Case Report

Mycotic aneurysm and it's not so silent presentation – A case report

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ABSTRACT

We report a rare case of mycotic aneurysm of the aortic arch caused by *Salmonella typhi*, initially manifesting as hoarseness of voice secondary to left recurrent laryngeal nerve compression. A 58-year-old diabetic and hypertensive male presented with intermittent fever, initially managed as a urinary tract infection with diabetic ketoacidosis. Progressive symptoms led to the diagnosis of a saccular mycotic aneurysm of the aortic arch with purulent pericardial effusion and bilateral pleural effusions. Serology confirmed typhoid etiology. The patient was stabilized on antibiotics and referred for thoracic endovascular aortic repair. This case highlights the importance of considering vascular pathology in patients with unexplained vocal cord palsy and systemic infection, and underscores the need for early recognition and multidisciplinary management.

Key words: Hoarseness of voice, Mycotic aneurysm, Pericardial effusion, *Salmonella typhi*, Vocal cord palsy

Mycotic aneurysms are localized abnormal dilatations of the arterial walls that develop secondary to an infective process that causes destruction of the wall [1]. The majority of patients with mycotic aneurysms due to *Salmonella* species had lesions that involved the abdominal aorta or iliac arteries; <10% of these lesions involved the intrathoracic vessel. The combination of clinical presentation, laboratory results, and radiographic results is used to make the diagnosis of a mycotic aneurysm of the aorta. The risks are higher in immune-compromised hosts, that is, human immunodeficiency virus, diabetes, high-dose glucocorticoids, or malignancy/chemotherapy. Fever, pulsatile mass, local discomfort, and arterial site inflammation (back pain in the aorta, headaches in cerebral arteries) are the most typical signs. Because these symptoms are non-specific, many individuals are classified as having a fever of unknown origin and go undetected until they develop severe symptoms of sepsis, thrombosis, hemorrhage, or rupture. To reduce the severe morbidity and mortality associated with this illness, timely identification and treatment are required. Cultures are positive only in 50–85% of the cases [1]. Surgery remains the definitive treatment, but perioperative mortality can be as high as 63% in patients with aneurysm rupture. Cardiovascular hoarseness, especially hoarseness due to an aortic arch aneurysm, is very rare. The bulging cardiovascular structures can frequently compress the left recurrent laryngeal nerve, leading to left vocal cord paralysis, rendering patients with hoarseness. Early diagnosis of Ortner's syndrome is essential for starting timely treatment, restoring vocal cord function, and avoiding permanent damage to the patients. Hoarseness was a sole symptom in 36.8% patients, a major symptom in 39.1% patients, and an accompanying symptom in 24.1% patients [2].

This case is reported due to its exceptional rarity, as a mycotic aneurysm of the aortic arch caused specifically by *Salmonella* Typhi is uncommon, with very few cases documented in published literature. Adding to its uniqueness, hoarseness of voice secondary to recurrent laryngeal nerve compression (Ortner's syndrome) served as the initial and sole presenting symptom, a rare and easily overlooked manifestation of an otherwise life-threatening vascular infection.

CASE DESCRIPTION

A 58-year-old male with a history of diabetes, hypertension, and asthma presented to us with intermittent fever for 3 weeks, accompanied by chills. He had been treated for a urinary tract infection and diabetic ketoacidosis (DKA) at a local hospital with antibiotics and supportive management. During his

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hospital stay, his family noticed hoarseness of voice, and further evaluation revealed left vocal cord palsy, which was confirmed by indirect laryngoscopy. After discharge, he returned to our emergency department the same day, complaining of shortness of breath, left shoulder pain, and chest pain, leading to his admission to the intensive care unit for further evaluation.

On initial assessment, he was found to be tachypneic with a respiratory rate of 40/min, with room air saturation at 89%, and tachycardic with a heart rate of 150/min, with elevated blood sugar levels of 326 mg/dL.

A 2D echocardiogram showed pericardial effusion measuring 21 mm posterior – lateral to the left ventricle, 24 mm anterior to the left atrium/left ventricle, and 20 mm anterior to the right atrium, with right atrial systolic collapse. His respiratory distress was supported with high-flow nasal oxygenation. A pericardial pigtail catheter was inserted, draining 500 mL of pericardial fluid that appeared purulent, with an analysis showing a total white blood cell count of 27,382 cells/mm^3 with 98% neutrophils.

