

Ruptured Pneumococcal Aortic Aneurysm Presenting as ST-Elevation Myocardial Infarction

Case Report and Literature Review

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Abstract

Ruptured mycotic aneurysms occur infrequently in current clinical practice, and a pneumococcal etiology is even more rare. This case report describes a patient who initially presented with catheter lab activation for an acute ST-elevation myocardial infarction, receiving a full Plavix load. She was subsequently found to have a ruptured aortic aneurysm and underwent emergency surgical repair, with intraoperative findings of an aorta seeded with *Streptococcus pneumoniae*. A retrospective evaluation of her history revealed clues of a previous upper respiratory infection and long-standing back pain. The subsequent literature review summarizes presentations and outcomes in previously reported, ruptured pneumococcal aneurysms and describes the relatively common occurrence of aortic conditions masquerading as acute myocardial infarctions. We provide recommendations to help approach similar situations in the future.

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Key Words

Mycotic • Aneurysm • STEMI • Streptococcal • Rupture

Introduction

Rapid diagnosis of myocardial infarction is vital for optimal treatment and patient outcome [1]. As such, in order to minimize door-to-balloon times, emergency medical services (EMS) are encouraged to

perform and evaluate electrocardiograms to assist in pre-hospital activation of the “cath lab” if suspicion is strong enough for an ST-segment elevation myocardial infarction (STEMI) [2]. However, in a small but significant number of patients, suspected STEMI mask other disorders. In those cases, routine myocardial infarction management can be dangerous and cause catastrophic outcomes. We describe a unique case of a ruptured mycotic aortic aneurysm due to *Streptococcus pneumoniae* presenting with STEMI, which led to the patient receiving antiplatelets and anticoagulants before being sent to the operating room for emergency aneurysm repair.

Case Report

The patient was a 56-year-old African American woman brought in by EMS from home for mid-sternal chest pain, with cath lab activation from the field for ST elevation in V4-V6 and Q waves in the inferior wall. She reported a several month history of intermittent chest pain with multiple evaluations, but her current episode began the night before with associated dyspnea, nausea, and back pain worse on inspiration. She otherwise denied fever, syncope, cough, abdominal pain, or vomiting. Her medical history was pertinent for HIV with unknown CD4 count and viral load, hypertension, ½ pack per day smoking for the past 4



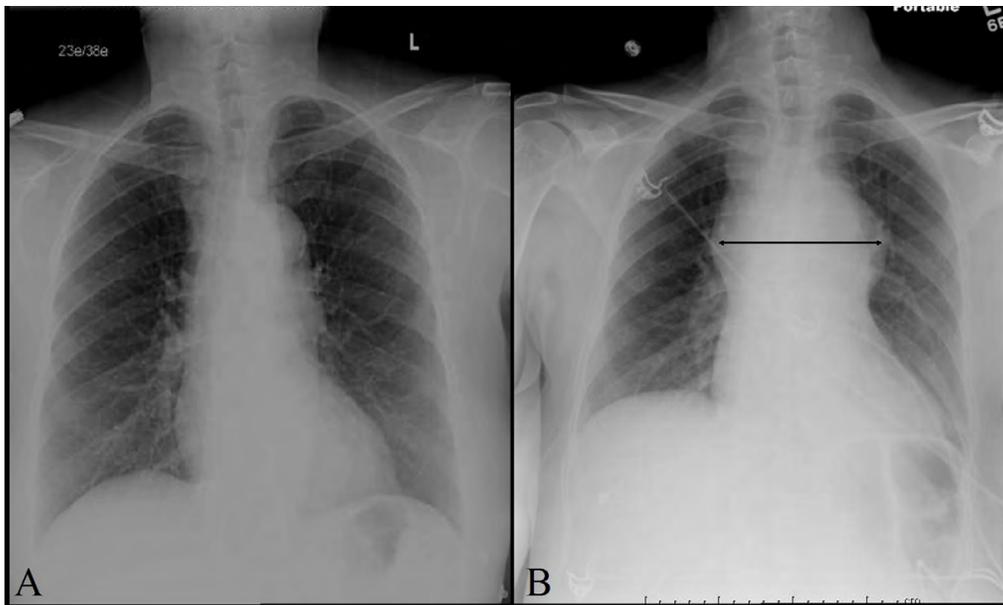


Figure 1. Chest plain film revealed new widening of the mediastinum (B, double-headed arrow) compared to three weeks prior (A).

years, active crack cocaine use approximately once a week and no alcohol consumption.

