Thoracic Aortitis Due to *Salmonella enteritidis*

Aortitis torácica por Salmonella enteritidis

To the Editor,

Mycotic aortic pseudoaneurysm is a rare clinical disorder that accounts for a very small proportion of all arterial aneurysms. It is characterized by inflammation of the arterial wall due to the presence of microorganisms. Infective endocarditis, one of the pathogenic mechanisms that leads to vascular infection, is a major cause of arterial pseudoaneurysms. Valvular and ventricular aneurysms also occur but they are less common. The clinical manifestations of infectious aortitis are nonspecific and a high index of clinical suspicion is thus necessary to establish a diagnosis. Early diagnosis and prompt initiation of empirical antibiotic therapy are crucial as pseudoaneurysms are associated with rapid growth and consequently a high risk of aortic rupture and death. We present the case of a 70-year-old Caucasian woman with a pseudoaneurysm due to *Salmonella enteritidis* treated with endovascular aneurysm repair (EVAR) and targeted antibiotic therapy. The patient had several cardiovascular risk factors (hypertension, dyslipidemia, and diabetes mellitus type 2) and had visited the emergency department 3 times in the space of 4 weeks reporting physical weakness accompanied by considerable asthenia and generalized myalgias. Her past history included chronic ischemic cardiomyopathy. On her last visit to the emergency department, she continued to show signs of general deterioration but in addition had dysphagia, dysphonia, and episodes of choking. She was admitted to hospital and her laboratory workup showed elevated acute phase reactants (C-reactive protein, 304 mg/L; leukocytes, 14 x 10^3 cells/μL; erythrocyte sedimentation rate, 120 mm/h), accompanied by normocytic and normochromic anemia (hemoglobin, 10.3 g/dL; mean corpuscular hemoglobin concentration, 28.5 pg; mean corpuscular volume, 89 fL). Tumor markers (carcinoembryonic antigen and carbohydrate antigen 19-9) and autoimmune markers (antinuclear antibodies, antinucleoprotein antibodies, and antineutrophil cytoplasmic antibodies) were within normal ranges. A gastroscopy and a computed tomography (CT) scan of the chest, abdomen, and pelvis were ordered to investigate the causes of the dysphagia. The findings of the gastroscopy were unremarkable, but the CT scan showed focal esophageal wall thickening and an aneurysm measuring 16 × 8 mm in the aortic arch. One week after admission, the patient experienced episodes of diarrhea and fever (38 °C) and we therefore ordered blood cultures and a stool examination. Both the blood and stool cultures grew *S enteritidis* that was sensitive to several antibiotics. Despite antimicrobial treatment with ciprofloxacin, the fever persisted and the laboratory studies continued to show elevated C-reactive protein (205 mg/L) and erythrocyte sedimentation rate (106 mm/h). Suspecting an endovascular infection, we ordered a second CT scan, which showed progression of the aortic aneurysm (24 × 15 mm) (Figure A). On confirmation of the diagnosis of a pseudoaneurysm due to *Salmonella enteritidis* and in view of the high comorbidity associated with this condition, the patient was treated with EVAR consisting of the insertion of a Zenith endovascular graft (34 × 77 mm) in the thoracic aorta (Figure B). Following the intervention, the patient was treated with intravenous ceftriaxone and ciprofloxacin for 4 weeks and progressed favorably. Bacteria of the *Salmonella* genus are the main cause of infectious aortitis and have been implicated in 33% to 50% of all cases.[1] Approximately 5% of *Salmonella* gastroenteritis cases are thought to cause bacteremia, which is complicated by arteritis in approximately 1 in 10 cases.[2] Vascular infections due to *Salmonella* species are much more common in the aorta than in other peripheral arteries, and the thoracic zone is involved in 17% of cases.[1] Despite the radical treatment approach, bacterial aortitis is associated with high mortality (up to 36%).[3] The most common risk factors are diabetes mellitus and a history of coronary artery disease and hypertension. The signs and symptoms vary according to the site of the mycotic aneurysm and therefore tend to be nonspecific. The most common manifestations, however, are fever, back pain, abdominal pain, and chills. We have included a review of 30 cases of aortitis caused by *Salmonella* species treated with EVAR since 2003. Of the 30 patients reviewed, 27 (90%) survived and showed no signs of recurrent infection and 3 (10%) died (Table of the supplementary material). There have been recent reports of improved survival rates in patients treated with a combination of EVAR and antibiotics, which can be considered an adequate treatment approach in this setting.[4] The optimal duration of antimicrobial treatment following EVAR, however, is still a topic of debate. While some authors recommend treatment for life,[4] others have

