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Case Report

Achromobacter endocarditis in native cardiac valves — an autopsy case report and review of the literature

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1. Introduction

Achromobacter xylosoxidans is a motile, aerobic, Gram-negative bacterium that causes nosocomial infections such as bacteremia, endocarditis, pneumonia, and wound infections. Most *A. xylosoxidans* infections occur in immunocompromised patients with indwelling catheters [1, 2]. There have been 18 cases of *Achromobacter* spp. endocarditis described in the English literature to date, with majority of the cases having prior cardiac surgeries or severe premorbid valve disease [3–19]. We present an autopsy report of *Achromobacter* endocarditis involving the native valves in a woman with end-stage renal disease (ESRD) on hemodialysis (HD).

2. Case Presentation

The patient was a 66-year-old African–American woman with history of hypertension, diabetes mellitus, and ESRD on HD for 6 months. The patient developed shaking chills and fever during routine dialysis (day 1). The blood culture drawn on day 1 was positive for *Enterobacter agglomerans*, which was susceptible to levofloxacin, ciprofloxacin, meropenem, cefepime, and trimethoprim/sulfamethoxazole, and the patient was given a 7-day course of cefepime IVPB (Fig. 1A). Meanwhile, the patient was afebrile with three successively negative blood cultures, followed by a blood culture positive for *A. xylosoxidans*, susceptible to levofloxacin, ciprofloxacin, piperacillin/tazobactam, trimethoprim/sulfamethoxazole, and meropenem (minimal inhibitory concentration, MIC=2 μg/ml) but resistant to cefepime. After the cefepime treatment was completed, the patient had several episodes of fever; another blood culture drawn on day 15 was negative (Fig. 1A). On day 42, the patient again had fever and developed diarrhea, nausea, and vomiting. A blood culture drawn on that day grew *A. xylosoxidans* susceptible to meropenem (MIC=2 μg/ml) (Fig. 1A). The patient was then admitted on day 44 and started on meropenem for the *A. Xylosoxidans* and vancomycin P.O. for the treatment of colitis as she had a positive *C. difficile* Toxin A and B test. The blood culture drawn on the day of admission was still positive for *A. xylosoxidans*, susceptible to levofloxacin, ciprofloxacin, piperacillin/tazobactam, and trimethoprim/sulfamethoxazole but intermediate to meropenem (MIC=6 μg/ml). Three blood cultures drawn on the 46th, 47th, and 48th day were negative, but the blood culture drawn on the 49th day was positive for *A. xylosoxidans* resistant to meropenem (MIC≥16 μg/ml) (Fig. 1A). The patient was then started on levofloxacin and trimethoprim/sulfamethoxazole.

Troponin I was 4.54 ng/ml (reference <0.01 ng/mL) on the day of admission (day 44) and was persistently high during the hospitalization. The brain-type natriuretic peptide was also elevated (6895 pg/ml; reference. <125 pg/ml). A transesophageal echocardiogram showed mitral regurgitation and vegetation on the mitral valve (Fig. 1B). A plan was made for mitral valve replacement surgery when the patient was sufficiently stabilized. The hospital course was complicated by several episodes of hypotension. On the morning of day 63, the patient was found unresponsive, in cardiopulmonary arrest, and was later pronounced dead after unsuccessful cardiopulmonary resuscitative efforts.

An autopsy was performed 30 h postmortem. On gross examination, the decedent was an overweight African–American woman. The heart was large in size with a cardiothoracic ratio of 1:2 and weight of 560 g (reference 270–360 g). Upon sectioning of the heart, there was a vegetation with associated abscess, measuring 1.5×1.0×1.0 cm, on the anterior leaflet of the mitral valve (Fig. 2A). The base of the abscess showed perforation, through which the vegetation extended to involve the left and posterior leaflets of the aortic valve (Fig. 2B). The mitral and aortic valves were dilated with circumferences of 12.0 cm (reference 8.2–9.1 cm) and 7.5 cm (reference 5.7–6.9 cm), respectively. The valve

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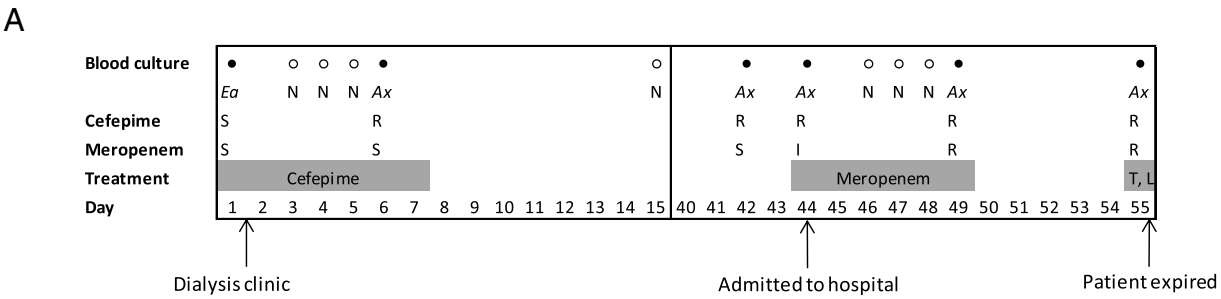

