

Native Valve Endocarditis: A Case Report of Classical Skin Signs and Review of Literature

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Abstract

Infective endocarditis, though an infective lesion of the heart, can lead to systemic complications. Embolisation and subsequent stroke can be a major manifestation apart from other vascular phenomena. Mortality in infective endocarditis depends on various factors including the type of organism and the extent and type of valvular involvement.

Case presentation: We report a case of a young male with native valve infective endocarditis, brought to attention by the systemic complications namely ischemic stroke and other vascular phenomena. The valve pathology leads to sepsis and subsequent mortality.

Conclusion: Native valve infective endocarditis should be approached as a multisystem based disease, as its morbidity and mortality are as a result of the systemic complications apart from cardiovascular pathology per se.

Keywords: Rheumatic heart disease; Infective endocarditis; Embolism; Vascular phenomena; Echocardiography

Introduction

Infective endocarditis is a rare disease, with an incidence of two to six episodes per 100,000 habitants/year. Incidence is higher in elderly people; besides, this group is often affected by much comorbidity [1].

Complications of infective endocarditis may involve cardiac structures when the infection spreads within the heart or extra cardiac ones when the cause is usually from embolic origin; they may also be due to medical treatment or to the septic condition itself [2].

Coagulase negative *staphylococci* (CNS) were a rare cause of native valve endocarditis. However, they are emerging as an important cause of native valve endocarditis (NVE) in both community and healthcare settings [3].

Case Presentation

A 27 years old male came to the emergency OPD with history of fever of 7 to 10 days duration, which was mild grade, intermittent and was followed by sudden onset right sided hemiplegia with loss of speech and loss of bowel and bladder function. On examination, he had multiple purple spots on palms of both hands and subungual linear hemorrhages. The patient was drowsy and had right sided hemiplegia (grade 0/5 power) with extensor plantar response on right side. On auscultation of chest, a pansystolic murmur (grade 3/6) was heard, with radiation to ipsilateral axilla. A trans-thoracic 2-D Echocardiography revealed rheumatic heart disease with moderate mitral stenosis (Mitral valve area-1.7 cm/sq) and severe mitral regurgitation (jet area-8 cm/sq.). A computed tomography scan of brain revealed an area of infarct in the region of left para-central lobule. His blood culture was positive for staphylococcus hominis ssp hominis. In spite of initiating appropriate antibiotic therapy, the patient showed deterioration in terms of leucocytosis and azotemia, which resulted in fatality (Figure 1).

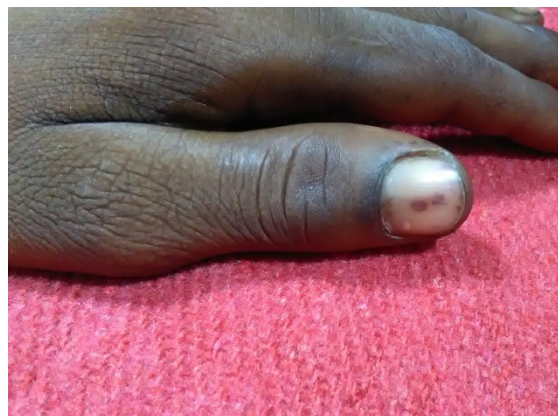


Figure 1: Splinter Hemorrhages.

