Infective Endocarditis Complicated with Cerebral Infarction and Osler’s Nodes

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A 25-year-old man had a 6-year history of rheumatic heart disease with mitral regurgitation. He had experienced persistent reddish and painful Osler’s nodes on the fingertips for 2 months. One week before admission, he developed an acute onset of slurred speech. Physical examination showed a grade V/VI pansystolic murmur at the cardiac apex with transmission to the left axillary region. Blood cultures revealed *Streptococcus viridans* infection. Echocardiography showed a vegetative growth over the mitral leaflet of about 1.0 × 0.5 cm, with severe mitral valve regurgitation. Magnetic resonance imaging of the patient’s brain revealed multiple lesions scattered primarily in the cortical layers of both cerebral hemispheres and cerebellum, with a bright signal intensity on diffusion-weighted and contrast-enhanced T1-weighted images. A diagnosis was made of infective endocarditis with cerebral infarction and Osler’s nodes. Emergency cardiotomy, debridement, and mitral valve replacement were performed. After surgery, he received anticoagulation therapy (5 mg/day coumadin) and intravenous penicillin G (24 million units per day) for 6 weeks, and recovered without sequelae.

Key words: cerebral infarction, infective endocarditis, Osler’s node, *Streptococcus viridans*

INTRODUCTION

Systemic embolization occurs in 22%-50% of patients with infective endocarditis. Emboli often involve major arterial beds, including the brain, lungs, coronary arteries, spleen, bowel, and extremities. Up to 65% of the embolic events involve the central nervous system and more than 90% of such emboli lodge in the distribution bed of the middle cerebral artery1. Osler’s nodes are a form of peripheral embolic lesion in patients with infective endocarditis, and occur infrequently during the course of the disease2. However, few cases of infective endocarditis combining cerebral infarction with Osler’s nodes have been reported recently in Taiwan1. A lower mortality rate has been reported if early surgical intervention is performed for infective endocarditis with neurological complications3.

Here we report a case of rheumatic heart disease with mitral regurgitation complicated with infective endocarditis, concomitant with multiple cerebral embolic infarctions and Osler’s nodes. After surgery and postoperative antibiotic therapy, the patient recovered without sequelae.

CASE REPORT

A 25-year-old man had a 6-year history of rheumatic heart disease with mitral valve regurgitation. He had no history of cardiac or cranial surgery, and no history of dental surgery, intravenous drug use, or recent skin infections. He was admitted because of swelling, reddish, and painful nodes on all his fingers and intermittent episodes of fever, chills, and general malaise for 2 months. He had also developed a sudden onset of slurred speech for 1 week before admission.

On admission, a physical examination found that the patient had a body temperature of 37.5°C, a pulse rate of 78/min, a respiratory rate of 18/min, and a blood pressure of 120/70 mmHg; the heart rhythm was regular. A grade V/VI pansystolic murmur was heard at the cardiac apex with transmission to the left axillary region. Swelling, reddish, and tender nodes were noted on all fingertips (Fig. 1). A
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A neurological examination revealed that the patient showed alert consciousness, mild dysarthria, no aphasia, no facial palsy, no dysphasia, and no motor or sensory limb dysfunctions.

A complete blood count revealed a white blood cell count of 11,370/mm³ with 83.5% neutrophils, 10.3% lymphocytes and 5.5% monocytes, a hemoglobin level of 11.6 g/dL, a hematocrit of 35.0 and a platelet count of 237,000/mm³. The C-reactive protein level was 8.22 mg/dL, and the erythrocyte sedimentation rate was 58 mm/h. An electrocardiogram showed a normal sinus rhythm. Echocardiography showed a growth measuring about 1.0 × 0.5 cm over the mitral leaflet, with severe mitral valve regurgitation and enlargement of the left atrium and ventricle (Fig. 2).

Infective endocarditis with septic embolism was suspected from the serial examinations and clinical features. The patient was therefore treated with intravenous penicillin G (4 million units every 4 h) and gentamycin (80 mg every 8 h). On the second day of hospitalization, brain magnetic resonance imaging revealed multiple lesions scattered primarily in the cortical layers of both cerebral hemispheres and cerebellum, with a bright signal intensity on diffusion-weighted and contrast-enhanced T1-weighted images (Fig. 3). From these imaging characteristics and multiple locations, acute embolic infarctions of cardioovascular origin caused by infective endocarditis were considered most likely.

On day 3 of hospitalization, the patient’s symptoms of slurred speech got worse and he developed limb weakness. Repetitive embolization even under antibiotic therapy was considered likely, so emergency cardiotomy was performed. The operative findings showed thickening and shrinkage of both mitral valve leaflets, many vegetative growths over the mitral leaflets, and a small growth over the non-coronary cusp of the aortic valve. Because of the rheumatic changes and massive destruction of the mitral leaflets, we carried out total valve replacement using a 33 mm metal valve (CarboMedics Y-H-Z, USA), aortotomy, debridement, and obliteration of the left auricle.

