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*.

*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 IU/l; creatinine 0.9 mg/dl; haemoglobin 12.5 g/dl; leukocyte count 11.5 x 10\(^9\)/l.
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, anti-cardiolipin 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 angiotomography 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. 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).

Review of published series in the literature revealed a total of 306 cases of *S. bovis* endocarditis. About 50 to 70.6% of these had no previous cardiac valvular disease. Mortality rate varied from 0 to 75% and was almost always associated with neurological events.

There are no clinical characteristics that distinguish endocarditis caused by *S. bovis* from other aetiologies of infective endocarditis. Fever is usually present in all patients, 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 days.

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-
formation, cardiac septal or valvular ring abscess and suppurative meningitis. Such valvular lesions frequently need surgical repair. The rate of cardiac surgical intervention varied in the previous reports. For example, Kuperwasser et al. reported that 73% of their 22 patients with S. bovis endocarditis needed surgery, whereas Carfagna et al. 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. These included diplopia, hemiplegia, hemiparesis, transient arterial ischemia, amaurosis fugax, convolution, meningitis and coma. 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 Sylvius, 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 and enterococcal endocarditis. A case-control study 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. 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).

REFERENCES