Cardiology Cases and Systematic Reviews

Case Report

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Complications after Implanting Cardiac Assistants

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Volume 3	Issue 1
Pages	25-27
Received	February 17, 2021
Accepted	March 23, 2021
Published	March 25, 2021

Citation: Gómez HL, Blanco AP, García-Perrote SC, Bigot SR (2021) Complications after Implanting Cardiac Assistants. Cardiol Cases Syst Rev 3:008.

Keywords

Assistance, Valvular heart disease, Cardiac assistance, Complications

Ventricular assist devices are increasingly used in the hemodynamic management of the postoperative period of cardiac surgery, either as bridging therapy to transplantation or until recovery from ventricular dysfunction. However, the use of these right-only assistive devices is rare [1,2].

Local thrombosis and systemic embolism continue to be important postoperative complications. Its incidence increases when the flow through the device is low, which occurs during weaning or native ventricular recovery, and when it is located in the right heart chambers, due to its low pressure system with respect to the left heart, so it is more frequent thrombosis of the tricuspid prosthesis, and especially in mechanical prostheses [3]. It usually occurs in the early postoperative period (first three months), when endothelialization of the ring suture is not complete.

The diagnosis is based on echocardiography, by visualizing an intraprosthetic thrombus or by indirect signs such as an increase in the mean transvalvular gradient, a reduction in the prosthetic area, a decrease in the motility of the prosthetic leaflets or their thickening [2,4].

Possible treatments consist of: Fibrinolysis in hemodynamically unstable, symptomatic patients or with large thrombi (> 0.8 cm^2), reserving anticoagulation for the rest of patients (NYHA I-II, small thrombi) or surgery if fibrinolysis is contraindicated or fails.

We present the case of a 76-year-old woman with a history of dyslipidemia and osteoporosis, diagnosed with moderate mitral regurgitation, severe tricuspid regurgitation, and atrial fibrillation (AF) after an episode of heart failure, with preserved biventricular function on echocardiographic study. We underwent programmed intervention for mitral valve replacement with mechanical prosthesis (St. Jude No. 25), tricuspid valve replacement with biological prosthesis (Magna Ease No. 31) and closure of the left atrial appendage with atriclip®.

Upon admission to the ICU, she presented hemodynamic instability that required vasoactive support with norepinephrine and dobutamine; and arrhythmic storm that was treated with amiodarone and lidocaine infusion. Analytically, refractory hyperlactacidemia and impaired kidney and liver function stood out, with total bilirubin levels > 4 mg/dl, as well as coagulopathy. In the transesophageal echocardiogram, severe right ventricular (RV) dilation and systolic dysfunction were observed, with normally functioning tricuspid and mitral prostheses.

Due to multi-organ failure, it was decided to place a right ventricular assist device, achieving a right cardiac output of 3 L/min (2000 rpm).

During the following days, the patient presented electrical stability that allowed the withdrawal of antiarrhythmic drugs, remaining dependent on ventricular pacing by epicardial pacemaker due to slow AF. It progressed with a progressive decrease in inotropic and vasoactive

support, and normalization of signs of hepatic congestion and hypoperfusion. The control echocardiogram revealed persistence of great dilatation and dysfunction of the RV, and limitation of opening of the tricuspid prosthesis with a progressive increase in the transvalvular gradient (Figure 1) despite reduced circulatory support.

