RIGHT-SIDED ENDOCARDITIS IN A NON-DRUG ADDICT

Sajeeth Kumar1, Prashanth Poulose2

1Professor, Department of Medicine, Government Medical College, Calicut, Kerala.
2Junior Resident, Department of Medicine, Government Medical College, Calicut, Kerala.

ABSTRACT

BACKGROUND

Infection localised to right heart valve (Right-sided infective endocarditis) occurs in about 5 - 10 percent of cases of infective endocarditis.[1,2] This is a case report of a 50-year-old diabetic, hypertensive male patient with no history of IV catheterisation or IV drug abuse who presented with multilobar consolidation with sepsis.

KEYWORDS

Right-sided Infective Endocarditis, Multilobar Consolidation, Sepsis.


BACKGROUND

The incidence of right-sided endocarditis has been steadily increasing over the years.

Intravenous drug abusers, cardiac device carriers and immunosuppressed patients are at maximum risk for right sided endocarditis.[1] Right-sided IE should be suspected in the presence of fever, recurrent respiratory events, anaemia, and microscopic haematuria.[3] A high index of suspicion will lead to early recognition and prompt treatment which can reduce complications like pulmonary infarction, abscesses, bilateral pneumothoraces, pleural effusion, and empyema. Mycotic aneurysms of the pulmonary arteries may be complicated by potentially fatal pulmonary haemorrhage. Multiple pulmonary emboli may result in right-sided chamber dilatation, right heart failure, and a worsening of tricuspid regurgitation. Cardiac complications also include supraventricular arrhythmias including atrial flutter and fibrillation secondary to chronic right atrial dilatation.[4]

CASE REPORT

A 50-year-old male patient with history of uncontrolled diabetes mellitus and hypertension, Ex-alcoholic and smoker, presented with fever which was initially low grade, developed high grade fever 3 days prior to admission associated with breathlessness and dry cough. On admission to hospital, patient also had severe myalgia and fatigue. Patient became restless and developed disorientation to time, place and person at night on the day of admission to our hospital. There was no history of haemoptysis, altered bowel habits, headache, dysuria, or joint pain.

On examination, patient was conscious, appeared ill. He was moderately built and nourished. He had Pallor, icterus on general examination. Pulse - 110/min., blood pressure 150/100 mmHg, Temperature 101 F, Respiratory rate 44/minute. Respiratory system examination revealed Tachypnoea, accessory muscles of respiration active, breath sounds diminished in right infra-axillary and infra-scapular areas. Stony dullness elicited in these areas. Crepitations heard bilaterally over mammary, infra axillary and infra scapular areas. Per Abdomen Tip of spleen palpable. Cardiovascular system, Jugular venous pulsations absent. Systolic murmur heard in the tricuspid area. From the history and examination findings, possibilities considered were multilobar consolidation with right-sided suppneumonic effusion, Leptospirosis with ARDS, Infective endocarditis, Enteric fever.

Investigations showed high TC 25200 (P82, L5, M13), Hb - 8.5, Platelet - 2.37 lakh, ESR - 130 first hour, RBS - 463, Total/Direct bilirubin - 3.5/1.6, Albumin - 2.5, SGOT/SGPT -45/94, Urine routine - normal. Initial Chest X-ray PA view revealed multifocal patchy alveolar opacities involving the right middle and bilateral lower zones. Pleural fluid study showed TC - 600 (DC - PMN100), Protein 5.1 g, Sugar - 67, Albumin 2.4 g, Pleural fluid LDH - 2407, ADA - 84. These findings were consistent with an exudative pleural effusion. Sputum AFB, Culture and sensitivity showed no organisms. Pleural fluid gene expert, Culture and sensitivity and Mantoux test were negative. Blood culture yielded methicillin-sensitive staphylococcus aureus. Urine and Bone marrow cultures were sterile. C-reactive protein was 90.3 mg/L (<6 mg/L), Serum ferritin 2048. Peripheral smear – Dimorphic RBC DCT, ICT, stool occult blood were negative. Subsequent Chest radiographs showed caviation in right middle zone. Plain and Contrast enhanced CT thorax showed right-sided pleural effusion, subsegmental collapse of right lower lobe and multilobar consolidation. ECG Sinus tachycardia. Widal, Lepto IgM, Wel-Felix, Monospot, HIV, HBsAg, Anti-HCV ELISA were negative. Thyroid function test, Troponin I quantitative, ANA quantitative titre were within normal limits.