Cultures were sent for analysis, and he was started on polymyxin 15 lakh unit bolus dose followed by 7.5 lakh 12th hourly initially to cover multidrug resistant organism in view of previous hospital admission and partially treated urinary tract infection with meropenem 1 g IV 8th hourly in prior hospital and later deescalated to ceftriaxone 2 g 12th hourly after the *Salmonella* serology report became positive. His total white blood cell count was 27,000 cells/mm^3, and procalcitonin levels were elevated at 3.8 ng/mL. Tropical workup results were negative, with non-reactive serology and no growth in blood, urine, and pericardial fluid cultures.

A computed tomography (CT) scan of the neck and chest was performed to investigate the cause of the vocal cord palsy. The neck CT confirmed left vocal cord palsy (Fig. 1), while the chest CT (Figs. 2 and 3) indicated potential issues with the demarcation of the aortic arch walls, revealing air-fluid pockets in the perivascular space. A contrast-enhanced CT aortogram (Fig. 2a) demonstrated disruption of the mid-arch wall with a saccular/mycotic aneurysmal outpouching, along with adjacent collections and air pockets in the perivascular region extending into the aortic recess and left perihilar area. There was also moderate pericardial effusion and bilateral pleural effusion, with a small segment of wall enhancement in the distal ileum (Fig. 4).

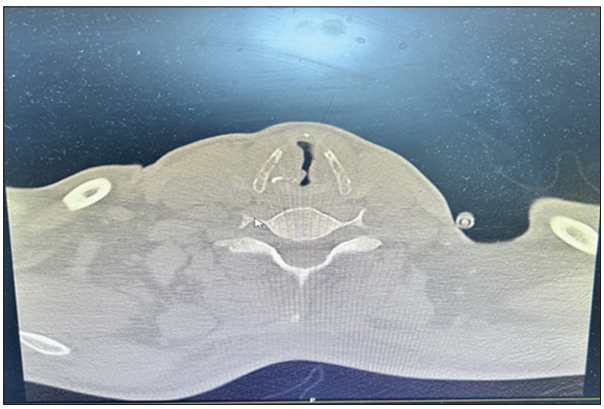

Figure 1: Left vocal cord palsy during valsalva-computed tomography

To investigate the etiology of the mycotic aneurysm, a Widal test was performed, showing an *S.* Typhi O antigen titer of 1:320 and H antigen titer of 1:80, with normal paratyphoid titers and a positive typhi immunoglobulin M (IgM). The patient was subsequently evaluated by the cardiothoracic surgery team, who recommended thoracic endovascular aortic repair.

DISCUSSION

This case presents a rare and diagnostically challenging manifestation of typhoid fever, a mycotic aneurysm of the

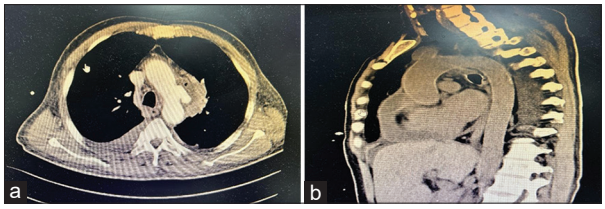

Figure 2: (a) Contrast-enhanced computed tomography (CT) showing aortic arch aneurysm - interruption of the wall of mid aortic arch with mycotic aneurysmal pouching (2.8 × 1.0 × 1.6)-adjacent collection with air pockets in the prevascular region, aortopulmonary window, and left perihilar region; (b) Sagittal CT – Aortic arch aneurysm

