Tricuspid Valve Endocarditis with Septic Pulmonary Emboli in a Drug Addict

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Presented is a case of a young, polytoxicomaniac male with a history of intravenous drug abuse. He arrived at our department in a septic state with fever and showed signs of right-sided decompensated cardiac insufficiency. The patient tested positive for hepatitis C, and blood cultures were positive for Staphylococcus aureus. A thoracic computed tomographic scan revealed bilateral, multiple septic pulmonary emboli. Transesophageal echocardiography disclosed large mobile vegetations on the tricuspid valve associated with severe regurgitation. The infected tricuspid valve was replaced with a mechanical heart valve, and the patient recovered uneventfully from surgery.

Keywords: tricuspid valve, endocarditis, septic emboli, beating heart surgery, drug addiction

Introduction

Acute tricuspid valve endocarditis is rare and usually associated with habitual intravenous self-administration of drugs. In most cases of intravenous drug addicts (IVDA) with infective endocarditis (IE) the right side of the heart is involved, most frequently, the tricuspid valve (58%–80%) with a reported mortality of 5% to 10%.1 Patients presenting with human immunodeficiency virus (HIV), especially those with advanced immunosuppression are common. Poor hygiene with unsafe injection practices and diminished immune capabilities are two of the main causes of right sided endocarditis in intravenous drug abusers. Tricuspid endocarditis in the case of a drug addict can usually be treated using antibiotic therapy. Surgery is often the only option following a failed antibiotic treatment to avoid sepsis or severe right heart failure. The high risk of vegetations on the tricuspid valves is septic pulmonary emboli resulting in various pulmonary complications such as pneumonia and pulmonary abscess.1,2 Precise localization of the site of involvement is necessary, should antibiotic therapy fail and surgical therapy become indicated.

Case Report

A 28-year old male with a history of intravenous drug abuse was in a buprenorphine substitution program for 7 months prior to presenting to our hospital with fever. On examination, he was very pale, febrile and had severe pedal edema. His heart auscultation showed a right-sided parasternal 2/6 systolic murmur at the 5th intercostal space. The lung auscultation revealed bilateral fine bubbling crackles. The electrocardiography did not show any pathological changes. His white blood cell count was 9460 cells/μl with an elevated c-reactive protein of 155 mg/l; he also suffered from anemia with hemoglobin of 7.5 g/dl and thrombocytopenia of 111,000/μl. The blood
tests showed a negative result for HIV and a positive result for the hepatitis C virus. His vital signs upon admission were temperature of 33°C, blood pressure 130/80 mmHg, pulse 100, and respiratory rate 30 describing systemic inflammatory response syndromes (SIRS). A ubiquitous, spot-shaped opacity with bilateral pleural effusions were present in the thoracic x-ray. Clear hepatosplenomegaly with signs of evident liver parenchymal damage, explained by the diagnosed hepatitis C with perihepatic ascites, were seen in the abdominal ultrasound. Additionally, there was evidence of borderline congestion of the liver veins and the inferior vena cava. The following transesophageal echocardiogram (TEE) showed large vegetations surrounding all three valve leaflets of the tricuspid valve which prolapsed in the right atrium, causing a moderate to severe tricuspid valve regurgitation (Fig. 1A). Furthermore, a moderately impaired ejection fraction with anterior, anterolateral and apical hypokinesia was able to be seen.

Partially nodular infiltrates with small cavernous fusions in both lungs were recognized in the thoracic computed tomography (CT), in the context of multiple septic emboli with clear dystelectatic and atelectatic changes and pulmonary congestions symptoms. In addition, bilateral pleural effusions were demarcated in the interlobar fissures and dorsally (Fig. 1B).

The initially prescribed and begun in-patient antibiotic therapy for a primary pulmonary infection (Piperacillin/Tazobactam, Erythromycin, Fluconazole) was changed according to the antibiogram to Gentamycin, Ceftriaxone, and Flucloxacillin following the blood culture verification of a Staphylococcus aureus. Because the CT images were not able to exclude a fungal pneumonia, Voriconazole was added to the therapy. An intensive diuretic therapy was commenced for the right-sided cardiac decompensation.

As the vegetation on the tricuspid valve progressed (larger than 1cm) throughout the course of the intensive intravenous antibiotic therapy, the systemic sepsis remained unrelenting, and bilateral pulmonary septic emboli with deteriorating respiratory status developed, surgical intervention was recommended.

The chest was opened through a routine median sternotomy incision, and the operation was without complications. Cardiopulmonary bypass was established through direct cannulation of both venae cavae and the ascending aorta. The body of the dilated right atrium was opened in beating heart modus. Huge vegetations were present on all leaflets of the almost completely destroyed tricuspid valve and extended down the tips of the papillary muscles (Fig. 2). The annulus and the interventricular septum were not involved. The valve was excised as well as the entire subvalvular chordal structures. Small, almost endocarditis-like deposits surrounding the free wall of the right ventricle were removed, along with the endocardium. Afterwards, the tricuspid valve was replaced by a mechanical heart valve prosthesis (33 mm diameter) in the beating, perfused heart.

