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RESEARCH ARTICLE

A SILENT INVADER: CUTIBACTERIUM ACNES ENDOCARDITIS PRESENTING WITH OBSTRUCTIVE MITRAL VEGETATION AND CATASTROPHIC EMBOLIC COMPLICATIONS

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Abstract

Background: Infective endocarditis (IE) is a life-threatening condition, particularly in elderly patients with high mortality rates if not treated promptly. An uncommon but severe manifestation of IE is the development of obstructive vegetation on the native mitral valve, leading to systemic embolization including coronary embolism, splenic and renal infarction with hemorrhagic transformation of cerebral stroke. While cases of prosthetic mitral valve endocarditis with resultant stenosis have been reported such as cases involving Cutibacterium acnes infection, stenotic complications in native mitral valve endocarditis remain exceptionally rare. The management of such cases is particularly challenging due to the critical balance between the urgent need for surgical intervention and the heightened hemorrhagic risk that may contraindicate anesthesia, thus future guidelines should establish specific criteria to optimize surgical decision-making in these high-risk scenarios.

Case presentation: We present a case of infective endocarditis affecting the native mitral valve characterized by an obstructive vegetation caused by Cutibacterium acnes. The condition was complicated by multiple embolic events involving the coronary, cerebral, renal, and splenic circulation. The development of embolic phenomena in infective endocarditis is not uncommon, but their occurrence as a result of a vegetation obstructing the mitral valve is an atypical presentation, requiring immediate intervention.

Conclusion: While embolic complications are a well-recognized consequence of infective endocarditis, their occurrence secondary to a mitral valve obstruction remains an unusual presentation, necessitating urgent intervention.

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Introduction:

Infective endocarditis (IE) is a severe condition associated with high in-hospital mortality rates (15-20%) [1], with stroke being the second most frequent cause of death in affected patients [2]. While IE primarily predominantly involves the aortic and mitral valves, its clinical presentation can be highly variable, posing significant diagnostic

and therapeutic challenges. While IE is most commonly caused by *Staphylococcus aureus* and *Streptococcus* species, *Cutibacterium acnes* is a rare causative pathogen, typically associated with prosthetic valve infections and postsurgical endocarditis [3,4]. Native mitral valve involvement by *Cutibacterium acnes* is exceptionally rare, and its indolent course can delay diagnosis, increasing the risk of severe complications [5] with a mortality rate of 16 % [6]. We present a rare case of obstructive vegetation on a native mitral valve caused by *Cutibacterium acnes*, an infrequent etiology in clinical practice. The resulting vegetation led to significant functional mitral stenosis, complicated by coronary, cerebral, renal, and splenic embolism, as well as hemorrhagic transformation of an ischemic stroke.

Case report:

A 56-year-old patient with unknown cardiovascular risk factors or notable medical history presented to the emergency department with a two-week history of fever, asthenia, and progressive weakness associated with febrile low back pain.

On examination, the patient was conscious, hemodynamically stable, and able to tolerate the supine position. He was eupneic with a respiratory rate of 14 cycles per minute and a peripheral oxygen saturation SpO₂ of 94%. Notably, he was tachycardic (heart rate: 140 beats per minute) but normotensive (blood pressure: 120/55 mmHg). The patient was febrile at 39°C, and oral examination revealed poor dental hygiene. There were no signs of congestive heart failure, and pulmonary auscultation was unremarkable. Cardiovascular examination revealed a murmur suggestive of mitral stenosis. Neurological assessment was unremarkable, and the neck was supple.

The initial electrocardiogram (Figure 1) demonstrated sinus tachycardia at 100 bpm with a normal axis and signs of left ventricular hypertrophy (LVH) with ST segment depression in lateral and inferior leads. A skin examination revealed track marks over the left antecubital fossa and Janeway lesions on the fingertips. A chest X-ray was unremarkable. Given the high clinical suspicion of infective endocarditis (IE), two sets of blood cultures were obtained.

A transesophageal echocardiography (TEE) identified an 11 mm obstructive vegetation attached to the distal third of the anterior mitral valve leaflet, with fine added elements but no evidence of intra-atrial or intraventricular thrombus. Left ventricular ejection fraction was preserved (Figure 2).

Given the potential risk of atrioventricular (AV) block progression, the patient was transferred to the cardiac intensive care unit for continuous monitoring.