On presentation to the resuscitation bay, her vitals were within normal limits except for a blood pressure of 96/61 and a respiratory rate of 22. She was in mild distress but cooperative and her exam was notable only for distended jugular veins. Her labs showed an elevated white blood cell count of $13,800/\text{mm}^3$ with left shift, mild anemia with hemoglobin of 11.3g/dl and a hematocrit of 33.9%, and negative troponins. Her electrocardiogram (ECG) confirmed ST-elevation in leads II, III, and aVF and the patient received a Plavix load of 600 mg and aspirin and was started on a heparin infusion with PTT aimed for between 60 to 80 seconds in preparation for coronary angiogram. As she was waiting to go to the cath lab, chest radiograph revealed a widened mediastinum (Figure 1).

The patient was emergently sent for computed tomography with angiogram (CTA) as per dissection protocol, which revealed a pseudoaneurysm of the aorta and a large mediastinal hematoma (Figure 2). There were also prominent lymph nodes in the chest and nonspecific hypodensities in the spleen raising the possibility of mycotic disease. She was emergently transferred to the operating room (OR) for resection and repair of the ruptured aneurysm.

The patient was put on bypass via groin vessel cannulation with systemic cooling prior to sternal opening. After opening the sternum, the ruptured aneurysm was immediately evident even without dissection of the mediastinal fat. Operative findings included a large intrapericardial ruptured ascending aortic and arch aneurysm involving the bovine arch. The patient's dynamic ECG changes could be attributed to physiologic compromise of coronary ostial blood flow by the aneurysm itself. Transesophageal echocardiogram did not show evidence of vegetation or endocarditis on any of the heart valves. The rest of the aorta was normal in caliber.

The entire arch and the ascending aorta up to the sinotubular junction were resected and replaced with a Hemashield Platinum graft under deep hypothermic arrest, and the arch vessels were implanted as an island. Due to the saccular nature of the aneurysm with anterior projection, OR cultures and pathology of the resected tissue were sent to the lab. The wound culture was eventually positive for *S. pneumonia*, and the patient was treated with intravenous ceftriaxone. She was discharged 2 weeks later on four more weeks of intravenous antibiotics coverage (for a total of 6 weeks of ceftriaxone, 2 grams daily). At last follow up 4 months after the inciting event, the patient remained asymptomatic.

