Acta Cardiologia Indonesiana

pISSN:2460-5700
eISSN:2579-4345

Web page: jurnal.ugm.ac.id/v3/jaci

Right-Sided Infective Endocarditis, Pulmonary Embolism and Infarction in Adult with Uncorrected Ventricular Septal Defect

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Keywords:
right-sided infective endocarditis;
pulmonary valve;
pulmonary infarct,;
ventricular septal defect

Manuscript submitted: January 15, 2020
Revised and accepted: February 1, 2020

ABSTRACT

Right-sided infective endocarditis (IE) is very rare and mostly occurs in the tricuspid valve, which pulmonary valve involvement in very rare. Most IE of the pulmonary valve occurs in patients with congenital heart defects. we report a case of the complications of pulmonary valve IE and pulmonary embolism which then causes pulmonary infarction in patients with VSD who have not been corrected. This case successfully underwent surgery for endocarditis/vegetation removal and septal defect closure.

INTISARI

Endocarditis infektif sisi kanan jarang dijumpai dan sebagian besar terjadi pada katup trikuspid, sedangkan keterlibatan katup pulmonal sangat jarang. Sebagian besar endokarditis infecktif dari katup pulmonal terjadi pada pasien dengan kelainan jantung bawaan. Dilaporkan sebuah kasus komplikasi dari endocarditis infektif pada katup pulmonal dan emboli paru yang kemudian menyebabkan infark paru pada pasien dengan defek septum ventrikel yang belum diperbaiki. Kasus ini berhasil menjalani operasi untuk pengangkatan endokarditis/vegetasi dan penutupan defek septum.

Introduction

Right-sided infective endocarditis (IE) is very rare and mostly occurs in the tricuspid valve. Pulmonary valve IE is even less common than tricuspid valve and accounts for less than 2% of hospital endocarditis events. Differences in hemodynamic pressure gradients that pass through the valve, low incidence of congenital and valve abnormalities and lack of oxygen content in the right heart can all contribute to the low incidence of right-sided IE, as compared to left-sided IE, especially those involving pulmonary valves.[1] Most IE of the pulmonary valve occurs in patients with congenital heart defects.[2] In this case, pulmonary valve IE may be preceded by a jet or turbulent flow resulting from a ventricular septal defect (VSD) which then spreads to the pulmonary valve.[3]

The incidence of embolism is a frequent and life-threatening complication of IE related to the migration of cardiac vegetation. The brain and spleen are the most common places where emboli occur on the left-sided IE, whereas pulmonary embolism often occurs on the right-sided IE. Here we report a case of the complications of pulmonary valve IE and pulmonary embolism which then causes pulmonary infarction in patients with VSD who have not been corrected. This case successfully underwent surgery for endocarditis/vegetation removal and septal defect closure.

Case Report

A 19 year old male patient was referred to our hospital due to main complaint of left-sided chest pain. He felt unwell since one month previously, with dyspneu on activity, dry cough and slight fever. He restrained his activity, however the symptoms progressed into high fever and chill, intense chest pain especially during inspiration and movement to the leftside and more frequent coughing, sometime accompanied by blood (haemoptysis). The patient was diagnosed as VSD perimembrane type (defect size 3-5 mm) and pulmonary stenosis (PS) (infundibular gradient 93 mmHg) since childhood and has been undergone pulmonary balloon valvuloplasty. He was planned to have surgery for VSD and PS, however until current admission he refused surgery.

In our hospital, the vital sign stable. Physical examination showed cardiomegaly with pansystolic murmur indicating VSD. The ECG examination showed right axis deviation and

right ventricle hypertrophy. Laboratory examination showed haemoglobin 12.4 g/dL, haematocrit 38.3%, leukocytes 10,800/mm3, creatinine 0.78 g/dL. Other laboratory examination showed normal findings. Chest X ray indicated bilateral pleural effusion, especially in the left side (figure 1).