Laboratory assessment showed a significant infectious syndrome with a C-reactive protein CRP of 369mg/dl, procalcitonin of 115 ng/mL, leukocytosis (17,000 cells/mm³), and thrombocytopenia (platelet count: 80,000/mm³), the troponin level was elevated to 5000 ng/ml (Table 1).

A thoracoabdominal computed tomography (CT) scan was performed as part of the embolic workup, revealing multiple splenic and renal infarctions (Figure 3), which explained the patient's low back pain.

On the second day of hospitalization, the patient developed a sensorimotor deficit characterized by dysarthria and left-sided hemiparesis. An urgent cerebral CT scan identified an acute ischemic stroke in the right frontoparietal region. Twenty-four hours later, the patient exhibited a decline in Glasgow Coma Scale (GCS) score, prompting an emergent follow-up neuroimaging, which revealed a large hemorrhagic transformation involving the right frontoparieto-occipital region (fig 4).

The clinical course rapidly deteriorated with the onset of bilateral mydriasis and signs of brain herniation, necessitating urgent endotracheal intubation. Unfortunately, the patient suffered a sudden cardiorespiratory arrest on the spot, which remained refractory to resuscitative measures. Blood cultures grew *Cutibacterium acnes* ten days after they were drawn.

Variable	Results
Sodium (mmol/L)	139
Potassium (mmol/L)	4.4
Chloride (mmol/L)	98
Blood glucose (g/L)	1.20
Serum bicarbonate (mmol/L)	27
Urea (g/L)	0.32
Creatinine (mg/L)	10.3
ASAT (U/L)	16
ALAT (U/L)	19
GGT (U/L)	23
CRP (mg/dL)	369
Procalcitonin (ng/mL)	115
Troponin (ng/ml)	5000
White blood cells (/mCL)	17000
Neutrophils	12000
Hemoglobin (g/dL)	12.9
Platelets (/mCL)	80000

Table 1: Biological parameters on admission to emergency room

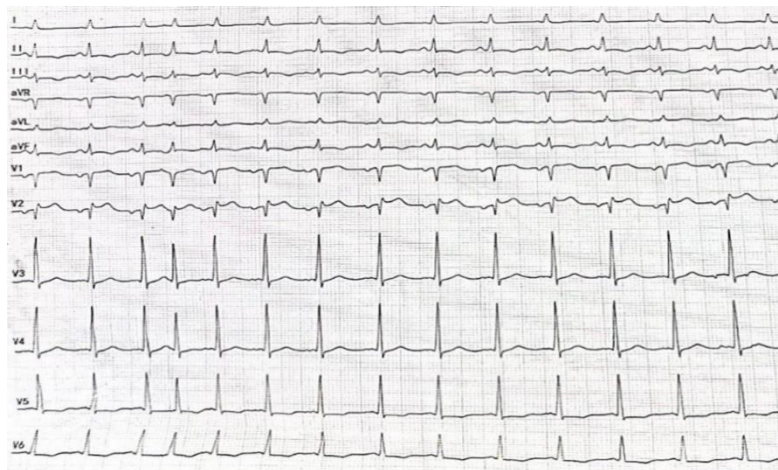


Figure 1: Electrocardiogram demonstrating sinus tachycardia at 100 bpm with a normal axis and signs of left ventricular hypertrophy (LVH) with ST segment depression in lateral and inferior leads

