

CEREBRAL ISCHEMIA CAUSED BY *Streptococcus bovis* AORTIC ENDOCARDITIS

Case report

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ABSTRACT - Cerebral ischemic processes associated with infective endocarditis caused by *Streptococcus bovis* are rare; only 2 cases having been reported. Here we report a case of a 50-year-old man with *S. bovis* endocarditis who presented signs of frontal, parietal and occipital lobe cerebral ischemia. This is the first case reported in which the presence of hemianopsia preceded the endocarditis diagnosis. Initially, the clinical manifestations suggested a systemic vasculitis. Later, vegetating lesions were identified in the aortic valve and *S. bovis* grew in blood cultures. Antibiotic use and aortic valve replacement eliminated the infection and ceased thromboembolic events. A videocolonoscopy examination revealed no mucosal lesions as a portal of entry in this case, although such lesions have been encountered in up to 70% of reported cases of *S. bovis* endocarditis.

KEY WORDS: cerebral ischemia, hemianopsia, infectious endocarditis, *Streptococcus bovis*.

Isquemia cerebral causada por endocardite aórtica pelo *Streptococcus bovis*: relato de caso

RESUMO - A associação de isquemia cerebral e endocardite por *Streptococcus bovis* é um evento raro, tendo sido publicados apenas 2 casos anteriormente. Nós relatamos o caso de um homem de 50 anos com endocardite por *S. bovis* que apresentou sinais isquêmicos nos lobos frontal, parietal e occipital. Este é o primeiro caso em que a hemianopsia precedeu o diagnóstico de endocardite. Inicialmente, o quadro foi confundido com vasculite. Posteriormente, foi confirmada a presença de vegetações na válvula aórtica e a hemocultura identificou *S. bovis*. Os eventos tromboembólicos foram controlados com o uso de antibióticos e a troca da válvula aórtica. Estudo videocolonoscópico não identificou nenhuma lesão, apesar de lesões colônicas serem descritas em até 70% dos casos de indivíduos com endocardite por *S. bovis*.

PALAVRAS-CHAVE: isquemia cerebral, hemianopsia, endocardite, *Streptococcus bovis*.

Streptococcus bovis is an important cause of bacteraemia and infectious endocarditis (IE) in adults. It is frequently accompanied by valvular abscess formation and systemic thromboembolism. The bacterium usually attacks healthy cardiac valves and is frequently associated with colon neoplasm and other gastrointestinal mucosal lesions which are believed to provide the portal of entry into the systemic circulation for the *S. bovis*. It is not usually considered to be part of oral or colonic flora.

We report a patient with cerebral ischemia in frontal, parietal and occipital lobe territories caused by *S. bovis* endocarditis.

CASE

A 50-year-old Caucasian man presented with a weight-loss of 3 kg, myalgia, asthenia and dizziness over a three-week period. The subject drank alcoholic beverages in moderate amount and told that he did not smoke or used illicit drugs. Fever was not detected at any moment. Temporal hemianopsia was diagnosed in the right eye. Physical examination revealed a systolic hypertension (150 mmHg), but was otherwise unremarkable. He presented fleeting rash on the left leg, but skin biopsy was inconclusive. The brain MRI showed infarcted areas in left temporal and occipital regions (Figure). Complementary blood tests revealed the following: gamma-GT 80 UI/l; ASP 41 UI/l; ALT 81 UI/l; creatinine 0.9 mg/dl; haemoglobin 12.5 g/dl; leukocyte count 11.5 x 10⁹/l

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Fig. Axial T2 flair MRI image of the brain showing a tumescent lesion, measuring 5.0 x 2.2 cm of extension in the cortical and subcortical regions of the left occipital lobe (arrow). This location suggests an ischemic event in a branch of left posterior cerebral artery.

with 80% neutrophils. Erythrocyte sedimentation rate was 67 mm/h (normal 20 mm/h); and the urine exam was normal. Serologic tests for lupus anticoagulant, anticardiolipin antibodies, ANA, anti-dsDNA, ANCA, HBV and HIV serology were all negative. Rheumatoid factor was positive.

The presumptive initial diagnosis was vasculitis and a prednisone and aspirin 100 mg/day therapy was initiated while study continued on an outpatient basis. Mesenteric artery angiogram did not show any microaneurysm. After 3 weeks, the subject returned with partial improvement of the symptoms. At this occasion, a diastolic murmur on the aortic area and digital clubbing were noted. Transthoracic echocardiography showed deformed leaflets and vegetations on the aortic valve. The left ventricular ejection fraction was 74% and the left ventricle was mildly enlarged (62 mm diameter). Soon afterwards, *S. bovis* grew in 3 blood cultures. Corticosteroid therapy was discontinued, but at that point the patient was hospitalised with fever (39.5°C) and extensive palpable purpura in his legs.