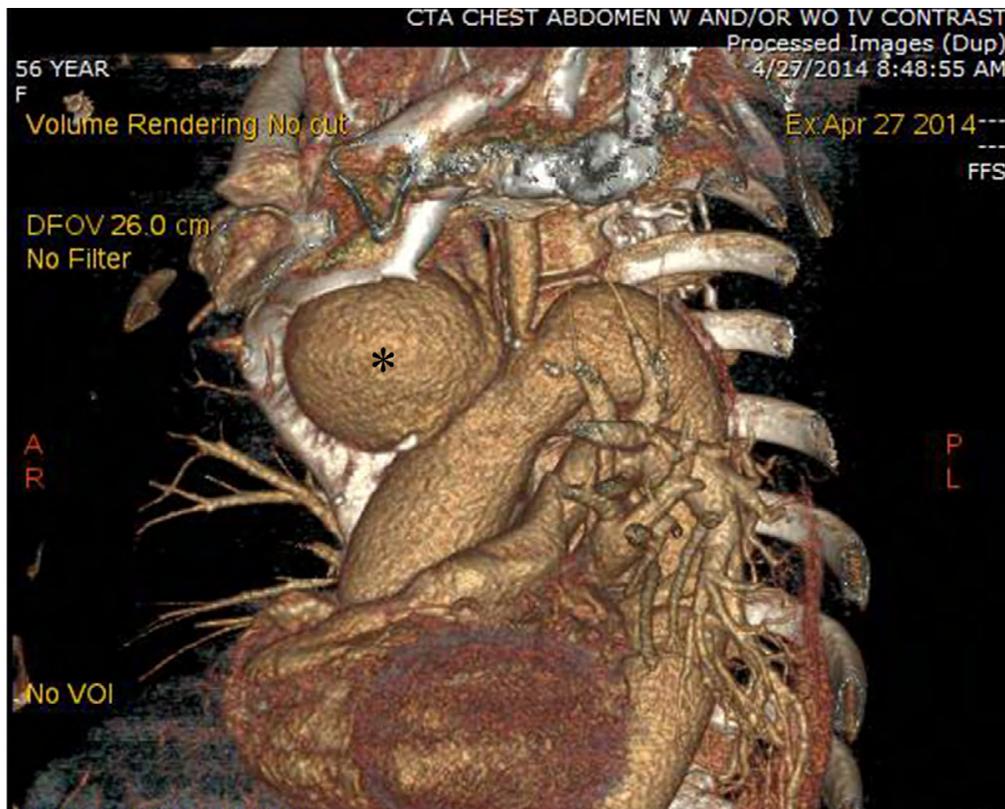


Figure 2. Emergency CTA showed a 5.2cm by 4.2cm pseudoaneurysm (*) arising at the origin of the common trunk of the right brachiocephalic and left common carotid arteries.

Literature Review

Between 1.4 to 15% of patients presenting with acute chest pain with STE do not have an acute MI (AMI) [3] and approximately 1 to 3% of aortic aneurysms are of mycotic etiology [4-6]. We report a unique case of a combination of these two relatively rare occurrences in a ruptured *S. pneumoniae* mycotic aortic aneurysm presenting as a STEMI.

Commonly described conditions misdiagnosed as AMI generally fall into four categories: (1) transient STE from acute coronary syndrome, (2) cardiac conditions not affecting the coronary arteries such as peri- or myocarditis, (3) vascular events including pulmonary emboli and aortic dissections, and (4) other etiologies like acute cholecystitis or pancreatitis [3]. The pathophysiological processes of these conditions may manifest with cardiac symptoms, such as when coronary ostia are compressed during a dissection or when inflammation mimics AMI via viral infections,

immunological change, or induced platelet abnormalities [7, 8]. In the pre-antibiotic era, this mimicry may have been more common as infected aneurysms were overwhelmingly due to endocarditis, with a smaller subset being due to lung and bone infections. The most commonly perpetrated organism was non-hemolytic *Streptococcus* although *Staphylococcus*, *Pneumococcus*, and *Gonococcus* also were implicated. Another common pre-antibiotic era mycotic aneurysms included syphilitic mycotic aneurysms. Aneurysm formation was described at the time to be either due to the lodging of infected emboli into the vessel walls or a contiguous infection from affected valves [9].

With the onset of antibiotic use and decreased rates of endocarditis, the incidence of mycotic aneurysms is decreasing and the microbiological profile is changing. *Staphylococcus aureus*, *Salmonella* peripheral and other Gram-negative organisms are now more frequently seen [10-13]. However, the clas-

sification of mycotic aneurysms remains basically the same: primary bacteremic seeding (such as from endocarditis, pneumonia, and cellulitis) to a weakened vessel wall, secondary traumatic inoculation (such as from intravenous drug use), and contiguous infection from a nearby source [5, 14]. *S. pneumoniae* in particular presently accounts for approximately 9-36% of all mycotic aneurysms [4, 12, 15, 16]; Cartery et al. [14] found 52 cases reported in the literature from 1924 to 2007, and we found an additional 16 cases since 2007 [6, 16-23]. Perhaps not surprisingly, the majority of these patients have an antecedent history compatible with a lower respiratory infection in the near past [12, 14, 24].

Compared to typical aortic aneurysms, the tendency for a mycotic aneurysm to rupture is substantially higher, with sources citing a risk between 34% and 83% [6, 12, 16, 22, 25]. In our review, 12 patients since 1923, including our current case, had a ruptured *S. pneumoniae* aneurysm [10, 13, 14, 17, 19, 20, 25-28]. These aneurysms were located in the infrarenal abdominal aorta in five patients (41.7%), the descending aorta in three (25%), an unspecified abdominal aortic location in two (16.7%), and multiple locations including descending thoracic, suprarenal abdominal, and common iliac in one (8.3%). The case we described is the only reported such aneurysm of the aortic arch (Table 1). All patients had a recent history of infection that provided a primary source for the aneurysm and a nidus for an inflammatory response which may lead to mimicry of AMI. Thus, patients with ruptured mycotic aortic aneurysms in the right location can be misdiagnosed as AMI due to both coronary ostia compression and mimicry.

