Endocarditis of the Mitral Valve! *TEE Superiority!*

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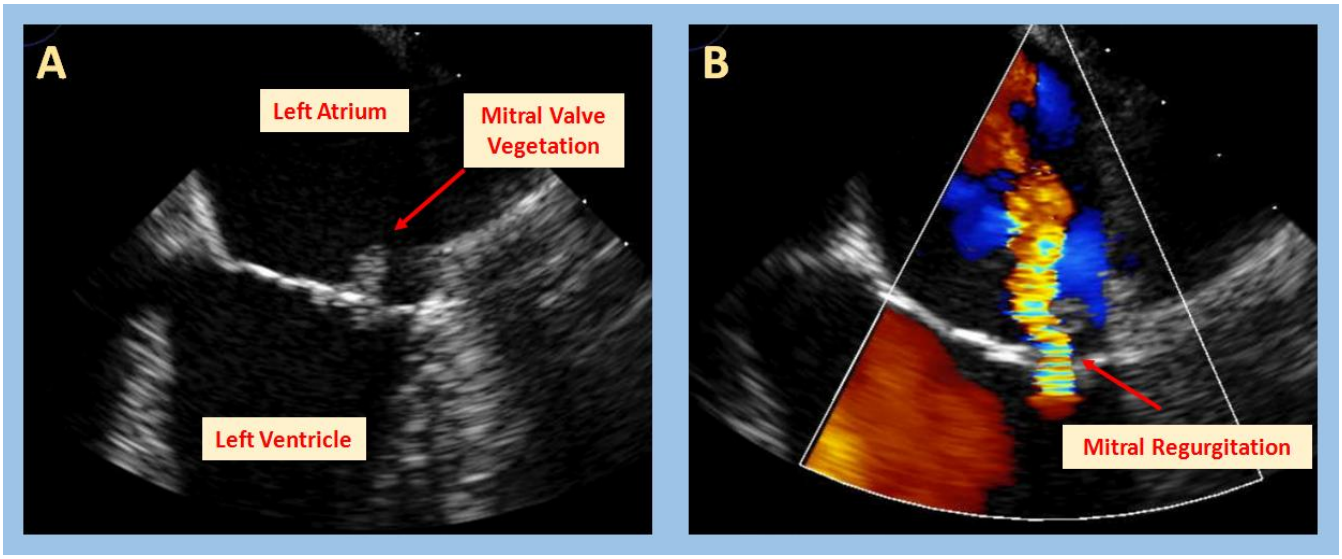

Description

The transesophageal echocardiography images and videos show mild thickening of the anterior and posterior mitral valve leaflets, along with a highly mobile oscillating mass that can be visualized on the atrial aspect of the posterior mitral valve leaflet seen in Image A (red arrow) and video A. The mass is consistent with a vegetation, commonly diagnosed in patients with Infective Endocarditis (IE). Auscultation of a new murmur on cardiac physical examination, which reflects valvular damage due to the infection and subsequent valvular regurgitation, is a sign of endocarditis. This phenomenon is illustrated by the mild to moderate mitral valve regurgitation seen in Image B (video B).

Manuscript submitted August 5, 2019, accepted August 6, 2019.

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https://cardiofellows.com/newsletter-august-2019.html

IE has a relatively rare occurrence – a recent study found the 2011 incidence in the United States to be 15 per 100,000 persons, a slight increase from an incidence of 11 per 100,000 persons in 2000 [1]. The endothelium lining the valves of the healthy heart is designed to resist infective processes, but when the inner lining becomes damaged or a particularly virulent microorganism invades the bloodstream, infective endocarditis may occur, most commonly on the aortic or mitral valves. Endothelial disruption on the valvular surface may be caused by congenital heart defects, valvular dysfunction, the presence of a prosthetic valve, or prior infection, amongst other things. The damage of the endothelial cells leads to activation of the clotting cascade and the release of various inflammatory cytokines, eventually forming a thrombus, composed primarily of platelets and fibrin. The newly formed thrombus serves as a nidus for bacterial colonization during any period of transient bacteremia. This colonization results in the release of more inflammatory and clotting factors, creating a feed-forward type of mechanism that ultimately ends with the formation of a vegetation. Alternatively, infection

ISSN 2689-291X https://doi.org/10.13140/RG.2.2.10987.98086

can occur on a relatively normal valve in the setting of bacteremia with a more virulent organism, most commonly *Staphylococcus aureus* [2]. The diagnosis of endocarditis is largely guided by the use of the modified Duke criteria. Using this approach 2 major, 1 major and 3 minor, or 5 minor criteria are necessary to definitively make the diagnosis of infective endocarditis. The major criteria consist of specific parameters surrounding positive blood cultures as well as information supporting the involvement of the endocardium, most commonly provided through echocardiography, although other imaging modalities such as cardiac CT can be used [2,3]. Like any serious infection, endocarditis can be accompanied by numerous complications such as heart failure, abscess formation, and heart block, due to compromise of the cardiac conduction system in the setting of perivalvular extension [4]. Traditionally, IE is treated with parenteral antibiotics, although recent investigation has shown that transitioning to oral antibiotics in clinically stable patients may be acceptable [5]. Surgical intervention may be indicated in certain scenarios including a large vegetation (typically >10 mm) which carries a high risk for embolization, a resistant and progressive infection or, most commonly, in the event of severe valvular dysfunction [2,3]. The mortality rate associated with IE has shown minimal improvement throughout the years, remaining close to 30% in most cases. However, rates vary from case to case and differ based on the location, the infectious organism, the acuity, and associated comorbid conditions [3].

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KEYWORDS: Vegetation; Mitral Valve; Endocarditis

Reference this article as:

Roberts M, Garriga G, Sachdev S, Omar B. Endocarditis of the Mitral Valve! TEE Superiority! Cardiofel Newslet 2019 August; 2(8): 36-37.