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CASE REPORT

Isolated pulmonary valve endocarditis masquerading as community-acquired pneumonia

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Isolated pulmonary valve endocarditis in intravenous drug users is a rarely reported phenomenon. We present the case of a 25-year-old male with history of intravenous drug use who presented with respiratory symptoms after failing outpatient treatment for community-acquired pneumonia. Further investigations identified multiple lung lesions with early cavitation, concerning for septic pulmonary embolism on computerized tomography scan, positive blood cultures with methicillin-susceptible *staphylococcus aureus*, and isolated vegetation of the pulmonic valve on transthoracic echocardiography. The patient had a complete recovery after being treated medically with intravenous oxacillin for a total of 6 weeks.

Keywords: *native pulmonary valve*; *bacterial endocarditis*; *staphylococcus aureus*

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Isolated pulmonary valve endocarditis (PVE) is an extremely uncommon entity that accounts for less than 2% of all reported cases of endocarditis (1). Common risk factors include intravenous drug use, indwelling catheters, congenital heart disease, pacemakers, or prosthetic valves. Diagnosis is challenging mainly because of non-specific signs and symptoms at presentation. Common clues include presence of lung infections due to septic pulmonary emboli or new onset pulmonary valve insufficiency in patients with underlying risk factors. Furthermore, echocardiograph views used in the assessment of pulmonic valve are often limited, and as a result vegetation of the pulmonic valve can frequently be missed. This case highlights the importance of careful evaluation of the pulmonic valve in patients with risk factors for right-sided endocarditis despite the presence of structurally normal, non-infected tricuspid valve.

Case presentation

A 25-year-old male presented to a community hospital with the chief complaints of productive cough and shortness of breath on exertion for 2 weeks, which was associated with fever and chills. Other review of systems was unremarkable. He was initially evaluated by his primary care physician who started him on a course of azithromycin for 5 days, but his symptoms worsened after the antibiotics were done. He had a history of intravenous drug abuse in the past, but he admitted to being clean for the last 2–3 months. For the last 2 months prior to his admission, he installed television and internet routers in residences for a job, with most installations in basements that were damp and moldy. He denied history of recent travel or incarceration.

On presentation, he had temperature of 101°F, pulse of 124/min, respiratory rate of 18/min, and blood pressure of 108/58 mmHg. Oxygen saturation was 84% on room air. Physical examination revealed bibasilar crackles on auscultation of chest. Cardiac examination revealed first and second heart sounds with no murmurs, rubs, or gallops. Rest of physical examination was within normal limits.

Laboratory investigations revealed white blood count of 17,100/UL (normal 4,000–11,000/UL) with absolute neutrophil count of 15,570/UL. Chest x-ray showed bibasilar infiltrates. Computerized tomography (CT) scan of chest showed multiple cavitary and pre-cavitary lesions in both lung fields measuring 1–3 cm, as shown in Fig. 1. Differential diagnoses for the bilateral cavitary lesions were considered including septic emboli from endocarditis, tuberculosis, fungal infection as well as wegener granulomatosis. He was started empirically on vancomycin and cefepime, and the hypoxia was treated with non-invasive ventilation. The initial blood cultures were positive for methicillin-sensitive *staphylococcus aureus*. Sputum was negative for Acid-Fast Bacilli. Serum cryptococcal