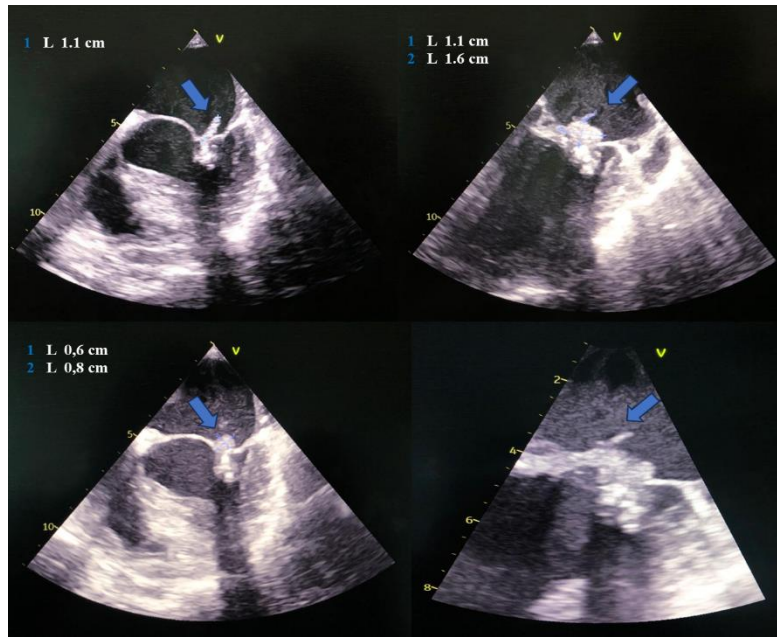


Figure 2: Transesophageal echocardiography (TEE) demonstrating an 11 mm obstructive vegetation attached to the distal third of the anterior mitral valve leaflet, with fine added elements (blue arrow)

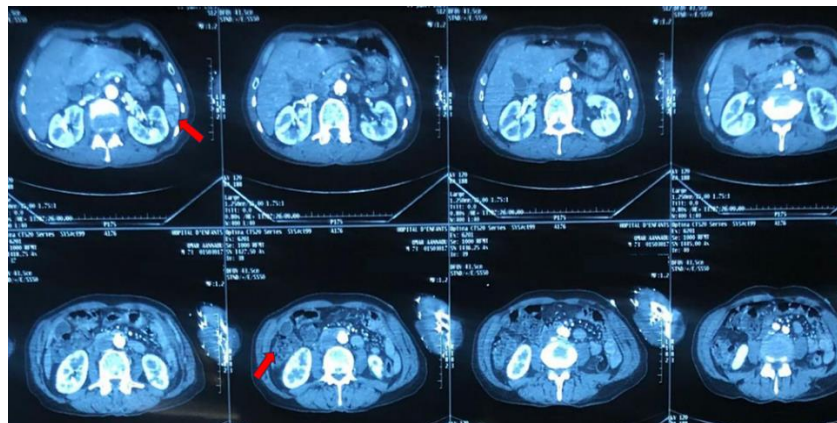


Figure 3: Thoracoabdominal computed tomography (CT) scan revealing multiple splenic and renal infarctions (red arrow)

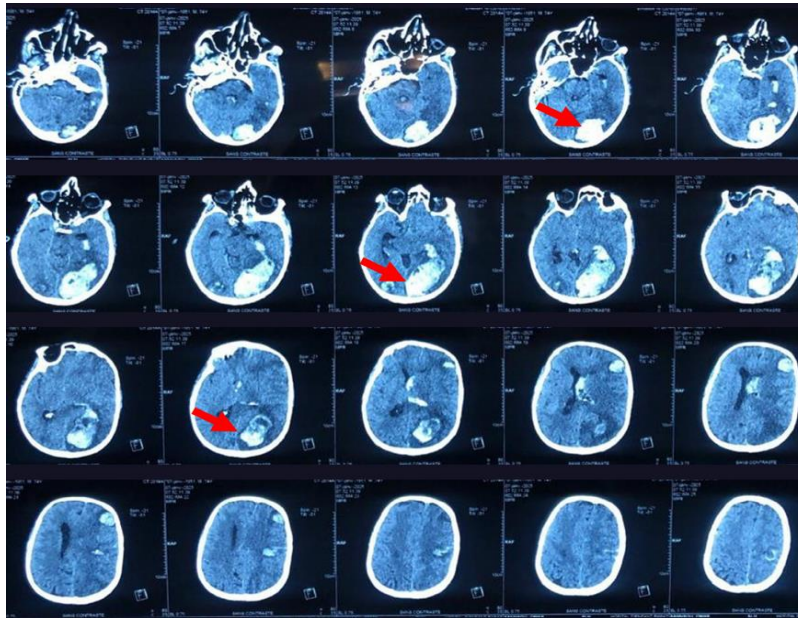


Figure 4: Cerebral tomography scan revealing a large hemorrhagic transformation involving the right frontoparieto-occipital region (red arrow)

Discussion:

Infective endocarditis (IE) presenting primarily as valvular stenosis is an exceptionally rare phenomenon. The estimated annual incidence of IE in developed countries ranges between 3 and 10 cases per 100,000 individuals, a figure that has remained relatively stable over the past two decades [1]. While valvular regurgitation is the most frequently observed hemodynamic consequence of IE, mitral stenosis patent hemodynamically remains an unusual finding [2]. Few cases have been documented in which mitral valve obstruction resulted from extensive vegetation, and only two cases have described diffuse thickening of a prosthetic valve leaflet leading to stenosis [2].

