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CASE REPORT

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A rare case of mycotic aortic aneurysm with *Clostridium perfringens* culture

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Key Clinical Message

As only early diagnosis, prompt surgical intervention, and appropriate antibiotic therapy can decrease clostridial MAA mortality rate; keeping in mind a broad differential diagnosis in a patient with sepsis and unusual vascular symptoms is important.

Abstract

Mycotic aortic aneurysm (MAA) is an infrequent but very consequential condition characterized by the pathological disruption of the aorta due to infection. *Clostridium perfringens* is a bacterium that falls under the taxonomic classification of the genus Clostridium. Although mycotic aneurysm is often not commonly linked with this infection, there are instances when it may function as a causative agent for MAA. Timely diagnosis and thorough therapeutic techniques, including surgical intervention and quick administration of appropriate antibiotics, can potentially reduce the mortality rate associated with clostridial MAA. In this study, we presented a clinical report detailing the diagnosis of a mycotic aneurysm caused by *C. perfringens* in the thoracic aorta in a 66-year-old male patient with a history of diabetes mellitus and a recent prostate biopsy. Furthermore, we discussed the surgical approach and overall management strategy to address this case.

KEYWORDS

aorta, *Clostridium perfringens*, computed tomography, infection, mycotic aneurysm

1 | INTRODUCTION

Mycotic aortic aneurysm (MAA) is a rare but lethal disease that can involve a localized or a large area and progress slowly or rapidly.[1] Mycotic thoracic aortic aneurysm (MTAA) is commonly caused by bacteria and rarely by fungi. Different mechanisms of seeding of the aortic wall by organisms include bacterial endocarditis emboli, contiguous extension of infection to the thoracic aorta, bacteremia or systemic sepsis, or direct introduction of bacteria into the wall of the artery due to trauma.[2] *Salmonella, Streptococcus* species, and *Staphylococcus aureus* are the

most common infection causes.[3,4] The presence of arterial injury and seeding of the organisms causes the intima to infect. After the infection of the vessel wall by microorganisms, degradation of the deeper layers occurs, and results in the development of an aneurysm.[5] MAA can involve any part of the aorta, yet the management is more challenging if it involves the thoracic region. Successful management of MTAA can be achieved by early diagnosis and antibiotic and surgical treatment.[8,9] Herein, we report a case of a patient diagnosed with a mycotic aneurysm of the thoracic aorta caused by *Clostridium perfringens* and the surgical management of the case.

2 | CASE PRESENTATION

A 66-year-old male patient with a medical history of diabetes mellitus (DM) and hypertension, who had undergone a transrectal ultrasound-guided prostate biopsy (TRUS) about 1 month before the current admission and reported on and off fever in the past month, was admitted to our hospital with complaints of sudden hemiparesis in the left side and dysarthria for the preceding 2 h. In physical examination, the heart rate was 113 beats/min, the oral temperature was 38.9°C, the blood pressure was measured at 150/110 mmHg, and the room air oxygen saturation level was 91%. The lab findings revealed a blood glucose level of 785 mg/dL, a WBC count of 17,600/μL, and a C-reactive protein above 150 mg/L.

The patient received a diagnosis of Ischemic cerebrovascular accident (CVA) based on findings from physical examination, as well as brain computed tomography (CT) scan and brain magnetic resonance imaging (MRI). Consequently, he sought treatment in the stroke care unit (SCU), where he received medical oversight from neurologists. The following day, the individual continued having tachycardia and decreased oxygen saturation, prompting immediate intubation. The new laboratory findings revealed a WBC count of 27,700/μL, a PRO-BNP level of 2300 Pg/mL, and a Pro Calcitonin concentration of 4.4 ng/mL. The administration of meropenem (MEPM) was started at a dosage of 3 g/day for the patient. The patient had further evaluation with the use of echocardiography. The transthoracic echocardiogram revealed a left ventricular ejection fraction (LV EF) of 35% and no obvious wedge.

The chest CT scan indicated the presence of gas-filled regions with emphysematous changes in the posterior mediastinum. (Figure 1) This finding was crucial for distinguishing between the potential occurrence of a mycotic aortic aneurysmal rupture and an esophageal perforation. The surgeons requested a CT angiography (CTA), which revealed the presence of a ruptured aortic pseudoaneurysm. This pseudoaneurysm was located in the thoracic aorta, distal to the left subclavian artery, and above the celiac artery. (Figure 2).

