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CASE REPORT

Ruptured Thoracic Aortic Aneurysm Infected with *Listeria Monocytogenes*: A Case Report and a Review of Literature

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Abstract: A 75-year-old male with a history of alcoholic liver cirrhosis, sigmoid colon cancer, and metastatic liver cancer was admitted to our institution with a complaint of a prickly feeling in his chest. On admission, a chest radiograph revealed a normal cardio-thoracic ratio of 47%. Echocardiography revealed pericardial effusion and blood chemical analyses revealed elevated C-reactive protein levels (14.7 mg/dL). On day 3, chest radiography revealed cardiomegaly with a cardio-thoracic ratio of 58% and protrusion of the left first arch. Contrast-enhanced chest computed tomography revealed a saccular aneurysm in the aortic arch with surrounding hematoma; thus, a ruptured thoracic aortic aneurysm was suspected. Emergency surgery was performed, which revealed a ruptured aortic aneurysm with extensive local inflammation. The diagnosis of an infected aortic rupture was therefore confirmed. The aneurysm and abscess were resected, followed by prosthetic graft replacement and omental packing. Histopathology of the resected aneurysm revealed gram-positive bacilli; and *Listeria monocytogenes* was confirmed as the causative organism by culture. Postoperative course was uneventful; on postoperative day 60, the patient was ambulatory and was discharged. Here we report the case of a male with a ruptured thoracic aortic aneurysm infected with *L. monocytogenes*.

Keywords: Thoracic aortic aneurysm, *Listeria monocytogenes*

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Introduction

The incidence of infected aortic aneurysms has increased over recent years. This type of aneurysm is critical in nature and is associated with both high morbidity and mortality. Moreover, this condition is associated with rapid clinical deterioration, and thus rapid diagnosis and treatment are essential to improve patient survival. The clinical symptoms of infected aortic aneurysms may include fever, abdominal pain, or general fatigue. Symptoms, however, may be non-specific or entirely absent as well. The organisms most commonly isolated from infected aortic aneurysms include *Salmonella, Staphylococcus* species, *Treponema pallidum,* and *Chlamydia*;[1–4] *Listeria monocytogenes,* as detected in this case, is rarely isolated. Here we present a male with a thoracic aortic aneurysm infected with *L. monocytogenes.*

Case Presentation

A 75-year-old male was admitted to our institution with a complaint of a prickly feeling in his chest. The patient had a history of alcoholic liver cirrhosis and sigmoid colon cancer for which he had undergone laparoscopic colectomy at the age of 72. Thereafter, the patient was diagnosed with metastatic liver cancer at the age of 75 and was treated with transcatheter arterial chemoembolization. Both sigmoid colon cancer and metastatic liver cancer were almost cured. Two weeks before admission, he was diagnosed with type 2 diabetes mellitus and the administration of oral hypoglycemic agents (voglibose 0.6 mg/day and sitagliptin 50 mg/day) was initiated. During the same time, he had experienced a prickly feeling in the chest, but the sensation resolved within 30 minutes. In the middle of August 2012, the patient was admitted to our institution for further examinations. He admitted to a habit of heavy alcohol with the use of about 700 milliliters of whisky a day, but denied tobacco use. On admission, his blood pressure was 124/80 mmHg; heart rate was 90 beats/min; body temperature was 36.0 °C; and oxygen saturation was 98% on room air. Initial clinical examination revealed a weight of 60.4 kg, height of 167.1 cm, and a body mass index of 21.6. Inspection of the palpebral conjunctiva revealed no evidence of anemia. Auscultation of the chest revealed no evidence of abnormal heart murmurs and no rale or other lung sounds. Physical examination of the patient revealed no edema or cyanosis. Blood chemistry analyses revealed the following (Table 1): normal white blood cell counts (4200/μL; neutrophil differential counts: 64%); mild anemia (red blood cell count, 378×10^4/μL; hemoglobin, 11.4 g/dL); elevated C-reactive protein levels (8.8 mg/dL); mild hypoalbuminemia (2.9 g/dL); elevated total bilirubin levels (2.3 mg/dL); elevated gamma-glutamyl transpeptidase levels (228 IU/L); elevated alkaline phosphatase levels (535 IU/L); evidence of coagulation dysfunction (prothrombin time, 61%; activated partial thromboplastin time, 37.5 s; fibrin/fibrinogen degradation products, 7.8 μg/mL; and D-dimer levels, 2.9 μg/mL); elevated glucose levels (313 mg/dL);

Table 1. Blood chemistry analyses.

