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Case Report

Rapidly Expanding Infected Aortic Aneurysm Caused by Haemophilus Influenzae In a 51-year-old HIV- Positive Patient

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Abstract

Haemophilus influenzae has infrequently been reported to cause mycotic aneurysm formation. In this report, we describe an HIV patient who developed a rapidly expanding mycotic aneurysm caused by *H. influenzae* type f. She had presented with back pain only. Clinicians must have a high index of suspicion for mycotic aneurysm development in immune compromised patients who present with non-specific abdominal or back pain only. In addition to describing the diagnosis and treatment of this rare case we include what we believe to be the first comprehensive literature review on the subject.

Case Report

A 51-year-old Haitian female with a past medical history of HIV, hypertension, and breast cancer presented to the hospital complaining of a four day history of worsening back pain. She denied any constitutional symptoms. She had presented 6 months prior to this visit for abdominal pain and a computed tomography (CT) scan had demonstrated a normal appearing aorta without evidence of other intra-abdominal pathology (Figure 1a).

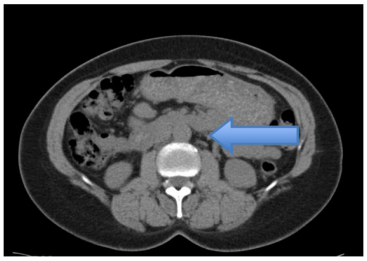

Figure 1a: August 2013 CT Scan Abdomen/Pelvis, no contrast.

She had traveled to Haiti also around that time without report of an illness or any sick contacts. The abdominal exam was limited due to her obese habitus, however, it revealed no palpable or pulsatile masses. Laboratory analysis revealed an inflammatory/infectious process with a white blood cell (WBC) count of 12,000 (reference range: 4.00-11.0 x10^9/L), C-reactive protein (CRP) level of 137 mg/L (reference range: 0-10 mg/L) and an erythrocyte sedimentation rate (ESR) level of greater than 145(reference range: 0- 29 mm/hr for women). A repeat CT scan showed a 5.3 x 5.2 cm saccular infrarenal aortic aneurysm (Figure 1b, 1c).

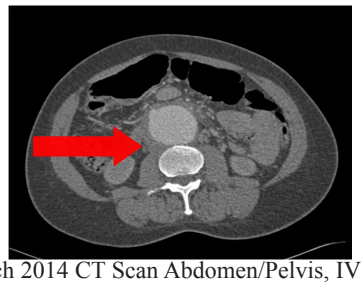

Figure 1b: March 2014 CT Scan Abdomen/Pelvis, IV contrast, axial image.

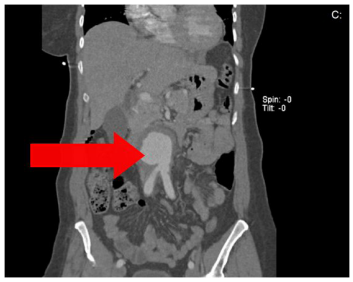

Figure 1c: March 2014 CT Scan Abdomen/Pelvis, IV contrast, coronal image.

Blood cultures were positive for *H. influenzae* serotype f, beta lactamase positive. The patient was admitted to the Intensive Care Unit and operative intervention was planned.

The patient underwent an excision of the aneurysm and in situ reconstruction with a CryoGraft^{R} (CryoLife Inc.). Using the transperitoneal approach, the aorta was exposed and no purulence was noted. The right-hand side of the aneurysm wall had a noticeably weak area in a focal segment indicating impending rupture. Certain portions of the aorta were thickened and appeared to have an associated rind. Pathology of the aortic wall revealed severely inflamed aneurysmal tissue consistent with mycotic aneurysm and small amounts of atheromatous material. Cultures of the pathology specimen further demonstrated *H. influenzae*. Latex agglutination as well as PCR- based capsular typing confirmed type f.

Postoperatively, the patient developed acute renal failure; however, in subsequent days her creatinine and urine output had returned to baseline and she was discharged from the hospital on antibiotics indefinitely. On postoperative visits the patient was doing well with a return to baseline activities and no evidence of aneurysmal expansion or infection.

Discussion

The earliest description of the concept, secondarily infected arterial disease, appeared in 1853 by Tufnell [1]. It was not until 1885 that the basic contribution in the evolution of the concept was made by Osler with his introduction of the term "mycotic" to describe an infected aneurysm [2]. Mycotic aneurysms have long been attributed to the presence of subacute bacterial endocarditis and septic embolization. This relationship and the resultant inflammatory arteritis were believed to be the sole cause of the formation of mycotic aneurysms. In 1937, Crane described the term, "primary mycotic aneurysm" to identify those lesions in the absence of endocarditis [3]. Since that time, the definition of mycotic aneurysms has expanded to include any infected aneurysm.

More recently, mycotic aneurysms remain a rare entity, but are becoming more common because of the increasing incidence of invasive procedures, vascular prostheses, intravenous drug use, and immunosuppression as in our case [4]. According to Lopes and colleagues, the incidence of this disease represents between 0.9 and 2.6% of aortic aneurysms [5,6]. Mycotic aneurysms develop primarily in the abdomen, with only one case being reported in the literature from the thoracic aorta [25] (Table 1).