Blood cultures showed the presence of a Streptococcus viridans group bacterium, but cultures from the vegetative growths showed no bacterial growth. We maintained anticoagulation therapy postoperatively (coumadin 5 mg daily) and continuous antibiotic treatment with intravenous penicillin G (24 million units per day) for 6 weeks. The patient was discharged on the 43rd hospital day without dysarthria or fever. His motor and sensory functions were normal at discharge. He continued to receive oral anticoagulation therapy regularly and has shown no sequelae.

**DISCUSSION**

Factors that increase the risk of mortality related to infective endocarditis are being aged more than 60 years, a diagnosis of staphylococcal infection, aortic valve involvement, prosthetic valve involvement, congestive heart failure, embolic phenomenon, and neurological complications. The overall mortality rate from infective endocarditis is 26%-40% and rises to 50%-100% in high-risk groups. Staphylococcus aureus, Staphylococcus epidermidis, Streptococcus pneumoniae, Streptococcus viridans, Group D streptococci, and beta-hemolytic streptococci are the major causative organisms in native and prosthetic valve infective endocarditis. Staphylococcus aureus infective endocarditis presents with an acute onset, a rapid, and progressive clinical course, whereas Strepto-
infective endocarditis presents with a subacute and indolent clinical picture\(^8\). The blood cultures from the patient showed evidence of \textit{S. viridans} infection that was compatible with his subacute clinical course\(^6,9,11\).

The average overall incidence of neurological complications in patients with infective endocarditis is 30%, with almost all of these complications in patients with left-sided valvular disease\(^12\). Embolic events are more common in patients with vegetative growths identified echocardiographically, especially with those growths greater than 10 mm in diameter\(^9\).

Infective endocarditis is a complex disease, often requiring both medical and surgical therapy\(^13\). The traditional indication for valvular surgery in infective endocarditis to avoid further embolization is that the patient has had two or more major embolic events\(^15\). This indication is arbitrary. It excludes cutaneous embolization, which is common, or embolism occurring before the institution of therapy\(^15\). At present, any chosen strategy for surgical intervention to avoid systemic embolization in cases of infective endocarditis remains specific to the individual patient. Benefit is greatest in the early phase, when embolisms are most likely and when other predictors of a complicated course are found, such as previous repetitive embolization, congestive heart failure, aggressive antibiotic-resistant organisms or prosthetic valve-associated infective endocarditis\(^1\). Surgery should be delayed for 2 to 4 weeks in patients with infective endocarditis complicated by recent neurological injury\(^16,17\). However, when repetitive embolization develops, early valve replacement is the only effective treatment to prevent embolic complications in patients with a risk of embolism from endocardial vegetative growths\(^1\). This patient showed rheumatic heart disease with mitral regurgitation that was complicated with infective endocarditis concomitant with persistent embolization: emergency surgery was imperative.

The risk of embolisms developing in patients with infective endocarditis is low (less than 5%) when infection is controlled\(^18\). Low-intensity anticoagulation may indeed be sufficient prophylaxis for many embolism-prone cardiac disorders\(^18\). However, for patients with infective endocarditis on native valves, the risk of hemorrhage seems to be increased if they are on anticoagulation therapy, when cerebral infarction might occur. Anticoagulation medications do not increase the risk of primary hemorrhage (mycotic aneurysms or septic arthritis), but they may increase the risk of hemorrhagic transformation of a brain infarct\(^19\). This patient, therefore, did not receive preoperative anticoagulation therapy and we opted for emergency surgery to remove the source of the emboli from the heart. Anticoagulation therapy was started 2 days after surgery to reduce the risk of embolisms. The patient showed no recurrent cerebral infarctions or symptomatic brain hemmorhages after the use of coumadin.

Peripheral embolic events, including Janeway’s lesions, Osler’s nodes, Roth’s spots, and petechia, occur in 20%-25% of patients with infective endocarditis\(^2\). Gram staining and culture of specimens aspirated from Osler’s nodes are useful in the diagnosis of infective endocarditis\(^20\). Osler’s nodes present most frequently in cases of left-sided infective endocarditis and originate from microvascular septic emboli\(^20\). Osler’s nodes appear in 10%-23% of patients with infective endocarditis, and their presence is noted most often during endocarditis secondary to infection with \textit{Streptococcus viridans}, as in this patient\(^21\). At present, there is no study available to reveal an association between cerebral infarction and Osler’s nodes. Whenever there is cutaneous manifestation such as Osler’s nodes in patients with infective endocarditis, the possibility of multiple embolisms should therefore be kept in mind.

The diagnosis of infective endocarditis remains a challenge to physicians providing primary care. Furthermore, infective endocarditis, particularly in its initial phase,
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often has an uncharacteristic presentation with signs and symptoms shared with many other frequent and often harmless diseases. We must therefore pay close attention to those patients with any of the clinical features of infective endocarditis, such as low-grade fever, cardiac murmur, multiple cerebral infarctions, and occurrence of Osler’s nodes.

REFERENCES