Treatment for his *S. bovis* endocarditis was initiated with intravenous gentamycin, 80 mg three times a day, and intravenous penicillin G, 12 million units daily, and continued for a total of 2 and 4 weeks, respectively. On the ninth day of hospitalisation he presented a new ischemic event in the left frontal lobe. On the sixteenth day of hospitalisation, the aortic valve was replaced with

a biological valve and warfarin was started and eventually continued for 3 months. Pathologic examination of the removed aortic valve disclosed extensive deformities of its leaflets, but no abscess was found. On the twenty-fifth day of hospitalisation day - or the ninth postoperative day - the subject developed atrial fibrillation, which was reverted with electric cardioversion and controlled with amiodarone. Two years after surgery, the patient's only sequel was a partial reduction of the right temporal visual field. A videocolonoscopy was normal.

DISCUSSION

This is the first report of cerebral ischemic infarct preceding the diagnosis of *S. bovis* endocarditis. The case evolution was unusual because of the signs of infective endocarditis (e.g. fever and murmur) appeared after the signs of cerebral infarcts. The cerebral infarcts were initially attributed to systemic vasculitis.

S. bovis represents 2-6% of the streptococci isolated from blood cultures of hospitalised patients¹ and 11-19% of the organisms isolated from patients with infective endocarditis¹⁻⁵. Although there is no gender predominance, about 80% of the patients with *S. bovis* endocarditis are above the age of 50². There seems to be a higher incidence of *S. bovis* endocarditis in patients with liver diseases and in immunosuppressed patients (e.g. those with acute kidney failure, diabetes mellitus, HIV infection, and those on immunosuppressants)⁵⁻⁷. In this case, none of these predisposing factors was present.

Review of published series in the literature revealed a total of 306 cases of *S. bovis* endocarditis^{1-5,7-11}. About 50 to 70.6% of these had no previous cardiac valvular disease. Mortality rate varied from 0 to 75%^{2,5,8-9}, and it was almost always associated with neurological events⁹.

There are no clinical characteristics that distinguish endocarditis caused by *S. bovis* from other aetiologies of infective endocarditis. Fever is usually present in all patients², but in our case it happened late in the natural course of the disease. The approximate time to reach the diagnosis of *S. bovis* endocarditis was 14 days¹².

Another uncommon finding in our case was that the patient did not develop suppurative complications or valvular abscess, despite receiving corticosteroids. The infection by this microorganism can be highly destructive, resulting in valvular per-

foration, cardiac septal or valvular ring abscess² and suppurative meningitis¹¹. Such valvular lesions frequently need surgical repair. The rate of cardiac surgical intervention varied in the previous reports. For example, Kupferwasser et al.¹⁰ reported that 73% of their 22 patients with *S. bovis* endocarditis needed surgery, whereas Carfagna et al.⁸ did not perform any valvular surgical repair, although none of their 14 patients had thromboembolic events to central nervous system.

Neurological manifestations can occur in 18 to 50% of patients of the *S. bovis* endocarditis^{5,9,11}. These included diplopia, hemiplegia, hemiparesis, transient arterial ischemia, amaurosis fugax, convulsion, meningitis and coma^{5,6,9,11}. Our case described here is the first report of ischemic infarct in occipital, parietal and frontal cerebral regions. There is one previous report describing ischemia from the obstruction of the aqueduct of Sylvius⁹, but no details of the case evolution were provided. In our case, the evolution was favourable after the introduction of antibiotic therapy and the successful valvular replacement. One year after the endocarditis, our patient was asymptomatic, except for a partial and discrete right temporal hemianopsia.

There is an association between *S. bovis* endocarditis or bacteraemia and the presence of colonic neoplasm^{2,3,5,7,12,13}. A case-control study¹⁴ showed that the relative risk of developing *S. bovis* endocarditis is 3.6 greater when a colonic tumour is present than when it is absent. Search for colonic lesions in these patients can disclose the presence of premalignant lesions, benign polyps, lymphoma, colitis, mechanical abnormalities, adenoma and adenocarcinoma^{1-3,5,7,14,15}. In our case, no colonic lesions were detected by a video-colonoscopy.

In summary, this case illustrates that one needs to have a high index of suspicion for endocarditis

in cases presenting with cerebral events, even without the common IE symptoms (fever, cardiac murmur).

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