To date, only 15 cases of bacterial endocarditis affecting a mitral prosthetic valve and causing stenosis by obstruction have been reported in the literature [3]. Despite its rarity, IE should remain a differential diagnosis in patients presenting with mitral valve obstruction, particularly in the presence of predisposing factors.

Infective endocarditis represents a clinical challenge, particularly when associated with large mitral valve vegetations that carry a significant risk of hemodynamic deterioration and embolic complications. The case presented herein describes an extensive vegetation leading to severe mitral stenosis and a cascade of systemic embolization, including ischemic stroke, renal infarction, and hepatic embolization.

The causative pathogen, *Cutibacterium acnes*, poses a significant diagnostic challenge due to its ubiquitous presence as a skin commensal [9], frequently leading to its misinterpretation as a contaminant when isolated from a single blood culture [10]. Unlike more common IE pathogens, *C. acnes* requires both aerobic and anaerobic culture conditions, with prolonged incubation to ensure reliable detection [11]. Several studies recommend extending blood culture incubation to 10 to 14 days to reduce the risk of false-negative results [12].

There have been reported cases of *Cutibacterium acnes* infective endocarditis in which patients remained afebrile and exhibited normal C-reactive protein levels, thereby complicating the clinical diagnosis [13]. This delay presents a critical therapeutic dilemma, as illustrated in the present case, where native mitral valve endocarditis progressed rapidly, culminating in multi-organ embolization and fatal neurological deterioration within 24 hours of diagnosis on transesophageal echocardiography, before the initiation of targeted antimicrobial therapy. This highlights the need for heightened clinical suspicion and revised microbiological protocols to enhance the timely detection and management of *C. acnes* endocarditis.

The diagnosis of IE remains primarily based on the modified Duke criteria [14], which emphasize key echocardiographic and microbiological findings such as vegetations, abscesses, prosthetic valve dehiscence, and

new valvular regurgitation. However, valvular stenosis is not currently recognized as a diagnostic criterion, even in the most recent updates to the European Society of Cardiology (ESC) guidelines [15]. While these guidelines provide essential diagnostic and therapeutic frameworks, certain cases such as the present one challenge existing classification paradigms and call for a more nuanced approach to defining IE-related complications.

Early cardiac surgery is recommended in cases complicated by acute heart failure, uncontrolled infection, or large vegetations [16]. However, the presence of concomitant intracranial hemorrhage introduces a significant management challenge, necessitating a careful risk-benefit analysis.

In patients with an unstable clinical course due to heart failure, refractory infection, or a persistent high embolic burden, urgent or emergent surgery should be considered, provided there is a reasonable expectation of neurological recovery [17].

Current ESC guidelines have become more restrictive regarding the indication for surgery based on vegetation size. In native aortic or mitral valve endocarditis, urgent surgery should be considered in patients with vegetations exceeding 10 mm when associated with severe valve stenosis or regurgitation, particularly in cases of low operative risk.

This raises the question of whether surgical intervention should be prioritized in all cases where the vegetation is mitral, even before embolic complications ensue. The case described herein illustrates the devastating consequences of delayed intervention, as the occurrence of hemorrhagic transformation, manifesting as meningeal hemorrhage, ultimately precluded surgery and led to a fatal outcome.

This underscores the urgent need for refined surgical criteria in IE, particularly in cases of mitral valve involvement, to optimize outcomes and prevent irreversible complications.

Conclusion:

In conclusion, this case of infective endocarditis on a native mitral valve, leading to mitral stenosis and cerebral and splenic embolizations with hemorrhagic transformation, highlights the complexities and challenges of managing such severe cases. The contraindication to surgery and the poor prognosis underlines the importance of early diagnosis and intervention. Future perspectives should focus on improving diagnostic techniques, optimizing medical therapies to control infection and prevent complications, and developing more effective surgical approaches for high-risk patients. These advancements could potentially improve outcomes and reduce mortality associated with infective endocarditis.

Abbreviations

EI	Infective endocarditis
C. acnes	Cutibacterium acnes
LVH	Left ventricular hypertrophy
TEE	Transesophageal echocardiography
CRP	C-Reactive protein
CT	Computed tomography
GCS	Glasgow coma scale

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None to declare

Declarations

Ethics approval and consent to participate

Not applicable

Consent for publication

Written informed consent was obtained from the patient for publication of this case report and accompanying images

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