The elevated rupture rate may partially account for the high mortality of 14% to 75% associated with mycotic aneurysms [4, 15, 16, 21], although this patient population tends to have a frailer baseline; up to 60% of the patients have an immunocompromising condition such as a history of prolonged steroid or immunosuppressive agent use, alcoholism, irradiation, chronic renal disease, diabetes, or malignant illness [4, 16]. In the 12 patients we described, all those with listed medical histories were immunocompromised, further suggesting atypical presentations of illnesses.

The literature lists frequent presenting symptoms as fever, abdominal pain, back pain, and palpable

pulsatile masses in the case of abdominal aortic aneurysms [15, 24, 25]. Of the patients we described, 72.7% (8/11) complained of back or flank pain, 54.5% (6/11) had a fever, and 36.3% (4/11) had abdominal pain. These symptoms had often been worsening for several weeks before presentation. Of note, our case was the only patient presenting with chest pain or shortness of breath. Diagnostically, the results obtained in these 12 patients agreed with those found in the literature, where increased markers of inflammation such as C-reactive protein (CRP) were seen in 50% and leukocytosis in 61.5% of cases [16]. For imaging, 80% (8/10) of patients had CT scans, all of which were diagnostic; of the two patients without CT, one had a positive MRI. Two of three patients had concerning findings on chest radiography, and TTE did not elucidate any abnormality in either of the two patients in whom it was performed. Other than our case presentation, electrocardiographic results were not noted for any patient.

Treatment of mycotic aortic aneurysms requires prompt surgical involvement, as sole medical management with antibiotics is almost inevitably fatal [14, 19, 23]. After surgical debridement and resection, generally accepted guidelines are for appropriate antibiotic treatment intravenously for at least 6 to 12 weeks until blood cultures clear [14]. There is little consensus for long-term oral antibiotic treatment but it has been suggested that erythrocyte sedimentation rate or CRP could be used to both guide response to and duration of antibiotic treatment [12, 25]. In our described patients, all but one underwent open surgery with resection and/or graft, as well as intravenous antibiotic treatment for 1 to 8 weeks. Of the survivors, 62.5% (5/8) continued oral antibiotics from 6 weeks to lifelong treatment after that. With an aggressive approach, survival is purported to reach 100%, compared to 62.5% prior to 1998 [14]. In these 11 cases post-2000, survival was 73% to discharge; however, in two of the three patients who died, surgical intervention had been delayed.

Conclusions and Recommendations

In this reported case, it was fortuitous that there was delay in communication with the catheter lab

Table 1. Characteristics of patients in literature with ruptured *S. pneumoniae* mycotic aortic aneurysms.

