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Echocardiography

(Abstracts and Case Report/Series)

Diagnosis of Left Ventricular Outflow Obstruction During a Liver Transplant

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Introduction:

Liver transplantation (LT) surgery involves significant hemodynamic fluctuations which can be challenging for the anesthesiologist. It is essential to identify the causes of hemodynamic instability in a timely manner as they can lead to acute kidney injury, graft failure and increased mortality.[1]One of the causes of hypotension can be systolic anterior motion of the mitral valve (SAM) causing Left ventricular outflow obstruction (LVOTO). This Inducible LVOTO can often be diagnosed during dobutamine stress echocardiography (DSE) for preoperative assessment of patients with end stage Liver disease (ESLD) with a reported incidence of (24.3%).[2] Development of LVOTO perioperatively compromises stroke volume, increases left ventricular end diastolic pressure, causes mitral regurgitation, culminating in hypotension.[2] Intraoperative Transesophageal echocardiography (TEE) during LT can help identify such causes of hemodynamic instability and guide its management. We report a patient who developed refractory hypotension secondary to SAM and LVOTO during LT.

Case Presentation:

A 63-year-old male with ESLD secondary to alcoholic cirrhosis and a sodium–MELD of 20 presented for an urgent LT. He had portal hypertension, refractory ascites, hepatic encephalopathy and Hepatocellular carcinoma. His preoperative Transthoracic Echocardiography (TTE) showed hyperdynamic left ventricular function with an ejection fraction of 70% and mild Mitral Regurgitation. The patient was taken to the operating room and a baseline Transesophageal Echocardiography (TEE) was performed after induction of General Anesthesia which concurred with preoperative TTE findings. Through the dissection phase he had massive bleeding but responded to blood products, albumin, crystalloids and a modest dose of norepinephrine and vasopressin. As soon as the Inferior vena cava was clamped, there was sudden, severe hypotension and a significant drop in the cardiac index without much response to very high doses of vasopressors and intravenous fluids. The TEE exam showed SAM with LVOTO and moderate mitral regurgitation. We then gave 40 mg of esmolol in boluses followed by an infusion at 50 micrograms/kg/minute. SAM and LVOTO resolved with this and we weaned off the esmolol. We encountered significant reperfusion syndrome with refractory hypotension. TEE again demonstrated SAM and we restarted the esmolol infusion, which again cured the LVOTO. Patient was transferred to the intensive care unit (ICU) on vasopressors along with esmolol infusion, all were discontinued a few hours later. He was hemodynamically stable for the rest of his ICU stay and was discharged home on day 5 postoperatively. His postoperative TTE did not redemonstrate SAM and LVOTO.

Conclusion:

Though classically described in patients with hypertrophied cardiomyopathy, LVOTO is also reported in other conditions.[3] LVOTO is unpredictable due to a complex interplay between preload, afterload, and heart rate in susceptible patients.[3] LVOTO during LT increases the incidence of perioperative cardiac arrest and requires significantly higher doses of vasopressors and fluids.[2] A rescue TEE can diagnose five prespecified causes of

hypotension including SAM/ LVOTO in 92% LT study patients.[4] Our case confirms that TEE during LT is an invaluable tool in prompt diagnosis of hypotension and LVOTO in a structurally normal heart during precipitating conditions and guides its immediate management.

References:

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Figure 1:

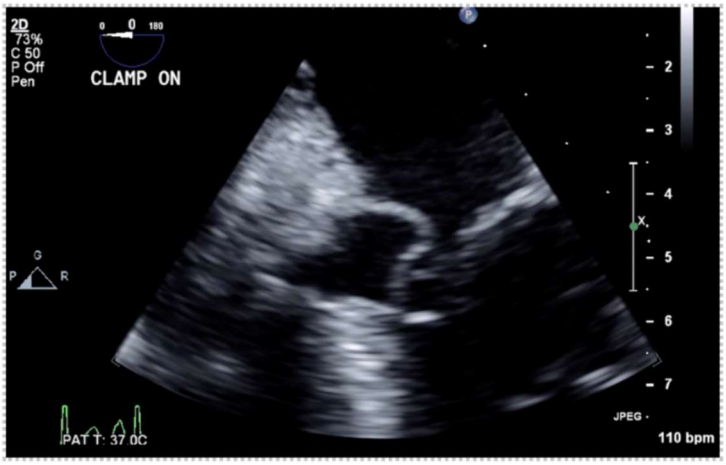

FIGURE 1: Mid esophageal 5 chamber view showing SAM/LVOTO during the Anhepatic phase of a Liver Transplant.