

Listeriosis Infection of an Abdominal Aortic Aneurysm in a Diabetic Patient

Spyros I Papadoulas, Stavros K Kakkos, Pantelis A Kraniotis¹, Maria E Manousi, Markos N Marangos², Ioannis A Tsolakis

Departments of Vascular Surgery, ¹Radiology, ²Internal Medicine, University Hospital of Patras, Patras, Greece

ABSTRACT

A rare case of an abdominal aortic aneurysm (AAA) infected by *Listeria monocytogenes* in a 72-year-old male diabetic farmer, is reported. Our patient had a history of a recent pneumonia that could have been caused by *Listeria* too. Aneurysm infection was manifested by fever and abdominal and back pain, which prompted investigation with CT scanning that revealed a 4.9 cm AAA with typical signs of infection. He underwent urgent AAA repair with aortobifemoral bypass grafting and had an uneventful course. Aneurysm content microbiology revealed *Listeria monocytogenes* and following a 9-week course of antibiotics our patient remains asymptomatic 11 months later.

Key words: Infected aneurysm, *Listeria*, Pneumonia

INTRODUCTION

Listeria monocytogenes can rarely infect atherosclerotic abdominal aortic aneurysms (AAA) in nonimmunocompromised patients.^[1,2] In contrast to Listerial meningitis and bacteraemia, where over 90% of patients have immunosuppression due to neoplasia or other reasons, it has been suggested that patients with Listerial endocarditis and infected arteries must have other types of deficiencies predisposing to infection.^[2] Herein, we present a case of AAA infected by *Listeria monocytogenes* following a recent pneumonia, the first to our knowledge, which could have been caused by the same pathogen. Pulmonary Listeriosis is extremely rare with a handful of cases being reported.^[3,4] Additionally, no more than two dozen cases of infected AAA caused by *Listeria monocytogenes* have been reported in the English literature.

CASE REPORT

A 72-year-old male hospitalized at an outside facility

2 months ago with lower-left lobe pneumonia treated with moxifloxacin for 12 days, and continuing symptoms of malaise, loss of appetite and weight loss, was referred to the emergency department with a symptomatic 4.9 cm AAA on CT scanning causing severe continuous lower abdominal and back pain requiring opiates and recurrent fever, up to 38.5°C. The latter symptom had started 12 days ago, although it had subsided 3 days later after receiving ciprofloxacin. Past medical history included COPD, hypertension, hyperlipidaemia, diabetes mellitus, and hyperuricemia. He was on formoterol and tiotropium inhalers, valsartan/hydrochlorothiazide, barnidipine, moxonidine, ezetimibe/simvastatin, sitagliptin/metformin, and allopurinol. He was an ex-smoker, while his occupation was sheep and goat keeper and slaughterer and he reported to consume raw dairy products, including home-made cheese made from unpasteurized milk. On presentation, he was hemodynamically stable, afebrile, while a tender AAA was noted. Leukocytosis (WBC 16,650/mm³ with a left shift) and raised CRP (29 mg/dL, reference value <0.8 mg/dL) were found. Blood cultures were negative. He underwent an abdominal CTA with contrast ([Figures 1a-c and 2a-b] with a 16 × MDCT scanner (Lightspeed 16, GE, Milwaukee, WI, USA). The nonenhanced scan revealed an infrarenal AAA with a maximum diameter of 4.9 cm, in the axial plane, aortic wall calcifications, some of them apparently discontinuous, and subtle

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Address for correspondence:

Dr. Stavros K Kakkos, E-mail: kakkos@upatras.gr

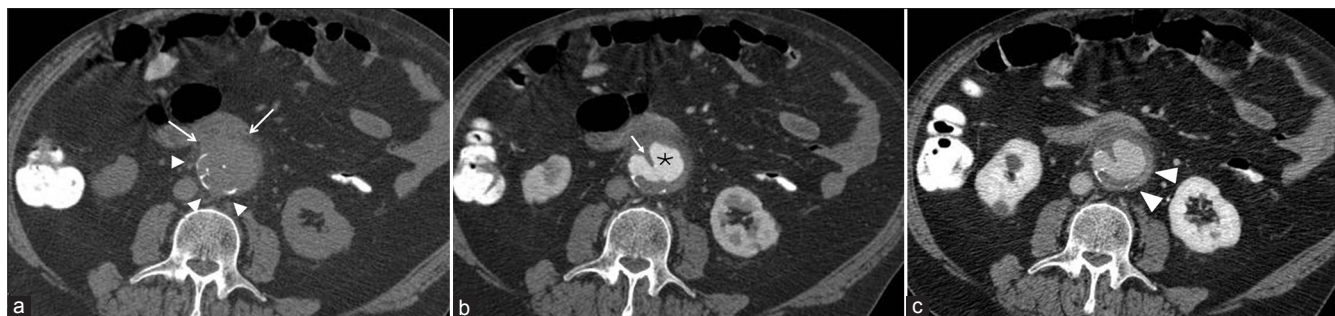


Figure 1: Unenhanced (a), arterial (b), parenchymal (c) axial CT. (a) There is periaortic fat stranding (white arrowheads). The fat plane between aorta and the duodenum is indistinct (white arrows). (b) The aortic lumen is irregular (black asterisk) with an internal flap (white arrow). (c) There is enhancement of the aortic wall (white arrowheads)