Hematology	
WBC	4,200/μL
RBC	378×10^4/μL
Hb	11.4 g/dL
Ht	33.3%
MCV	88.3 fL
MCH	30.2 pg
MCHC	34.3 g/dL
PLT	16.5×10^4/μL
Serology	
CRP	8.8 mg/dL
Blood chemistry	
TP	7.1 g/dL
Alb	2.9 g/dL
T-Bil	2.3 mg/dL
γ-GTP	228 IU/L
ALP	535 IU/L
AST	32 IU/L
ALT	25 IU/L
LDH	200 IU/L
CK	34 IU/L
BUN	17.2 mg/dL
Cr	0.82 mg/dL
Na	134 mEq/L
K	4.4 mEq/L
Cl	97 mEq/L
Coagulation	
PT	61%
APTT	37.5 sec
FDP	7.8 μg/mL
D-dimer	2.9 μg/dL
Sugar	
Glucose	313 mg/dL
HbA_{1c}	10.1%
Tumor marker	
CEA	3.5 ng/mL
CA19-9	9.7 U/mL

and elevated HbA_{1c} (10.1%). A chest radiograph revealed a normal cardio-thoracic ratio of 47%; moreover, there was no evidence of cardiomegaly, pulmonary congestion, or pleural effusion (Fig. 1A). An electrocardiogram revealed a normal sinus rhythm, a heart rate of 114 beats/min, mild Q-wave depression, and mild ST changes. A transthoracic echocardiogram revealed evidence of fluid retention in the pericardium of the anterior and posterior walls of the left ventricle, as well as a preserved ejection fraction of 55% (Fig. 1B). A parasternal long axis view revealed basal segment of the ascending aorta to be of normal diameter (42 mm). Soon after admission, the patient developed atrial tachycardia with a heart rate of 150 beats/min, which was evident on the monitoring electrocardiography. Subsequently, the patient was administered 5 mg verapamil intravenously, after which there was full restoration to sinus rhythm. In addition, warfarin potassium (2 mg/day) was administered to prevent thrombosis. Thereafter, no chest symptoms were observed. On day 3 of admission, blood chemical analyses revealed evidence of an inflammatory response with white blood count of 9400/μL and elevated C-reactive protein levels (14.1 mg/dL). At this time, the patient was afebrile with a body temperature of 36.3 °C. Two sets of blood cultures, a urine culture, and a sputum culture revealed no causative agents; therefore, the presence of infection was supposed to be deniable as a cause for the inflammatory response. On day 3, a chest radiograph revealed enlargement of the cardiac shadow (cardio-thoracic ratio of 58%) and the protrusion of left first arch (Fig. 2A).

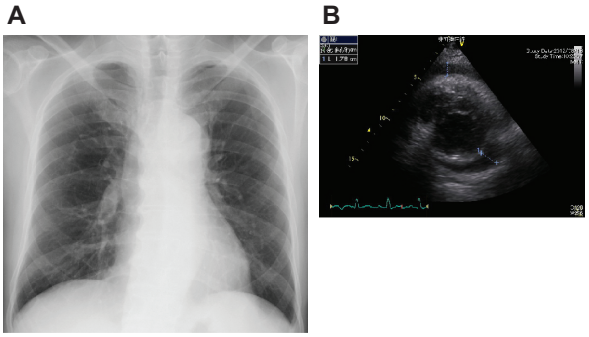

Figure 1. Chest radiography on admission revealing a normal cardio-thoracic ratio of 47%. There is no evidence of cardiomegaly, pulmonary congestion, or the retention of pleural effusion (**A**). On admission, echocardiography revealed fluid retention in the pericardium of anterior and posterior walls of left ventricular. Ejection fraction was preserved with 55% (**B**).