The edges of the atrial incision were approximated with 4/0 prolene sutures followed by the decannulation of cardiopulmonary bypass. Following the closure of the pericardium, both pleural cavities were examined, and one large bore chest drain was inserted into each cavity followed by a routine sternum wire-closure. After surgery, the patient fully recovered and received long-term intravenous antibiotics for six weeks postoperatively.
Discussion

Common symptoms secondary to right-sided endocarditis are persistent fever, bacteremia, and multiple pulmonary emboli. Other findings include pleuritic chest pain or other pulmonary findings which may aggravate the clinical diagnosis. Our patient presented with multiple septic pulmonary emboli seen in the CT images. In this context, the emboli can induce pulmonary infarction, abscesses, pneumothoraces, and purulent pulmonary effusions. In exceptional circumstances, right-heart failure can arise, generated by the increase in pulmonary pressure, severe tricuspid valve regurgitation, or obstruction of pulmonary circulation through multiple pulmonary emboli.

Surgery for right-sided infective endocarditis is recommended in the following situations: 1. right-heart failure due to severe tricuspid valve regurgitation 2. inability to eliminate bacteremia or organisms resistant to culture-directed antibiotic treatment, within 7 days. 3. tricuspid valve vegetations >20 mm.

Echocardiographic vegetation imaging is most telling of an IE in febrile IVDA. In the majority of cases, tricuspid valve vegetations are large due to low pressure in the right ventricle allowing them to grow, possibly exceeding 2 cm. Occasionally parts of the vegetations can be visualized floating in the right ventricle or entrapped in the subvalvular apparatus. Transthoracic echocardiography (TTE) usually allows assessment of tricuspid valve involvement because of the valve's anterior location and usual large vegetations. TEE imaging is more sensitive to detect vegetations than TTE imaging, especially in case of abscesses, and associated left-sided involvement. In our case, TEE was proven the best examination technique to diagnose right-sided endocarditis, clearly recognizing the tricuspid valve vegetation. In summary, the combination of typical right-sided echocardiographic structures and positive blood cultures can be considered as major criteria for right-sided endocarditis.

Right-sided endocarditis in IVDA is commonly caused by Staphylococcus aureus, Pseudomonas aeruginosa, and other gram-negative organisms, fungi, streptococci, and enterococci have also been found. Ruotsalainen et al. underlined the increased incidence of infective endocarditis being 46% among IVDA. Additionally, others demonstrated a high infestation rate of methicillin-resistant Staphylococcus aureus (MRSA) in IVDA.

However, uncomplicated tricuspid valve endocarditis can be successfully, medically treated in 80% of patients; in the remaining 20% with very large vegetations and expectably poor antibiotic penetration like in the presented case, surgical treatment is required with an appropriate postoperative course of culture-directed antibiotics, although IVDA run the high risk of reinfection. Because

Fig. 2  Intraoperative view of the severely destructed tricuspid valve with huge vegetations on all valve leaflets.
of this, patients presenting with right-sided endocarditis should receive an antibiotic treatment spanning Staphylococcus aureus, streptococci, and enterococci and should include penicillinase-resistant penicillins or vancomycin, depending on the local prevalence of MRSA.¹)

The present approach for right-sided endocarditis was based on the following principles: 1. radical excision of the infected region 2. valve repair when practicable, and 3. if valve repair is not possible, excision of the tricuspid valve followed by prosthetic valve replacement.¹)

Rizzoli¹⁰) and Carrier¹¹) demonstrated comparable results in long-term survival and reoperation-free survival for mechanical and bioprosthetic valves in the tricuspid valve position. Valve replacement usually gives better long-term hemodynamic results when repair is not an option or comes with high risk, for example, in cases of large or multiple leaflet destructions. In case of annular destruction major postoperative complications are common like high-grade av-block. The implantation of large prostheses (>30 mm) guarantees low transvalvular gradients. The incidence of prosthetic thrombosis is low, even though compliance with long-term anticoagulation in IVDA is unpredictable and bioprosthesis degeneration develops more slowly owing to the low pressure conditions in the right ventricle.¹²,¹³)

Conclusion

In summary, right-sided IE is most frequently observed in IVDA. TEE is of major value in these patients. Right-sided IE has a high recurrence rate in IVDA secondary to continued drug abuse even though the disease itself has a low in-hospital mortality. The optimum management of such cases is not defined, as IE remains a diagnostic and therapeutic challenge. In cases with extraordinary findings such as septic emboli, severe right heart failure, large vegetations, or recurrent/intractable sepsis, surgery should be recommended.

Disclosure Statement

None.

References