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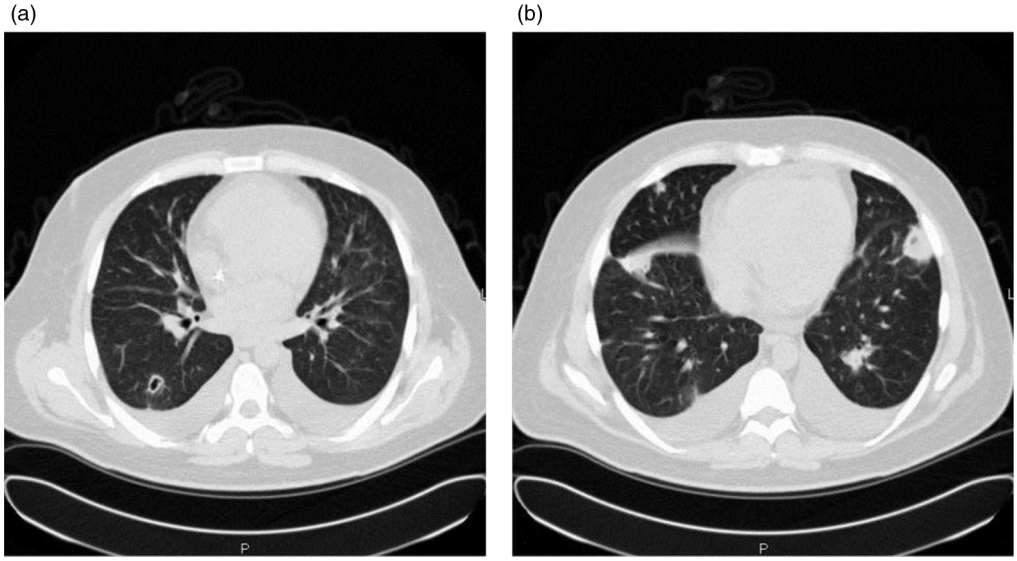

Fig. 1. CT chest: Panels a and b show multiple cavitary and pre-cavitary lesions, respectively, in both lung fields suggestive of septic emboli.

antigens were negative. His serum histoplasma antigen was negative while the urine histoplama antigen was incidentally positive, although he never lived in an endemic area for the histoplasma. He was switched to intravenous oxacillin. The transthoracic echocardiogram (TTE) was done which revealed large-sized, freely mobile vegetation on the pulmonic valve with moderate pulmonic insufficiency, as shown in Fig. 2. His subsequent blood cultures after 48 h on antibiotics were negative. Cardiothoracic surgery was consulted who decided to pursue medical management unless patient deteriorates. CT chest repeated after 2 weeks showed decrease in size of cavitary lesions. He was subsequently discharged to a skilled facility to complete 6 weeks course of intravenous antibiotics per infectious diseases recommendations. On follow-up, he was found to have complete recovery and full resolution of symptoms. Given the fact that he responded well to oxacillin, further workup such as bronchoscopy and lung biopsy for positive urine histoplasma antigen was deferred.

Discussion

Right-sided infective endocarditis (RSIE) is a rare disorder that predominantly involves the tricuspid valve, with or without the involvement of the pulmonic valve. However, isolated PVE is an extremely rare event that typically occurs in less than 2% of cases (1). From 1979 to 2013, there were only 70 reported cases of PVE (2). The mean age at presentation was 44 years and the majority of patients were male. Proposed mechanism for the low incidence of RSIE include the low pressure gradient within the right heart, lower oxygen content of venous blood, lower prevalence of right-sided congenital malformations, and

