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Megasphaera elsdenii Endocarditis

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A case of endocarditis caused by *Megasphaera elsdenii* is reported. This anaerobic gram-negative coccus has rarely been associated with human infections and has not previously been described as a cause of endocarditis.

Megasphaera elsdenii, a gram-negative anaerobic coccus, has been shown to be a normal inhabitant of the gastrointestinal tract in humans. It has rarely been implicated in human infections and has never been shown to be a primary pathogen. This paper is a case report of *M. elsdenii* endocarditis in an apparently immunocompetent host.

CASE REPORT

A 49-year-old white male was admitted to the medical service of Church Hospital on 3 July 1978 because of weight loss, fatigue, and edema of both legs. The patient denied having rheumatic fever; however, he recalled that he had a heart murmur as a child.

On physical examination the patient was a well-developed, thin white male. His temperature was 99°F (ca. 37.2°C) orally, his pulse was 92, his respirations were 18, and his blood pressure was 150/50. There was a grade IV/VI harsh holosystolic murmur along the left sternal border which was best heard in the second intercostal space. Also present was a short crescendo-decrescendo grade IV/VI diastolic murmur at the same location. An S_3 gallop and a systolic and diastolic thrill were present. The point of maximal impulse was at the sixth intercostal space, 2 cm left of the midclavicular line. The liver was enlarged and extended 6 cm below the right costal margin, with mild tenderness on deep palpation. There was 3+ pitting edema of the lower extremities to the level of the knee. Laboratory data on admission included: hemoglobin, 6.7 g; hematocrit, 21.6%; leukocyte count, 11,400 cells/mm^3, with 71% segmented neutrophils, 16% bands, 17% lymphocytes, 4% monocytes, 1% basophils, and 1% eosinophils. Toxic granulation was present. Immunoelectrophoresis was essentially normal. An admission chest X ray revealed cardiomegaly with prominence of the left atrium and right ventricle, a small pleural effusion, and minimal pulmonary congestion. The electrocardiogram showed a right bundle branch block.

On the evening of admission, the patient spiked a fever of 102.5°F (ca. 39.2°C). Blood cultures were drawn, and two of two bottles grew a gram-positive anaerobic coccus which was subsequently identified as *M. elsdenii.* During the following few days the patient continued to spike fevers of 102 to 103°F (ca. 38.9 to 39.4°C). An additional set of blood cultures drawn on 7 July also grew *M. elsdenii* in three of three bottles. The patient was begun on intravenous infusion of 24×10^6 U of penicillin and 1 g of streptomycin daily. A subsequent echocardiogram was interpreted as showing vegetations of the aortic valve with evidence of aortic incompetence. On antibiotic therapy the patient's fever subsided. Although weak, the patient appeared to be improved clinically. However, on 14 July he became acutely short of breath with clinical and radiographic evidence of pulmonary edema. That afternoon the patient was transferred to Johns Hopkins Hospital. A cardiac catheterization was performed and revealed a significant left to right shunt which was interpreted as being consistent with a ventricular septal defect. The patient began showing increasing signs of congestive heart failure and was scheduled for emergency valve replacement and possible repair of a ventricular septal defect. During surgery, examination of the aortic valve revealed almost complete destruction of the valve by bacterial vegetations. A 0.5-cm ventricular septal defect was also noted just below the aortic valve. The defect had some friable infected-looking material associated with its superior aspect. While a 25-mm Hancock prosthesis was being sutured in, a small aortic root abscess was noted in the area of the previously described friable material. The abscess was drained, and the prosthesis was placed. The remaining operation was uneventful, and the patient was taken to the Intensive Care Unit in critical but stable condition. Subsequently, the patient developed increasing hypotension and congestive heart failure and expired

on 17 July.

