รายงานผู้ป่วย : ผู้ป่วยเยื่อบุหัวใจอักเสบที่ลิ้นหัวใจพัลโมนิก และมีภาวะแทรกซ้อนเยื่อหุ้มปอดอักเสบเป็นเลือดจากลิ่มเลือดอุดตันที่ปอด

Case Report: A Rare Case of Isolated Pulmonary Valve Infective Endocarditis Complicated by Bloody Pleural Effusion from Septic Pulmonary Embolism

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บทคัดย่อ

โรคติดเชื้อของเยื่อบุหัวใจ ที่ลิ้นหัวใจพัลโมนิก เป็นโรคติดเชื้อของเยื่อบุหัวใจด้านขวาที่พบน้อยในผู้ใหญ่ มักพบเป็นเพศชาย อายุน้อย ส่วนใหญ่รอยโรคเกิดที่ลิ้นหัวใจไตรคัสปิด ผู้ป่วยที่ใช้ยาเสพติดทางหลอดเลือดดำมีความเสี่ยงต่อโรคนี้เพิ่มขึ้น การวินิจฉัยเป็นเรื่องที่ท้าทายเนื่องจากมีอาการไม่จำเพาะเจาะจง (เช่น มีไข้ หายใจลำบาก ไอเป็นเลือด และเจ็บหน้าอกจากเยื่อหุ้มปอดอักเสบ) และยากจะตรวจพบจากการตรวจร่างกายและการตรวจหัวใจด้วยคลื่นเสียงสะท้อน กรณีเคสศึกษาเป็นผู้ป่วยชายอายุ 40 ปี มีประวัติใช้ยาเสพติดทางหลอดเลือดดำมาด้วย ไข้ หายใจลำบาก ระบบหายใจล้มเหลวเฉียบพลัน และพบมีภาวะแทรกซ้อนจากลิ่มเลือดอุดตันหลอดเลือดที่ปอดร่วมด้วย ภาพรังสีทรวงอกแสดงให้เห็นน้ำในเยื่อหุ้มปอด เจาะน้ำมาตรวจพบเป็นน้ำสีเลือด ไม่พบการติดเชื้อ จากการตรวจเอกซเรย์คอมพิวเตอร์ปอดยืนยันพบเส้นเลือดอุดตันที่ปอด และพบลิ้นหัวใจพัลโมนิกรั่วจากการตรวจหัวใจด้วยคลื่นเสียงสะท้อน ผลเพาะเชื้อในเลือดขึ้นเชื้อแสตปไฟโลคอคคัสออเรียส ได้ให้การรักษาด้วยยาคลอกซาซิลลินฉีดเข้าเส้นเลือดดำ แต่ผู้ป่วยยังคงมีไข้สูงและพบมีเส้นเลือดที่ปอดอีกข้างอุดตัน ผู้ป่วยรายนี้ไม่ตอบสนองต่อยาปฏิชีวนะภายใน 4 สัปดาห์ ดังนั้นจึงส่งต่อผู้ป่วยรายนี้ไปผ่าตัดเพื่อเปลี่ยนลิ้นหัวใจต่อไป

คำสำคัญ: เยื่อบุหัวใจด้านขวาอักเสบจากการติดเชื้อ เยื่อบุหัวใจอักเสบจากลิ้นหัวใจพัลโมนิก เยื่อหุ้มปอดอักเสบเป็นเลือด

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Abstract

Isolated pulmonary valve endocarditis (PVE) is an extremely rare form of right-sided infective endocarditis (RSIE) in adults. PVE tends to affect younger patients and most are male. The majority of cases involve a tricuspid valve. Intravenous drug users are at increased risk for developing RSIE. Diagnosis can be challenging because of non-specific symptoms (fever, dyspnea, hemoptysis, and pleuritic chest pain) and the difficulty of detection by physical examination and echocardiography. We describe a case of PVE in a 40-year-old male, who was a current intravenous drug user. He presented with fever, dyspnea, and acute respiratory failure, complicated with bloody pleural effusion from septic pulmonary emboli. The chest radiography showed pleural effusion. Diagnostic thoracocentesis showed sterile bloody pleural effusion. Septic pulmonary embolism in both lungs was found from a computed tomography scan of the chest. Transthoracic echocardiography revealed large vegetation with pulmonic insufficiency. Blood cultures showed the presence of Staphylococcus aureus (MSSA). Cloxacillin was administered intravenously, respectively. The patient still had a high-grade fever, repeated septic pulmonary embolism and unresponsiveness to antibiotics within 4 weeks, thus this case was referred for cardiothoracic surgery for pulmonary valve replacement.

