CARDIOLOGIE TUNISIENNE

CASE REPORT

Endocardite mitrale à Streptococcus sanguinis révélée par une fièvre prolongée et une diarrhée

Streptococcus sanguinis Mitral Endocarditis Presenting with Fever and Diarrhea

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Résumé

Streptococcus sanguinis est une cause peu fréquente mais importante d'endocardite infectieuse, touchant le plus souvent les valves natives. Nous rapportons le cas d'un patient de 50 ans admis pour une fièvre prolongée et une diarrhée évoluant depuis 15 jours, initialement traité sans succès par amoxicilline-acide clavulanique et traitement symptomatique. L'échocardiographie a montré une volumineuse végétation mobile de 2 cm implantée sur le feuillet antérieur de la valve mitrale, associée à une insuffisance mitrale sévère. Les hémocultures ont isolé S. sanguinis avec diminution de la sensibilité aux β-lactamines. L'antibiothérapie a été adaptée par vancomycine et gentamicine, puis le patient a bénéficié d'un remplacement valvulaire mitral par sternotomie mini-invasive. L'évolution postopératoire a été favorable, avec une amélioration clinique et biologique durable. Ce cas souligne l'importance d'un diagnostic précoce, de l'adaptation du traitement antibiotique selon l'antibiogramme et d'une prise en charge chirurgicale en temps opportun chez les patients présentant de volumineuses végétations du cœur gauche et une dysfonction valvulaire sévère.

Mots-clés

Endocardite infectieuse ; Streptococcus sanguinis ; valve mitrale ; régurgitation mitrale ; hémoculture; tests de sensibilité microbienne.

Summary

Streptococcus sanguinis is an uncommon but important cause of infective endocarditis, most often involving native heart valves. We report the case of a 50-year-old patient who presented with a 15-day history of fever and diarrhea and was initially treated with amoxicillin-clavulanate and symptomatic therapy without improvement. Echocardiography revealed a 2-cm mobile vegetation on the anterior mitral leaflet associated with severe mitral regurgitation. Blood cultures yielded S. sanguinis with reduced susceptibility to β-lactams. Antimicrobial therapy was escalated to vancomycin and gentamicin, and the patient subsequently underwent mitral valve replacement through a minimally invasive ministernotomy. Postoperatively, the patient showed sustained clinical and biological improvement. This case highlights the importance of early recognition of infective endocarditis, susceptibility-guided adjustment of antimicrobial therapy, and timely surgical intervention in patients with large left-sided vegetations and severe valvular dysfunction.

Keywords

Infective endocarditis; Streptococcus sanguinis; Mitral valve; Mitral regurgitation; Blood culture; Microbial sensitivity tests.

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INTRODUCTION

Streptococcus sanguinis, a member of the viridans group streptococci, is a recognized cause of subacute infective endocarditis (IE), typically involving native heart valves [1,2]. Although viridans group streptococci have historically been considered highly susceptible to penicillin, recent epidemiological studies suggest increasing rates of reduced β-lactam susceptibility among non-beta-hemolytic streptococci, which may complicate empirical therapy and reinforce the importance of susceptibility-guided treatment [3]. Early diagnosis remains challenging when the initial presentation is dominated by non-specific symptoms such as prolonged fever or gastrointestinal complaints, including diarrhea, which may initially direct the workup toward more common infectious conditions [4]. Recognition of these atypical patterns is essential to prevent delayed diagnosis and progression to severe valvular involvement, large mobile vegetations, and embolic complications [5,6].

CASE PRESENTATION

A 50-year-old patient presented with a 15-day history of persistent fever and diarrhea. Before admission, the patient had received amoxicillin-clavulanate and symptomatic treatment without clinical improvement. There was no prior history of cardiac disease or recent dental procedures; the most recent dental evaluation had taken place 6 months earlier. On admission, the patient was hemodynamically stable and had no peripheral stigmata of IE, including petechiae, splenomegaly, Osler nodes, or other peripheral embolic signs.

Laboratory investigations showed leukocytosis of 17,000/mm^3 and an elevated C-reactive protein level of 70 mg/L, with preserved renal function. Three sets of blood cultures were obtained, and all yielded S. sanguinis. Transthoracic echocardiography revealed a 2-cm mobile vegetation attached to the anterior mitral leaflet, associated with severe mitral regurgitation (Figure 1). Transesophageal echocardiography confirmed these findings. A thoraco-abdominal-pelvic computed tomography scan showed no extracardiac complications, and a positron emission tomography scan performed during antibiotic therapy showed no embolic foci or occult malignancy.

Empirical treatment for native valve endocarditis was initiated with intravenous ceftriaxone, ampicillin, and gentamicin while awaiting susceptibility testing. On day 5, microbiological susceptibility testing demonstrated reduced β-lactam susceptibility, prompting escalation to vancomycin and gentamicin. Despite this therapeutic adjustment, follow-up echocardiography showed persistence of the large vegetation and ongoing severe mitral regurgitation. Given the large and highly mobile mitral vegetation, coupled with severe valve dysfunction, the patient fulfilled guideline-based criteria supporting urgent surgery [5–7].