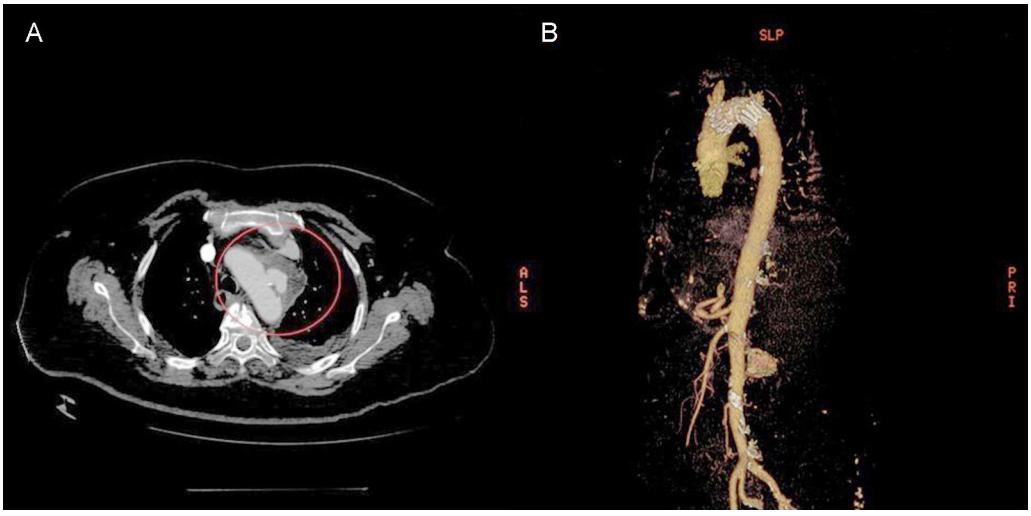

Figura. A, Computed tomography contrast scan showing an infected inflammatory aneurysm measuring 24 × 15 mm in the aortic arch. B, Three-dimensional reconstruction showing endovascular repair following the insertion of a thoracic stent graft.

reported resolution after 12 months.[5] In conclusion, aortitis due to *Salmonella* species is rare, but it can be fatal. A better understanding of the clinical manifestations of this condition is necessary to ensure prompt and effective treatment and improved survival outcomes. EVAR could be a particularly interesting option for patients with a high risk of perioperative complications.

SUPPLEMENTARY MATERIAL

Supplementary material associated with this article can be found in the online version available at doi:10.1016/j.rec.2017.06.021.

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Therapeutic Hypothermia, Propofol, and High Lactate Levels: A Suspicious Combination

Hipotermia terapéutica, propofol y lactato elevado: una combinación sospechosa

To the Editor,

A healthy 59-year-old man experienced sudden cardiac death caused by ventricular fibrillation while jogging in the street. Cardiopulmonary resuscitation was initiated with return of spontaneous circulation after 11 minutes and 4 defibrillations. He had no cardiovascular risk factors or family history of heart disease. He had been running 5 km daily for more than 5 years and had never had any symptoms.

He was intubated and received mechanical ventilation. Initial blood pressure was 154/110 mmHg. Pupils were isochoric and reactive. No myoclonus was observed. The first electrocardiogram showed sinus rhythm at 98 beats per minute, narrow QRS and nonspecific conduction disturbances.

A therapeutic hypothermia protocol was started using the CoolGard 3000 device and Icy catheter. At that moment, the lactate level was 0.9 mmol/L. It took 7 hours to reach 33 °C. Propofol was used at a rate of 0.4-1.2 mg/kg per hour to maintain a bispectral index value between 40 and 60.

Mean blood pressure was higher than 70 mmHg and urine output was about 1.8 mL/kg/h. There was a typical rise and fall of plasma creatinine kinase-isoenzyme MB and troponin I (Figure A). A first 24-hour electroencephalogram showed nonspecific diffuse slow activity and response to all stimuli. The first 24-hour serum neuron-specific enolase level was 14.8 ng/mL. At 6 hours after propofol infusion, blood analysis revealed a lactate level of 4.3 mmol/L, which peaked at 6.1 mmol/L 4 hours later. Due to suspicion of potential propofol toxicity, the drug was discontinued (12 hours after initiation of propofol perfusion). Lactate levels dropped rapidly to a normal range 8 hours later (Figure B). Creatinine kinase activity increased progressively until reaching a peak of 5843 UI/L 48 hours after propofol discontinuation (Figure C). Increased lactate dehydrogenase and transaminase levels were also detected after propofol discontinuation (Figure D).

The patient's clinical course was satisfactory. Coronary angiography was performed, revealing severe 2-vessel disease. A cardiac magnetic resonance scan showed an ejection fraction of 60% with a subendocardial infarct in the territory of the left anterior descending artery. At 3 months, the patient was fully recovered, and gave written consent to this publication.

Increased blood lactate levels may be found in all clinical conditions involving tissular hypoperfusion. The most common type, known as type A lactic acidosis, may be due to conditions that decrease oxygen delivery, usually caused by hypoxemia. Type B lactic acidosis is due to excess demand for oxygen or metabolic problems.

In our patient, systolic heart failure was ruled out due to hemodynamic conditions (mean blood pressure $>$ 70 mmHg, urine output $>$ 1.5 mL/kg/h spontaneously). Intestinal ischemia could reasonably be ruled out, despite the effects of sedation-analgesia, with a soft abdomen and no fecal incontinence. Sepsis and postcardiac arrest syndrome are scenarios in which increased lactate levels occur. However, this immediate effect on increased lactate rules out these causes. The initially normal creatinine-kinase activity made the findings unlikely due to trauma or compartment syndrome. Renal function was not altered, thereby eliminating acute renal failure as a potential cause. Liver function tests demonstrated mild elevated transaminases with a total bilirubin level and parameters of the blood coagulation system within normal ranges. This pattern does not support hepatocellular failure as a cause of increased lactates in our patient. Finally, generalized convulsions, another cause of raised lactate levels, were not observed.

Propofol-related infusion syndrome (PRIS) is a rare but life-threatening complication in patients receiving propofol. Briefly, the pathophysiological mechanism consists of a failure of adenosine triphosphate production by the inhibitory effect at the mitochondrial electron transport chain and the conversion of free fatty acids to acyl-coA, resulting in an increase in lactic acid