Intraoperative Echocardiographic Diagnosis of Inferior Vena Cava Stenosis After Cardiopulmonary Bypass

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THE INFERIOR VENA CAVA (IVC) is responsible for about two thirds of the total venous return to the right atrium (RA).[1] Obstruction of the IVC can be due to chronic disease or an acute insult. The etiology of chronic IVC obstruction includes congenital abnormalities, indirect compression of the IVC (retroperitoneal tumors, hepatic tumors, giant left atrium), or intravascular tumor extension (metastatic renal tumors).[2,3] Chronic obstruction of the IVC is associated with findings of hepatic congestion, ascites, lower extremity edema, and venous thrombosis.[4-7] The slow progression of disease results in collateral venous return to the superior vena cava (SVC) via the azygos, intrathoracic, and vertebral veins.[5,6] In contrast, acute stenosis of the IVC can result in hepatic and renal dysfunction, severe hemodynamic instability, and cardiovascular collapse if left untreated.[5-8] Acute IVC obstruction is rare and has been reported as a complication of operative procedures, including bicaval heart transplantation, orthotopic liver transplantation, mitral valve replacement, and extrinsic compression from implantation of a Nuss bar for repair of pectum excavatum.[1,9-11]

The authors describe a case of acute IVC stenosis at the RA junction that was diagnosed by intraoperative transesophageal echocardiography after cardiopulmonary bypass (CPB). The intraoperative echocardiographic findings included a localized area of narrowing at the IVC-RA cannulation site, turbulent flow into the RA, underfilled cardiac chambers with hyperdynamic biventricular activity, and dilated hepatic veins. The echocardiographic findings were correlated with invasive hemodynamic measurements, and a therapeutic intervention with return to CPB was performed. *The authors recommend the routine examination of the IVC at the IVC-RA junction with color-flow Doppler and spectral Doppler interrogation of flow after cardiac surgery cases, especially in the case of a bicaval venous cannulation technique.*

CASE PRESENTATION

A 74-year-old man with symptoms of worsening shortness of breath and dyspnea on exertion was brought to the operating room for elective mitral valve repair and atrial fibrillation ablation. The patient's past medical history was significant for severe mitral insufficiency, chronic atrial fibrillation, permanent pacemaker for symptomatic bradycardia, hypertension, hyperlipidemia, obstructive sleep apnea, and coronary artery disease. Following placement of a radial arterial line, the patient was intubated after uneventful induction of general anesthesia. A pulmonary artery catheter was placed through the right internal jugular vein. Intraoperative transesophageal echocardiography (TEE) revealed severe mitral regurgitation from anterior leaflet prolapse, mild-to-moderate tricuspid regurgitation, normal right and left ventricular systolic function, and a severely dilated left atrium measuring 6.5 × 6.1 cm.

The patient underwent median sternotomy and uneventful cannulation of the IVC, SVC, and coronary sinus (for retrograde cardioplegia) before bypass. After extracorporeal circulatory support was initiated, the right and left pulmonary veins were ablated using bipolar radiofrequency (ATS CryoMaze; ATS Medical, Minneapolis, MN). The IVC and SVC were isolated using snares, and a right atriotomy was performed to complete the right atrial ablation protocol. The interatrial septum was incised, and ablation lesions were performed in the left atrium. Examination of the mitral valve pathology confirmed isolated prolapse of the anterior leaflet at the A2 scallop. The prolapsed portion of the anterior leaflet was repaired with a GORE-TEX neochord (W. L. Gore & Associates, Newark, DE), and a 30-mm Profile 3D Annuloplasty Ring (Medtronic, Minneapolis, MN) was placed. Before closure of the interatrial septum, a static test demonstrated restored competency of the repaired mitral valve. After de-airing, an infusion of epinephrine (2 μg/min) was started, and the patient was weaned successfully from cardiopulmonary bypass; the total cross-clamp and bypass times were 54 minutes and 130 minutes, respectively.