The patient's vital signs became unstable, and norepinephrine infusion started. The surgical procedure was conducted on an urgent basis. It began with administering general anesthesia, ensuring that the patient was adequately prepped, and draped for the surgical intervention while being continuously watched by anesthesiologists.

Stabilizing the patient through serum therapy (30 cc/kg) allowed us to initially proceed with the axillofemoral bypass, considering the complexities involved in ligating the aorta directly, therefore the vascular surgery team first conducted an exploration of the right subclavian artery at the right deltopectoral groove and performed an axillofemoral bypass with an externally enforced polytetrafluoroethylene (PTFE) graft with a diameter of 8 mm and a length of 80 cm.

Then, we positioned the patient in right lateral decubitus, and a classic left posterolateral thoracotomy was performed. During the thoracic exploration, the surgical team encountered an estimated 1 L of purulent hemothorax and an area of infection in the posterior mediastinum. The adhesions between the lungs and the infective sac were meticulously removed, revealing the severe infection and total necrosis of the aortic wall. The aorta was doubly suture ligated with 2/0 Prolene sutures distal to the left subclavian and proximal to the celiac artery. (Figure 3)

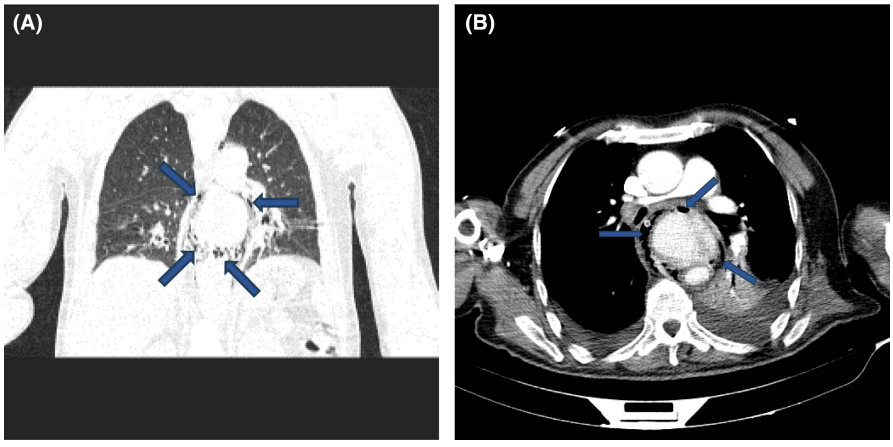

FIGURE 1 (A and B): Chest CT Scan indicating Pneumomediastinum (The Blue Arrows).

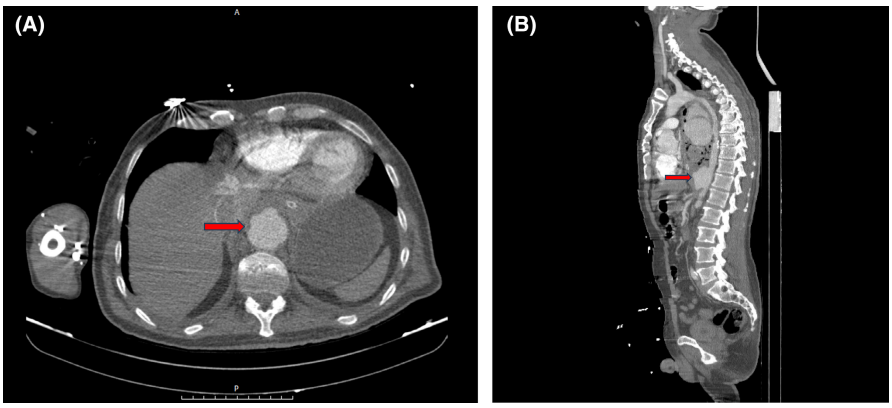

FIGURE 2 (A and B): CT angiogram shows pseudoaneurysm in the thoracic aorta, distal to the left subclavian artery, and above the celiac artery. (The Red Arrows).

