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Case report

A ruptured mycotic aortic aneurysm in a patient with urinary retention: A case report ☆,☆☆

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ABSTRACT

Symptomatic abdominal aortic aneurysm (AAA) is a diagnosis that is a true emergency. Since AAAs are typically asymptomatic prior to rupturing, they can easily be missed. When an abdominal aortic aneurysm becomes symptomatic and ruptures, the ramifications can be catastrophic for the patient. We present a case of a 55-year-old male who presented with urinary retention and suprapubic pain. Computerized tomography demonstrated a rapidly expanding AAA and signs of impending rupture. Emergent vascular surgical repair was performed successfully. There was concern for mycotic nature of the AAA with recent COVID-19 infection and possible bacteremia. This case demonstrates the need for maintaining a wider differential when examining patients and avoiding anchoring bias and serves as a point of discussion for potential complications of COVID-19 infection.

Introduction

Abdominal aortic aneurysm (AAA) is a pathologic process where the aorta dilates due to chronic wall inflammation from increased proteinase activity and degradation of the connective tissue that holds the aorta together [1,2]. This can occur at any point along the aorta, however, approximately 80% are located infrarenal [2]. An AAA is defined by either 50% dilation greater than the average size of that aortic segment or an aortic diameter >3 cm [3].

AAAs are usually asymptomatic until they rupture, and the most common symptom experienced initially is out of proportion abdominal pain [2]. Patients may also have flank pain, groin pain, hypotension, hemodynamic instability, syncope, or death [2]. Rupture is a feared complication due to the high morbidity and mortality rate associated with it [4]. About 30%-50% of patients with a ruptured AAA die

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before hospitalization and an additional 30%-40% die after arrival [4].

AAAs typically expand at a rate of roughly 0.3 cm/y [3]. However, not all of them expand at this rate [3]. As the aortic diameter increases, the likelihood of rupture increases as well [2,5]. Wall tension, stiffness and peak wall stress may contribute to the possibility of rupture as well [2,5]. AAAs are diagnosed by abdominal ultrasounds (U/S) and computed tomography angiography (CTA) of the chest, abdomen, and pelvis. CTAs are considered the most reliable method of detecting aortic rupture [6,7]. First line treatment for AAA rupture is typically with endovascular aortic repair or open surgical repair of the aorta [7]. In the following case report, we discuss a man diagnosed with a 3.9 cm AAA with impending rupture.

Case report

A 55-year-old male with past medical history of non-ST-elevation myocardial infarction, ischemic cardiomyopathy, congestive heart failure with AICD placement, hypertension, and a motor vehicle accident in 2014 with subarachnoid hemorrhage, multiple fractures, residual bilateral lower extremity paresis, and hemicolectomy presented to the emergency department for urinary retention and suprapubic pain. He had been discharged from the hospital earlier the same day of presentation. He was hospitalized for a week for diarrhea, acute kidney injury, and dehydration secondary to COVID-19 infection that was confirmed by a PCR test on a nasopharyngeal specimen. The patient reported that at home, he developed suprapubic abdominal pain and was unable to urinate. He reported he had had a foley catheter placed for urinary retention during the previous hospitalization. The patient also reported hematuria over the past few days. He denied any fevers, chest pain, dyspnea, back pain, or lower extremity swelling.

The initial physical exam revealed an ill appearing man in acute distress from pain. His lungs were clear to auscultation. He had 2+ pulses in the bilateral upper and lower extremities. He had normal S1 and S2 heart sounds with good capillary refill. His abdomen was tender in the periumbilical and suprapubic area and had guarding with rebound on examination. Initial vital signs were a temperature of 97°F (36.1°C), a heart rate of 56 beats per minute, a respiratory rate of 24 breaths per minute, a blood pressure of 147/77 mm/hg, and an oxygen saturation of 98% on room air. A bladder scan was completed at bedside that showed 23 cc of urine. Intravenous fluids (500 ml of lactated ringers solution) and morphine (4 mg) were given.

Pertinent labs in the ED were a white blood cell count of 15.9 thou/cmm (normal 4-10.5 thou/cmm) and absolute neutrophils of 13.8 thou/cmm (normal 1.8-7.8 thou/cmm), lactate of 4.1 mmol/L (normal 0.5-2.1 mmol/L), Pro-bnp of 23,795 pg/mL (normal <125 pg/mL), troponin of 0.06 ng/ml (normal <0.05 ng/mL), and creatinine of 1.51 mg/dL (normal 0.53-1.30 mg/dL). The CT scan showed that the patient's AAA diameter had increased to 3.9 cm as compared to 3.0 cm seen on CT scan performed six days prior during his previous hospitalization (Fig. 1). His CT also revealed soft tissue stranding along the AAA, which was concerning for impending rupture (Fig. 2). The cranial and caudal extents of the AAA are highlighted in Figures 3A and B, respectively. Vascular surgery was consulted and they immediately took him to the operating room where he underwent emergent repair using hybrid approach: endovascular repair with aorto-uni-iliac endografts and left-to-right femoral-femoral bypass. Hybrid approach was necessary due to iliac artery occlusive disease. Open repair was not possible due to emergent nature of the presentation and hostile abdomen from previous injury and subsequent abdominal surgeries. Initial intraoperative aortogram showed evidence of contained rupture of the AAA. The patient was then transferred to the intensive care unit in stable condition after successful repair. There was concern that

