



An Oral Abscess as the Entrance Leading to Endocarditis: A Sexual Behavior Role

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Abstract

We report a case of atypical endocarditis for which the transmission mode has long been debated among cardiac surgeons, microbiologists, and infectious disease specialists. This is the first reported case of *Streptococcus agalactiae* being responsible for the probable sexually transmitted case of endocarditis in a healthy, 49-year-old female.

Keywords: *S. agalactiae*; Infective endocarditis; Aortic valve

Introduction

The risk factors for infective endocarditis (IE) in the population are changing [1]: the reported worldwide increase in the incidence of IE has been associated with the widespread use of medical devices and procedures responsible of health care associated infection [2,3]. Endocarditis incurred by *Streptococcus agalactiae* in adults (non-pregnant females and men) has increased in recent years [4,5], even though the source of infection due to this bacterium and the portal of entry are unclear and not often well documented.

Concerning the possible sexual port of entry in the cause of endocarditis, this mode of transmission should be suspected in a search of clinical history, especially in recurrent endocarditis.

Case Report

We report on a 49-year old female with recent adjustment or orthodontic appliances one month after which probably due to the breakdown of mucocutaneous barriers she had an oral abscess, apparently treated successfully with amoxicillin/clavulanate. Microbiological culture of the abscess and blood cultures made at another hospital in Milan revealed the presence of *S. agalactiae*. After non-improvement, the patient was admitted one month later to our emergency department with shoulder pain on movement that did not change for 5 days. Physical examination revealed a low grade fever (38°C). Laboratory testing revealed leukocytosis (13,540/ μ l), platelet count (474,000/ μ l), C-reactive protein (50 mg/L), and D dimer (286 ng/dl). Empirical therapy with ciprofloxacin was begun after collecting two sets of blood cultures and a urine sample.

During hospitalization in the Infectious Department, she underwent transesophageal and transthoracic echocardiography (Figure 1) that confirmed the presence of endocarditis on the anterior (A2) and posterior (P2-P3) leaflets of the mitral valve and with a free floater mass in the left ventricle having a base of about 1 cm and a diameter of 2 cm.

The suspected endocarditis and septic peripheral embolization was confirmed. Blood cultures had been negative during admission to our hospital, thus the patient was treated with gentamicin and ampicillin in vain.

According to the modified Duke criteria for infective endocarditis (IE), the patient was taken to the emergency operating room based on a potential embolism.

The native mitral valve was repaired by two triangular resection of A2 and P2-P3. Leaflets reconstruction and anuloplasty with Carpentier Edwards Physio II n.28 (Edwards Lifesciences Corp, Irvine, CA) completed the procedure that was performed through a small right thoracotomy. The resected leaflet and vegetations were sent to the microbiological laboratory for the analysis.

A traditional culture with a 5-day subculture was performed with negative results for bacteria (aerobes and anaerobes) and fungi; a *dithiothreitol* procedure obtained during surgery was positive for *S. agalactiae*.

The postoperative course was free from major complications, and the patient was discharged and sent home with instructions to continue antibiotic therapy with ceftriaxone for 4 weeks. However, approximately a month post-surgery, the patient had an increase in C-reactive protein (70 mg/L) and fever (38°C), entailing a reassessment of the patient with a possible diagnosis of postsurgical pericarditis.

Further microbiological investigation was performed. Discussion with the patient and her husband, asymptomatic for urogenital discomfort or discharge, revealed that the couple had had oral sex while the husband was under orthodontic treatment a few days before the first emergency room episode. An oral swab determined he was colonized orally by *S. agalactiae*. We hypothesize that the vaginal infection due to *S. agalactiae* pre-surgery and during the dental work was caused by the entrance of the pathogen into the patient's circulation and therefore was responsible for infection of the valve.