Transthoracic echocardiography was performed during admission and showed the perimembranous VSD with diameter 3-5 mm with left-to-right shunt, right ventricle dilatation, pulmonary stenosis infundibular type (pressure gradient 90 mmHg) with vegetation in pulmonary valve dimension of 13 mm x 8 mm. Suspicious of infective endocarditis was arrised, therefore blood culture from different sites was collected (the result several days later indicated no bacterial growth). Since the patient also complained of pleuritic chest pain and haemoptysis, we also suspect pulmonary embolism and performed MSCT pulmonary angiography scan. The MSCT scan revealed pulmonary infarction in the lingula and the anterobasal and inferior lobes of left pulmo; suspicious of thrombus at lingular artery and common basal artery (branch of left pulmonary artery) (figure 2). The diagnosis of right-sided infective endocarditis (IE), pulmonary embolism and infarction was made with underlying disease of left-to-right perimembraneous VSD and infundibular stenosis.

The patient was given empirical antibiotics for IE for 14 days, unfractionated heparin and bridging with oral warfarin. During treatment course, the patient felt reduced chest pain (VAS 7 to VAS 2), diminished hemoptysis and subsided fever. On day 14, the transthoracic echocardiography showed the vegetation in pulmonary valve with dimension of 10 mm x 5 mm. The MSCT cardiac scan was performed and showed cut-off in the left pulmonary artery posterior branch and pulmonary infarction in anteromedial segment of inferior lobe of left pulmo. We consulted with cardiothoracic surgeon and the surgeon decided to perform VSD closure, infundibular resection and vegetation evacuation. As for pulmonary infarction, the infarction evacuation was planned three months afterwards.

After operation, the MSCT cardiac scan was performed and showed thrombus in inferior branch (A5/A6 branch) of left pulmonary artery, pulmonary infarct in anterobasal and laterobasal inferior segment of left pulmo, no vegetation was identified in pulmonary valve. Compared with previous MSCT scan, the size of thrombus was reduced 22.2% and the size of pulmonary infarct was reduced 42.4%. Transthoracic echocardiogram showed residual VSD 1 mm, residual infundibular stenosis (pressure gradient 29 mmHg) and no vegetation. After three month follow-up, the patient felt better however left chest pain was still persisted despite in lower intensity. He refused to have decortication and pulmonary infarction evacuation.

Discussion

Righr-sided IE occurs in about 5-10% of all cases of IE, mainly involving the tricuspid valve, where involvement of the pulmonary valve is rare.[4] Right-sided IE in non-drug users occurs in only 9% of cases, where intravascular catheters, pacemaker wires and intracradial devices (catheters for hemodialysis or tricuspid prosthetic valves) are the main predisposing factors.[5]