Keywords: Right-sided infective endocarditis, Pulmonary valve endocarditis, Bloody pleural effusion

Introduction

Infective endocarditis (IE) remains an important clinical problem. It has a 1-year mortality rate of 30%. Right-sided infective endocarditis (RSIE) is uncommon (5–10% of total IE) (Shmueli et al., 2020). The majority of RSIE 90% of cases involve a tricuspid valve. PVE is an extremely rare condition, affecting less than 1.5–2% of patients suffering from all cases of IE (Tekin, Acet, Ertas, Tekin, & Albudak, 2012). Reasons for this include the lower transvalvular pressure gradients and venous oxygen concentration in the right chambers (Frontera & Gradon, 2000). Isolated pulmonary valve endocarditis (PVE) tends to affect younger patients, and more than 80% of affected subjects are male (Seraj, Gill, & Sekhon, 2017). Most cases of pulmonic valve endocarditis in children are secondary to the presence of a congenitally abnormal pulmonic valve and in adults secondary to intravenous drug abuse (IVDUs) (Mansour et al., 2016). The common precipitating factor for PVE

includes IVDUs, immunosuppression, valvular replacement, congenital heart diseases, alcoholism, sepsis, catheter-related infections or pacemaker implantation with subsequent lead infection (Ranjith et al., 2013). In 28% of cases, the predisposing factor was not identified. (Bamford, Soni, Bassin, & Kull, 2019). IVDUs are the most common cause of IE on the right side of the heart and the major valve involves was the tricuspid valve (>90%) (Acharya, Anwar, Iannuzzi, Anugu, & Ghavami, 2020), responsible for the increasing incidence of IE in developed countries. The overall incidence of IE among IVDUs patients ranges between 2% and 5% per year (Shmueli et al., 2020). It has been estimated that up to 76% of cases of IE among IVDUs occur on the right side, compared with only 9% in nonaddicts. In IVDUs, the tricuspid valve is usually involved 40–69%, the aortic and mitral valves 20–30%, and multiple valves 5–10% of cases. Although the mortality rate associated with RSIE is lower than the LSIE, cardiopulmonary, neurologic, renal, ophthalmologic, and abdominal and extremity vascular complications can cause significant morbidity (Frontera & Gradon, 2000). The clinical presentation of PVE is nonspecificity of symptoms and lack of typical peripheral findings usually subtle and easily missed. The diagnosis of PVE is made based upon a synthesis of clinical, microbiological, and echocardiographic findings. The modified Duke criteria are the standard criteria used to guide the diagnosis of IE (Chan, Hsieh, Chen, Huang, & Chuang, 2019). The two major criteria for a definite diagnosis of IE are positive blood cultures and evidence of endocardial vegetations on either a transthoracic (TTE) or transesophageal echocardiogram (TEE). The sensitivity of TTE has been estimated at 30–63% with a specificity of 91–100% (Zhang et al., 2020), and TEE has 87–100% sensitivity with 91–100% specificity (Bamford et al., 2009). The most commonly isolated causative agents in PVE include Staphylococcus aureus (60–90% of cases) (Shmueli et al., 2020), coagulase-negative Staphylococci, and group B Streptococci (Saleem et al., 2019). The response of Staphylococcus aureus IE to antimicrobial therapy is slow, and many patients remain persistent bacteremia for weeks. Traditionally, beta-lactamase-resistant penicillins, first-generation cephalosporins, and aminoglycosides besides vancomycin for patients allergic to penicillins have been used as the main drugs in the treatment of staphylococcal endocarditis (Chan et al., 2019). Response to antibiotics and prognosis in RSIE tend to be better than in its left-side counterpart (Raja, Kasim, & Zainal, 2020). Most patients with PVE can be treated using medical therapy alone (Saleem et al., 2019). Overall mortality for RSIE is between 5–15% but could be as high as 30% in those lacking the

