Native Valve Endocarditis Due to Corynebacterium striatum: First Reported Case of Medical Treatment Alone

The first case of native valve endocarditis due to Corynebacterium striatum required a combination of medical and surgical treatments for cure [1]. We describe the first patient with native valve endocarditis due to C. striatum who received only medical treatment.

A 24-year-old man was admitted to the hospital because of a persistent unexplained fever. His medical history was remarkable for congenital hydrocephalus that led to complete paraplegia and required a ventriculoperitoneal shunt at the age of 2 months. The shunt catheter was replaced when he was 16 years old because the distal extremity had migrated into the pulmonary artery. He had had an isolated fever 7 weeks before the current admission.

Providencia stuartii, C. striatum, and Escherichia coli were successively isolated from cultures of urine. The patient received therapy with ceftriaxone and cefixime for 1 week each; his fever resolved with therapy, but it returned as soon as the antibiotics were discontinued. A chest radiograph obtained 8 days before admission revealed a localized alveolar infiltrate in the lower lobe of the left lung. On admission, the alveolar infiltrate was not apparent.

C. striatum was isolated in three sets of blood cultures. A trans-thoracic echocardiogram revealed a 10-mm vegetation on the pulmonary valve that was close to the distal extremity of the ventricular shunt catheter and that was fluttering in the pulmonary artery. A transesophageal echocardiogram confirmed the pulmonary valve vegetation and did not reveal vegetation on the catheter.

The MICs and MBCs of amoxicillin, ceftriaxone, and netilmicin were 0.25 µg/mL and 0.25 µg/mL, respectively. The MICs of vancomycin and teicoplanin were 0.25 µg/mL and 0.25 µg/mL, respectively, and the MBCs of these drugs were 2 µg/mL and 0.25 µg/mL, respectively. The patient’s initial treatment included amoxicillin and netilmicin. Therapy with netilmicin was discontinued after 2 weeks. A sacral bedsore, which was considered the portal of entry of C. striatum, was treated surgically.

Six weeks after the treatment was started, while the patient was still receiving iv amoxicillin, he developed a sudden fever, chills, thoracic pain, and dyspnea. A localized alveolar infiltrate in the lower segment of the right lung was noted on a chest radiograph. Three sets of blood cultures remained sterile. A transthoracic echocardiogram revealed a 10-mm vegetation on the pulmonary valve that was close to the distal extremity of the ventricular shunt catheter and that was fluttering in the pulmonary artery. A transesophageal echocardiogram confirmed the pulmonary valve vegetation and did not reveal vegetation on the catheter.

The plots for A, B, and C are superimposed.

Figure 1. The effect of antibiotics alone or in combination on the Corynebacterium striatum strain isolated from a patient with native valve endocarditis as determined on the basis of bactericidal kinetics. ▲ = control; ■ = amoxicillin (1 mg/L); ○ = teicoplanin (0.25 mg/L); □ = netilmicin (0.06 mg/L); ● = teicoplanin (0.25 mg/L) + netilmicin (0.06 mg/L); □ = amoxicillin (1 mg/L) + teicoplanin (0.25 mg/L); △ = amoxicillin (1 mg/L) + netilmicin (0.06 mg/L). The plots for ▲, ●, and △ are superimposed.

References

transesophageal echocardiogram did not reveal any vegetations. Three sets of routine blood cultures yielded the strain that was initially isolated (C. striatum). This episode of bacteremia was interpreted as being due to the persistence of organisms on the ventriculoatrial shunt catheter. As surgery was not performed, the patient was discharged from the hospital with instructions to continue his long-term oral antibiotic therapy.

Cases of endocarditis due to Corynebacterium other than C. diphtheriae were recently reviewed [1, 2]. Most patients were treated with penicillin or ampicillin alone or in combination with gentamicin, with the exception of patients with endocarditis due to Corynebacterium group JK, which required treatment with a glycopeptide alone or with a glycopeptide combined with gentamicin [1]. Few cases of C. striatum infections have been reported [3], and only one case of native valve endocarditis due to C. striatum has been well documented [1].

Most of the isolated strains of C. striatum appear to be susceptible to a wide variety of antibiotics, including penicillin G, vancomycin, gentamicin, rifampin, and ciprofloxacin. In one case [3], a strain that was resistant to penicillin G and ciprofloxacin was responsible for central venous catheter-related infection during bone marrow transplantation. The patient was cured after the catheter was removed and amikacin and vancomycin were administered. Another strain that was resistant to many agents (i.e., rifampin, ciprofloxacin, and erythromycin) was responsible for a fatal pulmonary infection in a young man who was recovering from a cerebral hemorrhage in an intensive care unit [4].

Few synergy tests have been performed with C. striatum. The fractional inhibitory and fractional bactericidal indexes, which were determined for the strain responsible for endocarditis [1], showed slight synergy between ampicillin and gentamicin. There was no evidence of synergy with the combination of vancomycin and gentamicin. In vitro time-kill curves have been shown to be predictive of therapeutic results in experimental endocarditis [5]. Time-kill curves plotted for the strain isolated from our patient (figure 1) showed that the most rapid killing occurred with netilmicin alone. The addition of amoxicillin or teicoplanin or both did not increase the rate of killing obtained with netilmicin. This finding is consistent with findings of the few time-kill studies performed with corynebacteria other than C. diphtheriae isolated from patients with endocarditis [6, 7].

More information is needed concerning medical treatment of C. striatum infections since few cases of these infections have been reported. Nevertheless, the findings in our case suggest that a combination regimen that includes an aminoglycoside could help control this difficult-to-treat infection.

Pierre Tattevin, Anne-Claude Crémiex, Claudette Maller-Serieys, and Claude Carbon
Service de Médecine Interne and Service de Microbiologie, Hôpital Bichat-Claude Bernard, Paris, France

References