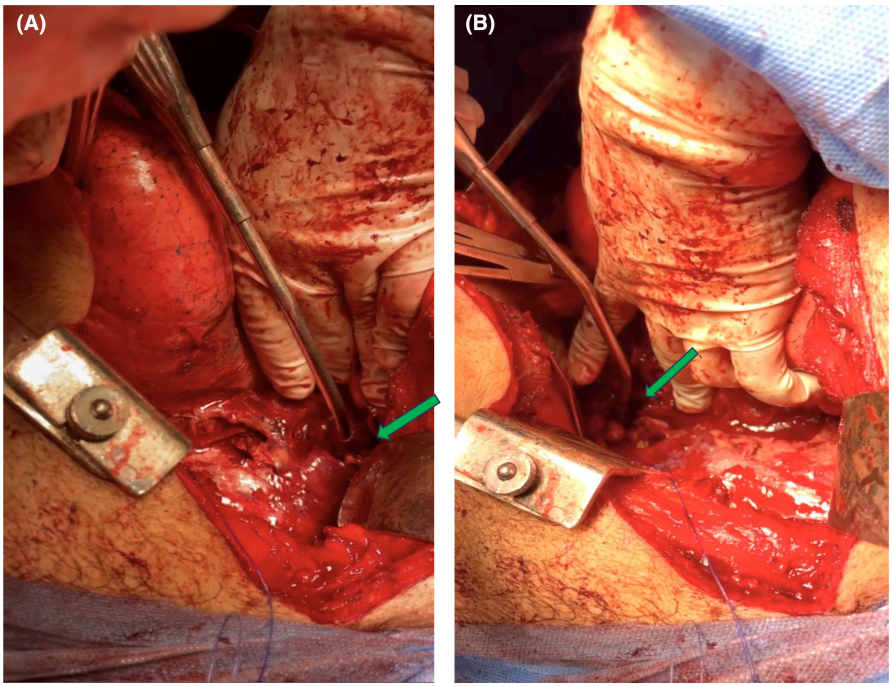

FIGURE 3 A. Thoracic Aorta defect (distal to left subclavian Artery). B. Thoracic Aorta defect (proximal to celiac artery).

Pulses of the right and left femoral arteries were palpable. The affected tissues were excised entirely, and a sample of the aorta wall was obtained for tissue culture and biopsy. The surgical site underwent a comprehensive cleansing process, ensuring the removal of all purulent material. To effectively regulate postoperative drainage, two chest tubes were placed.

Despite diligent attempts, it is regrettable that the patient's hemodynamic status worsened on the following day, ultimately resulting in the patient's untimely mortality. The subsequent examination of the samples confirmed that *C. perfringens* was the etiological agent.

3 | DISCUSSION

C. perfringens is a bacterium belonging to the genus *Clostridium* and is characterized by its gram-positive nature, rod-shaped morphology, anaerobic metabolism, and ability to generate spores. While often not associated with mycotic aneurysms, it may sometimes be a pathogen in this context. Similar to what we observed in our case, gas formation encircling the aorta or peripheral arteries is common in CT scans with clostridial mycotic aneurysms.[6] The proliferation of clostridia in tissues may occur in conditions of decreased oxidation–reduction or lower pH levels. This can be seen, for instance, in cases of vascular damage, tissue necrosis, or tissue anoxia leading to the buildup of lactic acid. Due to this rationale, clostridial infection is often linked to gastrointestinal (GI) or hematopoietic-system malignancies.[7] Therefore, in cases where there is suspicion of *C. perfringens* infection, it is crucial to use blood tests, CT scans, or endoscopic procedures to establish or exclude the diagnosis, mainly due to its association with problems related to malignant tumors.[8]

In our case, there was no known history of GI malignancies, but our patient did have a TRUS prostate biopsy a month before admission. In 2019, Al-Ani et al.[9]

reported two cases of mycotic abdominal aortic aneurysms after TRUS prostate biopsy occurring less than 10 days after the procedure. A male patient, aged 63, who exhibited symptoms of sepsis and back discomfort 9 days after a prostate biopsy, was diagnosed with an ectatic abdominal aorta measuring 27 mm. Over 1 week, despite receiving antimicrobial medication, the aorta enlarged to 59 mm. The patient had a surgical procedure in which the infected aortic aneurysm was successfully removed and reconstructed using a vein graft. Another male patient, aged 55, had the same symptoms, occurring 7 days after a prostate biopsy, indicating the presence of a 60 mm aortic aneurysm. The patient had a rupture of his aneurysm 2 days before the scheduled surgery, resulting in his unfortunate demise during an emergency repair procedure. In both cases, *Escherichia coli* was detected in analysis of aortic tissue samples. To our knowledge, no cases were previously reported with clostridium culture of aortic tissue sample following prostate biopsy.

