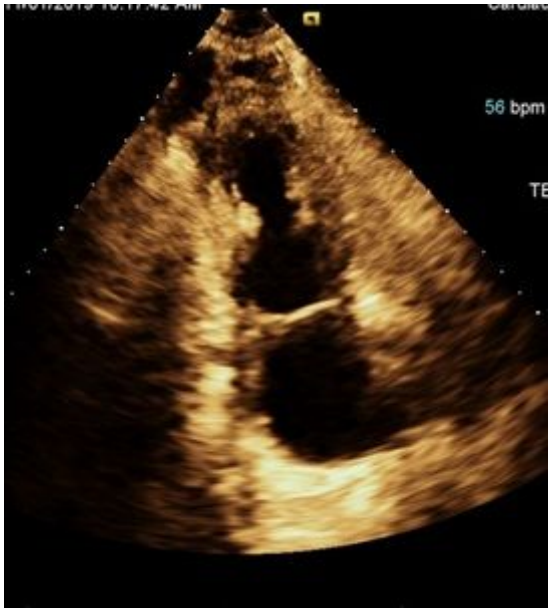

Figure 3

Echo revealed severe concentric left ventricular hypertrophy (LVH) and ejection fraction (EF) of 55-60% with moderate diastolic dysfunction

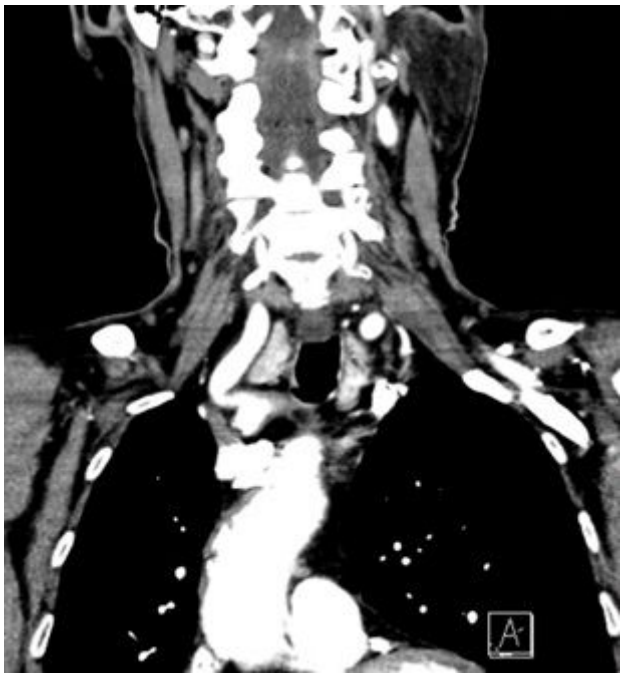

Figure 4

CT angiogram of head/neck revealed an irregular mural hypoattentuation of the lateral aspect of the mid to distal ascending aorta and to which contrast extended.

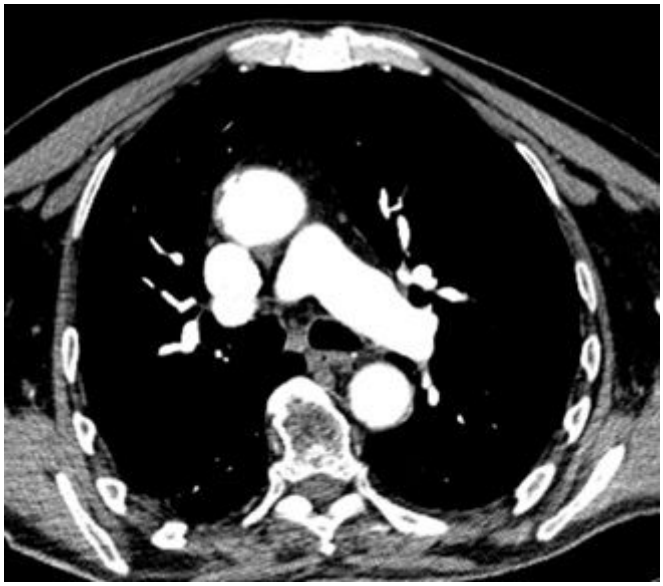

Figure 5

Contrast extension into anterior lateral aspect of ascending thoracic aorta representing PAU measuring 0.9 x 0.4 x 1.3 cm.

Supplementary Files

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- PAUCAREChecklistPDFTIMELINE.docx