IE risk factors have been identified [6], and they include age over 60 years and a diagnosis of diabetes, cancer, chronic renal disease, and

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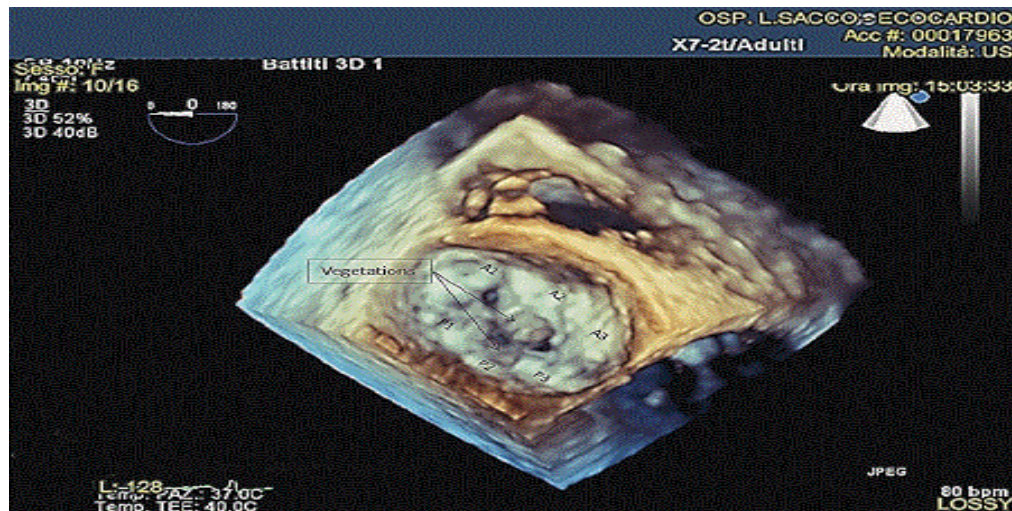


Figure 1: Real-time 3D transoesophageal echocardiography volume rendering of the mitral valve. Two vegetations on the anterior and posterior mitral leaflet (arrows). A1, A2, A3, anterior mitral valve scallops; P1, P2, P3, posterior mitral valve scallops.

neurological vessel disorder; however, in evaluating the medical history, one should consider sexual habits and dental status.

Even though it has been reported [7] that IE can be induced by bacteremia attributable to dental procedures as happened to our patient this is the first time that colonization/vaginal infection of a woman has been suspected of being transferred through oral sex.

Comment

Although *S. agalactiae* is responsible for various clinical forms of skin, soft tissue, bone, and urinary tract infections [8] most commonly occurring between mother and neonate more rarely it could be a pathogen involved in endocarditis. Even if, in the past few years, there have been epidemiological changes in the bacterium responsible for the infections [9], *S. agalactiae* is a rare cause of IE (1.7% of all cases) [10]. We believe that the change of risk factors for endocarditis should factor in sexual habits, especially in view of the increased frequency of sexually transmitted bacterial endocarditis. To support our theory, we call attention to 18 papers in the literature reporting that *S. agalactiae* and other pathogens have been suspected of being sexually transmitted. However, these cases have never been investigated as causative of IE. *Neisseria gonorrhoea* has been reported as a cause of endocarditis, especially in young males, although not necessarily reporting urogenital discomfort or urethral discharge [11]. *Chlamydia trachomatis*, *Mycoplasma hominis* [12], like *Ureaplasma urealyticum* and *Gardnerella vaginalis*, can induce cardiovascular disease, and endocarditis following respiratory tract infection as an entrance door. Transmission of endocarditis through sex has never been documented [13].

The Centers for Disease Control and Prevention has campaigned in an effort to raise awareness among adolescents (aged 13-24) about the sexual behaviors at risk for venereal diseases, infections, and unintended pregnancies due early coitarcia.

Therefore, our work should focus on the new population at risk for endocarditis, other than the injecting drug users dying of endocarditis, which have been typical of the past decade; sexual behavior should not be omitted in evaluating a medical history.

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