Nosocomial *Acinetobacter* Genomic Species 13 TU Endocarditis Following an Endoscopic Procedure

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**Abstract**

We report a rare case of prosthetic valve endocarditis caused by *Acinetobacter* genomic species 13 TU. This patient had rheumatic heart disease and received prosthetic mitral valve replacement eleven years previously. He was admitted due to tarry stool. Endoscopic procedure showed two gastric ulcers and some mucous breaks at the distal esophagus. He had a fever on the eleventh hospital day. Persistent *Acinetobacter* bacteremia was noted with conjunctival hemorrhage. The pathogen was identified as *Acinetobacter* genomic species 13 TU by PCR-based method. According to his whole course of disease, the most possible portal of entry was via the endoscopic procedure.

**Key words:** acinetobacter, endocarditis, endoscopy

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**Introduction**

*Acinetobacter* is a gram-negative bacterium responsible for 1-3% of hospital-acquired infection (1). This pathogen is resistant to many antibiotics and often causes infections of the respiratory, urinary tracts and blood stream (2, 3). Nosocomial bacteremia due to *Acinetobacter* spp. occurred in patients hospitalized in the intensive care unit undergoing invasive procedures, ventilator-associated pneumonia or surgery (2, 4). Cases of endocarditis due to *Acinetobacter* are very rare, especially *Acinetobacter* genomic species 13TU which is one species of *A. calcoaceticus-A. baumannii complex* (4-7). We present a very rare case of prosthetic valve endocarditis caused by *Acinetobacter* genomic species 13TU after the endoscopic procedure for his gastric ulcer.

**Case Report**

A 74-year-old man had been diagnosed to have rheumatic heart disease and received prosthetic mitral valve replacement eleven years previously. He had been taking warfarin to maintain a prolonged prothrombin time. He was admitted due to tarry stool for one week. The endoscope was performed to the duodenal 2nd portion. All lumen was clean without clots or blood. Two small healing ulcers on the surface of greater curvature near pyloric region and some mucous breaks at distal esophagus were found (Fig. 1). Hemostasis was not performed during the endoscopy. Anticoagulant therapy was discontinued and he received vitamin K1, proton pump inhibitor, and hydration. His tarry stool passage resolved gradually.

On the eleventh hospital day, he had fever, chills and tachycardia. No specific infectious focus was detected after the careful examination, including repetitive physical, radiographic and urinary examination. A peripheral hemogram revealed a leukocyte count of 12.58×10⁴/μL with 14% band form neutrophils. Other blood examinations for liver function and renal function were within the normal range.

Empirical antibiotics of ampicillin and gentamicin were used but intermittent fever and chillness persisted. On the fifteenth hospital day, two sets of blood culture yielded *Acinetobacter baumannii*, *Klebsiella pneumoniae* and *Enterococcus faecalis*. His abdominal computer tomography did not show any abnormalities. Antibiotics were shifted to ceftazidime and ampicillin-sulbactam, which were discontinued after a course of 14 days due to afebrility. Fever recurred on the thirtieth hospital day. Two sets of blood cul-
Figure 1. Transthoracic cardioechography showed a thrombus-like mass lesion (arrow) in the left ventricle (a) and peri-valvular leakage (arrow) (c). The endoscopy showed two small healing ulcers (arrows) on the surface of greater curvature near pyloric region (b) and some mucous breaks (arrow) at the distal esophagus (d).

Day 0. The endoscopy was performed due to tarry stool.

Day 11. Fever occurred and the blood culture yielded Enterococcus faecalis, Klebsiella pneumoniae and Acinetobacter baumannii.

Day 30. Fever occurred again and blood culture yielded Acinetobacter baumannii.

Day 33. The blood cultures still yielded Acinetobacter baumannii.

Figure 2. The whole clinical course.

tures yielded Acinetobacter baumannii and the same pathogen was isolated again from the two sets of blood cultures collected after two days. The conjunctiva hemorrhage was noted in the repetitive physical examination. Transthoracic echoechocardiography showed a thrombus-like mass lesion in the left ventricle and mild perivalvular leakage (Fig. 1). Intravenous gentamicin and ciprofloxacin were used on day 31 and fever subsided. The pathogens isolated from the blood cultures collected during this hospitalization were identified as Acinetobacter genomic species 13TU by analysis of the amplified intergenic spacer region of 16S-23S rRNA genes according to the method described by Chang et al (8). After 4-week course of intravenous antibiotics, the antibiotics were shifted to oral ciprofloxacin and trimethoprim-sulfamethoxazole and he was discharged in stable condition on day 61. He remained well after 3 months of follow-up. The whole clinical course is listed in Fig. 2.

