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Case Report

Superior Vena Cava Syndrome with Massive Thrombosis after Aortic Valve Replacement

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Abstract

Background: Superior vena cava syndrome is uncommon after cardiac surgery, because the formation of thrombus after cardiopulmonary bypass rarely occurs.

Case presentation: Herein, we present a case of a patient with dynamic severe mitral regurgitation after aortic valve replacement. Whereas the treatment of this disorder was a challenge, the real complication, an early-onset and massive thrombosis of the superior vena cava, was hidden and misdiagnosed.

Conclusion: Although superior vena cava syndrome after cardiac surgery has been reported previously, a large thrombus as the one described here is unusual. We have to keep in mind this complication in the presence of central vein catheter when we do no achieve our goal despite correct postoperative management.

ABBREVIATIONS

SVC: Superior Vena Cava; SAM: Systolic Anterior Motion; LVOT: Left Ventricular Outflow Tract

INTRODUCTION

Superior Vena Cava (SVC) syndrome is a well-known problem that is rare after cardiac surgery [1]. Patients with SVC syndrome usually have an extrinsic compression of the superior vena cava or infiltration by malignant diseases [2]. In rare cases it can be produced by chemotherapy catheters or permanent pacemakers electrodes [3,4]. Clinical manifestations are well-known, and are similar to cardiac tamponade caused by postoperative bleeding. But while both cardiac tamponade and SVC syndrome may present as elevated central venous pressure with hypotension, the presence of differential cyanosis is not usual with cardiac tamponade. Furthermore, the adverse effects of cardiopulmonary bypass on the factors that maintain hemostasis, mainly platelet dysfunction and activation of the fibrinolytic cascade, make the formation of thrombus unlikely in the immediate postoperative period. In this scenario, the formation of a thrombus that occludes the superior vena cava rarely occurs, unless there are ruptures or lacerations during surgery.

CASE PRESENTATION

A 73-year-old woman without comorbidities, with a recent diagnosis of symptomatic critical aortic stenosis was referred to our Department for aortic valve replacement. Transthoracic echocardiography demonstrated a calcified tricuspid aortic valve with a medium gradient of 86mmHg, a contact between the anterior mitral leaflet and the interventricular septum during systole with mild mitral regurgitation (the called "pseudo" systolic anterior motion (SAM)), and a left ventricular outflow tract (LVOT) too narrow (16mm). The left ventricle was severely hypertrophied with an interventricular septal thickness of 20mm and good systolic function. The coronary arteries were normal at angiography.

At operation, the aortic valve was excised and the LVOT measuring 19mm with Hegar dilator. A sutureless bioprosthesis Perceval S™ (Sorin, Saluggia, Italy) size small was implanted. The operation was uneventful.

Two hours after arrival to Postoperative Care Unit, the patient began to suffer episodes of hypotension. Central venous pressure increased up to 21mmHg while urine flow decreased. Although the patient was not bleeding, a cardiac tamponade was suspected. A transesophageal echocardiography revealed severe mitral regurgitation due to SAM of the mitral valve, with obstruction of the LVOT during systole, whereas cardiac tamponade was excluded (Figure 1A).

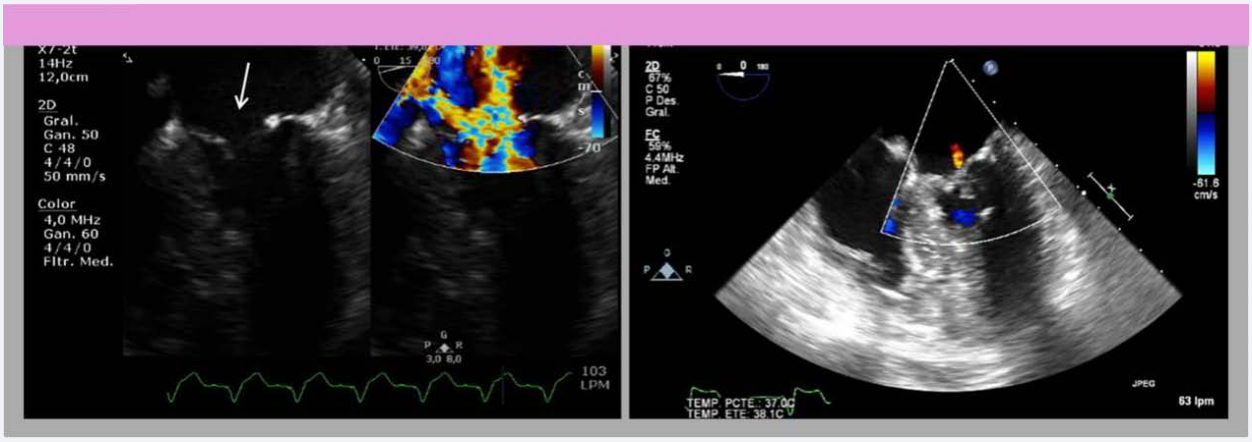

Figure 1 (A) - Transesophageal echocardiography revealed severe mitral regurgitation due to SAM of the mitral valve (white arrow), with obstruction of the LVOT during systole. (B) After betablocker treatment, both SAM and mitral regurgitation had resolved, without LVOT obstruction.