The patient underwent mitral valve replacement through a minimally invasive ministernotomy. The postoperative course was uneventful, with sustained apyrexia and normalization of inflammatory markers and leukocyte counts. On postoperative day 30, the patient remained hospitalized to complete the intravenous antibiotic course under close clinical surveillance. Follow-up imaging confirmed satisfactory prosthetic valve function without residual regurgitation or paravalvular leak.

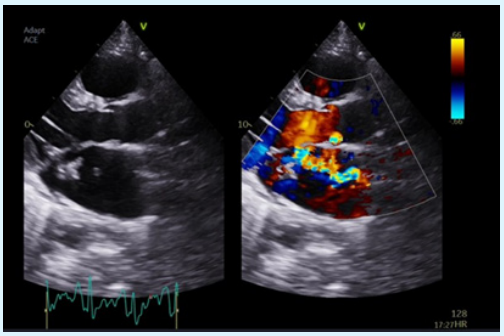

Figure 1. Transthoracic echocardiography, parasternal long-axis view, showing a large vegetation attached to the anterior mitral leaflet, associated with severe mitral regurgitation.

DISCUSSION

This case highlights that Streptococcus sanguinis, although classically regarded as an oral commensal and a recognized cause of subacute infective endocarditis, can be associated with severe left-sided valvular infection and a potentially complicated clinical course [1,2]. This case is notable because it combines a misleading initial presentation, a large mobile mitral vegetation with substantial embolic potential, and reduced susceptibility to first-line β-lactam therapy, ultimately requiring early surgical intervention [5,6].

A first important lesson from this case is diagnostic. The initial combination of prolonged fever and diarrhea was nonspecific and could reasonably divert attention

toward gastrointestinal or systemic infectious causes before IE was considered [4]. This observation reinforces a practical point recognized in contemporary IE guidance: the diagnosis should be considered in patients with persistent fever and inflammatory syndrome without a clear source, even when classical peripheral stigmata are absent [6]. In addition, several reports have shown that S. sanguinis IE may present with unusual extra-cardiac manifestations, including iron-deficiency anemia, vasculitis syndromes, or occult neurological complications, underscoring the protean nature of this infection [8–10].

The 2-cm mobile mitral vegetation was the major prognostic feature in this patient. Large left-sided vegetations, particularly those involving the mitral valve and exceeding 10 mm, are associated with an increased risk of embolic events, especially early in the course of treatment [5,11]. The 2023 ESC Guidelines support urgent surgery in patients with large vegetations associated with severe valve dysfunction or other surgical indications [6]. In our patient, the size and mobility of the vegetation, combined with severe mitral regurgitation, strongly supported a strategy of early surgery to reduce the risk of embolic and hemodynamic complications [6,7].

From a pathobiological perspective, the severity of infection observed in this case is biologically plausible. S. sanguinis possesses several surface-associated and biofilm-related factors that may facilitate adherence to platelet-fibrin aggregates and persistence within the intravascular environment [1,2]. These mechanisms may contribute to the organism's ability to establish valvular infection and form large vegetations, although no strain-specific virulence analysis was performed in our patient. This interpretation should therefore be regarded as supporting biological plausibility rather than a direct mechanistic explanation of the individual clinical course.

A second major message of this case concerns antimicrobial management in the setting of changing susceptibility patterns. Although viridans group streptococci have historically been regarded as broadly susceptible to penicillin, reduced β-lactam susceptibility has increasingly been reported among non-beta-hemolytic streptococci in recent epidemiological studies [3]. Our case underscores the importance of systematic susceptibility testing and prompt reassessment when the response to standard therapy is suboptimal. The escalation to vancomycin- and gentamicin-based therapy was justified by both the susceptibility profile and the high-inoculum nature of the 2-cm vegetation. Large vegetations may represent a relatively protected site in which delayed sterilization increases the risk of ongoing valvular damage and embolic complications [5,6].

Early surgery was central to the favorable outcome in this patient. Beyond reducing embolic risk, it addressed the mechanical consequences of severe mitral valve infection and removed an infected mass unlikely to be sterilized rapidly with antibiotics alone [7,12]. This approach is consistent with a multidisciplinary Heart Team strategy integrating vegetation size, valve dysfunction, embolic risk, and microbiological evolution into surgical decision-making [6,12]. The use of a minimally invasive ministernotomy also provided adequate surgical exposure while potentially limiting postoperative morbidity [12].

This case adds to the limited literature on S. sanguinis IE presenting with misleading non-cardiac symptoms and complicated by reduced β-lactam susceptibility. It also illustrates the practical importance of combining early echocardiographic assessment, microbiological susceptibility testing, and timely surgical referral in patients with severe mitral valve involvement. Taken together, these features support the view that apparently indolent viridans streptococcal endocarditis can evolve into a high-risk surgical disease if recognition and definitive management are delayed [3,6].

CONCLUSION

This case shows how Streptococcus sanguinis infective endocarditis may initially mimic a non-cardiac febrile illness yet progress to advanced mitral valve infection requiring combined medical and surgical management. It adds to the limited literature on reduced β-lactam susceptibility in streptococcal IE and highlights the importance of maintaining diagnostic suspicion in patients with persistent fever and non-specific symptoms. Once large left-sided vegetations, significant valve dysfunction, or reduced β-lactam susceptibility are identified, management should be guided by microbiological results and contemporary surgical criteria, with early multidisciplinary discussion to reduce the risk of irreversible embolic or hemodynamic complications [5,6].

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