Reference, Year	Age, Sex	Location	Serotype	Predisposing condition
1994- Adlakha et al.	72, F	Infrarenal Aorta	Type f	Unspecified
2010- Takashi et al.	59, M	Descending Thoracic Aorta	Type f	Vertebral osteomyelitis
2013- Wheeler et al.	58, M	Infrarenal Aorta, Right Common Iliac	Type f	Unspecified
2013- Saurez et al.	58, M	Infrarenal Aorta, Right Common Iliac	Type f	Unspecified
Our Case	51, F	Infrarenal Aorta	Type f	HIV

Table 1: Review of published cases of mycotic aneurysm due to *H. Influenzae* type f.

It is predominately a male disease and the average age is in the sixth decade [6-9]. Infection generating from a healthy aorta is exceedingly rare due to the natural resistance of the intima [6-9]. The pathophysiology is centered on the hypothesis of bacterial seeding of an already altered aortic site, alteration via atherosclerotic plaques or a pre-existing aneurysm These pathogens arrive as minute septic emboli in the vaso vasorum or by surface implantation [6,7]. This combination of factors give rise to a suppurative arteritis and weakening of the vessel wall with eventual arterial pressure increase, aneurysm formation, and ultimately rupture [6,7,10].

The two most commonly implicated organisms are Salmonella and Staphylococcus species; however, approximately 20% of the cases are culture negative [6,8,9]. *H. Influenzae* serotype f is an extremely rare cause of mycotic aneurysms. We present what we believe to be only the fifth documented case in the literature (Table 1). *H. influenzae* is a small, Gram-negative bacillus and is either encapsulated or nonencapsulated. Encapsulated strains are more virulent and are broken down into serotypes a-f depending on its capsular polysaccharide composition. Before the development of a vaccine, type b was the most prevalent and virulent type. Now, type f and non-typable strains are increasing in prevalence and have been suggested as the most common strains in the post-Hib vaccination era [11,12].

HIV has been known to cause arterial aneurysms since 1989 and was described as a distinct "clinic pathological entity" in 1999 [13,14,]. In a group of young African patients without atherosclerosis, aneurysms were found in multiple locations leading researchers to associate the two [15]. Since that time researchers have proposed hypotheses regarding the mechanism of aneurysm

formation: 1) the virus directly damages fibroblasts in the arterial wall; 2) the virus induces an autoimmune response in the vaso vasorum thereby causing ischemia of the arterial wall and aneurysmal formation; 3) immunodeficiency allows bacteremia to occur and subsequent invasion of preexisting arterial atheromatous material [13,16,17].

Diagnosis is difficult. Imaging plays a central role, with computed tomography, CT scan, being the standard because of its availability and ability to make a differential diagnosis with other abdominal emergencies [18]. Clinical presentation is often nonspecific and most commonly marked by abdominal or back pain [19,20]. This case represents a unique presentation because there was no clinical suspicion for an aneurysm due to the patient's relatively young age and the fact that her obese body habitus made it difficult to detect [21-25]. Additionally, this presentation is unusual because the aneurysm rapidly expanded in a six-month interval because her immunocompromised state led to bacteremia and subsequent bacterial invasion of her preexisting atheromatous plaque causing aneurysmal degeneration (Figure 1a, 1b). This conclusion is supported by laboratory, microbiological and pathological analysis: elevated ESR and CRP, positive preoperative blood cultures, the aneurysm's saccular nature and rapid expansion, and its severe inflammation [26].

The standard operative approach is excision of the infected aneurysm with reconstruction via an in situ or extra anatomic bypass repair. Endovascular stent grafting has recently provided an alternative treatment for mycotic aneurysms. However, in patients with active infections, endovascular treatment had a 12-month survival rate of only 39% [25,27]. Endovascular repair can be used for repair largely as a bridge to open repair. Endovascular repair was not an option in our case secondary to the known infection, but also secondary to the anatomical landing zone between the renal arteries and common iliac arteries being too short. In situ repair was chosen for this patient given her young age and obese body habitus placing her at higher risk of groin infection. In addition, extra anatomic bypass has been shown to have a higher morbidity and mortality as compared to in situ repair, higher risk for aortic stump blow out and lower extremity amputation rate while having a similar re-infection rate [9].

Conclusion

In conclusion, we report only the fifth case to be documented of a mycotic aneurysm caused by *H. influenzae* type f. This unique patient presentation of an HIV patient who had a rapidly expanding mycotic aortic aneurysm who presented with back pain only and denied any constitutional symptoms contributes to the already scarce literature on such an entity. The cause of her rapid expansion was likely multi factorial in that her baseline immunosuppressive state facilitated bacterial growth leading to invasion of her atheromatous plaque and aneurysmal degeneration. Although rare, clinicians must have a high index of suspicion for mycotic aneurysm formation and therefore impending rupture in immune compromised patients who present with abdominal or back pain only.

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