TEE performed after the intervention demonstrated a successful repair with no residual mitral regurgitation. The patient subsequently was administered protamine to reverse heparin anticoagulation, decannulation was performed, and hemostasis was achieved. A final TEE examination after decannulation demonstrated an abnormality at the IVC-RA junction not seen preoperatively (Fig. 1A and B). This incidental finding was noted in the RA during routine inspection of the tricuspid valve with color-flow Doppler, and it was not related to any hemodynamic instability at the time of detection. Further investigation with color-flow Doppler in the midesophageal bicaval view demonstrated a high-velocity turbulent diastolic inflow jet at an area of apparent restriction. Diagnoses of tricuspid regurgitation, patent foramen ovale, atrial septal defect, and coronary sinus pathology were ruled out by location of the color-flow jet and significant narrowing at the IVC-RA junction. Acquisition of a spectral Doppler pressure gradient technically was limited. However, two-dimensional examination revealed a stenotic area at the site of IVC cannulation. The diameter at the IVC-RA junction measured 7.1 mm in the midesophageal bicaval view, which was decreased significantly compared with the preoperative value of 15 mm.

Invasive pressure measurements were obtained by withdrawing the pulmonary artery catheter into the RA and redirecting it down the IVC under direct TEE guidance. A pressure gradient of 5 mmHg was measured across the IVC-RA junction. This finding was confirmed by transducing a central venous pressure (CVP) both from the pulmonary artery catheter and from the side port of the right internal jugular venous introducer. Based on the abnormal Doppler finding, apparent two-dimensional pathology at the RA-IVC junction, and pressure discrepancy, the decision was made to attempt surgical correction.

The sternum was reopened, and external adjustment of the IVC cannulation site was attempted without resolution of the problem. In preparation for bypass, an SVC and a femoral venous drainage cannula were placed. Atriotomy at the site of previous IVC cannulation was performed, and direct surgical visualization confirmed a very large "sail-like eustachian valve" drawn across the

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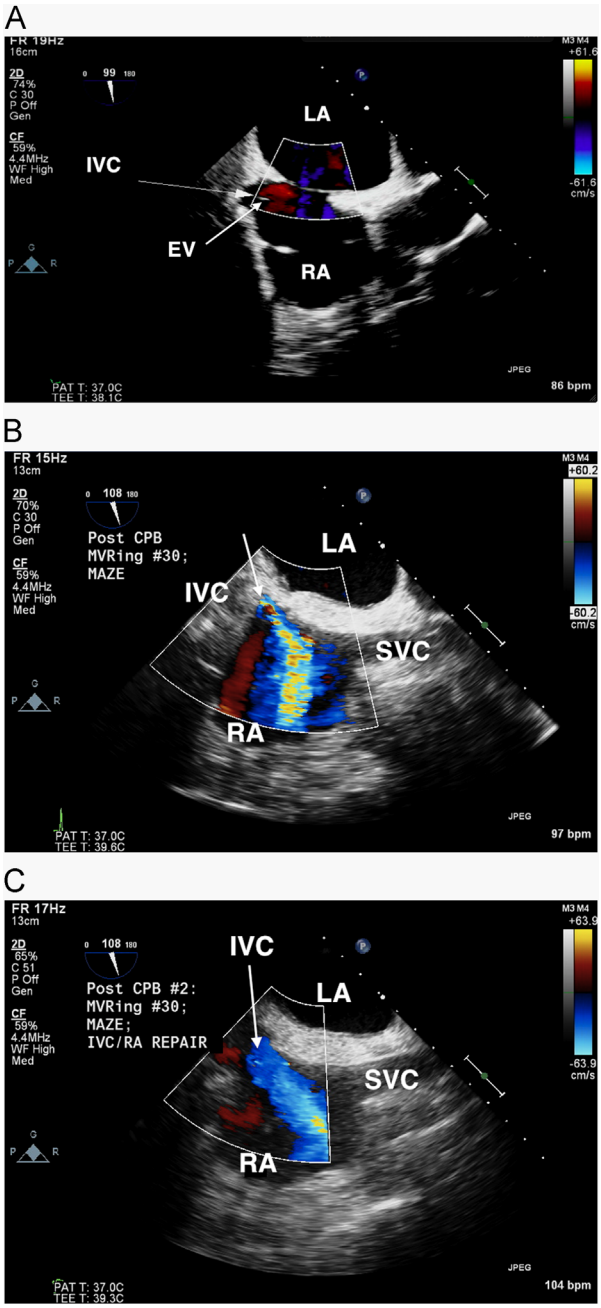

