Case Report

Septic Thrombophlebitis Causing Pulmonary Valve Endocarditis and Septic Pulmonary Emboli: A Rare and Troublesome Trifecta

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Abstract

Pulmonic valve endocarditis is a rare entity with a prevalence of 1.5-2.0% in all cases of endocarditis. As few as 45 cases were reported in patients with structurally normal hearts between 1960 and 2005 [1-4]. Several cases have been reported with unique etiologies and have been seen in patients with PDA, sickle cell, Valsalva sinus aneurysm, skin infection of the hallux, VSD, and following a pulmonary artery catheterization [7-12]. We present a case of septic thrombophlebitis inducing pulmonary endocarditis and septic pulmonary emboli.

CASE DESCRIPTION

A 25-year-old female with history of IV drug abuse with a recent IV use to the left lower extremity was admitted with septic shock endorsing a 1-week history of fatigue, fevers, chills and left lower leg pain. Physical exam revealed a febrile, ill appearing female with a split S2 and lower extremity swelling with multiple ecchymoses. Broad spectrum antibiotic therapy was initiated along with intravenous vasopressors. Five temporally separated blood cultures were obtained and all were negative. Initial imaging showed diffuse left lower extremity deep vein thrombosis. CT angiogram on hospital day 2 revealed multiple septic emboli in the lungs. A three-dimensional transesophageal echocardiogram on hospital day 3 showed moderate pulmonic valve regurgitation. Three dimensional images were obtained in the high esophageal view showing vegetations involving the right and non-coronary cusps. The echocardiographic findings with a predisposition (IVDA), fevers and vascular phenomena met the Duke criteria for a diagnosis of infective endocarditis. The patient clinically improved with intravenous antibiotic therapy, completing a 28 day course. Surgery was deferred given clinical response and likelihood of continuing intravenous drug abuse. A transthoracic echocardiogram was performed at one month follow up which did not show evidence of pulmonic valve vegetations. In addition, the pulmonic regurgitation improved from moderate to trace (Video 1). Systolic function and size of both ventricles remained normal.

DISCUSSION

Pulmonic valve endocarditis may go undiagnosed for some time due to its often indolent course. Because of non-specificity of symptoms and the lack of typical peripheral findings that associated with left-sided endocarditis, the diagnosis of pulmonic valve endocarditis may be delayed for up to 6 months, with a mean of delay noted in one study of 65 days. [7]. Pulmonic valve endocarditis is rare compared with the other cardiac valves, and usually only seen in the setting of tricuspid endocarditis [7]. Causative organisms are similar to other valves with staphylococcus aureus being the most common microorganism detected in blood culture. Approximately 10% of reported cases

are culture negative [13,14]. The development of pulmonary valve endocarditis sparing the tricuspid valve complicated by septic pulmonary emboli following septic thrombophlebitis represents an unusual and interesting case. In culture negative endocarditis, parenteral antibiotic therapy is generally recommended for 4 to 6 weeks, while indications for surgery remain the same as for endocarditis of the tricuspid valve, i.e. locally invasive infections, including abscess formation, progressive valve obstruction, incompetence, and relapsing infection despite full-dose antibiotic therapy. Studies have suggested that vegetation less than 1-2 cm long usually responds well to medical therapy, which occurred in this case [5,6]. Of importance, there is a surgical indication in patients with severe valve regurgitation and mobile vegetations greater than 10 mm to prevent emboli, although it is generally not advisable in the IV drug abuse population [15]. In our case, as the patient already demonstrated septic emboli, the decision was made to undergo a prolonged course of antibiotics in a controlled hospital setting. As three-dimensional echocardiographic technology continues to improve, there will likely be greater elucidation of the clinical picture of pulmonic valve endocarditis.

REFERENCES


