



Case Report

Iatrogenic Superior Vena Cava Syndrome: A Rare Cause of Hemodynamic Compromise After Cardiac Surgery

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ABSTRACT

Given the broad array of potential complications following cardiac surgery, specialized care is critical to ensuring good patient outcomes. Herein, we present a rare case of severe hemodynamic compromise, following cardiac surgery, found to be due to iatrogenic superior vena cava syndrome in the setting of baseline biventricular dysfunction. Hemodynamics improved immediately upon addressing the syndrome, highlighting the importance of multidisciplinary collaboration in the postoperative care of cardiac surgery patients.

RÉSUMÉ

Compte tenu du large éventail de complications potentielles après une chirurgie cardiaque, des soins spécialisés sont indispensables afin de garantir de bons résultats pour les patients. Nous présentons ici un cas rare de compromis hémodynamique grave à la suite d'une chirurgie cardiaque, causé par un syndrome iatrogène de compression de la veine cave supérieure dans un contexte de dysfonction biventriculaire existante. Le compromis hémodynamique s'est immédiatement amélioré après la prise en charge du syndrome, ce qui souligne l'importance de la collaboration multidisciplinaire dans les soins post-opératoires des patients en chirurgie cardiaque.

Case

Patients undergoing cardiac surgery are at risk of postoperative hemodynamic instability and circulatory shock. However, such instability is typically due to blood volume loss, baseline or new postoperative ventricular dysfunction, arrhythmias, or tamponade. Herein, we present a first case of critical hemodynamic compromise resulting from iatrogenic superior vena cava syndrome (SVCS) caused by iatrogenic dissection.

A 67-year-old woman was referred to our center for decompensated heart failure and severe mitral regurgitation. Ten years prior, the patient had benefited from a mitral valve annuloplasty. The patient also had a history of remote non-ST-elevation myocardial infarction (NSTEMI), with placement of a coronary stent in the proximal right coronary artery. Her baseline left ventricular ejection fraction (LVEF) was 45%.

In the preceding months, she had reported a progressive decline in her functional class, to New York Heart Association (NYHA) III, with orthopnea. Her preoperative

echogram revealed a now-dilated left ventricle with a markedly reduced LVEF of 25% and severe eccentric mitral regurgitation, as well as a hypokinetic right ventricle. Coronary angiography was notable for a patent right coronary artery stent and the absence of significant stenoses elsewhere.

In the operating room, a 7 French-triple lumen catheter and a Swan Ganz catheter were placed in the right internal jugular vein by the anesthetist. The patient subsequently underwent redo sternotomy for mitral valve replacement with a mechanical prosthesis. Bicaval cannulation cardiopulmonary bypass was then performed with a straight 22 French cannula placed in the superior vena cava (SVC). After weaning from bypass, a progressive increase in hemodynamic support requirements was observed, initially thought to be a combination of cardiogenic and vasoplegic shock. Transesophageal echocardiography (TEE) showed severe biventricular dysfunction, with an LVEF of 20%, with normal mitral prosthesis function. By the end of surgery, the patient required significant doses of dobutamine, norepinephrine, and vasopressin.

Once the patient was dedraped, marked swelling and plethora progressing rapidly to cyanosis of the face, upper torso, and upper extremities was noted. Manipulation of the Swan Ganz catheter allowed determination of an SVC gradient of 25 mm Hg. TEE confirmed the stenosis, but surgical intervention was not considered necessary at the time given the echographic patency of the vein, the initial impression that the cause was external compression due to tissue edema and that the central venous lines were likely

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Ethics Statement: The research reported has adhered to the relevant ethical guidelines.

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See page 1402 for disclosure information.

Novel Teaching Points

- SVC dissection is a potential complication of central venous catheterization or caval cannulation during cardiopulmonary bypass surgery.
- In rare cases, SVC dissection can lead to SVCS and, in susceptible patients, hemodynamic compromise.
- As physical examination during surgery is limited, transesophageal ultrasound remains the principal method to rapidly assess central venous structures.
- A multidisciplinary approach is critical to ensuring rapid troubleshooting and efficient management of SVC complications in the perioperative period.

contributing to the measured gradient, and the impression that the patient's significant biventricular dysfunction in the setting of a high-risk redo mitral valve replacement was primarily driving inotrope and vasopressor requirements. The patient was therefore transferred to the intensive-care unit for continued care, where an initial postoperative cardiac index of 0.80 L/min per m² was documented, and vasopressor and inotrope requirements rose dramatically. The patient was then transferred to the catheterization laboratory for hemodynamic assessment and possible mechanical support. Upon arrival, femoral venous and radial arterial access were established, and the jugular Swan Ganz catheter placed at the time of surgery was removed to allow placement of a femoral Swan Ganz catheter. Venography showed significant SVC narrowing without thrombus and a prominent azygos vein (Fig. 1). The gradient across the stenosis was then measured at 10 mm Hg, and the patient's hemodynamics improved dramatically, requiring rapid down-titration of vasopressors. With a cardiac index then calculated at 2.35 L/min per m², it was determined that the patient did not require mechanical support or any other intervention at that time.

