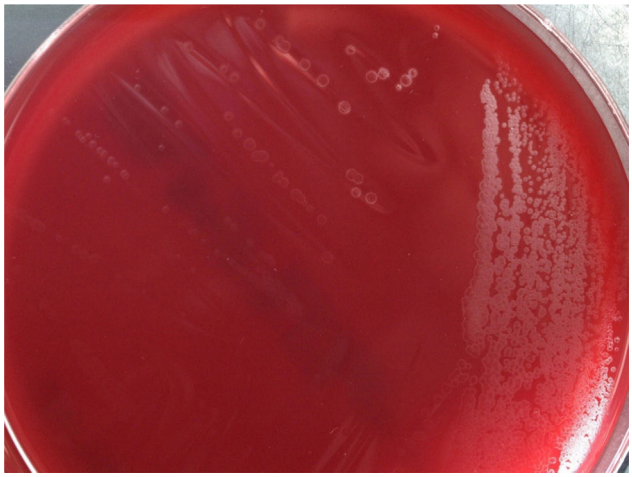

Fig. 1. Appearance of the colonies in blood agar after 24 h of incubation.

The bottle of blood culture was positive after 34 h of incubation. After the Gram stain, which revealed Gram-negative coccobacilli, a subculture was prepared in chocolate agar and blood agar at 37 °C and 5% CO_2. After 24 h of incubation, the colonies grown presented the same morphological and biochemical characteristics as the placenta culture. The automated Vitek 2® System (bioMérieux, Spain) was used to identify both microorganisms, but it failed to do so. Presumptive identification was performed using clinical chemistry tests (negative indole, positive ornithine decarboxylase and prolyl arylamidase, no carbohydrate fermentation). Finally, the 16S ribosomal RNA gene was amplified and sequenced, confirming both bacteria to be *E. corrodens*.

The sensitivity test was performed using antibiotic gradient strips in Müeller-Hinton agar, supplemented with 5% blood and NAD (MH-F, Oxoid). According to the CLSI M45 guidelines,[2] the isolated strains were sensitive to penicillin (MIC: 1 μg/ml), amoxicillin–clavulanic acid (MIC: 1 μg/ml), cefotaxime (MIC: 0.06 μg/ml), erythromycin (MIC: 4 μg/ml), ciprofloxacin (MIC: 0.006 μg/ml), cotrimoxazole (MIC: <0.002 μg/ml) and doxycycline (MIC: 1 μg/ml), intermediate to gentamicin (MIC: 4 μg/ml) and amikacin (MIC: 16 μg/ml), and resistant to clindamycin (MIC: >256 μg/ml) and metronidazole (MIC: >256 μg/ml), consistent with the antibiotic sensitivity described in the bibliography for this microorganism.[1]

The bacteria *E. corrodens* has traditionally been included in the so-called HACEK group of slow-growing, fastidious microorganisms, which are made up of the following genera: *Haemophilus*, *Aggregatibacter*, *Cardiobacterium*, *Eikenella* and *Kingella*.

Penicillin G or amoxicillin-clavulanic acid are considered the treatment of choice. In our case, after obtaining the antibiogram, intravenous penicillin G was administered (50,000 IU/kg/dose every 12 h for 7 days), with favourable progression and sterile blood culture at 5 days of life.

Very few cases of neonatal sepsis caused by *E. corrodens* have been published.[3–5] It is usually isolated in mixed infections, in patients with some degree of immunosuppression. In the case presented, *E. corrodens* chorioamnionitis was caused by genital colonisation of the bacteria during pregnancy, that ascended from the vagina. Some authors have suggested that oral sex during pregnancy may trigger chorioamnionitis by this microorganism.[6–9] It is presumed that the neonatal sepsis was vertically transmitted.

Infections caused by *E. corrodens* may be underestimated because of the bacteria's slow growth and due to the fact that they may be present in mixed infections, thereby hindering their microbiological identification.

Conflicts of interest

The authors declare that they have no conflicts of interest.

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2529-993X/

Intracranial mycotic aneurysm in a 5 month-old infant with pneumococcal meningitis☆

Aneurisma micótico intracraneal en lactante con meningitis neumocócica

A previously healthy 5-month-old boy vaccinated according to the official vaccination schedule (including the 23-valent pneumococcal vaccine) was admitted to the paediatric intensive care unit with symptoms of sepsis and generalised seizures. A lumbar puncture was performed, which showed fluid compatible with bacterial meningitis (1200 leukocytes, 80% neutrophils, glucose 2 mg/dl and proteins 2 g/dl), and Gram-positive diplococci was observed on the Gram stain. Antibiotic treatment was started with cefotaxime (300 mg/kg/day) and vancomycin (60 mg/kg/day). Penicillin-sensitive *Streptococcus pneumoniae* (MIC 0.008 μg/ml) and cefotaxime (MIC 0.012 μg/ml) were isolated in the blood and