traditional risk factors (intravenous drug use and cardiac devices) (Shibru, Greffie, Abay, & Muhie, 2020). RSIE implies a better prognosis than LSIE; the previous study revealed the mortality of right-sided IE is 12% in-hospital patients and 0–7.3% for surgical patients. However, these percentages increase at least twice in patients with intensive care unit admission (Adrian & Daniela, 2019). Surgical treatment is needed in 5–16% of RSIE (Shibru et al., 2020), and it has to be considered in the following situations (Meel, 2019) intractable right-sided heart failure with poor response to diuretics, persistent bacteremia despite the use of appropriate antimicrobial therapy, large vegetation (>20 mm) that does not diminish in size despite repeated embolism, fungal endocarditis, concomitant LSIE, and prosthetic valve endocarditis. The most common complications were valvular insufficiency, embolic events, abscess formation and pulmonary embolism (Acharya et al., 2020). Pulmonary involvement occurred in 80% of these cases and varied from minor atelectasis to large infiltrates, pleural exudates, and cavitation, generally involving the lower lobes (Shmueli et al., 2020).

PVE commonly manifests clinically as fever, persistent bacteremia, and septic emboli to the lungs. In the setting of predominantly pulmonary symptoms such as dyspnea, pleuritic chest pain, cough, and hemoptysis (Saleem et al., 2019). Pulmonary insufficiency is a late development of PVE and is detected on physical examination in about 50% of patients with PVE as a low-pitched, diastolic murmur that is easily missed. The absence of signs and symptoms of the cardiac disease often leads to delayed diagnosis, especially in patients lacking traditional risk factors or concurrent involvement of other valves (Saleem et al., 2019). Septic shock, renal failure, and uncontrolled disseminated infection are common features of patients with PVE without notable risk factors. Septic pulmonary embolism (SPE) is a rare type of pulmonary embolism in which emboli containing pathogens embolize to the pulmonary artery and cause pulmonary embolism (Magnaye, 2015a), most of these were intravenous drug users (Ye, Zhao, Wang, Wu, & Yan, 2014). SPE cloud presented with bloody pleural effusion. The history should focus on differentiating pulmonary etiologies from cardiovascular and other causes of effusion. When the pleural effusion is suspected, chest radiography, diagnostic thoracocentesis, computed tomography of the chest should be performed to confirm the diagnosis.

We describe a case of isolated pulmonary valve endocarditis in a 40-year-old male, who was a current intravenous drug user. He presented with fever, dyspnea, and acute respiratory failure, complicated with bloody pleural effusion from septic pulmonary emboli.

Timeline

Day 1 He presented with fever for 2 weeks, chills, dyspnea, and acute respiratory failure on intubation. Empirical antibiotics with ceftriaxone and azithromycin to treat bacterial pneumonia were prescribed.

Day 6 He had dyspnea and hypoxemia. His oxygen saturation was 90%. Chest radiography revealed left pleural effusion. Scrub IgG antibody was positive. His treatment was doxycycline.

Day 7 His blood culture reported MSSA, then switched antibiotics to cloxacillin intravenous.

Day 17 He developed dyspnea with hypoxemia. Chest radiography showed an increased left pleural effusion. Thoracocentesis was done and found to have bloody pleural effusion. Computed tomography showed septic embolism of the lungs. Percutaneous drainage (PCD) on the left chest was done.

Day 20 He was extubated.

Day 22 He again had dyspnea. Chest radiography revealed right pleural effusion and improved left pleural effusion. A thoracocentesis of the right chest revealed a bloody pleural effusion, which was suspected to be a septic pulmonary embolism. A computed tomography scan of the chest was done.

Day 28 A transthoracic echocardiogram was performed. (Delay caused by the COVID-19 situation). Finally, isolated pulmonary valve endocarditis was diagnosed.

Day 31 He still had persistent fever, then was referred to cardiothoracic surgery.

