An unusual microorganism, **Aerococcus viridans**, causing endocarditis and aortic valvular obstruction due to a huge vegetation

Endokarditin nadir bir nedeni **Aerococcus viridans** ve aort kapak tıkanıklığına neden olan dev vejetasyon

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**Summary** – **Aerococcus viridans** is not a common pathogen, and endocarditis due to **A. viridans** is very rare. A 44-year-old woman with persistent atrial fibrillation and rheumatic valvular heart disease was admitted with fever, sweating, weakness, and progressive shortness of breath. Transthoracic echocardiography (TTE) demonstrated a 8x9-mm vegetation attached to the right coronary cusp of the aortic valve, causing aortic obstruction. Blood cultures yielded **A. viridans** susceptible to penicillin. Despite optimal antibiotic therapy, subsequent TTE controls revealed enlargement of the vegetation, reaching a size of 21x10 mm, and an increasing gradient across the aortic valve. The patient underwent successful aortic and mitral valve replacement and was stable in the postoperative period without any problem. This represents the first reported case of **A. viridans** endocarditis in which the size and location of vegetation caused obstruction to blood flow, indicating surgery.

**CASE REPORT**

A 44-year-old woman with persistent atrial fibrillation and known rheumatic valvular heart disease was admitted to our emergency service with a two-week history of fever, palpitation, sweating, weakness, and progressive shortness of breath. On admission, her fever was 38.8 °C, blood pressure was 110/60 mmHg, heart rate was irregular with 120 beats/min, and respiratory rate was 30/min. Cardiac examination revealed a 3/6 systolic murmur, loudest at the aortic area and radiating to both carotid arteries. Examination of the other systems was normal. Telecardiography was normal. She had atrial fibrillation with rapid ven-

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Tricuspid response on electrocardiography. Laboratory tests showed leukocytosis (12,200/µl), a high level of C-reactive protein (5 mg/l), and increased sedimentation rate (96 mm/hr). Transthoracic echocardiography showed rheumatic aortic and mitral valves and a mobile mass, 8 x 9 mm in size, on the right coronary cusp of the aortic valve (Fig. 1a). There was a high gradient across the aortic valve due to this mobile mass (maximum 70 mmHg, mean 35 mmHg). After TTE, transesophageal echocardiography was performed, which showed an oscillating mass, 11 x 10 mm in size, consistent with a vegetation on the aortic valve, and moderate mitral stenosis (Fig. 1b). Identification of the microorganism was made using the API test strips and two cultures of blood samples drawn >14 hours apart were positive for *A. viridans* susceptible to penicillin. Prophylactic antibiotherapy consisting of ampicillin/sulbactam and gentamicin was continued. As the patient was hemodynamically stable, and had no symptoms or findings of congestive heart failure, peripheral embolism, or any other complications, she was scheduled to a follow-up program with antibiotherapy and TTE. Three weeks later, however, TTE showed enlargement of the mass, reaching a size of 21 x 10 mm, that caused aortic valvular obstruction (maximum gradient 89 mmHg, mean gradient 51 mmHg) (Fig. 1c-d). The patient underwent surgery for aortic and mitral valvular replacement. She was stable in the postoperative period and was discharged after antibiotherapy without any problem.

**DISCUSSION**

*A. viridans* is a microaerophilic, gram-positive, catalase-negative, and coccus-shaped microorganism. It is a rare organism in population, is responsible for invasive infections and has been associated with meningitis, bacteremia, septic arthritis, and endocarditis. The organism can be found as an indigenous inhabitant in the upper airways and skin of healthy individuals. Infections caused by *A. viridans* origin from a previously damaged tissue, and they are seen in patients...
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who are neutropenic, receiving long-term antibiotherapy, having an invasive procedure and long hospitalization. Dextran production of A. viridans promotes adherence to damaged endocardial surfaces. A. viridans is susceptible to penicillin, trimethoprim-sulfamethoxazole, and glycopeptides. It is not a common pathogen in population and endocarditis due to A. viridans is very rare. A. viridans endocarditis usually has a subacute course and there can be a significant diagnostic latency from three up to seven months. Popescu et al. reported four cases of A. viridans endocarditis, three of which had vegetations exceeding 10 mm, as in our case. In these three cases, vegetations decreased in size with medical therapy and only one required valve replacement because of severe mitral regurgitation. However, in none of these vegetations, obstruction was reported as a cardinal feature. In our case, the ultimate size and location of the vegetation caused obstruction to blood flow, and this was accepted as an indication for surgery. Echocardiographic follow-up demonstrated a progressive increase in the size of the vegetation, which constituted an additional indication for surgery.

The structure of IE vegetation consists of three layers: endocardium at the inner layer, pathogen microorganism at the mid layer, and aggregations of fibrin, platelet, and leukocyte at the superficial layer. These aggregations may prevent the penetration of antibiotics to the bacterium, decreasing the success of antibiotherapy. Despite optimal medical therapy, vegetations may enlarge and, it is recommended that, even if the clinical picture of the patient improves or blood cultures show no positivity for IE, enlargement of vegetation implies ineffective medical therapy and gives an indication for surgery.

To date, there has been no information about treatment of A. viridans endocarditis in textbooks or recent guidelines, but published case reports propose that treatment of A. viridans endocarditis be similar to that for endocarditis caused by Streptococcus viridans.

In conclusion, A. viridans is an unusual cause of IE and must be kept in mind in patients with rheumatic valvular heart disease and presenting with fever, weakness, and dyspnea. Delay in the diagnosis of A. viridans may result in rapidly enlarging vegetations that would lead to mechanical complications on the rheumatic heart valves, as in our case. Therefore, if there is clinical suspicion, it would be convenient to start prophylactic antibiotherapy with penicillin after taking blood cultures and perform frequent TTE follow-up to identify enlarging vegetations earlier, before they cause mechanical complications on the heart valves.

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**REFERENCES**


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**Anahtar sözcükler:** Endokardit, bakteriyel/cerrahi; gram-pożitif bakteri enfeksiyonu.