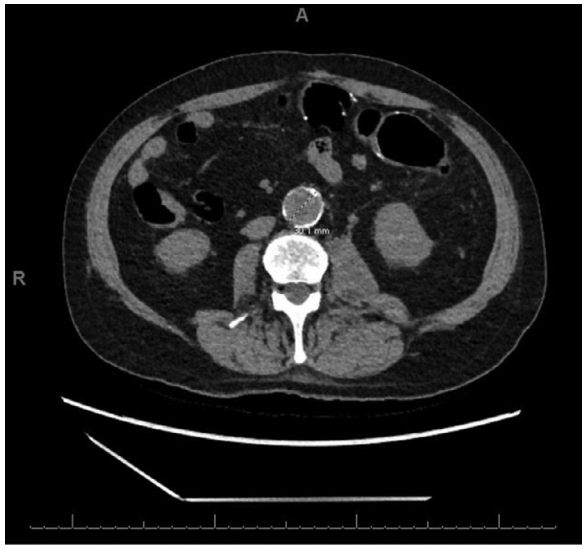

Fig. 1 – Computerized tomography scan showing extensive atherosclerotic calcification of the abdominal aorta, with a 3 cm infrarenal abdominal aortic aneurysm.

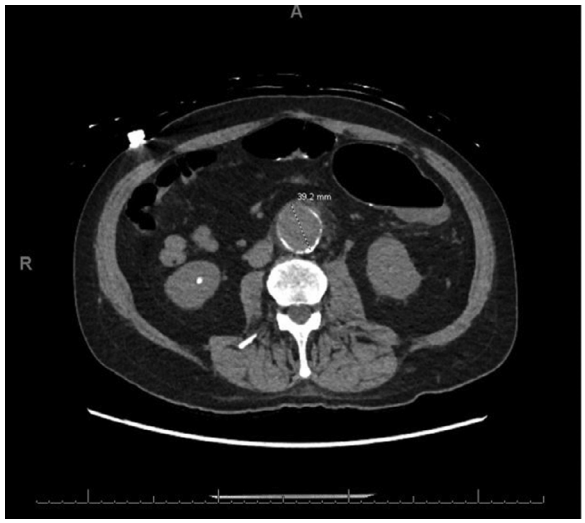

Fig. 2 – Computerized tomography scan taken six days after the prior scan showing new retroperitoneal soft tissue stranding, indicative of a plaque rupture and impending aortic rupture. Aneurysm currently measures 3.9 cm.

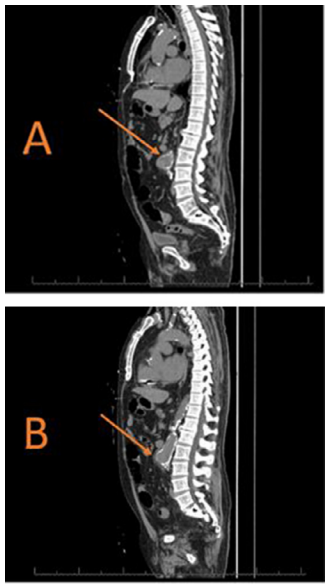

Fig. 3 – Images are a sagittal, non-contrast, computerized tomography scan showing the cranial (A) and caudal (B) extent of the AAA with impending rupture. CT angiogram with contrast was unable to be performed due to the patient's renal function.

mycotic nature of the AAA was the culprit for the rapid expansion and rupture of the patient's aorta. One of the two blood culture samples obtained was positive for *Staphylococcus epidermidis*. And the patient was symptomatic for COVID-19 infection at time of presentation. Infectious disease was consulted, and intravenous antibiotics was initiated. He was discharged home on suppressive antibiotics and close follow up.

Discussion

AAA rupture is an important diagnosis to make because if missed, the patient's prognosis is abysmal [4]. The patient in this case did not present in a typical way for AAA. His chief complaint was of urinary retention, and he had a recent CT scan prior to presentation that showed a new AAA at 3 cm, but with no signs of rupture. His risk of rupture was relatively low, given the diameter of his AAA and it was not anticipated that there would be an acute worsening of a 3 cm aorta within six days [8,9]. The most likely cause for this acute change was likely mycotic aorta, leading to rapid expansion and rupture. The culprit of his mycotic AAA is still unknown with confounding *Staphylococcus epidermidis* bacteremia and COVID-19 infection. However, literature is evolving that suggests COVID-19 as an etiology for acute AAA expansion [10]. Mycotic aortic aneurysms are rare, but if an abdominal aortic aneurysm does become infected, the risk of rupture increases [11].

This case highlights anchoring bias, a cognitive bias that causes individuals to rely heavily on the first piece of information given about a topic [12]. Initially the patient presented to the ED with urinary retention and a known history of urinary retention. Having recently been admitted with acute kidney injury, the initial differential focused on the potential for further injury to his kidney, a kidney stone, or urinary retention. A CT scan without contrast was initially ordered to look for kidney stones, but a rapidly expanding AAA with soft tissue stranding with concern for mycotic AAA with impending rupture was the unexpected result of the scan. It clearly illustrates the need to understand anchoring bias, and the dangers of narrowing a differential too early in an assessment. Given his recent history, AAA should have been considered given his sudden onset abdominal pain and renal issues.

Author contributions

All authors provided substantial contributions to manuscript content. All authors gave final approval of the version of the article to be published.

Patient consent

Patient consent was obtained for the publication of this Case Report.

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