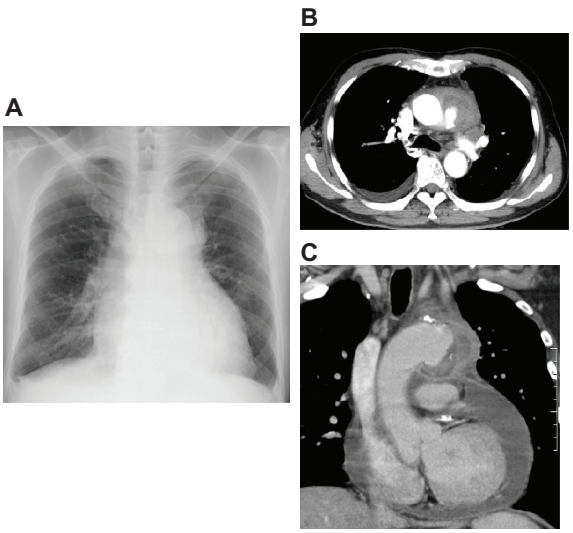

Figure 2. On day 3, chest radiography revealed enlargement of the cardiac shadow (cardio-thoracic ratio of 58%) and the protrusion of the left first arch (**A**). On day 3, contrast-enhanced chest computed tomography revealed an ulcer-like projection along the lesser curvature of the aortic arch surrounded by a hematoma. At this point, a ruptured thoracic aortic aneurysm was suspected (**B**). Coronal view enhanced computed tomography revealed pericardial effusion (**C**).

A thoracic aortic aneurysm was suspected, and contrast-enhanced computed tomography (CT) of the chest was performed. CT revealed an ulcer-like projection along the lesser curvature of the aortic arch surrounded by a hematoma (Fig. 2B and C). On the basis of these findings, a ruptured aortic aneurysm and cardiac tamponade were suspected. Although the patient's circulatory condition was stable, the possibility of deterioration of the patient's condition warranted emergent surgical repair.

Surgery (Fig. 3A–D) was performed under general anesthesia. Using a median sternal incision, the epicardium was incised exposing a large pericardial effusion, both clear and yellow in color. An aortic cannula of 24 Fr was then placed in the ascending aorta and venous cannulas of 24 Fr were placed in the superior and inferior vena cava, and cardio-pulmonary bypass was initiated. Thereafter, a left ventricular vent catheter (18 Fr) was placed in the right pulmonary vein. The wall of the aorta was severely thickened and a saccular aortic aneurysm with calcification was observed in the aortic arch. There was evidence of widespread inflammation that extended to the right brachiocephalic artery, the left common carotid artery, and the left subclavian artery. An incision of the aortic aneurysm revealed viscous blood with infection. Microscopic examination of the blood revealed