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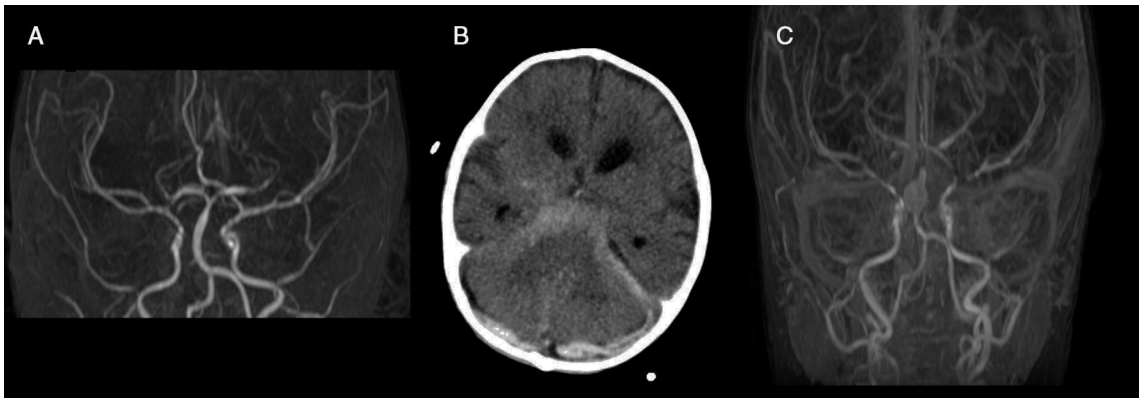

Fig. 1. (A) Image of coronal magnetic resonance angiogram: stenosis and dilations (string of bead) in the middle cerebral artery and basilar artery. (B) CT scan: subarachnoid and subdural haemorrhage. (C) Magnetic resonance angiogram with contrast: aneurysm of the basilar artery.

cerebrospinal fluid and were later identified as serotype 15C, which is not included in the vaccine.

24 h after initiating treatment, the infant was afebrile, with improved general condition and level of consciousness. Vancomycin was suspended. On the third day, the patient deteriorated and seizures reappeared. A cranial CT scan was performed that revealed ventricular enlargement and frontal effusions. An external ventricular drain was placed, which was replaced 3 days later due to obstruction. The infant improved slowly but on the twelfth day of admission he presented sudden deterioration with decreased level of consciousness, abnormal movements, pupillary changes, abnormal heart rate and increased blood flow due to the ventricular drain.

A new cranial CT scan was performed, which revealed ventricular and subarachnoid haemorrhage. With a suspected ruptured aneurysm, an angiography and magnetic resonance angiogram were performed. These tests confirmed the diagnosis of haemorrhage due to ruptured mycotic aneurysm of the basilar artery and multiple stenosis of most of the cerebral arteries studied (Fig. 1). The patient was not eligible for surgery and it was not possible to embolise the aneurysm due to a lack of collateral circulation. As such, life support was withdrawn and the patient died.

The term "mycotic" aneurysm was coined by Osler in 1885 to describe a mushroom-shaped aneurysm in subacute endocarditis.[1] Although some authors use the term "mycotic" to describe infected aneurysms of any aetiology, most limit use of this term to aneurysms that form when material from a distant site causes infection of the vascular wall and subsequent dilation.[2]

The pathogenic mechanism includes septic emboli that occlude the *vasa vasorum*, contiguous spread of infection from other foci of infection, haematogenous infection of the tunica intima or damage to the arterial wall caused by surgery or vascular devices.[3]

Subarachnoid haemorrhage secondary to a ruptured aneurysm is the most common cause of spontaneous subarachnoid haemorrhage in children. However, a recent review by Garg et al. found that out of 62 children with intracranial aneurysms, only 2 were mycotic.[4] Published cases of mycotic aneurysms in infants under the age of 1 year are extremely rare. In children, most mycotic aneurysms occur in the context of endocarditis, underlying vascular malformations, connective tissue disease or iatrogenesis, such as neonates with umbilical catheters.[5]

Before the advent of antibiotics, *Streptococcus pneumoniae* was a common cause of aneurysm infection. Although this fell dramatically with the arrival of penicillin, it may once again be an emerging pathogen in infected aneurysm.[6,7] In a review of the literature, we found 23 published cases of aneurysms (mycotic and infected) caused by *Streptococcus pneumoniae*, all of which were located in the aorta and in adults. We only found one case of a 65-year-old woman who was admitted for pneumococcal meningitis and died one week later due to a ruptured mycotic aneurysm.[8]

There is one published case of mycotic aneurysm and subarachnoid haemorrhage following tuberculous meningitis in an infant with congenital cytomegalovirus infection, in whom the vessels at the base of the cranium surrounded by inflammatory exudate developed infiltrative lesions. The granulomatous process in the subarachnoid space weakens and destroys the middle cerebral artery, causing aneurysmatic dilatation of the artery.[9]

This is the first case of pneumococcal intracranial mycotic aneurysm reported in the literature in infants.

