INTRODUCTION

Primary mural endocarditis is a rare form of intracardiac infection that occurs on endocardial surfaces independent of systemic valve involvement. More commonly, nonvalvular infective endocarditis occurs secondary to infected mural thrombus, intracardiac devices or prostheses, cardiac tumors, structural abnormalities including congenital defects, or valvular infective endocarditis.1 Risk factors for mural endocarditis otherwise include immunosuppression, intravenous drug abuse, prior cardiac surgery, and chronic debilitating disease.2 The clinical presentation of mural endocarditis is otherwise similar to that of infective valvular endocarditis.3 Mural endocarditis is most commonly caused by Streptococcus pyogenes and Streptococcus species, but any endocarditis caused by group A beta hemolytic Streptococcus is uncommon. 4,5 We describe a case of an immunocompromised patient with Streptococcus pyogenes bacteremia and clinical signs of myopericarditis who, after further evaluation, was diagnosed with primary mural endocarditis.

CASE PRESENTATION

A 37-year-old African American man with a medical history significant for HIV/AIDS and daily cannabis use presented to the emergency department reporting severe, stabbing, midsternal chest pain that started suddenly at rest. The patient reported an upper respiratory tract infection in the preceding weeks and described shortness of breath and subjective fever for several days. He also reported nonadherence to antiretroviral therapy within the year. On physical examination, he was normotensive and afebrile but notably tachycardic and tachypneic. Most notably, he had a prominent pericardial rub on auscultation, which was unlikely an isolated process.

The patient had symptomatic resolution on intravenous antibiotic therapy during his hospital stay, which was transitioned to ceftriaxone at discharge with a plan for completion of at least a 4-week course per infectious disease recommendations. Antiretroviral therapy was also restarted.

Transesophageal echocardiography was repeated 5 weeks after the initial study and after completion of the intravenous antibiotic course, revealing near resolution of the left atrial echo density (Figure 3, Video 5) and complete resolution of the pericardial effusion (Figure 4). The right atrial echo density had not significantly changed in appearance (Figure 5, Video 6). Blood cultures were repeated twice and showed no growth.

The patient was lost to follow-up after repeat transesophageal echocardiography, so further evaluation could not be performed, nor could we follow the patient to assess for further improvement.
DISCUSSION

Mural endocarditis is a rare manifestation of intracardiac bacterial or fungal infection that involves the nonvalvular endocardium and may involve any cardiac chamber. Generally, infective endocarditis occurs when pathogens adhere to damaged endothelial surfaces that are highly thrombophilic and activate procoagulant reactions, resulting in fibrin and platelet deposition, forming a nidus for microorganism adhesion and accumulation during bacteremia.1,6 Resulting plaque formation serves to promote the development of vegetations during transient bacteremia. Primary mural endocarditis is a rare form of intracardiac infection that more commonly occurs as an extension of infected mural thrombi, contaminated prosthetic materials, intracardiac tumors, or infected cardiovascular implantable electronic devices. In primary mural endocarditis, endothelial damage may be caused by high-velocity, eccentric regurgitant intracardiac jets produced by atrophicventricular valve disease that are directed toward the wall of a cardiac chamber.1,7,8

The most common causative organisms of mural endocarditis include S aureus, Streptococcus species, Candida species, and Aspergillus species.2 Our patient is the second reported case of primary mural endocarditis caused by group A beta hemolytic Streptococcus bacteremia; the first involved a 3-year-old girl with a history of developmental delay and Chiari malformation in 1992.9 S pyogenes is an overall rare cause of infective endocarditis in any age group.4,5 Although the primary source of infection in our patient is unclear, we suspect that his preceding upper respiratory tract infection was a predisposing factor, further complicated by his immunocompromised status.

Potential complications of mural endocarditis include peripheral embolization, abscess and fistula formation, papillary muscle or chordae compromise, and cardiac perforation.1 Thus, a high index of suspicion for mural endocarditis with early diagnosis is necessary. Given the scarceness of mural endocarditis and the unusual areas of vegetation involvement, diagnosis is challenging. Echocardiography is the principal imaging technique for diagnosis of valvular endocarditis and has been the primary modality used to diagnose mural endocarditis in the majority of reported cases.10 The overall diagnostic accuracy of echocardiography in the diagnosis of mural endocarditis specifically is unclear. Nonetheless, the transesophageal approach

**VIDEO HIGHLIGHTS**

Video 1: Midesophageal view on transesophageal echocardiography showing fibrinous material within the pericardial effusion at the time of initial diagnosis.

Video 2: Midesophageal four- and five-chamber view on transesophageal echocardiography shows the mitral annular mass and tricuspid annular thickening at the time of initial diagnosis.

Video 3: Midesophageal two-chamber view with biplane method shows a solid left atrial mass along the anterior mitral valve annulus (white arrow) that is independent of the valve leaflets and appears to be partially mobile. LA, Left atrium; LV, left ventricle.

Video 4: Apical four-chamber view with color Doppler showing trace to mild tricuspid and mitral regurgitation on initial transesophageal echocardiography.

Video 5: Repeat transesophageal echocardiography performed after completion of intravenous antibiotics shows near normalization of the anterior mitral valve annulus. Ao, Aorta; LA, left atrium; LV, left ventricle.