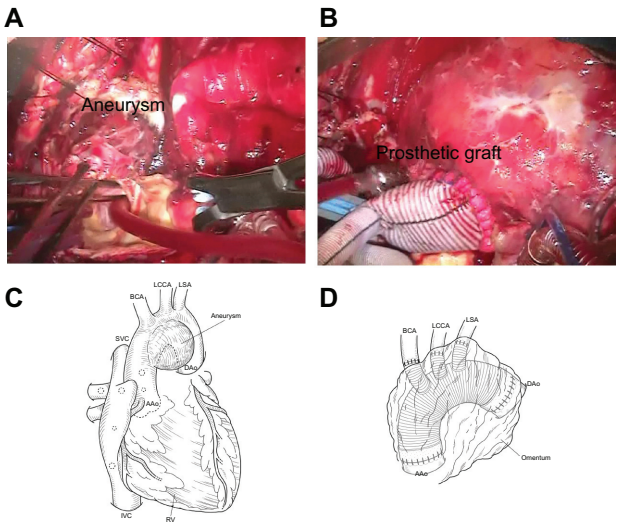

Figure 3. Surgical findings. The thoracic aortic aneurysm with infection was exposed (**A**). The aneurysm was resected and the rifampicin-soaked prosthetic graft was placed (**B**). Surgical findings of the scheme revealed a saccular aortic aneurysm with calcification at the aortic arch (**C**). A rifampicin-soaked prosthetic graft was placed and the omentum was packed into the aortic aneurysm and around the circumference of the prosthetic graft (**D**).

gram-positive bacteria. A diagnosis of infected aortic rupture was confirmed, and the placement of a prosthetic graft and omental packing were then planned. For performing antegrade selective cerebral perfusion, aortic cannulas of 16 Fr, 12 Fr, and 12 Fr were inserted into the right brachiocephalic artery, the left common carotid artery, and the left subclavian artery, respectively. After resection of the thrombus in the aortic aneurysm, the phrenic and left recurrent nerves were revealed. A pseudo aneurysm was suspected due to ruptured infected aortic aneurysm; however, viscous tissues were observed attached to the aortic wall and were resected. Consequently, the phrenic nerve was partially exposed. A prosthetic graft was selected (Gelweave Siena collared 4 branch plexus; Vascutek, Renfrewshire, UK), soaked in rifampicin, and sewn into the wall of the descending aorta. Thereafter, the ascending aorta was incised near its trunk, approximately 1 cm below the sinotubular junction. The thickened aortic wall with infection was subsequently resected. Next a rifampicin-soaked graft strip was placed circumferentially around the surface of ascending aorta and sewn to the ascending aorta. The prosthetic graft was sewn to the right brachiocephalic artery, the left common carotid artery, and the left subclavian artery. Finally, the omentum was packed into the aortic aneurysm and around the circumference of the prosthetic graft. Drainage tubes were then placed, and the chest was closed. Total operative time was 11 h and 4 min, with an extracorporeal circulatory time of 4 h 1 min, and circulatory arrest time of 1 h 44 min. Pathological examination of resected specimens revealed increased number of fibrous connective tissue with hemorrhage, proliferation of fibroblastic cells, and infiltration of lymphocytes and neutrophils around the aortic media and adventitia. In addition, there was evidence of neutrophils and abscess formation over a part of the resected specimen; these findings along with the presence of neutrophilic infiltration of the aortic wall and periaortic tissues were compatible with infective arteritis. After surgery, the patient was immediately administered a postoperative regimen of sulbactam/ampicillin (SBT/ABPC; 12 g/day).

On the postoperative day 3, a culture medium from the resected specimen of the aortic aneurysm revealed *L. monocytogenes*, which was sensitive to SBT/ABPC. On postoperative day 23, C-reactive protein levels had normalized to 0.26 mg/dL. The patient's postoperative course was uneventful, and he was ambulatory at discharge. The patient is now alive and visits our office regularly.

Discussion

In 1885, Osler first described a case of a ruptured infected aortic arch aneurysm complicated with infective endocarditis. An infected aneurysm comprises 0.85%–3% of aortic surgery[5,6] and tends to enlarge rapidly due to the fragility of the aortic wall. This category of aneurysm may easily rupture, with a risk of rupture as high as 50%–85%.[7] The mortality rate is as high as 23.5%–37%,[8] which is higher than the mortality rate of a non-infected aortic aneurysm.

