Acinetobacter Endocarditis in a Chronic Hemodialysis Patient

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Abstract

We report a 63-year-old patient with diabetes mellitus and end-stage renal disease on hemodialysis who was admitted for fever. A transthoracic echocardiogram revealed a large vegetation on the mitral valve. Blood cultures yielded *Acinetobacter baumannii*. Right parietal hemorrhage developed on the fourth hospital day. This is probably the first reported case of *Acinetobacter* endocarditis with cerebral embolism in a chronic hemodialysis patient.

KEY WORDS: *Acinetobacter baumannii*, bacteremia, cerebral emboli, infective endocarditis, hemodialysis

Introduction

The incidence of bacteremia in hemodialysis patients has been reported to range from 0.93 to 1.2 episodes per 100 patient months (1, 2). In 3-4% of bacteraemic episodes, the causative pathogen involved is *Acinetobacter* spp. (3, 4) The incidence of infective endocarditis (IE) complicating bacteremic episodes in hemodialysis patients ranges from 1.6% to 12% (1-4). The most common organisms are gram-positive pathogens including *Staphylococcus aureus* (range, 40-76.6%) and *Enterococcus* sp (range, 10-30%) (5-8). Metastatic septic emboli in the central nervous system are not common (5-7). We could find no previous report of *Acinetobacter* endocarditis with cerebral emboli in the hemodialysis population. We present the first case.

Case Report

The patient was a 63-year-old man with hypertension, type II diabetes mellitus, previous ischemic stroke with dysarthria and mild right-side hemiparesis, coronary artery disease, three-vessel disease status post-coronary artery bypass graft and end-stage renal disease secondary to diabetes on maintenance hemodialysis. Hemodialysis had been performed via a right internal jugular vein cuffed catheter, which was implanted one year prior to this admission. A left arteriovenous fistula over the upper arm was created nine months before admission, but it was used only once because of dysfunction and then left in place.

Two months prior to this admission, the patient was referred to our emergency room for fever and chills during the last hemodialysis session at a local dialysis unit. One of the two sets of blood cultures yielded *Acinetobacter baumannii*. Catheter-related infection was suspected and the cuffed catheter was then removed. The left arteriovenous fistula was used instead and endovascular intervention was successfully performed once. However, general malaise and anorexia persisted. One week prior to this admission, fever occurred again. Intravenous gentamicin sulfate and cephradine arginine were given empirically in the dialysis unit. One set of blood cultures yielded *Acinetobacter baumannii* again, which prompted the present emergency room visit. His initial vital signs showed a blood pressure of 121/59 mmHg, heart rate of 97 beats per minute, respiratory rate of 20 per minute and body temperature of 37.5°C. Pansystolic murmurs grade III/VI over the mitral area were heard on physical examination. The hemogram showed a white blood cell count of 16,080 cells/mL with 87.9% neutrophils. His serum C-reactive protein level was 14.8 mg/dL and procalcitonin was 2.0 ng/mL. He also reported general weakness, drowsiness and right-side
weakness. The initial computed tomography (CT) of the brain yielded no significant findings. A transthoracic echocardiogram (TTE) revealed a huge 1.64 × 1.07 cm vegetation on the mitral valve (Fig. 1). Two separate sets of venous blood cultures yielded *Acinetobacter baumannii*. Intravenous antibiotics with ampicillin/sulbactam 3000 mg once daily and amikacin sulfate 200 mg three times a week were given. Surgical valve replacement therapy was suggested but the patient and family refused. Loss of consciousness, bilateral eye deviation to the right and left-limb weakness developed on the fourth hospital day. Emergency brain CT imaging showed a hemorrhage over the right parietal region (Fig. 2). A craniectomy with hematoma removal was performed on the same day. To treat a possible intracranial infection, imipenem/cilastatin 250 mg three times a day was prescribed instead of ampicillin/sulbactam. Antibiotics were shifted to colistin methanesulfonate 33.4 mg once daily because of severe diarrhea. Follow-up brain CT imaging did not show an infectious focus. However, imipenem/cilastatin was resumed because of recurrent fever. The fever subsided subsequently. A follow-up TTE showed that the mitral valve vegetation had decreased to 0.7 × 0.99 cm, with decreased mobility and elevated echogenicity. The patient was treated with a 77-day course of intravenous antibiotics and was discharged after he had no fever for ten consecutive days.