Fig 1. TEE in the midesophageal bicaval view of the IVC-RA junction. (A) The IVC-RA junction before bypass measured 15 mm and demonstrated laminar flow. The eustachian valve (EV) is also seen. LA, left atrium. (B) Color-flow Doppler imaging after the first bypass demonstrates aliasing and turbulence of the venous return (arrow) from the IVC into the RA that is suggestive of restriction. Measurement of the IVC-RA junction confirmed narrowing at 7.1 mm (not shown). (C) Color-flow Doppler imaging after release of the entrapped eustachian valve shows disappearance of the high-velocity inflow jet from the IVC. Measurement confirmed an increased diameter at the IVC-RA junction of 14.7 mm (not shown).

IVC orifice by a cannulation suture. The IVC cannulation suture and eustachian valve were resected, and the previous cannulation and atriotomy sites were closed. The patient was weaned from extracorporeal support, and TEE documented resolution of the stenosis. Additionally, the diameter of the IVC-RA orifice measured 14.7 mm after the repair (Fig 1C), and transduction of the right femoral venous and SVC cannulae confirmed absence of a pressure gradient. The second surgical procedure required an additional 19 minutes of cardiopulmonary bypass time. The patient's subsequent postoperative course was unremarkable, and he was discharged on postoperative day 7.

DISCUSSION

Previously published cases of acute IVC stenosis in the setting of bicaval heart transplantation, orthotopic liver transplantation, mitral valve replacement, and extrinsic compression from implantation of a Nuss bar for repair of pectum excavatum reported delayed postoperative diagnosis after significant signs and symptoms became manifest.[1,9-11] Acute occlusion of the IVC causes decreases CVP and RA filling pressures, an increase in IVC pressure, transudation of retroperitoneal fluid, hypovolemia from fluid shifts, reduction in cardiac output, congestive nephropathy resulting in oliguric renal failure, congestive hepatic dysfunction, anemia, and thrombocytopenia related to splenic sequestration.[4-8] Postoperative physical findings of profound lower extremity edema coupled with ascites, hemodynamic instability, and hepatic and renal dysfunction suggested the differential diagnosis of right ventricular dysfunction and IVC pathology. Chronic obstruction may cause lower extremity pain and swelling, back pain, weakness, and venous stasis ulceration.[12] Chronic obstruction lasting 7 to 14 days can result in collateral circulation via the vertebral veins, ascending lumbar veins, renal veins, azygos vein, hemiazygous vein, and intercostal veins.[5,6] Progression of systemic disease depends on the severity of stenosis (Table 1). Therapeutic interventions for suprahepatic chronic IVC stenosis, as in the case of Budd-Chiari syndrome or anastomotic stenosis after orthotopic liver transplantation, include

Table 1. Signs and Symptoms of IVC Stenosis

Acute
Decreased CVP, RAP
Decreased cardiac output
Chronic
Hepatomegaly
Splenomegaly
Ascites
Lower extremity edema and pain
Back pain
Venous thrombosis
Portal hypertension
Right upper quadrant pain
Jaundice
Hepatic failure
Encephalopathy
Hematemesis
Engorgement of chest and abdominal wall veins
Congestive nephropathy (oliguric renal failure)
Anemia
Thrombocytopenia

Abbreviations: IVC, inferior vena cava; CVP, central venous pressure; RAP, right atrial pressure.