Year	Age/ Sex	Significant Med- ical History	Preceding Symp- toms	Presenting Symptoms	Infection Source	Aneurysm Location	Diagnostics	Treatment (in order performed)	Result
1988 [10]	51/M	N/A	"Flu-like illness" x 2 wk PTA, new onset DM	Lower back pain, abdominal pain, myalgias	Possible PNA	Abdominal	N/A	N/A	Died
2000 [13]	61/M	N/A	New onset AF, thyrotoxicosis	Lower back pain, fever, diffi- culty urinating x 4 wk	Mesenter- ic abscess	Infrarenal abdominal	+ CT ↑WBC	Resection Bypass IV abx (2 wk) Oral abx (24 wk)	Alive
2001 [25]	61/F	Smoking, alcohol abuse, HTN	PNA x 6 wk PTA	Low back pain x 1 wk	Possible PNA	Descending thoracic	+ CT ↑WBC	Dacron graft IV abx (8 wk) Oral abx (8 wk)	Alive
2001 [25]	67/F	MI, AF, MM, IPF	Rectovaginal fis- tula and pneumo- coccal meningitis x 8 wk PTA	Sudden onset abdominal and back pain	Possible menin- gitis	Descending thoracic	+ CT ↑WBC	Dacron graft IV abx (3 wk) Oral abx (6 wk)	Alive
2002 [28]	65/M	N/A	Meningitis x 2 years PTA	Fever, loss of consciousness	Menin- gitis	Descending thoracic	+ CXR, + CT ↑WBC, ↑CRP	IV abx Dacron graft Wedge lung resection	Alive
2003 [26]	52/M	COPD, alcohol abuse	PNA x 4 wk PTA	Flank pain, fever, dysuria, anorexia	Possible PNA	Descending thoracic, suprarenal abdominal, common iliac	- CXR, -TTE, + MRI ↑WBC	Dacron graft IV abx (6 wk) PO abx (life)	Alive
2007 [27]	65/M	N/A	PNA x 32 wk PTA	Hematemesis, abdominal pain, back pain x 8 wk	Possible PNA	Infrarenal abdominal	- US abdo- men, - Bone scan	Graft IV abx (1 wk)	Died
2010 [17]	77/F	DM, HTN, rectal cancer	N/A	N/A	N/A	Infrarenal abdominal	N/A	PTFE graft IV abx (5 days) PO abx (12 wk)	Alive
2010 [19]	59/M	N/A	N/A	Lower abdomi- nal pain, fever	N/A	Infrarenal abdominal	+ CT ↑WBC, ↑CRP	Declined surgery IV abx (5 wk)	Re- rupture
2011 [14]	75/M	Alcohol abuse, DM, HTN, alcohol-induced CM	Hypogastric pain x 3 wk PTA. Newly diagnosed bladder cancer.	Fever, pelvic pain, weight loss	PNA	Infrarenal abdominal	+ CT ↑ESR, ↑CRP	IV abx (1 wk) Surgical resection	Died
2011 [20]	63/M	DM, smoking	Fever without complications 8 wk PTA	Back pain, fever	N/A	Abdominal	- CT, + PET- CT, - TTE, - TEE, + CTA ↑WBC, ↑ESR, ↑CRP	Oral abx (5 days) IV abx (10 days) Dacron graft with liga- tion of renal arteries	Died
2014	56/F	HTN, HIV, drug abuse	Intermittent chest pain x several months	Chest pain, back pain, shortness of breath x 4 wk	Possible PNA	Aortic arch	+ ECG, + CXR, + CT ↑WBC	Graft IV abx (5 wk)	Alive

N/A, not applicable; DM, diabetes mellitus; AF, atrial fibrillation; MM, multiple myeloma; IPF, idiopathic pulmonary fibrosis; COPD, chronic obstructive pulmonary disease; CM, cardiomyopathy; HIV, human immunodeficiency virus; PNA, pneumonia; PTA, prior to admission; wk, week(s); CT, computed tomography; CXR, chest x-ray; TTE, transthoracic echocardiography; MRI, magnetic resonance imaging; US, ultrasound; PET, positron emission tomography; TEE, transe-sophageal echocardiography; CTA, CT angiography; WBC, white blood cell count; CRP, C-reactive protein; ESR, erythrocyte sedimentation rate; +, diagnostic results; -, normal; IV, intravenous; abx, antibiotics; PTFE, polytetrafluoroethylene (Gore Medical, Flagstaff, Arizona, USA).

upon patient arrival to the emergency department allowing for a chest radiograph to be obtained, albeit after full loading with antiplatelet agents. Had the patient received additional antiplatelets or anticoagulants, or had she undergone catheterization, her outcome likely would have been fatal. It has previously been reported that demographically similar patients with aortic dissections who were misdiagnosed as having AMI had a mortality rate double those with typical Type A dissections (36% vs 17%, respectively) [29].

On reflection, the patient's presentation was somewhat odd despite the classic STE seen on ECG; particularly, her relatively short past medical history and description of back pain worse on inspiration should prompt further workup. In one study of patients referred for primary percutaneous coronary intervention (PCI) for suspected STEMI who had a final non-MI diagnosis, those misdiagnosed individuals less often had traditional cardiac risk factors like smoking, symptoms of angina, or family history of cardiac disease [3]. Moreover, her history of chest pain for several months would have been important to indicate that this was not a conventional STEMI.