In the background of a prolonged fever, multikobar consolidation, systolic murmur in the tricuspid area, splenomegaly and staphylococcal septicaemia, a very high clinical probability of a right-sided Infective endocarditis was considered and transthoracic Echo done initially was normal. Due to a high index of suspicion of a right-sided endocarditis, a repeat transthoracic Echo was done after 10 days of initial Echo which showed freely mobile mass attached to tricuspid valve suggestive of a tricuspid valve endocarditis.
Patient was treated with antibiotics (Inj. Cefazolin, Inj. Amikacin), glycaemic control attained with basal bolus regime of insulin and antihypertensives were continued. Patient became afebrile, breathlessness and cough subsided, chest signs resolved, total count normalised, inflammatory markers (ESR, CRP) began to decrease. He was treated for a period of six weeks.

DISCUSSION

Right-sided endocarditis is commonly under-recognised and hence is an undetected entity. A high index of clinical suspicion in a patient with risk factors to develop right-sided IE is required for diagnosis. (3)

Most common predisposing factor for right-sided endocarditis is IV catheter related infection and intravenous drug abuse is the second most common cause. In about 80 percent of the cases, tricuspid valve is involved. Staphylococcus aureus is the most frequently isolated organism in about 70 percent of cases. Streptococci and Enterococci are the next most common pathogens. (3)

Right-sided Infective endocarditis should be considered in any patient with fever, respiratory symptoms (Especially Multilobar consolidation), immunosuppressed background and staphylococcal septicaemia in absence of signs of systemic embolisation seen in left-sided IE. Diagnosis can be grossly delayed, since right-sided murmurs often go undetected, while peripheral stigmata are absent. (4)

Pulmonary embolism occurs in 75 – 100% especially in the cases of tricuspid valve IE. If peripheral manifestations are present, an associated left-sided endocarditis or a paradoxical embolism should be suspected. (3)

DUKE’s criterion is not sensitive in diagnosing Right-sided Infective endocarditis. (4) Echo can be negative in 15% of infective endocarditis. In suspected Right-sided Infective endocarditis if the initial Echo is negative, the AHA recommendation is to repeat Echo after 7 - 10 days rather than to proceed with Transoesophageal Echo. (6)

Methicillin-susceptible S. aureus is best treated with a semisynthetic penicillin, nafcillin, oxacillin, flucloxacillin or cephalosporin (Cefazolin). Methicillin-resistant S. aureus should be treated with Vancomycin, Daptomycin. Uncomplicated cases (no evidence of renal failure, extrapulmonary metastatic infections, or simultaneous left-sided valvular infection) may be treated for two weeks, but complicated right-sided endocarditis should receive treatment for six weeks. (7)

Uncontrolled sepsis despite adequate antibiotic treatment, intractable right heart failure despite appropriate medical treatment, recurrent pulmonary emboli, if the size of a vegetation increases or persists at >1 cm are the most important indications for surgical intervention. (2) Surgical intervention will be needed in 5 - 16 % cases. (8)

Patients with right-sided IE should be screened for HIV, Hepatitis B, C co-infection. (2,4,9) Prognosis of Right-sided Infective endocarditis is much better when compared to left-sided endocarditis. (2)

CONCLUSION

Right-sided Infective endocarditis is a diagnostic challenge due to the complexity of its presentation and subtle clinical signs. Right-sided IE should be considered as a differential diagnosis in a patient presenting with multilobar consolidation. A high index of suspicion in a predisposed patient with meticulous history, clinical examination and necessary investigations will clinch the diagnosis.
REFERENCES