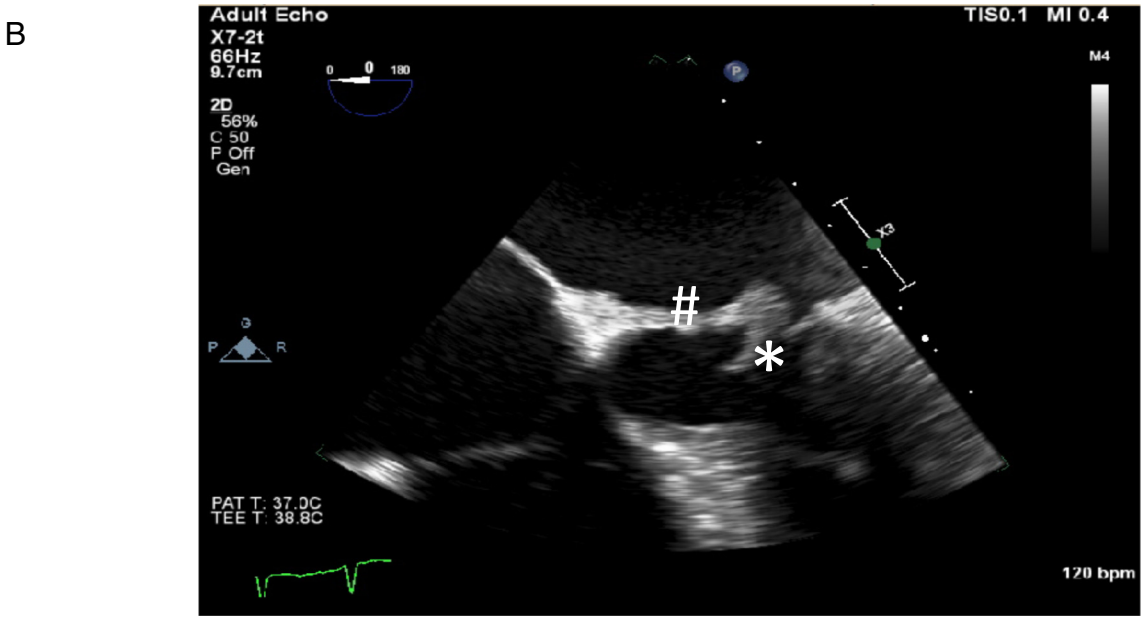

Fig. 1. Clinical findings. (A) Time course for the blood culture results and antibiotics treatments. *Ax, A. xylosoxidans*; *Ea, Enterobacter agglomerans;* N, negative culture; R, resistant; I, intermediate; S, susceptible; TMP-SMX, trimethoprim/sulfamethoxazole; LFX, levofloxacin. (B) Transesophageal echocardiography (TEE) showing long vegetation on mitral leaflet. * indicates the vegetation. # indicates the mitral valve.

leaflets uninvolved by the abscess appeared to be thin and delicate without abnormality. There were an area of hyperemia in the posterior interventricular septum (1.5×0.5 cm) suggestive of an acute myocardial infarction and white discoloration in the apex of the left ventricle consistent with old myocardial infarction. Marked atherosclerotic stenosis of the left anterior descending coronary artery (approximately 80%) and left circumflex coronary artery (approximately 80%) was noted. There was biventricular cardiac hypertrophy with a left ventricular thickness of 1.9 cm and right measuring 0.6 cm. Gross neuropathologic examination revealed hemorrhagic infarcts in the left cerebellar hemisphere, measuring 1.0×0.8×0.4 cm (Fig. 2C), and in the left occipital lobe, measuring 0.2 cm in greatest dimension.

Histological examination of the anterior leaflet of the mitral valve showed acute and chronic necrotizing inflammation (Fig. 3A) with destruction of the collagenous structure of the valve. The left atrial and ventricular wall immediately adjacent to the mitral valve abscess showed scattered acute inflammation, necrosis, and areas of mineralization, heaviest in the atrium adjacent to the mitral valve annulus. The aortic valve showed calcification and acute and chronic inflammation (Fig. 3B). There were areas of myocardial infarction in the posterior interventricular septum (7–12 days old) and in the papillary muscle (3–5 days old). Evidence of remote ischemic damage was noted in the papillary muscle with cardiomyocytes replaced by scar tissue. The lungs showed acute pulmonary congestion with red blood cells in alveoli and pulmonary edema. Postmortem cultures of the mitral valve collected during the autopsy were positive for *A. xylosoxidans* (Fig. 4A–C).

Neuropathology examination revealed embolic infarcts in the cerebellum. In the psoas muscle, there were focal necrosis and hemorrhage mixed with bacterial colonies, most likely due to septic emboli (Fig. 3C).

3. Discussion

Achromobacter xylosoxidans, previously known as *Alcaligenes xylosoxidans,* is a Gram-negative, aerobic, oxidase and catalase-positive, motile rod with peritrichous flagella. *A. xylosoxidans* possesses intrinsic characteristics, like a large genome rich in C-G sequences encoding the biofilm adhesin poly-β-1,6-N-acetyl-D-glucosamin and a range of antibiotic resistance genes coding efflux pump systems and antibiotic modifying enzymes, which enable the organism to be resistant to antibiotics and disinfectants and allow survival in chlorhexidine solutions, ultrasound gel, and intravenous fluids [20], leading to nosocomial infection. Although rare, endocarditis due to *A. xylosoxidans* is often fatal, especially in hosts with impaired immunity, chronic illnesses, or prosthetic devices. Among the 19 reported cases of *Achromobacter* endocarditis, including our case (Table 1), previous cardiac surgeries such as prosthetic cardiac valves or ventricle septal patch repairs are the most common premorbidity (63.2%) followed by valvular heart disease (26.3%). Only four cases, including our case, occurred on native valves without structural abnormalities [9, 11, 12]. Out of the 19 *Achromobacter* endocarditis cases, 7 cases involved the aortic valve, 1 case involved the mitral valve, and 6 cases involved both aortic and mitral valve. Other