Catecholamine infusion was ceased. A volume expansion to increase preload was done to control SAM and a norepinephrine was increased up to 0,1mcg/Kg/min to boost after load. An infusion of esmolol was started to reduce contractility and thus reduce the muscular component of the obstruction. After this, the patient improved. The central venous pressure dropped to 12mmHg. The mean arterial pressure was over 80mmHg and the urine flow was over 100mL/h.

Ten hours later, a routine sample of peripheral venous blood revealed venous oxygen saturation less than 35%. In order to evaluate the hemodynamic parameters, a Swan-Ganz catheter was inserted. Pulmonary artery pressure was 30/19mmHg, pulmonary capillary wedge pressure of 10mmHg, venous central pressure of 9mmHg and systemic vascular resistance of 2000 dyn*sec/cm5, but cardiac index was extremely low (0,9-1,3 L/min/m^2), due to a low ejection volume (15-20mL). A volume expansion was carried out once again, but we were not able to increase the cardiac index, despite medical management. The urine flow was adequate and the lactic acid in blood was less than 4mEq/L, without metabolic acidosis.

A new transesophageal echocardiography, 24 hours after surgery, showed that both SAM and mitral regurgitation (Figure 1B) had resolved, without LVOT obstruction. The filling of the right ventricle remained suggesting hypovolemia, despite repeated volume expansion.

A differential cyanosis limited to the head and neck were already established, which then suggested a SVC syndrome. A new echocardiogram was performed, that excluded the possibility of a localized tamponade causing SVC syndrome.

For a short period of time, the patient improved markedly and a cardiac index of 2.2L/min/m2 was achieved.

After this, the patient developed intermittent episodes of LVOT obstruction again, with severe pulmonary hypertension, without response to medical management. The patient was critically ill with important hemodynamic instability. We could not perform a CT scan or a cavography in this scenario. Unfortunately, 36 hours after surgery, the patient finally died due to multiple organ failure.

The necropsy showed aortotomy sutures and cannulation sites intact. It was noted a sutureless bioprosthesis in aortic position without obstruction. Severe hypertrophy was observed in the left ventricle (up to 2.2cm) and in basal interventricular septum (up to 3.4cm). This area showed fibrous degeneration, probably related with SAM. Also SAM related, hypertrophy of the posteromedial papillary muscle with anomalous fibrous adherences to the ventricular wall was found. A massive cavoatrial thrombus involving the jugular-subclavia junction and the left innominate vein was observed (Figures 2,3). Bilateral pulmonary embolism, partially obstructed (25%) was presented as well. There was no evidence of extrinsic compression or tumoral infiltration as possible causes of thrombus formation.

DISCUSSION

We present a well-known complication after open heart surgery, although a large thrombus as the one described here is unusual. Two main aspects have to be taken into consideration:

Firstly, it must be pointed out that the patient developed a dynamic LVOT obstruction in the immediate postoperative course due to true SAM. This condition should be considered after aortic valve replacement when the patient is hemodynamically unstable and does not respond to usual measures [5]. Although in the

Figure 2 Massive thrombosis of left innominate vein (*) and superior vena cava (**).

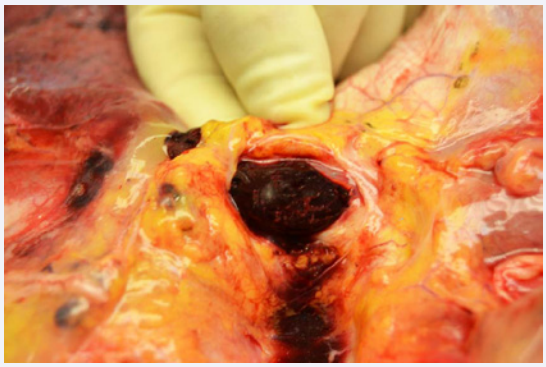

Figure 3 Longitudinal exposure of the superior vena cava with thrombus.

majority of cases this complication does not occur, the dynamic LVOT obstruction is caused by the aortic valve replacement. The end-systolic pressure in the LVOT is high in the presence of a stenotic aortic valve. Following relief of this obstruction, end-systolic pressure in the LVOT falls, exacerbating the tendency for obstruction. Even though it did not happen during cardiopulmonary bypass weaning under echocardiographic control, the patient had risk factors to develop it, such a severe septal hypertrophic of the left ventricle and preoperative pseudo-SAM (SAM without mitral regurgitation) with mild obstruction of the LVOT. Medical treatment is headed for filling the left ventricle to increase end-diastolic dimension with fluids, reduce in contractility with beta-blockers and increase afterload with vasoconstrictors [6]. While few patients develop severe complications, an isolated aortic valve replacement might be considered reasonable. But according to our experience, we can speculate if concomitant prophylactic septal myectomy could have prevented significant SAM in a patient with risk factors to develop it, taking risks of this nature added technique [7].