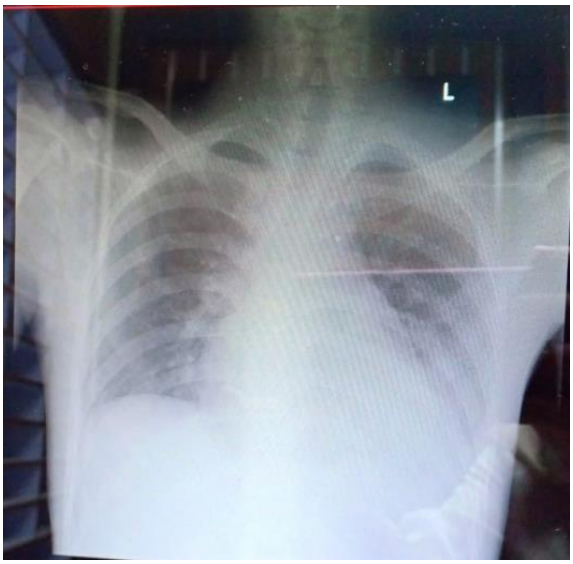

Figure 1. Chest x –ray

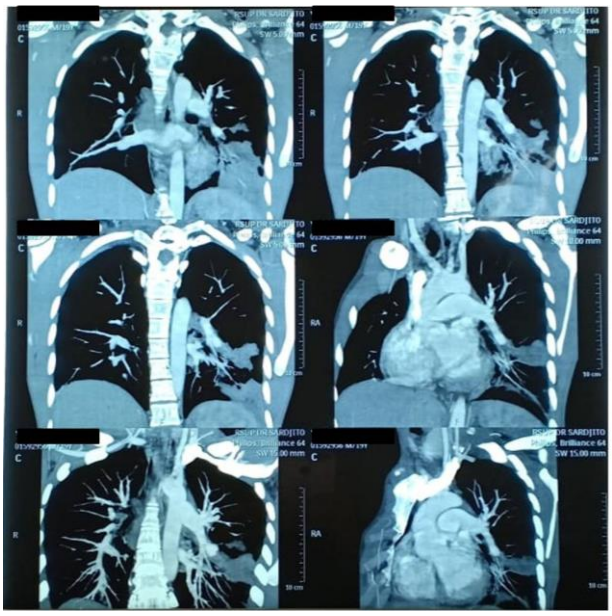

Figure 2. Multi slice CT scan

Disease history, clinical examination and a high index of suspicion are the main basis of diagnosis. The most common symptoms are respiratory symptoms followed by fever, anemia and microscopic hematuria.[6] The typical Duke criteria cannot distinguish between right-sided and left-sided IE. Pulmonary embolism occurs in 75-100% in right-sided IE.[7] As with left-sided IE, a positive blood culture combined with clinical and echocardiographic findings can confirm the diagnosis. However, pathogens that require certain media to develop or prior antibiotics can produce negative blood cultures.[8] Electrocardiograms and routine blood tests are not specific, but chest

radiographs can show pulmonary embolism due to septic pulmonary embolism from the right heart.[9]

Common manifestations of right-sided IE are persistent fever, multiple bacteremic and pulmonary septic emboli, which usually have manifestations such as chest pain, coughing or coughing up blood.[10] In our case, he complained intermittent fever, a day or two later recover. Fever is often accompanied by chills, cough has lasted for approximately 3 months, sometimes accompanied by blood. Our case had no history of intravenous drug use. Pulmonary valve IE is even less common than in tricuspid valves and occurs in less than 2% of hospital endocarditis events. Most endocarditis of the pulmonary valve occurs in patients with congenital heart defects.[2] (Nakauchi et al., 2007). Our case showed pulmonary valve vegetation and VSD, which considers a predisposing condition.

Right-sided IE can disappears on conservative therapy in 70-85% of cases and the remaining need surgical procedures. Due to the lack of official guidelines regarding indications for surgical management, and the small number of cases operated on, the latest recommendations vary. Some patients with IE need surgical treatment due to complications. The three main indications for surgery are heart failure, uncontrolled infections and prevention of embolic events. But it is still unclear when surgery should be performed and this is a topic that is still often discussed. Most of the observational studies stated that initial surgery was preferred. This is supported by a randomized controlled trial (RCT) from Kang et al.[11] which showed a significant reduction in deaths during hospitalization and the incidence of early embolism in patients who received initial surgery compared with those who received conventional therapy.[11]

Vegetation> 10 mm in size with 1 or more embolic phases, or associated with other complications, indicates early surgery. In cases with very large vegetation > 15 mm, surgery is indicated when valve repair may occur. Our case had a vegetation size of 14 x 9 mm according to the results of the last cardiac MSCT in the pulmonary valve. Pulmonary infarction is associated with coagulation ischemic necrosis.[12] This process occurs when perfusion of the parenchyma cannot meet tissue requirements because there is a vascular covering over the intrinsic or extrinsic lumen.[13] The end result is fibrotic scars.[14]

Pulmonary infarction has characteristics of intermittent pleuritic chest pain. Hemoptysis is sometimes found. Emboli often closes the peripheral pulmonary artery trees near the pleura. Tissue infarction usually occurs 3 to 7 days after embolism. Signs and symptoms are fever, leukocytosis, increase in mean erythrocyte sediment and radiological evidence of infarction.[15] The factors that link between pulmonary thromboembolism and infarction are not yet fully understood. In pathological factors, thromboembolism located in the distal artery and lower lobe is significantly associated with infarction. The size of the infarction is small and all complete infarcts are related to the pleura. In addition, the closure of small arteries in the lower lobes and close to the pleura is related to the rate of infarction.[15] Our case had pulmonary infarction as complication of right-sided IE. He eventually had surgery to remove the vegetation and to repair the congenital defect.

Conclusion

A 19-year-old patient with VSD with infundibular PS with main complaint of coughed for more than 3 months, chest pain in the last 2 weeks and intermittent fever. The patient was diagnosed with pulmonary valve IE and pulmonary embolism and infarction and given a complete course of antibiotics and anticoagulant. Finally, the surgery was performed after an evaluation of 14 weeks of persistent vegetation. The long-term evaluation with antibiotics shows good results.

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