Recurrent Staphylococcus capitis Prosthetic Valve Endocarditis Presenting With ST-Segment Elevation Myocardial Infarction: A Case Report

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Abstract

We report a case of ST-elevation myocardial infarction (STEMI) due to septic emboli secondary to Staphylococcus capitis endocarditis in a 32-year-old male patient with a past medical history of infectious endocarditis requiring mechanical aortic, mitral and tricuspid valve replacement presented with sharp chest pain and shortness of breath. Electrocardiogram demonstrated an acute inferior STEMI.

Coronary angiography revealed occlusion of the terminal left anterior descending (LAD) artery associated with a large apical wrap-around segment exhibiting TIMI 0 flow. Primary angioplasty was not performed given the distal location of the embolus. Clinical suspicion for septic or thrombotic coronary artery embolism was high given the patient’s history of mechanical valve prosthesis and in the setting of sub-therapeutic INR. Transesophageal echocardiography revealed a new mobile echodensity on the mitral prosthesis consistent with vegetation. S. capitis was isolated from blood cultures, confirming the diagnosis of endocarditis.

S. capitis is a rare cause of prosthetic valve endocarditis and should remain in the differential of septic coronary artery embolism among patients with features of infectious endocarditis.

Introduction

Prosthetic valve endocarditis (PVE) is a rare but rather serious and potentially life-threatening complication after valve replacement [1]. PVE significantly differs from native valve endocarditis in terms of both morbidity and mortality. Two main issues are critical when facing potential cases of PVE, establishing a diagnosis, and implementing effective treatment. Even though there is no characteristic clinical presentation that would be unique for PVE, patients might simply present with fever and loss of appetite, symptoms that otherwise are seen in the post-operative period and the patients might be neglected and not recognized early on. Surely, they can present with new or worsening heart failure, a new or changed heart murmur, heart block, or any other new conduction abnormality (LBBB), while others might have a cerebral embolic event or myocardial infarction [2]. It has been reported that PVE accounts for 20% of all cases of endocarditis with an incidence ranging from 0.3% to 1.2% per patient-year while the most common causative organisms are Staphylococcus aureus in early PVE (less than 12 months after valve replacement) and coagulase-negative Staphylococci in late PVE (greater than 12 months after valve replacement) [3].

Staphylococcus capitis is a coagulase-negative Staphylococci (CoNS) which is part of the normal flora of the human face and scalp [4]. CoNS are well known to possess the ability to form biofilms leading to their virulence in the development of device-associated infections [5]. But S. capitis in particular has a lower ability to attach foreign body surfaces as compared to other CoNS, which makes it a rare microorganism causing PVE [6]. In addition, it has not been reported to be associated with coronary artery embolism.

We report a case of ST-elevation myocardial infarction (STEMI) due to septic emboli secondary to S. capitis endocarditis. Due to the rarity of cases previously reported with S. capitis, we feel that this case will be a useful addition to the existing literature. It is important to identify the offending organism in any given case so that effective treatments can be effectively implemented in a timely fashion and for expansion of guideline-based management with emphasis on prophylaxis or prevention strategies if warranted in the future.

Case Presentation

A 32-year-old male patient with a past medical history of infectious endocarditis requiring mechanical
aortic, mitral, and tricuspid valve replacement in 2011 presented with sharp chest pain and shortness of
breath. The patient was afebrile, had a blood pressure of 100/44 mmHg, pulse 76 beats/min, and chest
auscultation revealed a systolic murmur and metallic click over the upper left sternal border.
Electrocardiogram demonstrated an acute inferior STEMI (Figure 1).

![Figure 1: EKG showing ST-elevation MI in inferior leads](image)

Coronary angiography revealed occlusion of the terminal left anterior descending (LAD) artery associated
with a large apical wrap-around segment exhibiting TIMI 0 flow (Figure 2a). Primary angioplasty was not
performed given the distal location of the embolus. Clinical suspicion for septic or thrombotic coronary
artery embolism was high given the patient’s history of mechanical valve prosthesis and in the setting of
sub-therapeutic INR. Transesophageal echocardiography (TEE) revealed a new mobile echo density on the
mitral prosthesis consistent with vegetation (Figure 2b). S. capitis was isolated from blood cultures,
confirming the diagnosis of endocarditis. The patient was treated with IV vancomycin for six weeks
according to organism susceptibility testing, was anticoagulated, and discharged without incident. A repeat
blood culture came back negative after three days of vancomycin. The patient was stable on discharge.
Follow-up TEE showed resolution of echo dense mass on the mitral prosthesis.

![Figure 2: (a) Yellow arrow points to distal LAD thrombus in LAO cranial view. (b) Red arrow points to vegetation on the mitral valve in TEE.](image)

**Discussion**

Coagulase-negative staphylococci, mainly *Staphylococcus epidermidis* is well documented in the literature to
cause PVE. Its role in native valve endocarditis is much smaller (<10%) [7,8].