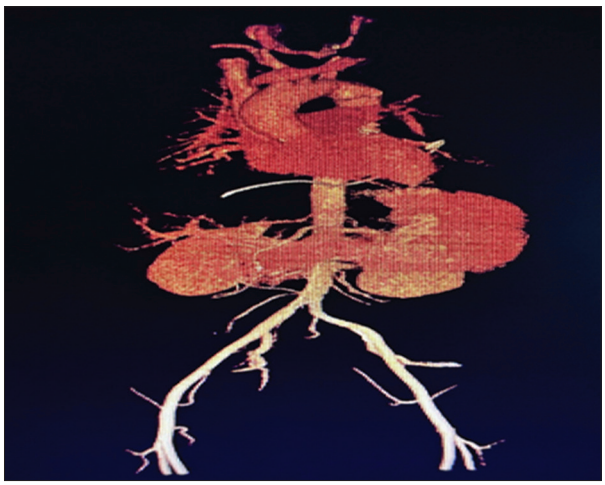

Figure 3: 3D reconstruction of computed tomography aortogram showing aneurysm

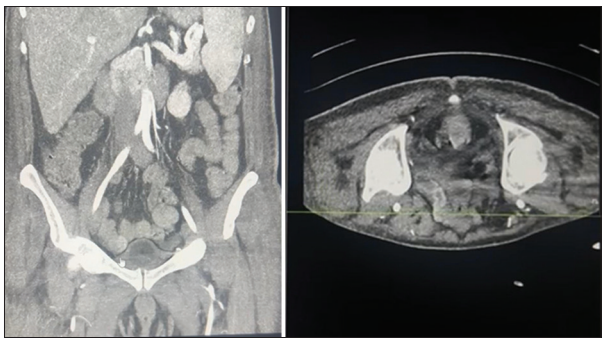

Figure 4: Computed tomography -small bowel (distal ileum) thickening

aortic arch complicated by purulent pericarditis, bilateral pleural effusions, and left recurrent laryngeal nerve palsy, in a 58-year-old male with diabetes, hypertension, and asthma.

Mycotic aneurysms are localized, infected dilatations of the arterial wall resulting from destruction of the vessel by an infective process. Despite the term "mycotic," coined by William Osler in his Gulstonian lectures to describe fungus-like vegetations on the aortic wall, the majority of these aneurysms are bacterial in etiology and are more accurately termed "infected aneurysms" [1]. A mycotic aneurysm due to a *Salmonella* infection is still very rare and a life-threatening disease, with an incidence of endovascular infections due to *Salmonella* ranging from 9% to 16.2% [3].

The pathogenesis involves one of several mechanisms: Bacteremic seeding of a pre-existing atherosclerotic plaque, contiguous spread from an adjacent infectious focus, septic embolization to the vasa vasorum, or direct inoculation following trauma or instrumentation [4]. *Salmonella* species are particularly known for their predilection toward atherosclerotic vessels, attributed to their ability to invade and replicate within endothelial cells and macrophages, triggering a destructive inflammatory cascade within the arterial wall [5]. Established risk factors include diabetes mellitus, hypertension, atherosclerosis, malignancy, and other immunocompromised states, all of which increase susceptibility to bacteremic seeding and impair the host's ability to contain infection [6]. Our patient had both diabetes and hypertension, placing him in a high-risk category for this rare vascular complication.

In the pre-antibiotic era, Gram-positive cocci, primarily *Staphylococcus* and *Streptococcus*, were the predominant pathogens in mycotic thoracic aortic aneurysms. In the post-antibiotic era, Gram-negative organisms now account for up to 40% of cases, with *Salmonella* species emerging as the most common pathogen in Asian countries [6]. Among *Salmonella* species, non-typhoidal strains such as *Salmonella* Enteritidis and *Salmonella* Choleraesuis account for the majority of vascular infections, while Salmonella Typhi is responsible for only approximately 2% of Salmonella-related mycotic aneurysms, making it an exceptionally rare etiological agent [7].

A systematic review of 57 reported cases of *Salmonella* mycotic aneurysms found that *S.* Typhi was documented in only two cases, with the abdominal aorta being the most commonly affected site (37/57 cases), and thoracic aortic involvement occurring in fewer than 10% of cases [8]. This makes our case, involving the aortic arch as the primary site, particularly noteworthy and among the very few reported in published literature. One of the most striking features of this case was the presentation of hoarseness of voice as the initial symptom, later confirmed to be due to left vocal cord palsy on indirect laryngoscopy and CT neck imaging. This phenomenon is explained by the anatomical course of the left recurrent laryngeal nerve, which loops around the aortic arch below the ligamentum arteriosum before ascending to innervate the larynx. Any mass lesion, aneurysm, or enlarged cardiovascular structure in this region can compress the nerve, resulting in left vocal cord paralysis and hoarseness, a clinical entity known as Ortner's syndrome or cardiovocal syndrome, first described by Ortner in 1897 [9].

While Ortner's syndrome is classically associated with mitral stenosis, it has since been described in association with aortic aneurysms, pulmonary hypertension, patent ductus arteriosus, and mediastinal tumors [10]. In a large review of patients with cardiovocal syndrome, hoarseness was the sole presenting symptom in 36.8% of patients, a major symptom in 39.1%, and an accompanying symptom in 24.1% [11]. This underscores the importance of recognizing isolated hoarseness as a potential harbinger of serious cardiovascular pathology, particularly in the context of systemic infection. In our case, the presence of hoarseness during an admission for fever and DKA should have prompted earlier thoracic imaging, which may have led to a faster diagnosis.

Our patient developed a large purulent pericardial effusion with right atrial collapse, necessitating emergency pericardial drainage via pigtail catheter, which yielded 500 mL of frankly purulent fluid with a white cell count of 27,382 cells/mm^3 and 98% neutrophils. Purulent pericarditis is a rare but serious complication of systemic bacterial infections, with mortality rates exceeding 40% if not promptly drained and treated [12].