The recommended surgical treatment for an infected aortic aneurysm includes resection of the aneurysm and abscess, followed by prosthetic graft replacement and omental packing. The surgical treatment of an infected aneurysm is associated with a mortality rate as high as 11%, attributable to both the invasive nature of the surgery as well as the poor physical condition of the patient.[9] Conversely, the surgical treatment for an infected aortic aneurysm employs the placement of aortic stent grafts more frequently.[10,11] Furthermore, percutaneous stent-graft placement is less invasive than surgical treatment;

therefore, the use of a stent-graft may be indicated for the elderly as well as for patients with poor performance status. Recent reports have confirmed the successful treatment of ruptured infected thoracic aneurysms with percutaneous stent-graft placement.[12,13] In our case, there was rapid enlargement of the cardiac shadow, and the aortic arch was revealed in plain chest radiograph within a few days of admission. A ruptured thoracic aortic aneurysm and cardiac tamponade were therefore suspected; however, an infected aortic aneurysm was not suspected. Although the patient's hemodynamics was stable at this time, the possibility of a rapidly deteriorating hemodynamic status was considered likely; therefore, emergent surgical intervention was necessary. We considered open heart surgery was more feasible than endovascular approach because our case of thoracic aortic aneurysm was accompanied by pericardial effusion, which was suspected signs of cardiac tamponade.

Because infected aneurysms frequently carry a high risk of rupture due to fragility, it is recommended that infected aneurysms should be surgically treated within 24 h after diagnosis, irrespective of the presence or absence of infection.[5] On the contrary, even if an infected aneurysm ruptures, it remains sealed as a pseudoaneurysm with preservation of hemodynamic stability; thus, some authors recommend that surgical treatment should be performed after infection is controlled by the administration of antibiotic therapy.[14] In our case, however, surgical findings during emergent surgery revealed a ruptured infected aneurysm; hence, an antibiotic-soaked prosthetic graft was used. This method should be a recommended measure to prevent further infection. With regards to infection-sensitive prosthetic grafts, rifampicin-soaked prosthetic grafts, homo-grafts, and polytetrafluoroethylene (PTFE) prosthetic grafts are commonly used. In addition, some authors report that rifampicin is sensitive to *L. monocytogenes*.[15] The use of an antibiotic-soaked prosthetic graft is known to be an effective measure for the treatment of an infected aneurysm,[16] decreasing mortality to 18.2% when used in the surgical treatment of an infected aortic aneurysm.[17]

The first line of Listeriosis treatment is the administration of broad spectrum penicillins and the co-administration of aminoglycosides, which have synergetic effects. As *L. monocytogenes* is an intracellular organism, rifampicin or trimethoprim-sulfamethoxazole, which transfer into cells, are effective. Cephem antibiotics, however, are ineffective.[18]

We reviewed 13 case reports of aortic aneurysms infected with *L. monocytogenes* that were reported from 1965–2012 (Table 2).[19–29] Although case reports involving stent grafts infected with *L. monocytogenes* were sporadically documented,[30–32] these reports were excluded in our review. The average age at presentation was 79.9 years (70–85 years), and men to women ratio was 11:3. Half of patients described in the case reports had a past history of diabetes mellitus, and some of the patients had liver cirrhosis or chronic liver dysfunction. The anatomical location of the infected aortic aneurysm reported in these cases varied: the abdominal aorta (nine cases; 64.3%); the thoracic aorta (five cases; 35.7%); the common iliac artery; and the popliteal artery. The chief complaint among the patients reviewed included fever (nine cases; 64.3%) and pain (six cases; 42.9%). However, no symptoms were present in the patient presented in this case report. This indicates that aneurysms infected with *L. monocytogenes* sometimes lack fever and pain, and thus, we should be careful during examination of such patients. Only three cases (30.0%) were positive for *L. monocytogenes* in blood cultures. In most cases, *L. monocytogenes* was not detected in blood cultures. Ten cases (71.4%) were treated surgically. Although infected aneurysms are occasionally complicated with infective endocarditis, aortic aneurysms infected with *L. monocytogenes* were not complicated with infective endocarditis, except in one case.