But although the medical management was effective, the patient was really unstable due to low cardiac output. Obviously, the management of this challenging complication might have confused us, and all the efforts were headed for the treatment of the low cardiac output. Low cardiac output associated with low filling pressures is a sign of hypovolemia and echocardiography still showed vigorous wall motion and a small chamber size in both ventricles. So we continued a volume expansion to increase preload in order to achieve hemodynamic stability, but the cardiac index never increased enough despite volume overload. We were not able to diagnose a large cavo-atrial thrombus with transesophageal echocardiography.

On the other hand, a SVC syndrome began to be evident 24 hours after surgery. Probably it started the day before, and was clinically evident the day after. As all efforts were aimed at medical treatment of LVOT obstruction, it was initially unnoticed. After diagnosis, an acute pericardial tamponade due to external compression of blood clot that caused obstruction of the superior vena cava was suspected, but echocardiogram excluded this possibility. But from this moment, the patient began once again with an important hemodynamic instability, refractory to all measures. Due to these reasons it was not possible to perform a CT scan or cavography, and the patient finally died.

As the cause of death was not clear, autopsy was requested by the family. A massive thrombus from the cavo-atrial junction towards de jugulo-subclavian junction involved all the superior vena cava and left innominate vein. A bilateral pulmonary embolism (partially obstructed) were observed as well.

Malignant diseases are the most common cause of SVC syndrome, mainly lung cancer and lymphomas. Iatrogenic is the next cause reported, in association with intravascular devices or catheters [8]. In the seventies some authors reported an iatrogenic superior vena cava syndrome as a complication of internal jugular venous catheter in patients with tumors, resolved after removing the catheter from the jugular vein or after open surgery. Rupture or lacerations that occur secondary to cardiac surgery or bi-caval heart transplantation are extremely rare, especially produced during cannulation of superior vena cava, with following stenosis and thrombosis [9].

The presence of an introducer catheter for right heart catheterization in cardiac surgery, even for a short time, is associated with an incidence of early-onset catheter-related thrombosis [10]. Approximately 15-30 % of patients in medical intensive care units might have a catheter-related thrombosis [11,12].

Our patient had an increased central venous pressure only during periods of LVOT obstruction, secondary to severe pulmonary hypertension and severe mitral regurgitation. But after controlling this, the central venous pressure decreased. After reviewing echo images, the tip of the venous catheter was free of thrombus. In fact, we could never increase the preload: firstly the venous introducer was placed in the superior vena cava, secondly the peripheral venous catheter was placed in the right forearm. Probably there were several short periods of transient filling of the right ventricle, and in those moments the hemodynamic situation improved. Finally, the hypovolemic status led to low cardiac output, renal failure and a LVOT obstruction and true SAM due to inadequate filling of the left ventricle, causing the death of the patient.

A CT scan or a cavography might help diagnose the cause of the SVC syndrome, but the patient was critically ill and it was not possible to perform them. Although thrombolysis therapy could be considered in such cases, this treatment is contraindicated in patients that occurs rapidly within a few hours of the surgery. A surgical re-exploration is also considered to carry a very high risk. Placement of intravascular stent device may be a valid approach in cases of SVC syndrome secondary to cardiac surgery.

The precise etiology of thrombus formation was not clear, but the association of postoperative low cardiac output and an introducer catheter in the superior vena cava might be fatal, although central venous catheters are widely used without complications.

In summary, we do not suspect SCV syndrome because the venous central pressure was not high and the central venous catheter had been placed recently. We could not properly measure the flow in superior vena cava with transesophageal echocardiography and the end of the superior vena cava in the right atrium was free of thrombi. So, the SVC syndrome could not be diagnosed in time, and timely intervention might have changed the fate.

Low cardiac index, despite aggressive volume expansion and low peripheral venous saturation, with transesophageal echocardiography supporting impaired right ventricle filling, with venous stasis, suggests the diagnosis of SVC syndrome.

CONCLUSION

Although this complication has been reported previously, there are important points in the management of this patient. Whereas the treatment of the initial disorder was a challenge, the real complication was hidden and undiagnosed. We have to keep in mind this complication in the presence of central vein catheter when we do not achieve our goal despite correct postoperative management.

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