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Bacterial Mural Endocarditis. A Case Series

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Mural endocarditis represents a rare subset of intracardiac infections, with potentially life threatening sequelae. Clinically alike, with many shared aetiologies, substrates and risk factors such as valvular endocarditis, it can be difficult to differentiate without the use of sophisticated cardiovascular imaging techniques. Despite high rates of complications, there are no definite strategies for management. Herein we present three interesting cases of left ventricular mural endocarditis, without valvular involvement, due to *staphylococcus aureus*.

Introduction

Infective endocarditis entails a diverse spectrum of intracardiac infections, including valvular endocarditis, and less commonly, nonvalvular structures such as prosthetic material, intimal surfaces of the great vessels and endocardial surfaces of the heart. Mural endocarditis forms a small subset, which involves the inflammation and disruption of the non-valvular endocardial surface, and has a presentation similar to valvular endocarditis [1]. Predominantly involving right and left ventricular free walls and apices, mural vegetations can develop in any cardiac chamber, including unusual locations such as left ventricular aneurysms [2], right ventricular moderator band [3], papillary muscles [4], and mural thrombi. They can be solitary or multiple [5] and have been known to mimic other masses such as cardiac tumours [6]. The differential diagnosis also includes thrombi and blood cysts. The true incidence of mural endocarditis is unknown.

In this series we present three cases with bacterial mural endocarditis of the left ventricular cavity, each highlighting the multifaceted presentations and complications of the disease, and the management predicament they pose. In all three, there was absence of valvular endocarditis.

Case 1

A 49 year-old male intravenous drug user, with a past history of hepatitis C, was admitted with methicillin sensitive *S. aureus* (MSSA) bacteraemia. There was a five-day prodrome of pyrexia, rigors, lethargy, and features of neurological deficit.

On examination he was obtunded and confused, with temperature 40.1 °C, heart rate 112 beats per minute and blood pressure 100/50 mmHg. There were peripheral stigmata of infective endocarditis, with prominent finger clubbing, Janeway lesions and splinter haemorrhages. Neurological examination revealed left-sided weakness of the upper and lower limbs and dysarthria. Praecordial examination was otherwise unremarkable with no valvular murmurs or evidence of heart failure.

Blood tests revealed raised inflammatory markers, (white cell count (WCC) $11.6x10^9$/L, neutrophils $10.7x10^9$/L, C-reactive protein (CRP) 106 mg/L), with acute renal failure (urea 14.8 mmol/L, creatinine 131 µmol/L).

Soon after admission, there was rapid clinical deterioration, with ensuing septic shock requiring haemodynamic support. An urgent transthoracic echocardiogram (TTE) revealed a large solitary vegetation (2.5cmx1.5 cm) encasing

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the anterolateral papillary muscle, and extending to the lateral ventricular wall. The mitral leaflets were spared with preserved valvular function (Fig. 1A and 1B). Computed tomography (CT) of brain revealed multiple subcortical septic emboli within both cranial hemispheres.

Appropriate antistaphylococcal therapy was commenced. Despite the size of the vegetation, a non-surgical approach to minimise the chance of prosthetic valve requirement was taken, in light of active sepsis, illicit drug use, and high risk of intracranial haemorrhage.

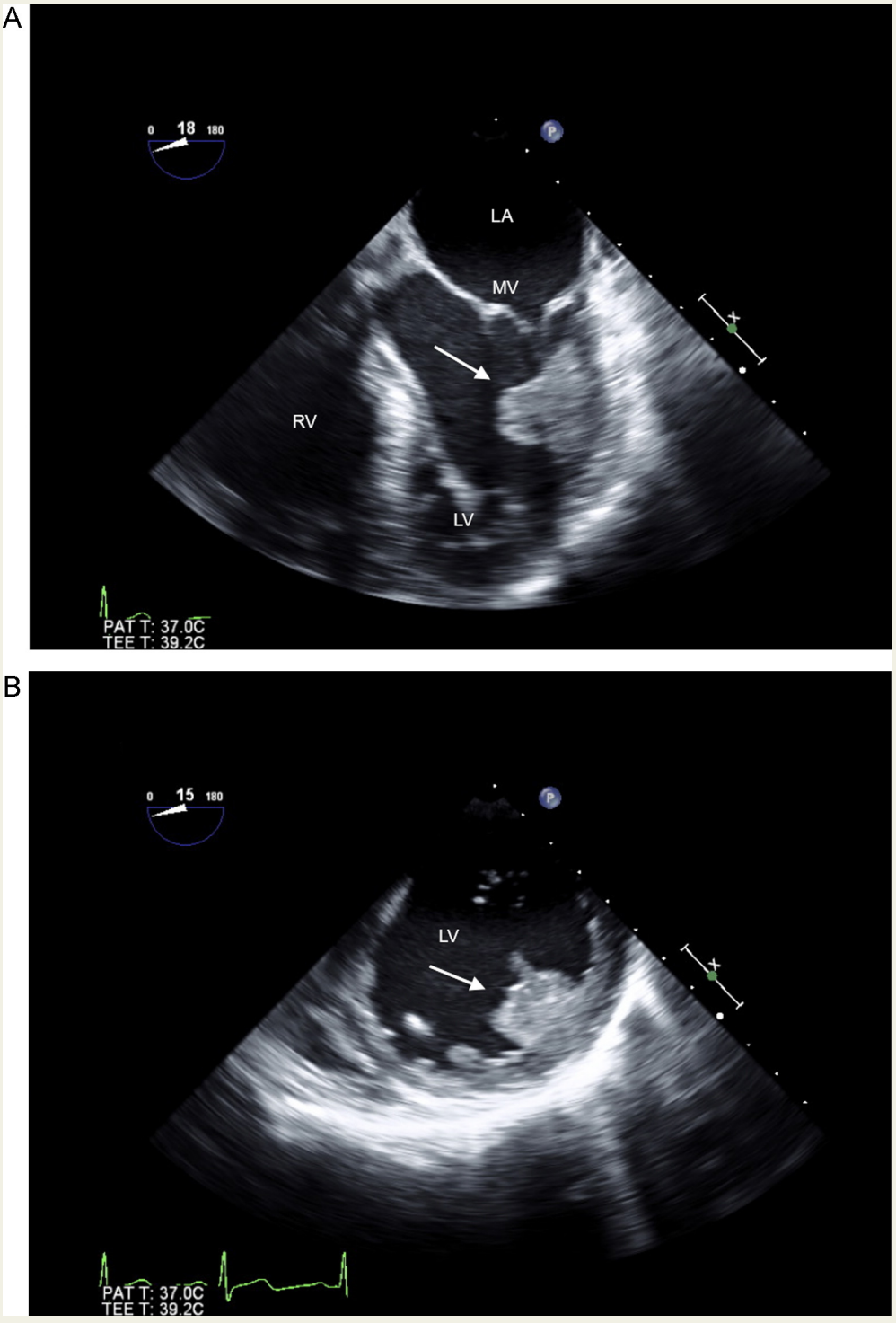

Figure 1 Large well-defined mass attached to the anterolateral papillary muscle (arrow) in the 5-chamber transoesophageal view of the left ventricle (A), and transgastric short axis view (B).