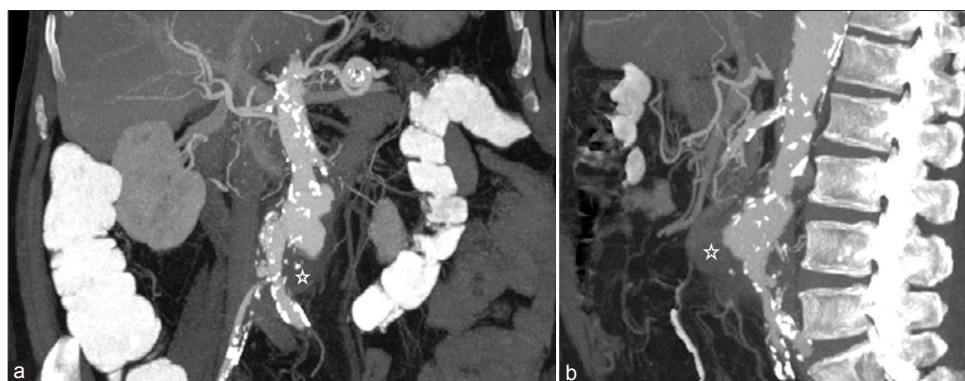


Figure 2: Coronal (a) and sagittal (b) thick-slab reformatted MIP, contrast-enhanced, arterial-phase image of the abdominal aorta. The aortic lumen is irregular, with saccular outpouchings. There is soft tissue attenuation (white asterisk) around the aortic lumen, representing the asymmetrically thickened, inflamed aortic wall

periaortic fat stranding. The contrast-enhanced CT scan underwent multiplanar reformat using the maximum intensity projection (MIP) algorithm. The aortic wall was markedly irregular, with the presence of multiple contrast-filled saccular outpouchings. There was also evidence of periaortic soft-tissue attenuation mass and/or luminal thrombus. The abnormalities extended distally up to the level of the aortic bifurcation, and the proximal part of the right common iliac arteries. There was no evidence of contrast extravasation or hematoma in the retroperitoneal space. There was no evidence of gas in the region. The abdominal CT scan was otherwise unremarkable.

Based on the clinical picture, the history of an infectious disease (pneumonia) and CT findings, an infected AAA was suspected. Our patient was started meropenem 1gr TID and vancomycin 1gr BID and underwent urgent AAA repair with typical aortobifemoral bypass grafting using a 16 × 8 mm bifurcated e-PTFE graft (Advanta SST, Atrium Medical Corp, Hudson, NH, USA). Operative findings included an infrarenal AAA extending to the aortic bifurcation. The right common iliac artery was pulseless. The left common iliac artery had a whitish color. The intestine was adhered densely on the aneurysm. A small amount of dirty fluid

inside the luminal thrombus was sent for culture along with aneurysmal wall samples and thrombus material for common bacteria, mycobacterium, and Brucella pathogens. Periaortic tissue and aortic wall were sent for histological examination. A part of the presumably infected aneurysmal sac was excised. The culture of the dirty fluid was positive for *Listeria monocytogenes*, sensitive to ampicillin, penicillin, vancomycin, teicoplanin, erythromycin, chloramphenicol, and ofloxacin. Additionally, a multisensitive *Staphylococcus epidermidis* was isolated. Histology showed acute sac inflammation. Postoperative course was uneventful and on postoperative day 10, our patient was transferred to his regional hospital to complete a further 4-week course of intravenous antibiotics (meropenem 2gr TID and vancomycin 1gr BID) and an additional 4-week course of oral antibiotics (trimethoprim/sulfamethoxazole 800/160 mg BID) as an outpatient. Eleven months later our patient remains asymptomatic.

DISCUSSION

A rare case of an AAA infected by *Listeria monocytogenes* is reported. Fewer than two dozen cases of infected arterial aneurysms caused by *Listeria monocytogenes* have been

reported in the English Literature.^[2,5] We assume that patient's pneumonia was due to the same pathogen, and we could not find in the literature similar cases of AAAs infected as a result of inoculation from a remote source.

In our patient, we believe that consumption of raw dairy products caused bacteraemia and seeding of avascular AAA contents, that is, thrombus and mural calcifications, although negative blood cultures, like our case, have been previously reported.^[2] We cannot prove if patient's pneumonia, which preceded the clinical manifestations of the infected AAA, was due to *Listeria* after hematogenous spreading, but we strongly suspect that this was the case because the patient never fully recovered. It is known that pulmonary listeriosis is extremely rare.^[3,4] Most likely both AAA infection and pneumonia were caused by the same bacteraemic episode(s) after ingestion of the pathogen and not inhalation of it.^[6]

Occupational risk and diabetes as a form of immunosuppression have possibly played a critical role in pathogen exposure and infection acquirement, respectively. In our area occupational exposure to *Brucella* sp has been reported to cause AAA infection,^[7] but no such mode of Listerial AAA infection has been reported in the literature to the best of our knowledge. On the other hand, occupational infection by *Listeria* sp has been reported by some authors,^[8] although this observation is not uniform.^[9]

Clinical presentation was typical of an infected AAA, while *in situ* repair following a 9-week course of antibiotics proved to be safe since eleven months later our patient remains asymptomatic. In most cases where the local inflammatory findings are not severe, *in situ* repair, as opposed to aortic ligation and extra-anatomical repair by means of axillobifemoral bypass grafting, is indicated,^[10] since its low rate of prosthesis infection outweighs the risk of stump blow-out.

CONCLUSION

A rare case of AAA infected by *Listeria* monocytogenes following a recent pneumonia is reported. The diagnosis of AAA infection should be considered in immunocompromised patients presenting with fever and AAA.

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The first two authors contributed equally.

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