CASE REPORT

Successful treatment of Aerococcus viridans endocarditis in a patient allergic to penicillin

Liang-Yu Chen a, Wen-Chung Yu b,c, Suang-Hao Huang b, Mei-Lin Lin a, Te-Li Chen a,c,*, Chang-Phone Fung a,c, Cheng-Yi Liu a,c

a Division of Infectious Diseases, Department of Medicine, Taipei Veterans General Hospital, Taipei, Taiwan
b Division of Cardiology, Department of Medicine, Taipei Veterans General Hospital, Taipei, Taiwan
c School of Medicine, National Yang Ming University, Taipei, Taiwan

Received 1 October 2009; received in revised form 30 July 2010; accepted 4 November 2010

KEYWORDS
Aerococcus viridans; Allergy; Cefotaxime; Endocarditis; Penicillin

Introduction

Aerococci are α-hemolytic, catalase negative, grampositive cocci that appear as staphylococci by gram stain in broth culture, but they have biochemical and growth characteristics of streptococci and enterococci. 1 Aerococci have been isolated from the environment as well as foods 1,2 because they can cause diseases in crustaceans 3 and animals.4 A. viridans also occasionally causes systemic infections in immunocompromised hosts, including meningitis,5 urinary tract infections,6 osteomyelitis,7 septic arthritis, wound infection,8 and, most commonly, bacteremia and endocarditis.1,7,9–11 Risk factors for infection have not been fully identified; but, granulocytopenia, oral mucositis, prolonged hospitalization, previous treatment with antibiotics, invasive procedures, and implantation of foreign bodies have been described as the major risk factors related to A. viridans systemic infection.8

Although Aerococci were reported to be of low virulence, it can still cause severe disease even in immunocompetent hosts. Here we presented a case without obviously immunocompromised factors but was diagnosed A. viridans endocarditis during the admission course.

Case report

A man aged 58 years was transferred to Taipei Veterans General Hospital in Taipei, Taiwan, with symptoms of
A. viridans endocarditis in a pt allergic to penicillin

Dysuria, spiking fever, and altered consciousness for 4 days. He had a history of type 2 diabetes mellitus and had received treatment with oral hypoglycemic agents for 15 years. The medications he took were rosiglitazone, glyburide, metformin, and rosuvastatin. He denied having recent dental work or exposure to illicit drugs.

He was febrile (up to 38.5°C) on admission and the initial vital signs were: BP, 124/78 mmHg; pulse rate, 117 beats/min; and respiratory rate, 22 per min. Physical examination revealed rales over the lower right lung, a diastolic murmur over the left-upper sternal border, and knocking pain over the bilateral costophrenic angle. The spleen was not palpable, and he had no signs of peripheral embolic and immunologic phenomena. The white blood cell count was 22,400 cells/mm³, with 92% segmented forms and 7% lymphocytes. The C-reactive protein level was 26.06 mg/dL, serum glucose was 227 mg/dL, blood urea nitrogen was 31 mg/dL, and creatinine was 1.05 mg/dL. Urine analysis showed the presence of microscopic hematuria, proteinuria, and pyuria.

Brain computed tomography (CT) revealed no organic lesion. Abdominal CT disclosed renal stones, increased infiltration at the bilateral perirenal fascia, and wedge-shaped areas of low density at the upper and middle poles of both kidneys. Transthoracic echocardiography failed to disclose any vegetation, but transesophageal echocardiography (TEE) revealed a vegetation of about 10 mm in size in the noncoronary cusp (Fig. 1) and mild mitral regurgitation.

Because the patient was allergic to penicillin, he was initially treated with cefotaxime (2 g, every 8 h) and vancomycin (500 mg, every 6 h). One of the three blood cultures yielded Aerococcus viridans, which was readily identified by using the Vitek 2 Compact System (bioMérieux, Marcy l’Etoile, France). The isolate was susceptible to penicillin, cefotaxime, moxifloxacin, tigecycline, linezolid, teicoplanin, and vancomycin, but was resistant to chloramphenicol, clindamycin, erythromycin, gentamicin, and trimethoprim/sulfamethoxazole. The minimal inhibitory concentration of the pathogen was 0.25 mg/L to both penicillin and cefotaxime. The identification of the pathogen was confirmed by analysis of the gene of 16S ribosomal ribonucleic acid (rRNA), in which amplification primers derived from conserved regions present at 16S rRNA (5’TGCTCGAGTTGACGTGCGG3’ and 5’TACCTTGGTA CGACCTACCCCA3’). He was diagnosed as having infective endocarditis due to A viridans based on the modified Duke’s criteria, including one major [vegetation on transesophageal echocardiography (TEE)] and three minor criteria (prolonged fever, glomerulonephritis, and positive blood culture). Vancomycin was discontinued after the culture result was available, and cefotaxime was administered for a total of 5 weeks.

The patient became fully alert at Day 3 and afebrile on Day 5 after initiation of cefotaxime therapy, with improvement of the dysuria and lower back pain. TEE performed 5 weeks after antibiotic therapy disclosed a decrease in the size of the vegetation to 5 mm in diameter. The patient remained well during 3 months of outpatient follow-up.

Discussion

Normally, A viridans endocarditis follows a chronic course, with symptoms lasting 12 weeks to 7 months. However, it occasionally causes fulminant disease, as it did in our case. Hence, a positive blood culture of Aerococcus in a patient who is not obviously immunocompromised deserves careful further examination with tools such as TEE to exclude endocarditis because delayed diagnosis leads to a poor prognosis.

In most reported cases, the isolate was identified by conventional biochemical testing and through use of commercial systems, such as the API 20 STREP and API Rapid ID 32 STREPT system (bioMérieux, Marcy l’Etoile, France). Using the 16S rRNA gene analysis as the standard, the identification rate of Aerococcus isolates from swine by API Rapid ID 32 STREPT was 75.9%. Although there are limited data regarding the efficacy of using the Vitek 2 system to identify Aerococcus, our case suggests that this system might correctly identify Aerococcus.

A viridans isolates are usually susceptible to penicillin, although some reports have suggested the emergence of penicillin-, or even chloramphenicol- and quinolone-resistant strains. The no standardized treatment regimen for this pathogen, and most cases have been treated with penicillin, according to the guidelines from the American Heart Association for the viridans group streptococcal endocarditis. Experience using other antibiotics in the treatment of A viridans endocarditis remains rare, although some sporadic cases have had a good response to combination therapy with amikacin and norfloxacin or pefloxacin and teicoplanin. In our case, monotherapy with cefotaxime was adopted because of our patient’s history of anaphylaxis to penicillin, and the clinical response was favorable. The follow-up TEE showed that the vegetation diminished in size of what was assumed to be aseptic after completing the 5-week course of antibiotic treatment.

Infective endocarditis should be highly suspected in a patient with A viridans bacteremia. Cefotaxime is an effective alternative option for therapy in the event that the patient is allergic to penicillin.

![Figure 1](image-url) Modified short-axis view demonstrates a small area of vegetation ~10 mm in size over the ventricular surface of the aortic cusp, which was compatible with typical findings of infective endocarditis. Arrow = vegetation; LA = left atrium; LV = left ventricle.
References