A subsequent computed tomography angiogram (Fig. 2) confirmed that dissection was the cause of the SVCS, with a minimum diameter of 3 mm at the cavoatrial junction. After consultation with angioradiology, and considering the complete resolution of the SVCS and hemodynamic compromise with removal of the jugular venous lines, the decision was made to manage the dissection conservatively.

Discussion

SVCS is a rare complication of cardiac surgery, and this report represents both the first case of critical, life-threatening circulatory compromise due to post-cardiac-surgery SVCS and the first case due to the combination of iatrogenic dissection and the placement of indwelling catheters across the stenosis.

SVCS is most often due to thrombosis or external compression in the context of a malignancy.¹ Nonmalignant causes of SVCS account for nearly 30% of all cases and are most often due to thrombus related to central venous catheters or pacemaker or defibrillator leads.² However, SVC dissection is a very rare finding and, it would seem, an even rarer cause of SVCS. It is unclear whether the dissection occurred as a result of bypass cannulation or at the time of jugular central line placement.

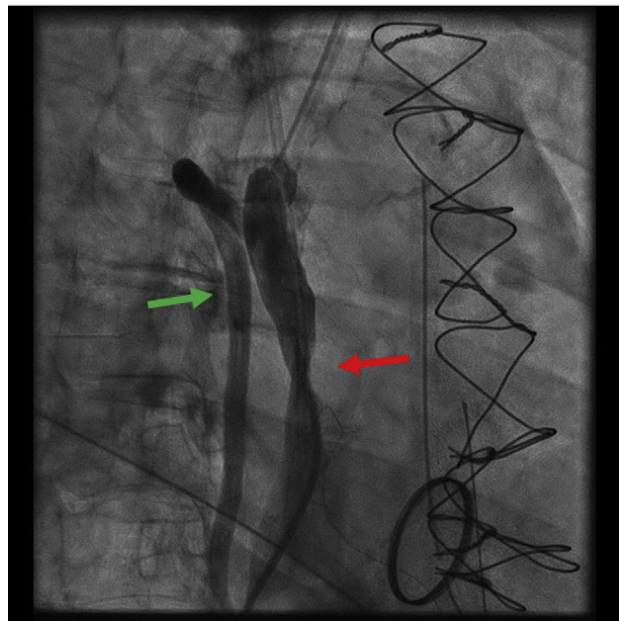


Figure 1. Venogram demonstrating superior vena cava stenosis (red arrow) at the cavoatrial junction and a prominent azygos vein (green arrow).

The patient in this report had significant baseline biventricular dysfunction. We believe that our patient was particularly preload-dependent, such that the reduction in venous return due to the SVCS had near-catastrophic consequences. Whether a patient with normal right ventricular function would have been as susceptible to the adverse hemodynamic effects of the SVCS is unclear.

The first clue that SVCS is occurring is typically the pathognomic distribution of swelling and plethora. However, as physical examination is limited during surgery, avoidance and early recognition of dissection when it occurs can stave off potentially catastrophic consequences. TEE verification of bypass cannulation sites and surface ultrasound guidance of jugular access are therefore recommended. In the advent of

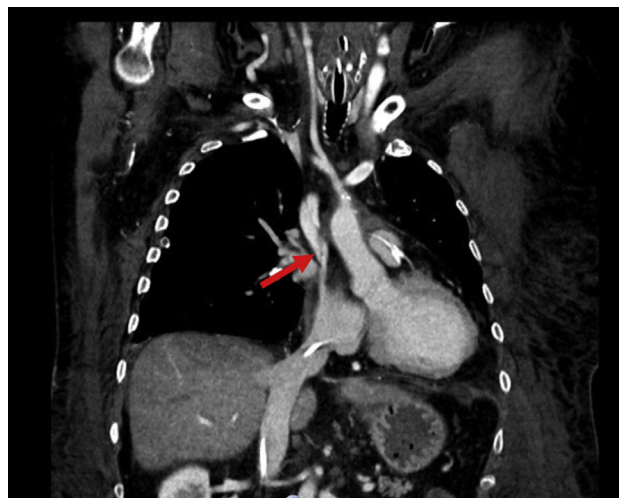


Figure 2. Computed tomography scan coronal view showing superior vena cava dissection (red arrow).

significant SVC narrowing, this case would caution against compounding the situation with the placement of central lines across the stenosis. Similarly, this case illustrates that prompt removal of such lines is likely to be a critical, yet simple intervention to alleviate the degree of obstruction to the venous return.

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Disclosures

The authors have no conflicts of interest to disclose.

References

1. Wilson LD, Detterbeck FC, Yahalom J. Clinical practice. Superior vena cava syndrome with malignant causes. *N Engl J Med* 2007;356:1862-9.
2. Kumar B, Hosn NA. Images in clinical medicine. Superior vena cava syndrome. *N Engl J Med* 2014;371:1142.