A previously healthy 40-year-old man was referred from another hospital with a two-week history of fever, chills, and dyspnea. He was admitted to the medical intensive care unit and subsequently intubated due to respiratory failure. He denied previous medication treatment. He was a current intravenous drug abuse user. He had no history of recent surgery. The patient had no other notable medical record. He also denied any recent invasive procedures or surgeries. He denied a history of cough, rhinorrhea, contact the COVID-19 patient. There were no known allergies or other conditions present. There was no record of a prior episode of infective endocarditis. On physical examination at admission, his vital signs showed a blood pressure of 116/67 mmHg, a heart rate of 127 beats per minute, and a respiratory rate of 28 per minute, and a temperature of 38°C. Oxygen saturation was 90% on room air. Cardiac auscultation revealed no murmur. There were also no bilateral rales or crackles at the basal of the lungs. Examination of his extremities and skin revealed no pitting edema. There were no peripheral stigmata of endocarditis present such as Janeway lesions or

Oslers' nodes. Neurological examination was unremarkable. Further clinical examination was unremarkable.

Laboratory analysis showed microcytic anemia with hemoglobin 8.7 g/dl, a white cell count of 24,130 /mm^3, and platelets count of 67,000 /mm^3. Liver function test were protein 7.6 g/dl, albumin 2.6 g/dl, TB 2.9 mg/dl, DB 1.7 mg/dl, AST 42 U/,L ALT 29 U/L, ALP 175 U/L, BUN 18 mg/dl, Cr 1.0 mg/dl, eGFR 93.7 ml/min, Na 128 mmol/L, K 4.1 mmol/L, Cl 96 mmol/L, CO_2 24 mmol/L. Urine analysis revealed slight leukocyturia of 3-5 cells/mm^3. Chest X-ray showed borderline cardiomegaly, minimal patchy infiltration at the right upper lung (Figure 1A). Tracheal suction and throat swab for COVID-19 not detected. A hepatitis serology documented negative hepatitis B surface antigen, positive hepatitis C antibody, and negative hepatitis A IgM antibody. HIV antibody was negative. Sputum acid-fast bacilli were negative for 3 days. Sputum gram stain revealed no organism and sputum culture reported no growth. Leptospirosis antibody was negative, and scrub typhus antibody IgG was positive. He had chronic hepatitis C viral infection and suspected liver cirrhosis. Further investigation to confirm the diagnosis was needed if clinically stable.

Initial investigations included laboratory tests and blood culture sets. An electrocardiogram showed a sinus rhythm without signs of underlying ischemia or atrioventricular block. Chest radiography showed borderline cardiomegaly, patchy infiltration at the right upper lung. An initial full blood count revealed leucocytosis, neutrophil predominates, left deviation, and microcytic anemia with thrombocytopenia. The patient presented with normal renal function. Liver function tests showed mild transaminitis and hypoalbuminemia. Electrolyte showed hyponatremia. A tracheal suction and nasal swab for the COVID-19 PCR test were reported to be negative. Empirical antibiotic therapy with ceftriaxone and azithromycin, according to considered bacterial pneumonia differentiated tropical infection such as leptospirosis or scrub typhus, was administered.

Six days after his admission, scrub typhus antibody IgG was reported positive. Then azithromycin was changed to doxycycline for covering scrub typhus and waiting for the second antibody titer to fourfold rise. The patient still had a high-grade fever, and he developed dyspnea and oxygenate desaturation 90%. Chest radiography showed increase right upper lung patchy infiltration and left lung effusion (Figure 1B). Diagnostic thoracocentesis on the left yielded 200 ml of bloody fluid (Figure 2), found to be a sterile exudate. Bloody pleural profiles were pH 8.0, RBC 1,130,000

