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CLINICAL INFORMATION

Anesthetic management for patient with severe cyanosis following bioprosthetic valve stenosis

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KEYWORDS
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Abstract We presented a 39-year-old female patient with life-threatening hypoxemia after tricuspid valve replacement because of Ebstein's anomaly. And the severe cyanosis is due to bioprosthetic valve stenosis and atrial septal defect. Anesthetic management of a patient with severe obstructive prosthetic valve dysfunction can be challenging. Similar considerations should be given to patients with Ebstein's anomaly to maintain the pressure equalized between the right and left atrial. Transesophageal echocardiography and cerebral oxygen saturation provided real time information in perioperative care.

PALAVRAS-CHAVE
Anestesia;
Cianose;
Estenose de valva bioprotética;
Anomalia de Ebstein

Manejo anestésico de paciente com cianose grave após estenose de valva bioprotética

Resumo Apresentamos o caso de uma paciente de 39 anos, com hipoxemia em risco de vida após a substituição da valva tricúspide devido à anomalia de Ebstein e cianose grave devido à estenose de valva bioprotética e comunicação interatrial. O manejo anestésico de um paciente com disfunção obstrutiva grave de prótese valvar pode ser um desafio. Os pacientes com anomalia de Ebstein também precisam de atenção especial para manter a pressão equalizada entre o átrio direito e o esquerdo. A ecocardiografia transesofágica e a saturação cerebral de oxigênio forneceram informações em tempo real nos cuidados perioperatórios.

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Introduction

Prosthetic valve thrombosis is a devastating complication resulting from the use of mechanical valves. Some different treatments of prosthetic valve stenosis were reported. Surgical treatment includes thrombectomy and valve replacement is still the only effective treatment for unstable patient. But the mortality ranges from 0% to 60% depending on the functional class at presentation and the urgency of operation.[1,2] We report a successful case of patient with severe cyanosis and life-threatening hypoxemia due to bioproshetic valve thrombosic stenosis and a large Atrial Septal Defect (ASD), requiring surgery of bioproshetic valve replacement.

Case report

A 39-year-old woman, weighing 50 kg, presented to the emergence department with one month history of increased fatigability, dyspnea on exertion. One year ago, she felt palpitation and was diagnosed Ebstein's anomaly, atrial septal defect combined with Wolff-Parkinson-White syndrome. She received radiofrequency catheter ablation therapy and had undergone tricuspid valve replacement 4 months ago. A 31 mm bioprosthetic valve was implanted and ASD was left open. She was given 100 mg of aspirin orally each day after surgery. The International Normalized Ratio (INR) on admission was 1.1. The physical examination revealed a severe cyanotic woman, regular heart rate 124 beats.min^{-1}, blood pressure 98/64 mmHg, respiration at 24 cycles.min^{-1} with pulse oximetry (SPO_2) 60%. Transthoracic echocardiogram (TTE) demonstrated vegetation on the bioprosthesis and causing prosthetic tricuspid valve stenosis with a mean gradient 5 mmHg, bilateral shunt across ASD (Figs. 1 and 2, Supplementary Material Video 1).

The patient was scheduled for the re-replacement of bioprosthetic valve and bidirectional Glenn surgery. On arrival in the operation room, the patient was in semireclining position with severe cyanosis. Initial hemodynamic statuses were blood pressure 110/70 mmHg, heart rate 58 beats.min^{-1}, pulse oximetry 32% on 4 L of oxygen by face mask. Left radial artery and right internal jugular vein were cannulated under local anesthesia. The arterial blood gas showed PH 7.463, PCO_2 34.5 mmHg, PO_2 28.2 mmHg, SO_2 43%, HCO_3 25.2 mmoL.L^{-1}. To evaluate the influence of anesthesia to the patient, 3% sevoflurane in 100% oxygen was given. After the patient loss of consciousness, we noted that the vital signs were stable, then sufentanyl 20 μg, rocuronium 50 mg and midazolam 2 mg were administrated intravenously. Combined with airway local anesthesia, the intubation was achieved successfully. To maintain the systemic vascular resistance and right ventricle function, norepinephrine and epinephrine of 0.02 ug.kg^{-1}.min^{-1} were infused continuously. Blood pressure and SPO_2 were almost as same as before. Transesophageal echocardiography confirmed the tricuspid valve stenosis (Supplementary Material Video 2). Cardiopulmonary bypass (CPB) was established via femoral artery and venous. At the initation of CPB, the SPO_2 was still low (52%) because of the inadequate draining and flow rate. After median sternotomy, the cannulation was switched to aorta and SPO_2 raised to 100%. In operation, multiple thrombosis were found on the bioprosthesis causing tricuspid valve stenosis. So the tricuspid valve was re-replaced with a 29 mm bioprosthetic valve, atrial septal defect was narrowed from 10 to 2 mm, along with the bidirectional Glenn surgery (Supplementary Material Video 3). There were no remarkable events in the operating room and in the cardiac ICU. The patient was given warfarin 1.95 mg per day after surgery and the INR was ranged from 2.43 to 2.58.