Discussion

Members of the genus Acinetobacter are now recognized as important nosocomial pathogens. Acinetobacter baumannii and the unnamed Acinetobacter genomic spp. 3 and 13 TU cause serious problems in the intensive care unit due to their antimicrobial resistance. They are very difficult to separate from A. calcoaceticus by manual and semi-automated commercial identification systems and commonly grouped in A. calcoaceticus-A. baumannii (9, 10). Twenty-five percent of Acinetobacter isolates belonging A. calcoaceticus-A. baumannii complex were misidentified as
A. baumannii by semi-automated commercial identification systems (11). Over 50% of A. baumannii isolates are resistant to currently used antimicrobial agents, such as ampicillin-sulbactam, ceftazidime and ciprofloxacin, while less than 5% of Acinetobacter genomic species 13TU (12). In the present case, A. baumannii was first identified as the pathogen. But it was susceptible to ceftazidime, ciprofloxacin, ceftazidime and trimethoprim-sulfamethoxazole not popular to common A. baumannii. Therefore, a PCR-based method was performed to identify the true species of this pathogen.

In this patient, persistent Acinetobacter spp. bacteremia with fever, subconjunctiva hemorrhage and prosthetic valve fit Duke’s criteria for the endocarditis. Cases of endocarditis due to Acinetobacter are very rare, especially Acinetobacter genomic species 13TU which is one species of A. calcoaceticus-A. baumannii complex (4-7). Endocarditis caused by Acinetobacter spp may involve normal, diseased, or prosthetic valves and usually affects patients with underlying structural heart disease (4, 5, 7). About half of them had risk factors for development of endocarditis, like septic abortion, dental extractions, drug addict and the recipient of an intravascular catheter. Other patients had no predisposing factors. The vegetations were commonly found on aortic or mitral valves. In 9 reviewed cases (4-7, 13) including the current patient, there was a survival rate of 88.9%. Compared with the native valve patients with Acinetobacter endocarditis (5), Acinetobacter endocarditis related to prosthetic valves seems to have a rather good prognosis. This finding is in contrast to the high mortality rates in the patient with prosthetic valve endocarditis caused by other pathogens.

There are many risk factors associated with Acinetobacter spp. bacteremia, like neutropenia, prolonged use of cephalosporins, invasive procedures, central venous catheters, surgery, ventilator support, severe decubital ulcer, parenteral nutrition and presence of a urinary catheter (14-16). Except for the endoscopic procedure, there were not any risk factors for Acinetobacter spp in our patient. Intraoperative contamination of the prosthetic material or perioperative bacteremia seeding the prosthesis is impossible due to the long interval between the timing of onset of infection and operation (17). The polymicrobial pathogens including Acinetobacter genomic species 13TU, Klebsiella pneumoniae and Enterococcus faecalis isolated from the first samples of blood culture suggested the gastrointestinal tract as the portal of entry. Visualization of gastric mucosa requires considerable manipulation of oropharyngeal areas and minor trauma to upper gastrointestinal mucosa often occurs (18, 19). Therefore, this procedure might be followed by an appreciable incidence of bacteremia. The reported incidence of bacteremia after diagnostic upper gastrointestinal endoscopy is about 2-5% (18, 19). In the present case, the panendoscopy may be the most possible even for his Acinetobacter genomic species 13TU endocarditis.

The common pathogens for the bacteremia associated with invasive endoscopic procedures are normal inhabitants of gastrointestinal tract including Streptococcus viridans, Streptococcus pneumoniae, Staphylococcus aureus and Cardiobacterium hominis (18). Acinetobacter is not a common inhabitant of the gastrointestinal tract but often presents on the hands of hospital staff (20, 21) and hospital environmental sources including ventilators, resuscitation bags, arterial pressure transducers, suction catheters, bed rails, mattresses and pillows. Two outbreaks with Acinetobacter 13TU have been reported recently (22, 23). Although a common environmental source for this pathogen was not found in the present case, the most possible source was the endoscope which was contaminated by the hands of staff or the hospital environment (20, 22). To deal with the patients with a high risk for endocarditis, it is more important to notice hand washing and disinfection of the endoscope to prevent this event.

In conclusion, a rare case of nosocomial Acinetobacter 13TU endocarditis occurring after endoscopic procedure is presented. A PCR-based method was beneficial to identify this pathogen. The most possible portal of entry in this patient was the gastrointestinal tract through a contaminated endoscope. Careful hand washing and disinfection of the endoscope is very important to perform endoscopy in patients with a high risk for endocarditis.

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