/mm^3, WBC 5,200 /mm^3, neutrophils 77%, mononuclear 23%, protein 5,500 mg/dL, sugar 81 mg/dL, albumin 1,300 mg/dl, LDH 742 mg/dL with pleural fluid hematocrit 6%. Gram stain and acid-fast bacilli smear were negative. Pleural fluid culture revealed no growth. Serum LDH was 252 mg/dl, serum protein 8.2 g/dl, and serum albumin 1.8 g/dl. Percutaneous drainage was done. Three weeks later, he again presented with dyspnea and chest radiography was performed, showing new right lung effusion but decreasing left lung effusion (Figure 1C). An exudative, sterile, bloody pleural effusion presented in drug users patients with prolonged fever should be considered possible causes such as septic pulmonary emboli and right-sided endocarditis. Computed tomographic of chest resulted in multifocal subpleural wedge-shaped consolidation and ground-glass infiltration at right lung, some of them have central cavities. Left pleural effusion causes passive left lung atelectasis (Figure 3). Seven days after admission blood cultures detection of methicillin-sensitive Staphylococcus aureus (MSSA) were reported, thus antibiotics had changed to intravenous cloxacillin. Transthoracic echocardiography (TTE) was done and revealed LVEF 47.4%, moderate thickening of the pulmonic valve, and large vegetation (16 x 10 mm) in the pulmonic valve area without other valvular abnormalities (Figure 4).

After treatment with intravenous cloxacillin that was sensitive from culture reported for 4 weeks, this patient still had a high-grade fever off and on with malaise. Re-evaluated echocardiogram revealed LVEF 69%, dilated RV with impaired RV systolic function moderate, dilated RA, vegetation size 4 x 9 mm, flail PV with severe PR, no ASD, VSD, no PDA, no pericardial effusion. Cardiothoracic surgery was consulted and referred him for pulmonary valve replacement. Further investigation and treatment of chronic hepatitis C infection might be evaluated.

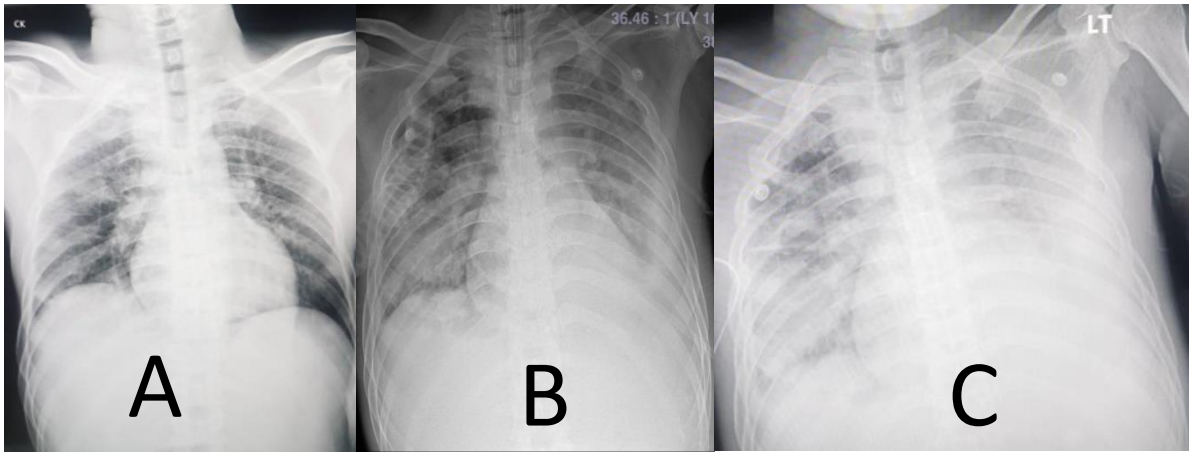

Figure 1 Chest x-ray (A) Day1 showed minimal patchy infiltrates at the right upper lung. (B) Day17 showed left pleural effusion and patchy infiltration at the right lung. (C) Day22 showed bilateral pleural effusion.

Figure 2 Diagnostic thoracocentesis showing bloody pleural effusion with pleural fluid hematocrit 6% (Blood hematocrit 25%).

