



Kissing Abscess Type Infective Endocarditis Caused by *Streptococcus tigurinus*: A Case Report

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Authors' contributions

This work was carried out in collaboration between all authors. Authors MS, LP, MF and RZV gathered patient information and wrote the manuscript. Authors LSA and GM cared for the patient, reviewed the manuscript and provided expert opinion. All authors reviewed and accepted the final manuscript for submission.

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Case Study

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ABSTRACT

Streptococcus tigurinus is associated with infective endocarditis and meningitis. Due to its similarity to other species it has been underreported. We report the case of a 46 years old patient with 3 months of intermittent fever and asthenia and a blood culture positive for *S. tigurinus*.

Keywords: *Infective endocarditis; Streptococcus mitis; Streptococcus tigurinus; kissing abscess.*

1. INTRODUCTION

The *Streptococcus tigurinus* (*S. tigurinus*) was described first in 2012 in a laboratory in

Switzerland as a causative agent of endocarditis [1]. Infective endocarditis (IE) is a pathology of considerable prevalence around the world, with a high rate of morbidity and mortality (up to

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20%). Early and adequate diagnosis is important for the prognosis and treatment of patients [2,3].

New laboratory and molecular analysis techniques have been important for recognizing previously unidentified species as etiologic agents of IE. One of these pathogens is *Streptococcus tigurinus*. According to its phenotype and molecular characteristic, it is closely related to *Streptococcus mitis* (*S. mitis*), *Streptococcus pneumoniae* (*S. pneumoniae*) *Streptococcus pseudopneumoniae*, *Streptococcus oralis* and *Streptococcus infantis* [4].

To our knowledge, 22 cases of IE with identification of *S. tigurinus* as the responsible pathogen (7 in Switzerland and Germany [3], 11 in USA [5], 3 in Japan [6,7] and 1 in Spain [8]) have been reported. Because there are few case reports of *S. tigurinus* as causative agent of IE and the importance of accurate identification of the pathogen for proper treatment, we present the case of a patient with bi-valvular infective endocarditis by *S. tigurinus*.

2. CASE PRESENTATION

A 46 years old male patient, from Lima-Peru, with no relevant past medical history, comes to the emergency department with fever for 3 months (up to 39°C), night sweats and malaise. During this time, he went to a private clinic where he was diagnosed with typhoid fever and treated with ciprofloxacin 500 mg. every 12 hours for 14 days without improvement of symptoms.

Upon physical examination the patient was hemodynamically stable, with vital functions in normal ranges, body mass index of 20 kg /m², slightly pale without skin lesions (no Janeway lesions or Osler nodes). The oral cavity presented gingivitis and multiple caries. Auscultation of the heart revealed a holosystolic murmur of III/VI of intensity, best heard over the aortic and mitral area. The rest of the physical examination was within normal limits. Patient was admitted for appropriate treatment.

On admission 3 blood cultures were taken. The first one was positive for *Granulicatella adiacens* sensitive to penicillins, cephalosporins, macrolides and fluoroquinolones and tetracyclines-resistant. The second and third ones were positive for *Streptococcus mitis* sensitive to penicillins, cephalosporins,

macrolides and fluoroquinolones and resistant to tetracyclines.

Funduscopy examination and retinography revealed Roth spots on the right retina. Transthoracic echocardiogram reported moderate mitral insufficiency due to anterior valve endocarditis with abscess of the segments A1 and A3; aortic valve endocarditis with perforation of the non-coronary sinus and right coronary sinus prolapse; moderate concentric hypertrophy of the left ventricle with diastolic dysfunction type 1 and normal systolic function. Transesophageal echocardiogram reported mitral valve endocarditis with multiple vegetations complicated with ruptured aneurysm of the anterior valve with significant regurgitation; bicuspid aortic valve endocarditis with anterior valve prolapse and moderate insufficiency; abscess formation in the mitral-aortic area (Fig. 1), findings related with "Kissing Abscess" type mitral endocarditis secondary to a primary aortic endocarditis.

Antibiotic therapy with intravenous ceftriaxone 2 gr every 24 hours and gentamicin 80 mg every 24 hours was initiated due to antibiogram results. At the seventh day of antibiotic treatment, two blood cultures were obtained and reported as negative. Patient responded rapidly to antibiotic therapy with resolution of symptoms and satisfactory clinical evolution.

Nonetheless, surgical management was decided due to the fact that the patient's echocardiogram showed multiple vegetations larger than 10 mm, moderate valvular insufficiency and perivalvular abscess. A double valve replacement and abscess drainage was performed and the patient recovered completely without complications.

Owing to the infrequency of *Granulicatella adiacens* and *Streptococcus mitis* as causatives agents of infective endocarditis, a blood culture that was done before starting antibiotics was sent for DNA sequencing. The pathogen reported by the DNA sequencing was *Streptococcus tigurinus* with an identity match of 99.21%.

