May positive U waves in V1-V3 leads predict left main coronary artery occlusion?

V1-V3 derivasyonlardaki pozitif U dalgaları sol ana koroner arter oklüzyonunu öngörebilir mi?

A 41 years old male presented with unstable angina pectoris. Electrocardiography on admission revealed marked positive U waves in V1-V3 chest leads on electrocardiography (Fig. 1). Transthoracic echocardiography revealed a normal left ventricular ejection fraction. Early coronary angiography revealed critical occlusion of the distal left main coronary artery (LMCA) (Fig. 2). Coronary artery bypass surgery was offered to the patient. The characteristic electrocardiography patterns suggestive of the LMCA as culprit vessel are (1): ST depression in leads II, III or aVF (highest sensitivity 88%), ST elevation in both aVR and aVL (highest specificity 98%), ST elevation in aVR less than the ST elevation in V1 and right bundle branch block and left anterior fascicular block. Yamaji et al (2) showed that ST elevation in aVR greater than or equal to that in V1 distinguished LMCA group from left anterior descending coronary artery disease group with 81% sensitivity, 80% specificity and 81% accuracy. Other sensitive criteria of LMCA disease are ST deviation in V6–V1≥0 and V6/V1≥1 described by Mahajan et al (3) in the largest series of electrocardiography analysis on acute coronary syndrome resulting from culprit LMCA lesion. However, to the best of our knowledge, the relationship between positive U wave and LMCA occlusion has not been reported in the literature so far. Our patient is the first case showing this relationship in the literature.

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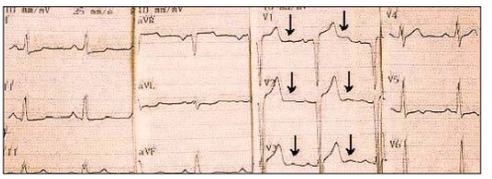

Figure 1. Electrocardiography on admission showing positive U waves in V1-V3 leads

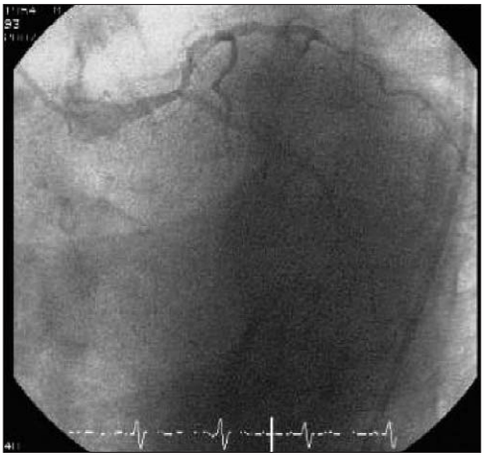

Figure 2. Left coronary angiogram showing critical occlusion of the distal left main coronary artery

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A rare pathogen causing endocarditis: Streptococcus constellatus

Endokardite neden olan ender bir patojen: Streptococcus constellatus

Herein we reported a 35-year-old man who developed endocarditis caused by Streptococcus (S) constellatus, a member of *S. milleri* group. Unlike other viridans streptococci, members of the *S. milleri* group rarely cause infective endocarditis.

A 35- year-old male patient was admitted to our clinic with complaints of tiredness, night sweating, cough and fever lasting two months. On clinical examination, vital signs except body temperature (39.5ºC) were within normal limits. We detected 2/4th grade diastolic murmur along left sternal border. His blood analysis revealed hematocrit 45%, leucocytes 16500/ mm3, ESR 75 mm/h. On his transthoracic echocardiographic examination severe aortic regurgitation, mild degree aortic stenosis (peak gradient 21 mmHg) and multiple vegetations on the aortic cusps (maximum diameter 9X5 mm) were seen (Fig.1). Two blood cultures for aerobic and anaerobic pathogens were immediately taken and therapy with penicillin G-gentamycin combination was initiated.

From blood cultures evaluated with The BacT/ALERT and the BACTEC 9240 systems viridans streptococci were isolated. Despite intensive antibiotic therapy, the patient's general condition was not improved, and hence, another set of blood cultures was obtained. The isolates grew well and had pinpoint colonies of alpha-hemolysis on 5% defibrinated sheep blood agar (Salubris, Turkey) in 5% CO2 and ambient air at 37°C. They were catalase-negative and gram-positive cocci. The isolate was identified as *S. constellatus* by both the API Rapid ID32 Strep system (bioMerieux, France) and REMEL Rapid STR system (Apogent-USA). The isolate was susceptible to ceftriaxone, chloramphenicol, erythromycin, ofloxacin, cefotaxime, tetracycline, levofloxacin, and vancomycin but resistant to penicillin G. We immediately changed the antibiotic treatment to cefotaxime 2x4 g IV per day. However, the persistence of vegetations on control transthoracic echocardiogram repeated three days later led us to transfer the patient to cardiac surgery clinic for aortic valve replacement with St Jude mechanical valve. Intraoperative and postoperative course was uneventful. We continued cefotaxime therapy for another six week and the patient discharged on the postoperative 20th day.