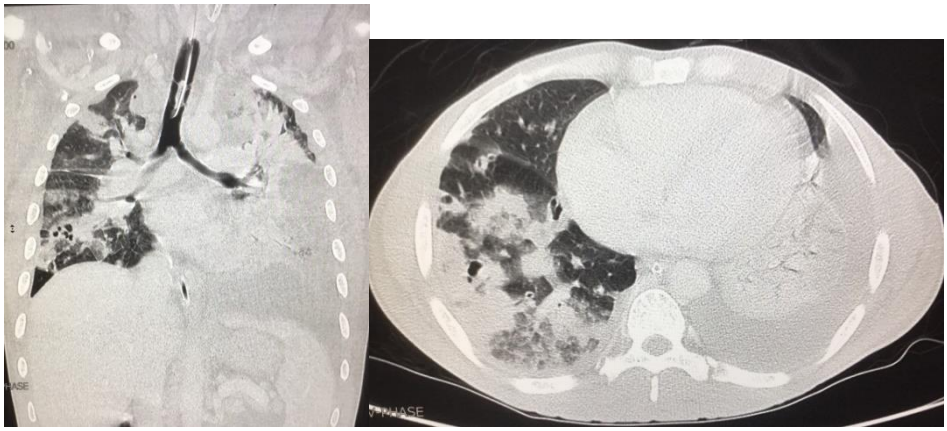

Figure 3 Computed tomography scan of the chest showing multifocal subpleural wedge-shaped consolidation and ground-glass infiltration at right lung, some of them have central cavities. Left pleural effusion causes passive left lung atelectasis.