It is evident that there needs to be a balance between speed and safety in diagnosis, especially for immunocompromised patients who tend to have atypical presentations for every disease process. Controversy exists over the evaluation and diagnostic algorithm for these patients. The majority of research is on misdiagnosed aortic dissections, likely because of the devastating effect of mainstream MI treatment in those patients, but the results may still be applicable.

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Some investigators suggest using TTE and D-dimer as screening tests if there is a concern for possible dissection [29], although neither of the two performed TTEs showed any pathology in the patients we described. Other authorities are more specific, suggesting aortic imaging in either those with STEMI and symptoms suggestive of an aortic dissection, or all non-STEMI cases because dissection is statistically more likely to occur with nonspecific ECG changes [8]. However, as classic symptoms often do not occur, more individualized recommendations are for a multimodal imaging approach with CT and ECG to evaluate patients with dynamic ST abnormalities that are suspicious for intermittent coronary artery occlusions [30].

Our review further shows that patients with mycotic aortic aneurysms tend to have heightened leukocyte counts and markers of inflammation, both of which are easily and quickly obtained via point-of-care testing and can help triage patients towards an infectious etiology. Finally, it is important to note that patients uniformly report a recent infectious event and that taking a good history overrides all diagnostic testing.

Conflict of Interest

The authors have no conflict of interest relevant to this publication.

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EDITOR'S COMMENTS

Ion S. Jovin, MD, Department of Medicine, Virginia Commonwealth University and McGuire VAMC, Richmond, VA, USA.

Reminding ourselves that uncommon things are uncommon

The report by Guo and Bonde presents a case of

a patient with ruptured mycotic aneurysm of the ascending aorta caused by *Streptococcus pneumoniae* presenting as an acute ST segment elevation myocardial infarction (STEMI). The authors then review the literature and present 12 other patients from other papers who presented with ruptured aortic aneurysms caused by the organism.

The report is interesting but raises several controversial issues: The main issue is that, while the authors recognize the difficulty of distinguishing entities that present with ST segment elevations on ECG yet are not true acute coronary syndromes from STEMI, they do not put matters adequately into perspective in terms of the relative (in)frequency of ruptured aortic aneurysms presenting as STEMI. Thus, their discussion does not account for the “needle in a haystack” nature of this case. Perhaps going into more detail on this aspect would help the reader interested in the topic. Moreover, while the authors state that between 1.4 and 15% of patients presenting with ST elevations on ECG do not have an acute myocardial infarction, they reference a paper from the Netherlands that reported only a 2.3% rate of patients presenting with ST elevations and taken for primary percutaneous intervention who did not have a myocardial infarction, among 820 consecutive patients. Of note, less than 1% of patients had aortic dissections [1]. Also, the authors do not discuss the feasibility of investigating such patients for alternative causes before they are taken emergently to the cardiac catheterization laboratory, a proven contemporary strategy that is associated with the best outcomes [2].

Moreover, the authors claim in the abstract that they “provide recommendations for ensuring a similar situation is not misdiagnosed in the future”. It would be more useful to provide a discussion of how

the large number of STEMI and the improved outcomes of patients treated with a primary PCI constitute a very powerful argument for striving for a short door-to-balloon time. The cost to pay for a strategy aimed at accomplishing the likely unattainable goal of eliminating all misdiagnoses of rare masquerading conditions may be the late diagnosis of the very frequent patients with true coronary syndromes presenting with appropriate features of STEMI. It would be unrealistic to expect all patients presenting with a typical coronary picture to have full laboratory studies including white cell count and CRP as well as chest X rays and/or computed tomographic angiography before deciding to activate the cardiac catheterization laboratory; such a requirement would significantly prolong door-to-balloon time, which is one of the important determinants of the outcomes of patients with STEMI.

Whether the patient described in this report got her chest X ray because of a delay in activating the cardiac catheterization laboratory or because the emergency room clinician had a clinical suspicion based on subtle atypical features of the patient is not clear. Ultimately, though, the authors’ suggested strategy to investigate every patient fully with the goal to avoid very rare misdiagnoses of unusual conditions is difficult to justify in terms of its risk : benefit ratio and thus not feasible in real life .

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