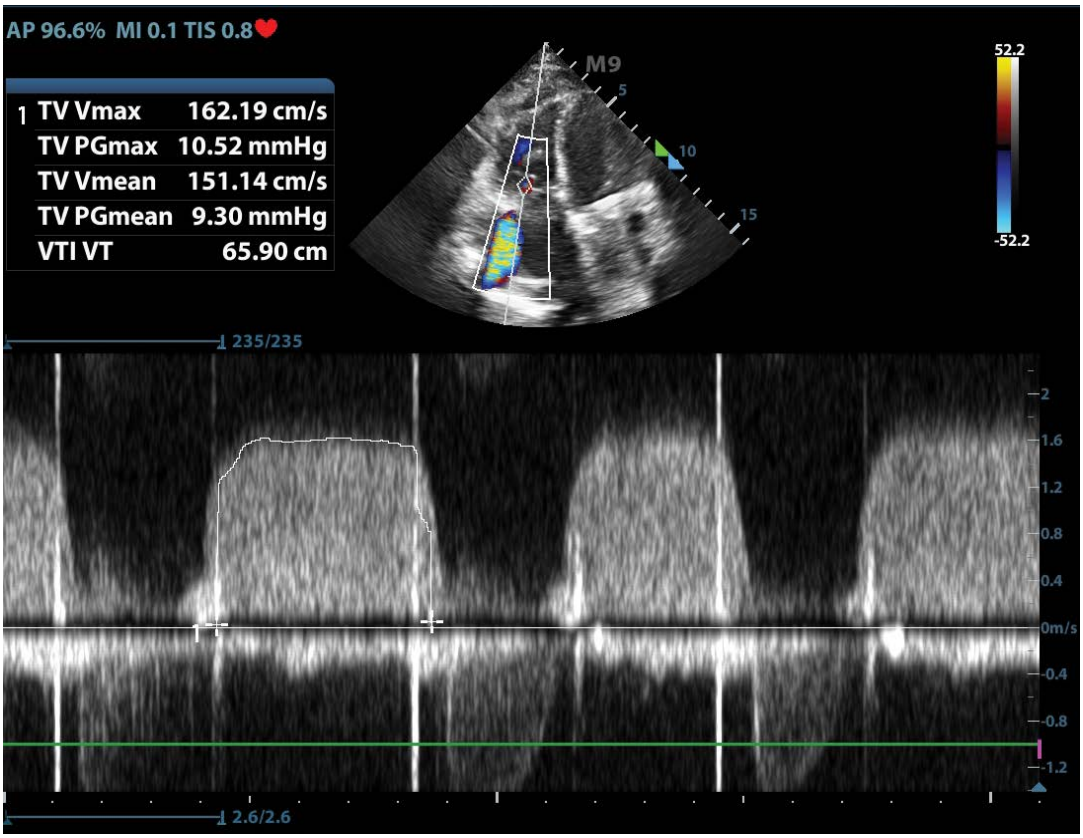

Figure 1: Severe limitation in tricuspid valve opening with significant stenosis.

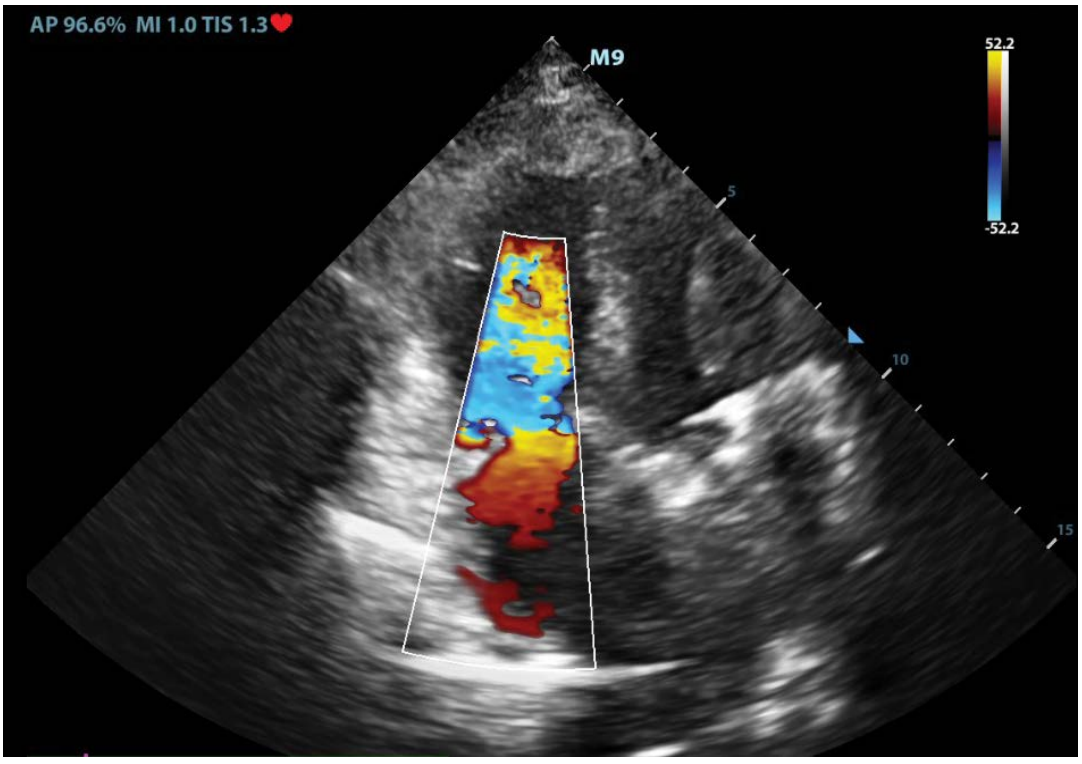

Figure 2: Normal tricuspid valve function after replacement.

Treatment with sodium heparin was started in the first 24 hours, in the range of anticoagulation that was suspended due to bleeding complications (upper gastrointestinal bleeding, hematic secretions through the orotracheal tube and femoral pseudoaneurysm after removal of the arterial catheter).

In echocardiographic control, an improvement in RV motility was observed with severe limitation in the opening of the tricuspid prosthetic leaflets, which caused significant stenosis (mean gradient of 9 mmHg). It suggested thrombosis of the bioprosthesis even without observing intraprosthetic thrombus.

After a week of admission, she was surgically revised, showing intraprosthetic tricuspid valve thrombosis. It was replaced by another biological one and the ventricular assist device was withdrawn.

Progressively, the evolution was favorable with improvement in right ventricular function and normal function of the new tricuspid prosthesis (Figure 2).

Discussion

In biological valve thrombosis, direct visualization of the thrombus is rare. The diagnosis is made from a high degree of clinical suspicion in conjunction with imaging tests suggesting valve obstruction [1,2,4]. In the case we present, the existence of thrombosis was suspected by indirect signs visualized by echocardiography: Increased mean transvalvular gradient and absence of prosthetic leaflet opening [2].

More than the absence of anticoagulant treatment, the early thrombosis of the bioprosthesis was favored by severe ventricular dysfunction and the presence of RV assistance with the absence of atrioventricular flow, even though it carried a mechanical valve, a priori, with a higher thrombotic risk [1,2].

In our case, we opted for surgical revision given the hemodynamic instability and the contraindication for fibrinolysis. The choice of treatment should be individualized in each case, since there is no evidence of the superiority of any of them in terms of morbidity and mortality [4-6].

We conclude that this complication should be suspected in patients with valve replacement due to a compatible clinical picture, especially in patients with ventricular assistance, due to the contribution to the presence of low intracardiac flows.

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