*S. capitis* is a coagulase-negative, novobiocin-sensitive, aerobic, and hemolysis-positive. It lacks the alkaline
phosphatase activity that is seen in *S. epidermidis* [9]. Coagulase-negative staphylococci (CoNS) are
constituents of our normal skin flora and have been shown to colonize foreign materials in the human body.
*S. capitis* mainly colonize the skin of the scalp, ears, neck, and face and it accounts for approximately 5% of
coagulase-negative isolates that cause pneumonia, urinary tract infection, cellulitis, and bacteremia [9-15]. The adhesion ability of \textit{S. capitis} to foreign surfaces is lower compared to most CoNS; however, \textit{S. capitis} as a cause of infective endocarditis is well documented in the literature but not that common. Additionally, there is a small number of cases that involve prosthetic valves [16-18]. In these cases, early removal of the prosthetic valve appears to be effective. Other cases with \textit{S. capitis} native valve endocarditis are successfully treated with antibiotics alone.

It is well documented in the literature that \textit{S. aureus} is the most common cause of PVE, followed by the CoNS \textit{Enterococcus} and \textit{Streptococcus viridans} [19]. The ability of CoNS to adhere and grow on prosthetic devices is very important in causing disease. However, \textit{S. capitis} has a weaker adhesion capability to smooth surfaces compared to other CoNS [20,21]. The adhesion ability of \textit{S. capitis} to prosthetic valves is due to biofilm production as well as secretion of exoenzymes [13,21]. Of note, \textit{S. capitis} has not been reported in the literature to be associated with coronary artery embolism.

Our patient is a young male who had infectious endocarditis requiring mechanical aortic, mitral, and tricuspid valve replacement, presented with sharp chest pain and shortness of breath. \textit{S. capitis} was isolated from blood cultures, confirming the diagnosis of endocarditis. The patient was treated with IV vancomycin for six weeks according to organism susceptibility testing, was anticoagulated, and discharged without incident. A repeat blood culture came back negative after three days of vancomycin. Treating PVE remains a challenge. PVE has a mortality rate of 21%-28.4% [20]. However, most of the patients with \textit{S. capitis} PVE reported in the literature as well as our current case showed improvement after antibiotic treatment only. Only a few cases required surgical intervention with antibiotics (Table 1).

| Case no | Age, gender | Valve affected | Management | Reference |
|---------|-------------|----------------|------------|-----------|
| 1       | 72, M       | Mitral         | Vancomycin + Gentamicin | [11]      |
| 2       | 53, M       | Mitral         | 2 weeks of amoxicillin + netilmicin followed by 4 weeks of ceftriaxone | [17]      |
| 3       | 63, M       | Tricuspid      | Cloxacillin | [14]      |
| 4       | 29, M       | Mitral         | Penicillin + Gentamicin | [15]      |
| 5       | 62, M       | Mitral         | Surgery and Vancomycin + Gentamicin. Later treated with Penicillin and Gentamicin and then Rifampin and Pefloxacin. | [15]      |
| 6       | 70, M       | Aortic         | Nafcillin + Gentamicin | [10]      |
| 7       | 73, M       | Mitral         | Ampicillin + Gentamicin + Cloxacillin | [7]       |
| 8       | 46, M       | Aortic         | Vancomycin + Rifampin | [22]      |
| 9       | 35, M       | Aortic         | Vancomycin + Rifampin | [22]      |
| 10      | 35, M       | Aortic         | Vancomycin + Rifampin | [23]      |
| 11      | 79, F       | Aortic         | Surgery + IV Vancomycin and oral minomycin | [24]      |
| 12      | 79, F       | Aortic         | Surgery + Vancomycin and Rifampin | [24]      |
| 13      | 76, M       | Aortic         | Surgery + IV teicoplanin and linezolid | [24]      |
| 14      | 68, F       | Mitral         | Surgery + IV Vancomycin and Gentamicin | [24]      |

**TABLE 1: \textit{S. capitis} PVE reported in the literature with treatment included.**

PVE: Prosthetic Valve Endocarditis

**Conclusions**

\textit{S. capitis} is a rare cause of prosthetic valve endocarditis and should remain in the differential of septic coronary artery embolism among patients with features of infectious endocarditis. Correct identification of coagulase-negative \textit{Staphylococci} is important to help in the decision to manage the disease either with medical or combined medical and surgical intervention.
Additional Information

Disclosures

Human subjects: Consent was obtained or waived by all participants in this study. Conflicts of interest: In compliance with the ICMJE uniform disclosure form, all authors declare the following: Payment/services info: All authors have declared that no financial support was received from any organization for the submitted work. Financial relationships: All authors have declared that they have no financial relationships at present or within the previous three years with any organizations that might have an interest in the submitted work. Other relationships: All authors have declared that there are no other relationships or activities that could appear to have influenced the submitted work.

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