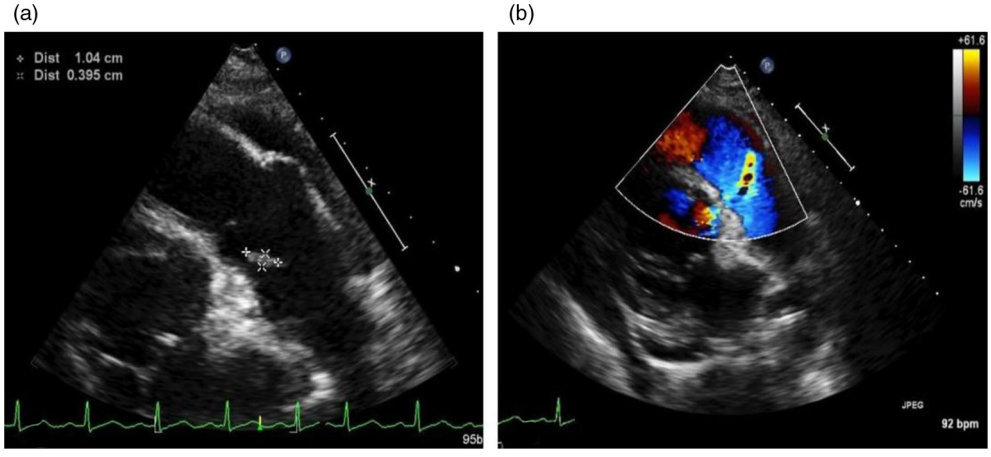

Fig. 2. TTE: Parasternal short axis view in Panel a shows a freely mobile vegetation greater than 1 cm in maximal diameter attached to the pulmonic valve. Parasternal view in Panel b shows mild to moderate pulmonic insufficiency.

differences in the covering and vascularization of the right heart endothelium (1). Risk factors for the development of PVE include intravenous drug abuse, indwelling catheters, alcoholism, *S. aureus* bacteremia, pacemakers, and congenital cardiac defects (1, 3). In our case, intravenous drug use and *S. aureus* bacteremia were the risk factors that led to the suspicion of pulmonic valve endocarditis.

The most common symptoms of PVE at presentation include shortness of breath, cough, and fever which are also present in other common pulmonary disease processes, such as pneumonia or pulmonary embolism. These patients often present with subtle clinical signs such as a pulmonic regurgitant murmur on auscultation which is present in 50% of cases or lung infections due to septic emboli from pulmonic vegetations. A significant delay often occurs in the diagnosis of pulmonic valve endocarditis due to the lack of typical diagnostic symptoms and signs unlike cases of a mitral or aortic valve endocarditis (3). For example, the patient described in this case was initially treated for pneumonia leading to a delay in diagnosis and presentation with hypoxemic respiratory failure secondary to multiple septic emboli. The bacterial pathogens most commonly identified include *S. aureus*, coagulase negative staphylococci, and Group B streptococci (1, 4–6). However, cases of fungal as well as other bacterial pathogens have also been reported in the literature. TTE is the most common modality used in the diagnosis of PVE and often requires multiple views to pick up the vegetation (2). In cases where TTE is negative despite high clinical suspicion, a transesophageal echocardiogram should be performed because of its better sensitivity and specificity.

Right-sided endocarditis has a better prognosis than left-sided endocarditis with majority of cases successfully treated with medical management and a reported mortality of less than 5% (7). Antibiotic treatment is targeted at the most likely causative organisms and should be initiated immediately after adequate blood cultures have been obtained. Empiric therapy should include an antistaphylococcal agent with activity against methicillin-resistant *S. aureus* such as vancomycin or daptomycin. The use of adjunctive gentamicin for the treatment of right-sided native valve endocarditis is discouraged (8). Once culture and sensitivity results of an identified microorganism are known, antibiotic therapy can be appropriately tailored. Surgical intervention for the management of RSIE is unclear. Based on pervious case reports, Akinosoglou et al. (7) suggested the following indications for surgical intervention, which include no response to antibiotics beyond 2 weeks, recurrent septic pulmonary emboli, septic shock, new onset renal and or hepatic failure, and development of secondary multivalvular endocarditis. However, no definite guideline exists due to complexity of RSIE and small number of operated cases. Experience of individual cardiothoracic surgeon and patient's clinical status are important in clinical decision-making. We proceeded with medical treatment due to the rapid clinical improvement with conservative management.

In summary, after reviewing the available literature and the clinical experience with our patient, we recommend that the diagnosis of pulmonic valve endocarditis should be strongly considered in patients with unexplained respiratory symptoms and underlying risk factors of RSIE. This often requires careful multiple view evaluation of pulmonic valve to visualize the valve vegetation.

Conflict of interest and funding

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