The most important factor to consider in terms of treatment is aneurysm rupture.[10] Unruptured aneurysms can only be treated with antibiotics, whereas ruptured aneurysms must be treated with antibiotics and surgery. Endovascular techniques may be useful in patients with ruptured infected aneurysms to contain the rupture until surgical treatment is viable.

Conflicts of interest

The authors declare that there are no conflicts of interest.

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Mycotic abdominal aortic aneurysm due to *Listeria monocytogenes*☆

Aneurisma micótico abdominal debido a Listeria monocytogenes

Listeria monocytogenes is a rare pathogen that principally affects neonates, pregnant women and immunosuppressed patients. It most commonly causes gastroenteritis in healthy adults and central nervous system infection in immunosuppressed patients. Cardiovascular infection due to *Listeria monocytogenes* is rare and tends to cause endocarditis.[1] This article presents the case of a mycotic aortic aneurysm due to *Listeria monocytogenes*.

A 76-year-old man with a history of hypertension, type 2 diabetes mellitus, dyslipidaemia and ischaemic heart disease attended the emergency department following 2 months of general discomfort, hyporexia, low back pain and 7 kg weight loss in the previous 2 weeks. The patient was afebrile and the physical examination revealed no significant findings. The vascular examination found distal pulses in both lower limbs. Bloods were normal, except for blood glucose at 460 mg/dl. The chest and abdominal X-rays were normal. After normalising the patient's blood glucose levels, he was discharged and referred to internal medicine with a diagnosis of constitutional syndrome to be investigated.

Internal medicine ordered a computed tomography scan (CT scan), which revealed an infrarenal saccular aortic aneurysm. He was admitted to the vascular surgery department, where a CT angiography confirmed the presence of aortic pseudoaneurysm on the left anterolateral side, 8 mm below the renal arteries.

Surgery was performed, with resection and placement of a straight graft (20 mm Silver Graft). Repeat blood cultures taken 6 and 3 days before surgery were negative and afebrile, and a transthoracic echocardiogram found no valve vegetation. An arterial tissue sample was taken during the surgery and sent to Microbiology. The culture in blood agar and chocolate agar showed growth of greyish colonies with beta-haemolysis, identified by Bruker® MALDI-TOF mass spectrometry as *Listeria monocytogenes* with a score of 2.2. The infectious diseases department started intravenous treatment with ampicillin 2 g/4 h and gentamicin 240 mg/24 h. Gentamicin was suspended after 2 weeks, while intravenous ampicillin was maintained for one further week. Sequential therapy was then started with oral amoxicillin 1 g/8 h and the patient was discharged, continuing treatment at home for 3 more weeks. Patient outcome was satisfactory, remaining asymptomatic one year after surgery.

Mycotic aortic aneurysms account for just 1–3% of all aortic aneurysms and may manifest in a previously healthy aorta, although the presence of a prior aneurysm has been reported to be a predisposing factor.[2] The microorganisms most commonly involved are *Salmonella* spp., *Streptococcus* spp. and *Staphylococcus* spp.[3] Aneurysms infected by *Listeria monocytogenes* are very rare.[4] This Gram-positive bacillus is usually transmitted through food. As the main infection pathway is the ingestion of contaminated food, it tends to produce self-limiting symptoms of gastroenteritis in healthy subjects.[5] In rare cases, predominantly in immunosuppressed or elderly patients, it can lead to severe conditions such as sepsis, central nervous system infections and endocarditis.[6] Nevertheless, cases have been reported in immunocompetent patients, and in the case presented here, the only immune deficiency was diabetes mellitus.

Diagnosis is established by contrast CT scan, together with *Listeria monocytogenes* isolation in blood cultures or arterial tissue biopsy.[4] Blood cultures are negative in more than 50% of cases, while the pathogen is isolated in the biopsy in more than 75% of patients.[7] In our case, the blood cultures were negative and *Listeria monocytogenes* was only isolated in the arterial tissue culture extracted during surgery.

The best therapeutic option is the combination of surgery (usually excision of the infected aorta segment and placement of an aortic prosthesis or graft), and prolonged antibiotic therapy.[4] Ampicillin continues to be the treatment of choice, although trimethoprim-sulfamethoxazole, erythromycin, vancomycin or rifampicin are also appropriate treatment options. Treatment duration has not been fully established, but at least 6 weeks is recommended as recurrences have been reported in up to 15% of patients.[5] The outcome of our patient was favourable 12 months after undergoing surgery and receiving intravenous ampicillin and gentamicin, followed by oral amoxicillin for a total of 6 weeks of treatment.

Finally, while excision and replacement by aortic prosthesis or aortic homograft is the most commonly-used procedure, use of endovascular prosthesis is also widely reported in the literature. Most of these cases are elderly patients with significant comorbidity or suprarenal involvement from South-east Asia (the majority involving *Salmonella* spp.). These cases go against the traditional "axiom" of not placing prosthetic material in sites of infection.[8,9]

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