Discussion

Infective endocarditis (IE) is the microbial infection of the endothelial lining of the heart which usually involves native or prosthetic valves but can also affect the adjacent structure of the valve, mural thrombus or cardio-vascular devices. In the presence of classical features such as fever, cardiac murmur, bacteremia and peripheral stigmata, the diagnosis of IE may be established easily. Unfortunately, in everyday clinical practice, this presentation is rarely seen and atypical presentation occurs more frequently. The clinical diagnosis of IE relies on microbiological, echocardiography and laboratory findings [4]. In a study conducted by Heiro et al. [5] neurological complications were identified in 55 episodes (25%), with an embolic event as the most frequent manifestation (23/55; 42%). In the majority (76%) of episodes, the neurologic manifestation was evident before antimicrobial treatment was started, being the first sign of IE in 47% of the episodes. Echocardiography has a strong predictive value in IE. Independently of other baseline characteristics, vegetation length has a major prognostic implications by predicting both embolic events under antibiotic therapy and mortality [6]. In a study conducted by Batool and Hussein [7], the causes of mortality (38.7%) included congestive heart failure (CHF), sepsis, surgery, stroke, cerebral haemorrhage, pulmonary embolism, sudden cardiac death, and hyperkalemia. In a study conducted by Reyn et al. [8], it was demonstrated that most cases were caused by *Viridans streptococci*, *Staphylococcus aureus*, or *Enterococci*. Enterobacteriaceae were absent and negative cultures infrequent (5%). Subgroups included nosocomial endocarditis (13%), usually with underlying valvular disease and invasive procedures; prosthesis endocarditis (12%); and cases requiring cardiac surgery (18%). Deaths were caused by heart failure, neurologic events, or superinfection. If untreated, Infective Endocarditis (IE) is a fatal disease. Major diagnostic (first of all echocardiography) and therapeutic progress (mainly surgery during active IE) have contributed to some prognostic improvement during the last decades. If the diagnosis is delayed or appropriate therapeutic measures postponed, mortality is still high [9]. Long term survival following infective endocarditis is 50% after 10 years and is predicted by early surgical treatment, age < 55 years, lack of congestive heart failure, and the initial presence of more symptoms of endocarditis (Figure 2) [10].

Review of Literature

Spectrum of IE in our country is different from west, but quite similar as reported from developed countries about 40 years ago. IE in our country occurs in relatively younger population with RHD as the commonest underlying heart disease [11]. The definitive incidence of endocarditis (IE) in the Indian population is variable, but the estimated incidence in western population is falling. *Streptococcus viridans* are responsible for 30-65% of native valve endocarditis (NVE) in India. Although *Staphylococcus* incidence is on rise [12].

Despite the introduction of new diagnostic tools, the cause of Infective Endocarditis has remained unchanged over a period of 16 years. Evidence of early dissemination of the disease to other

sites was associated with adverse outcome [13]. The diagnostic criteria employed for IE is the Modified Duke Criteria [14].

Definition of infective endocarditis according to the proposed modified Duke Criteria.

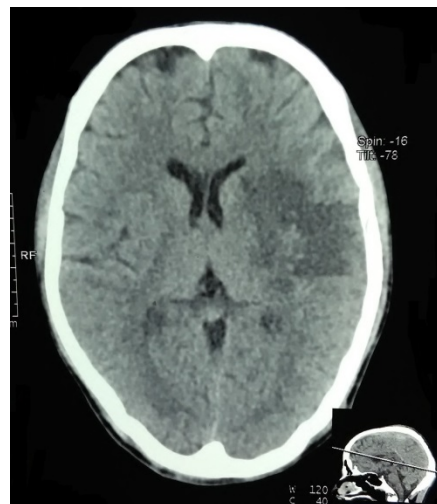


Figure 2: Infarct due to embolism as a result of endocarditis.

Definite infective endocarditis

Pathologic criteria

- Microorganisms demonstrated by culture or histologic examination of a vegetation, a vegetation that has embolized or an intracardiac abscess specimen; or
- Pathologic lesions; vegetation or intracardiac abscess confirmed by histologic examination showing active endocarditis

Clinical criteria

- (1) 2 major criteria; or
- (2) 1 major criterion and 3 minor criteria; or
- (3) 5 minor criteria

Possible infective endocarditis

- (1) 1 major criterion and 1 minor criterion; or
- (2) 3 minor criteria

Rejected

- (1) Firm alternate diagnosis explaining evidence of infective endocarditis; or
- (2) Resolution of infective endocarditis syndrome with antibiotic therapy for < 4 days; or
- (3) No pathologic evidence of infective endocarditis at surgery or autopsy, with antibiotic therapy for < 4 days; or
- (4) Does not meet criteria for possible infective endocarditis, as above

Definition of terms used in the proposed modified Duke criteria for the diagnosis of infective endocarditis (IE):

Major criteria:

Blood culture positive for IE

Typical microorganisms consistent with IE from 2 separate blood cultures:

Viridans streptococci, *Streptococcus bovis*, HACEK group, *Staphylococcus aureus*; or

Community-acquired *enterococci*, in the absence of a primary focus; or

Microorganisms consistent with IE from persistently positive blood cultures, defined as follows:

At least 2 positive cultures of blood samples drawn >12 h apart; or

All of 3 or a majority of >4 separate cultures of blood (with first and last sample drawn at least 1 h apart)

Single positive blood culture for *Coxiella burnetii* or antiphase I IgG antibody titer >1:800

Evidence of endocardial involvement

Echocardiogram positive for IE (TEE recommended in patients with prosthetic valves, rated at least "possible IE" by clinical criteria, or complicated IE [paravalvular abscess]; TTE as first test in other patients), defined as follows:

Oscillating intracardiac mass on valve or supporting structures, in the path of regurgitant jets, or on implanted material in the absence of an alternative anatomic explanation; or Abscess; or New partial dehiscence of prosthetic valve

New valvular regurgitation (worsening or changing of pre-existing murmur not sufficient)

Minor criteria:

- Predisposition, predisposing heart condition or injection drug use
- Fever, temperature >38°C
- Vascular phenomena, major arterial emboli, septic pulmonary infarcts, mycotic aneurysm, intracranial hemorrhage, conjunctival hemorrhages, and Janeway's lesions
- **Immunologic phenomena:** glomerulonephritis, Osler's nodes, Roth's spots, and rheumatoid factor
- **Microbiological evidence:** positive blood culture but does not meet a major criterion as noted above or serological evidence of active infection with organism consistent with IE
- Echocardiographic minor criteria eliminated

(Note: TEE=Transesophageal Echocardiography; TTE= Transthoracic Echocardiography).

In a study conducted by Gurusul et al. [15], the patients with Infective Endocarditis presented most commonly with fever

(67%), followed by weakness (49%), dyspnoea (24%), weight loss (24%) and chest pain (9%). On examination, cardiac murmur was most common finding (37%). Laboratory finding associated most commonly was high CRP (67%), followed by high ESR (62%) and anemia (61%). The distribution of patients according to microbiological findings was *Staphylococcus aureus* (11%), Coagulase negative *Staphylococci* (9%), *Streptococcus* (8%) and *Enterococcus* (7%). However in a study conducted by KPP Abhilash et al. [16], culture positive endocarditis was seen in 83.7%. *Streptococcus* species was the predominant etiological agent (44.7%), followed by *Staphylococcus* species (16.8%), *Enterococcus* (9.8%) and Gram-negative bacteria (9.3%). Native valve endocarditis was seen in 87.8% of patients while prosthetic valve and pacemaker endocarditis was seen in 10.4% and 1.75 respectively. Mitral valve was the most commonly affected valve (52.9%), followed by the aortic valve (23.2%).



Figure 3: Osler's Nodes.

Most (77%) patients presented early in the disease (<30 days) with few of the classical clinical hallmarks of IE. Complications were common: Stroke (17%), embolization other than stroke (23%), heart failure (32%), and intracardiac abscess (14%) [17]. In a study conducted by Servy et al. [18], 11.9% had skin manifestations, including 8.0% with purpura, 2.7% with Osler nodes, 1.6% with Janeway lesions, and 0.6% with conjunctival hemorrhages. Patients with skin manifestations had a high rate of IE-related extra cardiac complications than patients without skin manifestations, particularly cerebral emboli, without increased mortality.

The pathogenesis of Osler's nodes and Janeway lesions remain a mystery despite vigorous debate over the last 113 years. They are given great emphasis among the clinical signs of bacterial endocarditis but are seldom seen in practice. Although the appearance is usually consistent, it is not always possible to distinguish Osler's nodes from Janeway lesions based purely on clinical presentation (Figure 3) [19].

Conclusion

Native valve infective endocarditis should be approached as a multisystem based disease, as its morbidity and mortality are as a result of the systemic complications apart from cardiovascular pathology per sec.

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