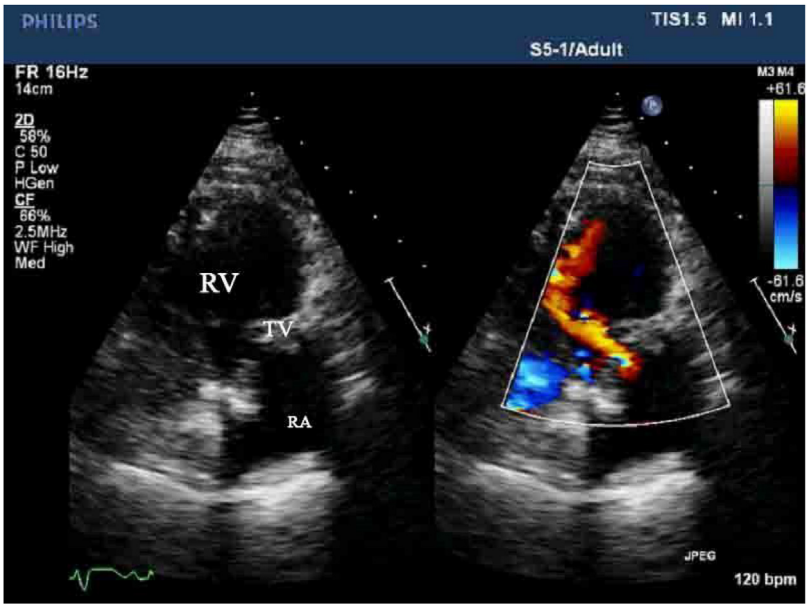

Figure 1 Transthoracic echocardiography (TTE) demonstrate bioprosthetic tricuspid valve and the color Doppler show narrow right ventricular inflow jet produced by the thrombosed tricuspid prosthesis (RA, right atrial; RV, right ventricle; TV, tricuspid valve).

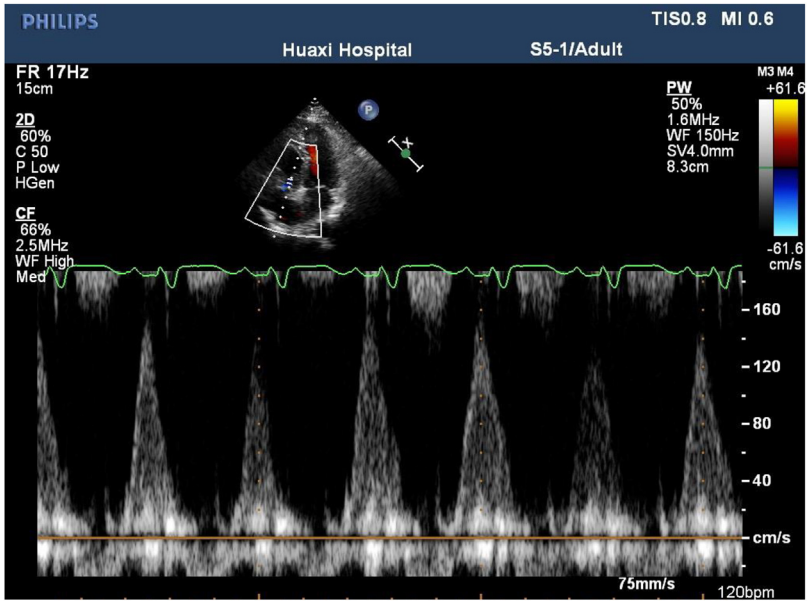

Figure 2 Increased Doppler gradient across the thrombosed tricuspid prosthesis.

Discussion

Thrombosis is a rare but severe complication of prosthetic heart valves, and the overall reported incidence of left-sided prosthetic-valve thrombosis ranged from 0.1% to 5.7% per year.[3] The incidence of valve thrombosis in the tricuspid position is higher than the left side valve, as the low velocity of blood across the valve makes it prone to thrombosis.[4] During last decades, thrombolytic therapy (TT) and intensified anticoagulation are the first choice of treatments in right-sided prosthetic valve thrombosis due to higher rates of mortality associated with surgery.[5] But for this patient with life-threatening cyanosis, emergency surgery is the only option to save the patient.

In the current case, the pathophysiology is similar to patient with Ebstein's anomaly and atrial septal defect. The severe cyanosis was caused by a large right-left shunt at atrial level because of the obstructive prosthetic valves stenosis at tricuspid valve site. While the systemic cardiac output rely on the amount of shunt. To anesthesiologist, the key point is to maintain the pressure equalizes between the right and left atrial. Intervention or circumstance that increases right atrial pressure should be avoided. Increases in pulmonary vascular resistance (acidemia, hypoxemia, hypercarbia) and overzealous intravenous fluid administration are examples of such circumstances. The use of Positive End-Expiratory Pressure (PEEP) should be avoided if possible because it may increase RV after load and compromise RV function.

To this patient, inotropes were necessary both before and after cardiopulmonary bypass because right ventricle was small and systolic function was impaired. And bidirectional Glenn surgery was performed to decreases volume load on right ventricle to preserve the right heart function. Vasoconstrictor such as norepinephrine may be a good choice because its predominant alpha-agonistic effect is combined with some limited beta agonism.[6]

Transesophageal echocardiography (TEE) is a standard monitoring for this patient. TEE provided real time information of ventricle function, severity of tricuspid valve stenosis and amount of atrial shunt. It also helps in preload optimization and assessment of postoperative RV and LV function thus guiding use of inotropes.

Conflicts of interest

The authors declare no conflicts of interest.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at doi:10.1016/j.bjane.2018.06.006.

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