In the context of *Salmonella* infection, purulent pericarditis is an uncommon but documented complication, arising from either hematogenous seeding of the pericardial space or direct extension from an adjacent infected focus, in this case, the mycotic aneurysm and its associated perivascular collections [13]. The contiguous extension of infection from the aortic arch aneurysm into the pericardial space and bilateral pleural cavities, as demonstrated on CT aortogram, represents a particularly aggressive and advanced disease course.

A significant diagnostic challenge in this case was the absence of microbiological growth in blood, urine, and pericardial fluid cultures, despite clear clinical and radiological evidence of an infectious process. Negative cultures have been reported in up to 25% of mycotic aneurysm cases and may result from prior antibiotic therapy, fastidious organisms, or anaerobic pathogens [1]. In our patient, prior treatment with meropenem at the referring hospital likely sterilized the cultures, masking the causative organism.

In this context, serological testing proved decisive. The Widal test demonstrated an *S.* Typhi O antigen titer of 1:320, and H antigen titer of 1:80, and Typhidot IgM was positive, collectively supporting active *S.* Typhi infection. The Typhidot IgM test detects specific IgM antibodies against a 50 kDa outer membrane protein of

S. Typhi and has been shown to be a rapid, sensitive, and reliable diagnostic tool, particularly valuable in culture-negative scenarios and resource-limited settings [14]. A prospective descriptive study performed by Ansari *et al*. on 125 pediatric patients demonstrated that Typhidot IgM has all the attributes of an ideal test for typhoid, which is simple, with a rapid turnover time and high sensitivity [15]. The study highlighted that early serological confirmation of typhoid fever using Typhidot IgM can facilitate timely initiation of appropriate antibiotic therapy, thereby reduce complications and improve patient outcomes. These findings are directly relevant to our case, where blood and pericardial fluid cultures were negative, and serological confirmation through Typhidot IgM played a decisive role in establishing *S.* Typhi as the causative organism of the mycotic aneurysm. This case reinforces the importance of serological testing as a complementary diagnostic modality when cultures fail to yield a pathogen.

CT imaging played a pivotal role in establishing the diagnosis. The contrast-enhanced CT aortogram demonstrated disruption of the mid-aortic arch wall with a saccular outpouching consistent with a mycotic aneurysm, accompanied by air-fluid pockets in the perivascular space, adjacent collections extending into the aortic recess and left perihilar region, moderate pericardial effusion, bilateral pleural effusions, and distal ileal wall enhancement. The presence of periaortic gas is considered highly specific for mycotic aneurysm and infected aortic conditions, as it reflects gas-forming organisms or fistulous communication with adjacent structures [16].

CT aortography remains the gold standard for diagnosis of mycotic aortic aneurysms, offering precise delineation of aneurysm morphology, extent of infection, and involvement of adjacent structures, which is essential for surgical planning [17]. The saccular morphology of the aneurysm in our case is characteristic of mycotic aneurysms, in contrast to the fusiform morphology more typical of degenerative aneurysms.

Empirical antibiotic therapy was initiated with polymyxin and meropenem, targeting multidrug-resistant organisms, given the patient's prior hospital admission and partially treated infection. Following positive *S.* Typhi serology, antibiotics were appropriately de-escalated to ceftriaxone 2 g intravenously every 12 h. Third-generation cephalosporins such as ceftriaxone are considered the first-line treatment for severe typhoid fever and *Salmonella* vascular infections [18]. Prolonged intravenous antibiotic therapy for a minimum of 6 weeks post-intervention is generally recommended to reduce the risk of recurrence and reinfection following vascular repair [9].

The CT scan also revealed small segment wall enhancement of the distal ileum, consistent with typhoid enteritis, a known manifestation of *S.* Typhi infection reflecting hematogenous seeding and lymphoid hyperplasia of the Peyer's patches [19]. This finding provided additional indirect evidence supporting *S.* Typhi as the causative organism and illustrated the systemic nature of the infection, with simultaneous involvement of the aorta, pericardium, pleura, and bowel.

CONCLUSION

Mycotic aneurysms caused by *S.* Typhi are rare but potentially life-threatening conditions that require prompt recognition and treatment. Early diagnosis, along with appropriate antibiotic therapy and possible surgical intervention, is essential for improving patient outcomes. Understanding the pathophysiology, risk factors, and clinical manifestations of these aneurysms can help clinicians identify at-risk patients to provide timely and effective care.

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