Nearly all microorganisms can cause infective endocarditis especially gram-positive cocci and the so-called HACEK microorganisms (1). Furthermore, viridans streptococci are the leading cause of native valve endocarditis especially in subacute cases (1).

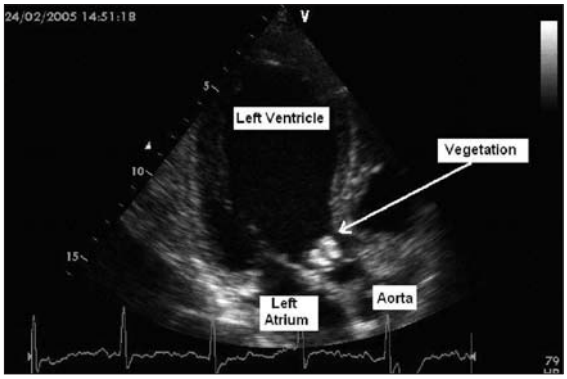

Figure 1. Transthoracic echocardiographic examination showing vegetation on aortic cusps caused by Streptococcus constellatus

The *S. milleri* group consists of three species: *S. constellatus, S. anginosus* and *S. intermedius*. This group of streptococci is part of the normal flora of the mucous membranes, but is often involved in suppurative infections especially in the presence of cirrhosis, diabetes, malignancy and immunodeficiency (2). They are also rare causes of infective endocarditis with S. anginosus predominance (3).

Although a high degree of clinical suspicion and correct interpretation of clinical findings still has paramount importance in infective endocarditis diagnosis, the possibility of rarely detected microorganism as a causative agent should be keep in mind. In our case, penicillin-gentamycin combination was unsuccessful which prompted for searching another organism. Moreover, the results of cefotaxime therapy were also unsatisfactory in the presence of persistent vegetations.

Early valve surgery with cefotaxime therapy, as in our case, may be more appropriate therapeutic approach in penicillin-resistant *S. constellatus* endocarditis cases (4, 5).

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A rare cause of dilated cardiomyopathy; Alström syndrome

Dilate kardiyomiyopatinin nadir bir nedeni; Alström sendromu

Alström syndrome (AS) is a recessively inherited genetic disorder characterized by; pigmentary retinal dystrophy, sensorineural hearing loss, obesity, non-insulin dependent diabetes mellitus. Other features reported in some but not all subjects include acanthosis nigrigans, hypogonadism, short stature, hepatic, renal and cardiac failure (1) (Table 1). The gene mutated in AS patients has recently been identified as ALMS1 (2).

We present a 21-year-old man who was referred to the hospital for routine examination before applying to military services. He had five brothers and three sisters. According to the history, one of his brothers also had almost the same features but could not be examined because of social causes. There was no history of consanguity and drug use in pregnancy. From birth, our patient suffered from progressive vision loss and nystagmus. On admission his blood pressure was 130/80mmHg, pulse rate 110/min. Weight was 55kg, height 144cm and body mass index (BMI) 26kg/m^2. He had thin hair and frontal hair loss. Eye examination showed nystagmus, bilateral posterior subcapsular cataract. Vision was at the level of perception but no projection in both eyes. His fundus examination showed pale discs and retinal pigmentary changes. He had acanthosis nigricans. Physical examination disclosed orthopnea, dyspnea, bilateral jugular venous distention and bilateral pretibial pitting edema. On auscultation, tachycardia, coarse lung sounds and bilateral rales below the scapulae were detected. He had gynecomastia, bilateral testicular atrophy and pretibial edema.

Laboratory analyses were as following; fasting blood glucose - 255mg/dl, insulin -36IU/mL, HbA1c - 11.7% (range, 4.8-6.0), creatinine - 1.5mg/dl (range, 0.6-1.2), serum glutamic-oxalacetic transaminase - 52 (range, 8-40), serum glutamic-pyruvic transaminase - 83U/L (range, 10-40), gamma-glutamyl transpherase -486 (range, 0-49U/L), alkaline phosphatase -127U/L (range, 38-94), thyroid stimulating hormone - 1.56 mcIU/mL (range, 0.27-4.2), luteinizing hormone - 30.28mıu/mL (range, 1.7-8.6), follicle stimulating hormone - 33.72 miu/mL (range, 1.5-12.4), prolactin 10.44 ng/mL (range, 4.04-15.2), testosterone - 1ng/mL (range, 2.4-9.5) 24-hour urinary albumin excretion- 250mg/24h. Electrocardiogram

Table 1. Clinical features and complications of Alström syndrome

Presentation	(*) Literature, %	Patient in the present study
Retinal degeneration	98	+
Sensorineural deafness	89	+
Diabetes	82	+
Obesity	98	-
Acanthosis nigricans	68	+
Hypergonadotropic hypogonadism	78	+
Hypothyroidism	17	-
Short stature	98	+
Hepatic dysfunction	92	+
Dilated cardiomyopathy	60	+
Renal dysfunction	49	+

*- Adapted from reference 1