While there is existing knowledge about risk factors associated with the formation and progression of MAA, a comprehensive understanding of the underlying pathobiology needs to be achieved. In our case, the precise source of the aneurysm and its development remains unknown. Organisms may infect the thoracic aorta by penetrating the aortic wall through many routes, such as the intima or adventitia, vasa vasorum, lymphatics, or direct involvement from nearby organs. Aortic infection is more likely to occur in individuals with risk factors such as atherosclerosis and preexisting aneurysms. Other factors that can contribute to the development of thoracic MAA include antecedent infections such as endocarditis, purulent pericarditis, pneumonia, periaortic lymphadenitis, osteomyelitis, soft tissue infection, periodontal infection, and sepsis.[10] Additionally, impaired immunity due to conditions like diabetes, chronic glucocorticoid therapy, posttransplantation, alcoholism, cirrhosis, chemotherapy, chronic hemodialysis, acquired immunodeficiency syndrome (AIDS), and malignancy can also increase the risk.[11] Furthermore, aortic injury resulting from iatrogenic injuries during cardiothoracic surgeries, cardiac catheterization, and blunt or penetrating traumatic injury to the thoracic aorta can also contribute.[1] From the above-mentioned risk factors, our patient had diabetes and a history of episodic fever during the month prior to his last admission.

The mortality rate associated with clostridial MAA may be decreased with timely diagnosis and comprehensive management strategies, including surgical intervention and prompt administration of suitable antibiotics.[12] Surgical approaches include open and endovascular repair techniques.[13] A lack of published randomized controlled studies comparing various surgical procedures necessitates an individualized strategy for each patient. Endovascular repair may serve as an interim intervention for patients in unstable conditions or as a conclusive repair method for a subset of patients who exhibit a favorable response to antibiotic treatment.[3,14,15] Our patient was unstable and endovascular facilities were not available at that moment. Moreover, there was not enough landing zone to celiac artery to place a thoracic stent graft. The available open surgical interventions include extra-anatomic bypass procedures and in-situ repair techniques with graft implantation. In this case, we used open surgery with an extra-anatomic axillofemoral bypass and debridement of the infected aorta and surrounding tissues. It is important to note that this strategy is associated with potential complications such as aortic stump rupture or the dissemination of infection to the newly implemented prosthetic bypasses.[14] Previous studies highlight the need to promptly administer antibiotics to manage clostridial infections. Penicillin is often regarded as the primary antibiotic of choice, whereas MEPM, imipenem, third- and fourth-generation cephalosporins, metronidazole, and vancomycin are considered secondary options. It is recommended that an extended course of antibiotic therapy be taken for a minimum duration of 6–8 weeks after surgical intervention.[12,16]

Taken together, it can be said that the prognosis of clostridial MAA is very poor, especially in the absence of proper intervention and antibiotic administration. As our patient was presented with unusual manifestations and the fact that he was primarily admitted as a case of CVA and MAA was accidentally found in his chest CT (he had no complaints of chest pain or ischemia in extremities), the diagnosis was made with delay, which left us no time for proper pre-op preparations and he opted for an urgent procedure.

AUTHOR CONTRIBUTIONS

Niki Tadayon: Conceptualization; project administration; supervision. **Saleh Shahsavari:** Methodology; project administration; writing – original draft. **Reyhane Mahya:** Writing – original draft. **Delaram Nourmohammadi:** Writing – original draft. **Faezeh Jadidian:** Writing – review and editing. **Masoud Babaei:** Writing – review and editing. **Mostafa Mousavizadeh:** Supervision. **Kimia Vakili:** Project administration; writing – original draft.

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CONFLICT OF INTEREST STATEMENT

The authors have no conflict of interest to declare.

DATA AVAILABILITY STATEMENT

Data supporting this study are included within the article and supporting materials. For more data please contact (kimiavakili1377@gmail.com).

CONSENT

Written informed consent was obtained from the patient's next of kin to publish this report in accordance with the journal's patient consent policy.

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