The mechanism by which an aortic aneurysm gets infected with *L. monocytogenes* begins when the organism migrates from the human intestine into the bloodstream and spreads hematogenously to adhere to the endocardium or the aortic wall. In the state of infective endocarditis, bacterial ooze of *L. monocytogenes* in the blood is attached to the aortic wall. Subsequently, *L. monocytogenes* spreads locally from cell-to-cell among the surrounding periaortic tissues.

In our case, the aortic aneurysm rapidly progressed and ruptured a few days after admission. Fortunately, the aneurysm in this patient was discovered in a contained status before complete rupture;

Table 2. Cases of an aortic aneurysm infected with *L. monocytogenes.*

References	Age	Sex	Risk factors	Type	Signs		Diagnosis	Surgery	Outcome
					Fever	Pain	Blood culture		
Navarrete–reyna et al[19]	79	F	Diabetes mellitus, hypertension, OMI	TAA	+	–	–	–	Died
Krol–van Straaten et al[20]	79	M	History of tuberculosis	AAA	–	–	–	+	Died
Gauto et al[21]	75	F	Diabetes mellitus	AAA	+	+	+	+	Died
Gauto et al[21]	85	M	Radiation therapy	AAA	+	–	+	–	Died
Gauto et al[21]	70	M	Hepatitis	AAA	+	+	–	+	Survived
Lamothe et al[22]	83	M	Diabetes mellitus	TAA	–	–	–	+	Survived
Clouse et al[23]	80	M	Diabetes mellitus, hypertension, IHD, COPD	AAA, CIA	+	–	–	+	Survived
Barkhordarian et al[24]	83	M	Gastric ulcer	TAA	–	–	NA	+	Survived
Goddeeris et al[25]	77	F	Hypertension, IHD	AAA	–	+	NA	+	Survived
Kida et al[26]	71	M	Alcholic hepatitis, hypertension chronic renal failure	Stanford IIIb	+	–	+	–	Survived
Papavassiliou et al[27]	72	M	Diabetes mellitus, atrial fibrillation peripheral artery disease, COPD	PAA	+	+	–	+	Survived
Sakamoto et al[28]	76	M	None	AAA	+	+	NA	+	Survived
Otoba et al[29]	72	M	Diabetes mellitus, alcholic hepatitis chronic pancreatitis	AAA	+	+	–	–	Survived
Present report, 2012	75	M	Diabetes mellitus, alcholic hepatitis colon cancer	TAA	–	–	NA	+	Survived

Abbreviations: OMI, old myocardial infarction; IHD, ischemic heart disease; COPD, chronic obstructive pulmonary disease; TAA, thoracic aortic aneurysm; AAA, abdominal aortic aneurysm; CIA, common iliac aneurysm; PAA, popliteal aortic aneurysm; NA, not available.

therefore, the circulatory hemodynamics of this patient remained stable. Although the pericardial effusion detected by CT was suspected to be a hematoma, the pericardial effusion detected during surgery was exudative in nature and probably the result of an inflammatory response triggered by the underlying infectious process.

Conclusions

This report presents a rare case of a thoracic aortic aneurysm infected with *L. monocytogenes*. The case presented with mild symptoms; therefore, confirming the diagnosis of infected aortic aneurysm was challenging. Moreover, this case illustrates the importance of careful evaluation when encountering a patient with evidence of pericardial effusion or markedly elevated C-reactive proteins levels. In such cases, a series of regular chest radiographs are necessary to detect acute changes in the cardiopulmonary status of the patient, who may present with no symptoms or with nonspecific complaints.

Author Contributions

Wrote the first draft of the manuscript: SM, NT, MT, KF, YN, TM. Contributed to the writing of the manuscript: SM, NT, MT, KF, YN, TM. Agree with manuscript results and conclusions: SM, NT, MT, KF, YN, TM. Jointly developed the structure and arguments for the paper: SM, NT, MT, KF, YN, TM. Made critical revisions and approved final version: SM, NT, MT, KF, YN, TM. All authors reviewed and approved of the final manuscript.

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Disclosures and Ethics

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