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Opinion
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Dynamic LVOT Finding in Patients with End Stage Liver Disease Candidates to Liver Transplantation Perioperative Assessment

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Opinion

Patients with End-Stage Liver Disease (ESLD) frequently present significant changes in hemodynamic parameters. These patients often, precisely in correlation with the liver disease, develop a circulatory hyper dynamic state that is characterized by: high Cardiac Output (CO) and a low Systemic Vascular Resistance (SVR) in association with cirrhosis cardiomyopathy, a complex medical condition characterized by increased cardiac output and a compromised ventricular response to stress. This cardiomyopathy is associated with other myocardial dysfunctions: Cardiac chamber enlargement, left ventricular hypertrophy, diastolic dysfunction, ventricular arrhythmias and QT prolongation [1,2]. Cardiovascular complications are a major cause of morbidity and mortality in patients with End-Stage Liver Disease (ESLD) undergoing liver transplantation. Identifying candidates at the highest risk of postoperative cardiovascular complications is the cornerstone for optimizing the outcome. The cirrhotic patient present increased arterial compliance with associated functional hypovolemia despite a volume overload in absolute terms. Splanchnic arterial vasodilatation unloads the ventricle and may mask the presence of ventricular insufficiency, with a great contribution by autonomic dysfunction and impaired volume and baroreceptor reflex. In fact, echocardiography, mandatory for all patients evaluated for liver transplantation, often show normal function, but in case of physiologic or pharmacological stress the cirrhotic cardiomyopathy it could be highlighted [3,4]. All patients who are candidates to liver transplantation should undergo a careful history, focused on the identification of cardiovascular risk factors, a complete physical examination, and a set of common blood tests. In the 1960s and 1970s, it was thought that patients with severe liver disease had a low incidence of CAD, based on a lower incidence of hypercholesterolemia, increased levels of circulating estrogen and decreased SVR thereby eliminating, at least in theory, hypertension as risks factors for CAD. However, there is increasing evidence that the prevalence of CAD in patients with ELSD is higher than previously thought and maybe even higher than in the general population (2.5% to 27%). Therefore, much emphasis is given to the diagnosis of CAD as the liver transplantation procedure creates a stress for the heart with virtually unavoidable episodes of often severe tachycardia ad hypotension. The stratification of the risk for CAD is therefore carried out through the execution of dobutamine stress test or myocardial perfusion scan. Dobutamine Stress Echocardiography (DSE) is used widely as a screening tool for cardiac risk stratification in patients with ESLD undergoing liver transplant evaluation [5,6]. While its overall sensitivity for detecting coronary artery disease is limited, the test has a good negative predictive value in this patient population, making it useful in excluding patients at risk for peri-operative cardiac events [7,8]. Dobutamine is a synthetic catecholamine with a strong affinity for β receptors. Its inotropic effect exceeds its chronotropic ability, pharmacologically, the basis of DSE is to induce a myocardial oxygen supply-demand mismatch and evaluate chronotropic competence [9]. Typically patients who develop regional wall motion abnormalities or cannot achieve a 85% or greater maximum predicted heart rate are subjected to further cardiac work up to evaluate extent of coronary artery disease, DSE is also useful in identifying the presence of Dynamic Left Ventricular Outflow Tract (DLVOT) obstruction [10]. The increase in heart rate combined with the vasodilating effects of dobutamine can unmask dynamic obstruction and increase the gradients identified by continuous-wave Doppler echocardiography across the LVOT. The observation of hypotension during DSE in the absence of regional wall motion abnormalities ultimately led to the description of DLVOT obstruction in 1992 [11]. The significance and prognostic implications of DLVOT obstruction were studied in a series of 106 consecutive patients who underwent DSE