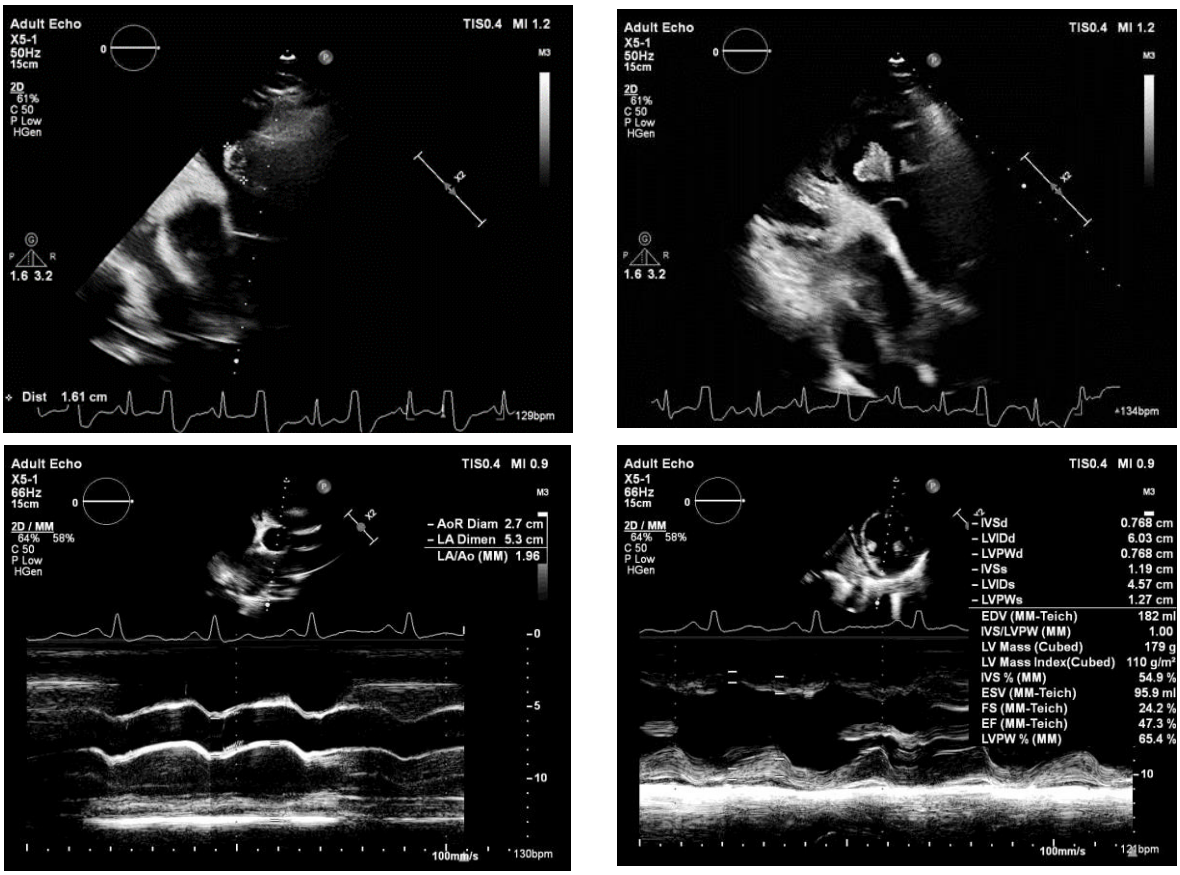

Figure 4 Transthoracic echocardiogram showing large vegetation 16×10 mm at the pulmonary valve area with pulmonic valve regurgitation.

Discussion

In this case, A 40-year-old male, who was a current intravenous drug user. He presented with fever, dyspnea, and acute respiratory failure at admission. Approximately in one-half of patients, there is a pulmonic regurgitant murmur in the cardiovascular examination (Tariq, Smego, Soofi, & Islam, 2003) but not found in our patient. It is difficult to diagnose without suspicion since the first clinical manifestations, this case suspected bacterial pneumonia empirical antibiotic with ceftriaxone and azithromycin was done. During admission, he developed dyspnea. Chest radiography showed repeated pleural effusion in the left and right chest. Diagnostic thoracocentesis found bloody pleural effusion with sterile exudate pleural fluid. Gram's stain and cytology were negative. A computed tomography scan of the chest corroborated the presence of septic pulmonary embolism. Respiratory complications

occur in 58% of cases and pleural complications have occasionally in 6% of cases. The embolic blood clot that leads to an infarction in 10% of cases occurs when a potential source of lung oxygenation was compromised (Magnaye, 2015b). Blood culture presented with staphylococcus aureus. Echocardiography showed large vegetation at pulmonic valve area, then the diagnostic of this case is PVE with IVDUs risk factor and complicated with bloody pleural effusion. To detect causes of PVE, repeated echocardiograms with a special focus on the pulmonary valve may be required, thus severe pulmonary regurgitation was diagnosed later. Usually, antibiotic treatment alone leads to recovery in PVE (Saleem et al., 2019). Surgical intervention may still be indicated in this case due to persistent fever despite antibiotics for 4 weeks, and recurrent pulmonary embolism was presented, even though echocardiography showed a decrease in the size of vegetation<20mm. We considered the option of surgery and referred him for cardiothoracic surgery for pulmonic valve replacement. A review of the published data indicated that the role of surgery in isolated pulmonic valve endocarditis is unclear (Ranjith et al., 2013). The main principles of surgery are radical debridement of vegetations and infected tissue as well reduction of regurgitation size. Reoperations were significantly more often observed in injection drug users.

It is difficult to diagnose without suspicion since the first clinical manifestations often mimic pulmonary infection caused by bloody pleural effusion due to septic pulmonary embolism. These delays appropriate treatment, thus careful physical examination and carefully getting medical history by a physician are very important. Also, thorough echocardiographic evaluation of all cardiac valves, including right-sided valves, should be carried out in all patients with suspecting infective endocarditis, especially when the patient has a risk factor. It is important to keep in mind that endocarditis can often be missed on a simple TTE and it is almost mandatory to get a transesophageal echocardiogram to visualize all the cardiac structures appropriately.

Conclusion

Isolated pulmonary valve endocarditis due to predisposing intravenous users remains a rare entity. It is important to consider the diagnosis might be challenging because symptoms are often not specific. Our case highlights the importance when assessing intravenous patients with persistent pyrexia and bloody pleural effusion for possible IE. Clinicians should take a complete history and

physical examination which are crucial to the proper and appropriate management of septic pulmonary embolism without delays to prevent further complications like death. Blood culture, chest imaging, and echocardiography are invaluable in the evaluation of a patient suspected of septic pulmonary embolism. In most cases, medical therapy led to successful treatment. Although the surgical treatment had a satisfactory outcome in this case management decisions should be made on a case-by-case basis with multidisciplinary coordination.

Ethical statement

Ethic No: SKH IRB 2021-Md-IN3-1046.

The study design and retrospective chart review were approved by Songkhla hospital.

Conflict of Interest

None

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