MATERIALS AND METHODS

Blood cultures are normally submitted to our laboratory in a series of three bottles, collected 1 h apart (100 ml; Trypticase soy broth with CO_2 and sodium polyethanol sulfonate; Baltimore Biological Laboratory). The second bottle in each set is vented. Subcultures and Gram stains are made at 24 and 72 h. All subcultures are made aerobically to chocolate agar (Baltimore Biological Laboratory) and anaerobically for 48 h to prereduced anaerobic blood agar. In this case, only two sets of cultures were drawn initially on 4 July. Two of two cultures gave positive smears from the bottles and colonies on the anaerobic plates after 72 h of incubation. A series of three subsequent blood cultures drawn on 7 July 1978 were also positive after 72 h of incubation. After ether extraction of a pure culture grown in peptone-yeast extract-glucose broth, gas chromatographic identification of the anaerobic coccus was made with a Hewlett-Packard model 5710A gas chromatograph.

RESULTS

The smears from the initial bottles revealed gram-variable pleomorphic coccobacilli, varying from 1.5 to 3.0 μm in length. The colonies on anaerobic blood agar were circular with entire edges, nonhemolytic, and translucent. They were catalase negative and 1 to 2 mm in diameter. Gram stains from the colonies also revealed pleomorphism, but more of the cells were gram negative. Some forms appeared as chains of gram-negative diplococci. The culture was sent to the Johns Hopkins Hospital Anaerobic Laboratory for gas-liquid chromatographic analysis for acid products and anaerobic susceptibility testing. Upon ether extraction of the peptone-yeast extract-glucose broth, the organism was identified as *M. elsdenii* by using criteria outlined in the Virginia Polytechnic Institute *Anaerobe Laboratory Manual* (4). The following fatty acids were identified: acetic acid, isobutyric and butyric acids, isovaleric and valeric acids, and caproic acid. Although many anaerobes produce somewhat similar fatty acid patterns, the presence of caproic and valeric acids is unusual and quite distinctive for *M. elsdenii*. Unfortunately, repeated attempts to obtain adequate growth for susceptibility testing and other biochemical tests were unsuccessful.

DISCUSSION

The normal oral flora viridans streptococci and staphylococci have been reported as the most common etiological agents of bacterial endocarditis (5). However, there have been increasing reports of less common causative agents of infection of the endocardium. These include *Haemophilus influenzae* and *H. parainfluenzae* (2), *Corynebacterium* species (11), *Neisseria catarrhalis* (6), and *Micrococcus* sp. (9). The present case of endocarditis is due to an anaerobic gram-negative coccus, *M. elsdenii*. This appears to be the first documented human infection due to this bacterium.

M. elsdenii was first recovered by Elsden in 1956 from the rumen of cattle, sheep, and pigs (8). Originally called *Peptostreptococcus elsdenii*, it was reclassified by Rogosa as a new genus, *Megasphaera* (7), in the family *Veillonellaceae*. Analysis of the cell wall of this bacterium revealed that its composition is that of a gram-negative organism, despite the fact that it frequently stains gram positive. *M. elsdenii* was isolated as a probable inhabitant of the human intestinal tract (12) and confirmed as such by Sugihara et al. in a study of human feces (10). The latter study first suggested a possible role of *M. elsdenii* in human pathological processes. This followed its isolation as part of the mixed anaerobic and facultative flora from the transtracheal aspirate of a putrid lung abscess. Two strains of *M. elsdenii* have been confirmed by the Virginia Polytechnic Institute Anaerobe Laboratory (E. P. Cato, personal communication) from human clinical sources. These were isolated from a rectal drainage (Emory University, Atlanta, Ga.) and from a frontal lobe tumor (Northwestern Memorial Hospital, Chicago, Ill.). To our knowledge, neither case has been documented, and the organism from the rectal drainage probably represents colonization rather than true infection.

Since this is the first reported case of *M. elsdenii* endocarditis and is in fact the first documented case of primary human pathogenicity, prognostic differences and generalizations would be difficult to make. However, in keeping with most cases of endocarditis in the post-antibiotic era, the patient's demise was more related to progressive congestive heart failure than to infection (1, 3).

The role of obligate anaerobic organisms in human infectious processes has only recently been appreciated. *M. elsdenii*, a normal inhabitant of the human gastrointestinal tract, is apparently very capable of attacking damaged heart valves. With rapid advancements in anaerobic methodology, investigators should expect to find increasing numbers and types of infections due to unusual anaerobic organisms.

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