**Discussion**

*Acinetobacter baumannii* is a gram-negative, nonmotile coccobacilli. It is ubiquitous in the envi-

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Fig. 1. Transthoracic echocardiogram on 2nd hospital day. An oscillating 1.64 × 1.07 cm mass (arrow) at the anterior mitral valve leaflet, with eccentric moderate mitral regurgitation.

Fig. 2. Computed tomography scan of the brain in the axial view. A. On admission, with non-contrast, there is no evidence of intracranial hemorrhage or a midline shift. B. On the fourth hospital day, with contrast, there is a hemorrhage (arrow) over the right parietal area with a perifocal edematous zone (arrow heads) and a deviated midline to the left side.
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Enronment and in the hospital setting (9). Infection is difficult to treat because of its ability to develop resistance to antimicrobials (10). Although *Acinetobacter* spp. have been isolated from various types of infections (9), a few very rare cases of native-valve infective endocarditis have been reported (11-14). Our patient fulfilled one major (positive endocardiogram with new mitral regurgitation) and three minor criteria (fever, intracranial hemorrhage and positive blood culture findings which did not meet a major criterion) of the modified Duke criteria, so the case was defined clinically as definite IE (15).

*Acinetobacter* vegetations are usually located on the surfaces of the mitral and aortic valves, which are common sites of the causative pathogens of common endocarditis in hemodialysis patients (8). Among 17 cases reviewed (11-14), the inciting events were dental work, intravenous drug abuse, septic abortion, stab wounds, burns, intravenous catheters, and open heart surgery. None of these events was noted in our patient except for the hemodialysis catheter in his right internal jugular vein.

An intravascular catheter is a common source of *Acinetobacter baumannii* bacteremia. In a study (16) of hemodialysis patients with bacteremia, 92% of cases were related to a tunneled cuffed catheter. The indwelling dialysis catheter might have been the source of bacteremia in our patient as seen in a recent case (12). However, the catheter was located in the right internal jugular vein and the septic vegetation developed on the mitral valve. We did not find an intracardiac right to left shunt on the echocardiogram. An arteriovenous fistula was another possible source but it was unlikely because no local inflammatory changes were documented in our case.

Hemodialysis itself is a risk factor for IE. The overall proportion of HD patients in a study population of 329 IE patients was as high as 20% (17). A one-year IE French survey (18) showed that the incidence of IE in HD patients was 1.7-2.0 cases/1000 patients, which is 50-60 times higher than the overall incidence of IE in France. Although aging-related valve calcification may play a role in the increased incidence of IE in HD patients, the most important risk factor seems to be the cuffed catheters (19).

Intracranial hemorrhage over the right parietal area developed in this case. No coagulation abnormalities, thrombocytopenia, anticoagulant use or uncontrolled hypertension was noted during hospitalization. Septic emboli-related intracranial hemorrhage was highly suspected although the bacteria culture and subsequent brain imaging revealed no bacteria or abscess. Our patient had a large heart valve vegetation longer than 10 mm, which was described as a risk factor for cerebrovascular complications in one study (20). In another study (21), 9 (7%) of the 133 episodes of native-valve left-sided endocarditis were complicated by symptomatic intracranial hemorrhage which was diagnosed by combined CT and clinical criteria. Mortality was as high as 67% (six of nine) in that study. Septic embolism, specifically an embolus to the brain resulting in stroke, is significantly associated with in-hospital mortality (8). Our case supports the mechanism that cerebral emboli in HD patients with IE occur in patients with left-side endocarditis and large vegetations. Although our patient had poor prognostic factors, his family refused valve replacement therapy. However, no surgical intervention was reported in a study of native-valve endocarditis due to *Acinetobacter* species (11) and a survival rate of 65% was reported.

Generally, imipenem is the most active agent against *Acinetobacter baumannii* (11, 22) Empirical broad-spectrum therapy (e.g., imipenem/cilastatin) for *Acinetobacter* infections should be considered in any patient with IE when gram-negative coccobacilli are isolated from blood cultures (11). In our case, the patient responded well to imipenem/cilastatin, even with failure of other antimicrobial agents.

In conclusion, the incidence of *Acinetobacter* endocarditis in hemodialysis patient is very rare and this is probably the first reported case with cerebral vascular complications. In addition, our report should remind physicians of two points. First, *Acinetobacter baumannii* is one of the causative pathogens in hemodialysis patients with IE. Second, chronic haemodialysis patients appear to be one of the most important subgroups who developed IE as a result of health-care associated procedures.

References

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