Video 6: Repeat transesophageal echocardiography performed after completion of intravenous antibiotics shows persistent thickening of the tricuspid valve annulus.

View the video content online at www.cvcasejournal.com.
alone approaches sensitivity rates for detection of valvular vegetations between 90% and 100%, so nondiagnostic transthoracic echocardiography does not preclude the diagnosis of any form of endocarditis. Transesophageal echocardiography resulted in the diagnosis of mural endocarditis in our patient.

Guidelines for treatment of valvular endocarditis suggest early surgical intervention when clinically appropriate, but a paucity of data pertaining to mural endocarditis limits guidance toward recommended treatment strategies. Previous cases have reported failure of antimicrobial therapy alone in resolution of mural endocarditis. In our case, however, the patient was successfully treated using conservative methods given no evidence for serious complications, and there was clinical improvement and resolution of vegetations with antimicrobial therapy. Annular thickening was presumably related to endocardial or myocardial inflammation in this case and remained unchanged. There was no prior cardiac imaging to compare and no clear indication for pathologic assessment to confirm this. Patients with large mural vegetations, myocardial abscess formation, and structural heart disease resulting in propensity toward infection (i.e., aneurysm with mural thrombosis) have had subsequent clinical deterioration in lieu of long-term antibiotic therapy. In these patients, surgical vegetectomy or aneurysmectomy have been pursued.

CONCLUSION

Primary mural endocarditis is rarely reported but presents similarly to infective valvular endocarditis, with complications that are potentially as severe. Even rarer is primary mural endocarditis caused by *S pyogenes* bacteremia. Diagnosis and management have not clearly been defined, with treatment recommendations suggested on a case-by-case basis. In our case, the patient had clinical and imaging resolution of the left atrial mural vegetation with long-term antibiotic therapy alone, although more complex cases may necessitate surgical

Figure 3 Midesophageal view on transesophageal echocardiography shows a mass on the anterior mitral annulus (white arrow) at the time of diagnosis (left). The right is a similar view after completion of antimicrobial therapy showing marked improvement in the appearance of the mitral annular mass (white arrow).

Figure 4 Midesophageal view on transesophageal echocardiography with a focus on the right heart showing complete resolution (right, white arrow) of a fibrinous pericardial effusion (left, white arrow) after completion of antimicrobial therapy. RA, Right atrium; RV, right ventricle.
intervention. Echocardiography is recommended to confirm diagnosis where there is a high index of suspicion.

SUPPLEMENTARY DATA

Supplementary data related to this article can be found at https://doi.org/10.1016/j.ccase.2019.09.001.

REFERENCES

1. Kearney RA, Eisen HJ, Wolf JE. Nonvalvular infections of the cardiovascular system. Ann Intern Med 1994;121:219-30.
2. Tahara M, Nagai T, Takase Y, Takiguchi S, Tanaka Y, Kunihara T, et al. Primary mural endocarditis without valvular involvement. J Ultrasound Med 2017;36:659-64.
3. Adel A, Jones E, Johns J, Farouque O, Calafiore P. Bacterial mural endocarditis. A case series. Heart Lung Circ 2014;23:e172-9.
4. Yeşilkaya A, Azap ÖK, Pirat B, Gültekin B, Arslan H. A rare cause of endocarditis: Streptococcus pyogenes. Balkan Med J 2012;29:331.
5. Burkert T, Watanakunakorn C. Group A Streptococcus endocarditis: report of five cases and review of literature. J Infect 1991;23:307-16.
6. Brinkman CL, Patel R. Molecular pathogenesis of infective endocarditis. In: Molecular medical microbiology. San Diego, CA: Academic Press; 2015: 811-22.
7. Gregory SA, Yepes CB, Byrne JG, D’Ambra MN, Chen MH. Atrial endocarditis—the importance of the regurgitant jet lesion. Echocardiography 2005;22:426-30.
8. Keynan Y, Rubinstein E. Pathophysiology of infective endocarditis. Curr Infect Dis Rep 2013;15:342-6.
9. Liu VC, Stevenson JG, Smith AL. Group A Streptococcus mural endocarditis. Pediatr Infect Dis J 1992;11:1060.
10. Shirani J, Keffer K, Gerszten E, Gbur CS, Arrowood JA. Primary left ventricular mural endocarditis diagnosed by transesophageal echocardiography. J Am Soc Echocardiogr 1995;8:554-6.
11. Casella F, Rana B, Casazza G, Bhan A, Kapetanakis S, Omigie J, et al. The potential impact of contemporary transthoracic echocardiography on the management of patients with native valve endocarditis: a comparison with transesophageal echocardiography. Echocardiography 2009;26:900-6.

Figure 5  Midesophageal view on transesophageal echocardiography with a focus on the right heart showing no significant change in the appearance of the tricuspid annulus (white arrows) at the time of diagnosis (left) and after completion of antimicrobial therapy (right). Although this finding suggested a possible alternative underlying process, the patient was lost to follow-up before further investigation could be pursued. The tricuspid valve leaflets are seen independent of the thickened annulus (black arrow). Varying quality between studies is likely responsible for increase brightness of the tricuspid annulus on posttreatment transesophageal echocardiography.