3. DISCUSSION

Streptococcus tigurinus was first reported as a causative agent of infective endocarditis. They were initially identified as viridans group streptococci. However, after the analysis

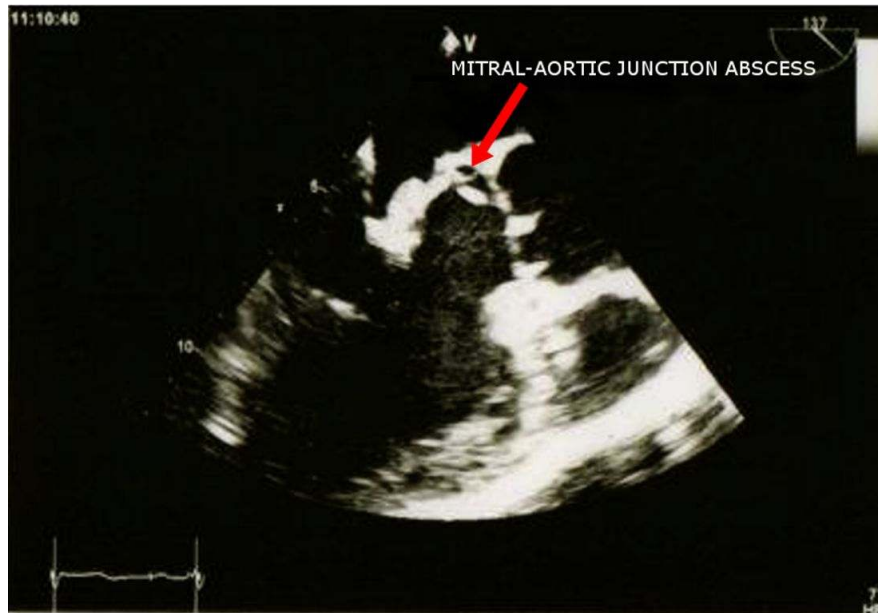


Fig. 1. Transthoracic echocardiogram. Arrow shows abscess in the mitral-aortic junction

of the 16S rRNA sequence, they were classified as members of the *streptococcus mitis* group [3].

Since the first case reported of *S. tigurinus* in 2012, it has been isolated from multiple sterile sites such as, cerebrospinal fluid heart valves and articular liquid [3,9]. *S. tigurinus* has been identified as the pathogen responsible for severe invasive infections such as infective endocarditis, infective endocarditis with negative culture, spondylodiscitis, bacteremia, meningitis, infection of articular prosthesis and thoracic empyema [7,9]. *S. tigurinus* has also been isolated from subgingival plaque of patients with periodontitis [10] and is the most common source of origin, although the skin and gastrointestinal tract have also been described as possible sources [5].

S. tigurinus is being underreported due to the limitations of routine laboratory exams [3]. The conventional phenotypic methods do not provide a precise identification as a result of its morphological resemblance to other pathogens. *S. tigurinus* forms S alpha-hemolytic colonies with a diameter of about 0.5 to 1 mm after incubation at 37°C for 24 hours on sheep blood agar [4]. *S. tigurinus* cannot be found in the VITEK 2 microbial identification system. Since this bacteria is not available in Biotyper (Bruker)

or Vitek MS (bioMerieux) database, it cannot be correctly identified by MALDI-TOF MS systems that are widely used [11]. Therefore, the analysis of the -5 end of the gene 16S rRNA is mandatory for correct identification of *S. tigurinus* as it was demonstrated that in this position there is a significant demarcated sequence [4,9].

The bacteria isolated in our patient was submitted for 16S rDNA sequencing and results were searched using the SmartGene Database. Sequence analysis showed that the organism was genetically consistent with *S. tigurinus*, based on a match length of 758 base pairs and an identity match of 99.21%, which corresponds to six base pair mismatch.

The antibiogram of the pathogen isolated in our patient demonstrated susceptibility to penicillin, cephalosporin, macrolides, fluoroquinolones and tetracycline resistance. Similar results were found earlier by Zbinden et al. who reported 13 cases, all susceptible to penicillin [3]. A review of previous reported cases of *S. tigurinus*, concluded that the pattern of antibiotic susceptibility of this pathogen is similar to other streptococci viridans sensitive to penicillin, and all recovered after receiving the appropriate medication [11].

Our patient also presented "Kissing Abscess" type aortic endocarditis, which is the subsequent infection of the mitral valve. There are several theories that could explain the spread of the infection; one of them is through the prolapse of vegetations of considerable size from the aortic valve during diastole that makes contact with the anterior leaflet of the mitral valve [12].

4. CONCLUSION

The specific identification of the causative agent in infectious diseases is important to determine the appropriate antibiotic treatment. It also help us to analyze the antibiotic susceptibility patterns and trends on resistance. This makes possible the knowledge of the actual prevalence of organism in different diseases and the creation of specific antibiotics schemes.

CONSENT

As per international standard or university standard written patient consent has been collected and preserved by the authors.

ETHICAL APPROVAL

It is not applicable.

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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