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during the workup for Orthotropic Liver Transplantation (OLT) [12]. Patients were considered to have a significant dynamic LVOT obstruction if they had a gradient greater than 36 mmHg with no evidence of hypertrophic cardiomyopathy. Significant dynamic LVOT obstruction was identified in 46 patients (43%). None of the patients in this group experienced any postoperative mortality. Furthermore, only 4 (8%) of the 46 patients with significant DLVOT obstruction developed significant intraoperative hypotension. These findings suggest that in patients undergoing OLT, the presence of a significant DLVOT gradient is a frequent finding that does not impact postoperative mortality but is associated with a higher incidence of intraoperative hypotension. Patients with ESLD undergoing DSE are particularly susceptible to DLVOT obstruction. The pathophysiologic disease-related factors include a combination of hyper dynamic circulation, marked reduction in systemic vascular resistance, and frequent use of diuretics. Beyond the preoperative evaluation it is necessary to plan the anesthetic management of the patient with DLVOT to be subjected to liver transplantation. In most cases patients are treated with β-blockers for the management of bleeding from oesophageal varies, therefore the beta-blocker therapy will not be interrupted during surgery [13]. In cases where patients are not undergoing β-blocker therapy, it will be necessary to introduce this drug at the beginning of the intervention, favoring short half-life molecules with the possibility of continuous infusion and a rapid modulation during surgery. During the surgery there may be important hemodynamic changes caused by reduced preload due to: sudden blood loss, manual compression of the inferior vena cava, clamping of the inferior vena cava to perform the piggy back technique in order to remove the native liver. In these patients, infusion fluids should therefore be administered as first line of treatment and vasoconstrictive drugs such as phenylephrine or nor epinephrine in continuous infusion should be used [14,15]. Ephedrine or other drugs with positive chronotropic action should not be used because in patients this leads to a paradoxical reduction in blood pressure due to outflow obstruction and severe mitral regurgitation. During surgery, hepatic reperfusion represents a particular clinical situation as Post Reperfusion Syndrome (PRS) can occur. The etiology of this cardiovascular depression is multifactorial and can result from several factors after unclamping of the portal vein, including: Systemic release of the allograft preservative solution (cold and hypercalemic fluid), the release of vasoactive mediators (such as inflammatory cytokines and free radicals), and acidic blood return from the liver and mesentery [16,17]. Preventative measures primarily consist of prophylactically administering fluids and vasoactive medications such as inotropes (e.g., epinephrine), vasopressors (e.g., phenylephrine), or calcium chloride, and flushing the allograft before reperfusion to eliminate vasoactive mediators and the preservative solution.

Arterial hypotension can however also be determined by acute pulmonary embolus (from an inferior vena cava or portal vein thrombus), venous air embolism, acute right ventricular failure, Dynamic Left Ventricular Outflow Tract Obstruction (DLVOTO), and preload limitations. An increment in HR and contractility from the treatment of the PRS with inotropes, compounded by the vasodilator state of reperfusion, and the relative hypovolemia (due to the allograft uptake of blood), may collectively precipitate the development DLVOTO35, 37If an expedient diagnosis of DLVOTO is not made, circulatory collapse may develop [18,19]. Thus, a critical diagnostic dilemma exists for the transplant anesthesiologist. This is especially true if intraoperative TEE monitoring is not utilized. Therefore during a liver transplant in which the LVOT presents is known or in case of suspicion following refractory hypotension it is necessary to add to the cardiovascular monitoring with Swan Ganz catheter, transesophageal monitoring. Based on the TEE findings, DLVOTO was diagnosed and allows formulating an intervention strategy that includes discontinuation of inotropes, administration of esmolol and phenylephrine, augmentation of preload through fluid challenge [20-25]. The drug of choice for this is phenylephrine because of its selectivity for -adrenergic receptors. Phenylephrine may also cause a reflex bradycardia and allow for a longer time in diastole to allow for improved filling. Adrenergic receptor blockers and calcium channel blockers have proven to be efficacious in the treatment of hypertrophic cardiomyopathy, and their efficacy is also valid in the management of DLVOTO without septal asymmetry. They act to improve the obstruction by decreasing both the chronotropic and inotropic state of the left ventricle [26-38]. Additionally, the discontinuation of epinephrine allowed for the heart rate to decrease to a rate that promoted diastolic filling and relief of SAM DLVOTO should be considered in the differential diagnosis of refractory hemodynamic instability during OLT based on the predilection of these patients to this condition [39-43].

Conclusion

In summary, the occurrence of DLVOT obstruction during dobutamine stress echocardiography is an expected finding in patients with ESLD. Therefore it is important to evaluate the patient together with the cardiologist because these patients do not require further clinical and instrumental studies prior to OLT listing and the knowledge that liver transplantation remains the only definitive treatment option. Patients safely can be listed for liver transplantation with the aforementioned precautions taken during their perioperative anesthetic management. The prevalence of latent LVOTO was 43%, significantly higher than the 12% to 21% prevalence previously reported in 5 non-cirrhotic populations. Therefore the knowledge of the possible onset of an outflow obstruction in determined clinical conditions during liver transplantation must lead the anesthesiologist to optimize the pharmacological strategy with the support where the clinical picture is complex of the transesophageal echography that allows discriminating between alterations of the volemic filling and heart disease. For this TEE played an invaluable role in the diagnosis and management of refractory hemodynamic instability in this patient.

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