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Hemodynamic TEE Guides Management in a Septic Patient

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Abstract

This case report describes how hTEE™, hemodynamic management guided by a miniaturized, disposable transesophageal ultrasound probe (ClariTEE®, ImaCor Inc., Garden City, NY), was used on a 57-year-old male with a history of liver and rectal cancer who underwent a colostomy and ileostomy. The patient became septic and hemodynamically unstable despite high-dose pressors three days post-op. Since the patient had a normal ejection fraction (59%) pre-op, hypovolemia was postulated as the cause for instability and aggressive fluid resuscitation was begun. hTEE further revealed moderately reduced LV and RV contractility, leading to a change in medical management: weaning from pressors and administering inotropes (dobutamine). Hemodynamics improved and a second imaging session 4.5 hours later revealed improved RV and LV contractibility. It is well known that sepsis can cause both RV and LV dysfunction; the present case demonstrated how hTEE can help detect RV and LV dysfunction and guide its management.

Case Report

A 57-year-old male with a history of liver and rectal cancer underwent a colostomy and ileostomy. Three days post-surgery, the patient appeared hemodynamically unstable on 25 mcg/min of levophed (BP 86/56, CVP 10 and HR 125 in sinus tachycardia). Since a pre-operative study had reported an ejection fraction of 59%, hypovolemia was postulated to be the cause of the patient's hemodynamic instability. Aggressive fluid resuscitation was begun, with a total of 1500 mL of lactated Ringer's solution and 500 mL of albumin.

In addition, at this time, a ClariTEE probe was inserted to directly visualize cardiac filling and function with the two standard monitoring views, the four-chamber mid-esophageal view and the trans-gastric short axis view (TGSAV). The four-chamber mid-esophageal view revealed mild to moderately reduced LV and RV contractility, normal LV size, and a dilated RV. The TGSAV confirmed reduced LV and RV contractility and increased RV size. The physician then weaned pressors and added dobutamine at 5 mcg/kg/min to address reduced contractility. A follow-up imaging session was scheduled.

The follow-up imaging session 4.5 hours later revealed improved LV and RV contractility. In addition, hemodynamics improved (BP now 105/70, CVP nearly unchanged at 11, and HR 120 in continued sinus tachycardia).

Discussion

Cardiac dysfunction in septic shock is now well known; see Vieillard-Baron's recent review article [1] for an excellent overview of our growing understanding of this phenomenon. We quote from[1] and include its first five references: "Reversible myocardial depression in patients with septic shock was first described in 1984 by Parker et al. using radionuclide cineangiography[2]. In a series of 20 patients, they reported a 65% incidence of left ventricular (LV) systolic dysfunction, defined by an ejection fraction <45%[2]. In 1990, using transthoracic echocardiography, Jardin et al. reported the same results[3]. In a canine model simulating human septic shock, Natanson et al. demonstrated that intrinsic LV performance was actually depressed in all animals and not corrected by volume expansion[4]. Finally, more recently, Barraud et al. confirmed the presence of severe depressed intrinsic LV contractility using LV pressure/volume loops in lipopolysaccharide-treated rabbits[5]. All of these studies, and many others not cited in this introduction, demonstrate the reality of the impairment of intrinsic LV contractility in septic shock. For many years, septic cardiac dysfunction was largely underestimated because the hemodynamic device used, i.e. the pulmonary artery catheter, was not appropriate for establishing such a diagnosis. Development of new hemodynamic tools at the bedside, such as echocardiography, allowed better characterization of the septic cardiomyopathy[6]." A recent study by Etchecopar-Chevreuil et al. further demonstrated the potential of TEE to reveal "Cardiac morphological and functional changes during early septic shock"[7].

The present case further illustrates the importance of diagnosing cardiac dysfunction, in this instance reduced LV and RV contractility, in management of patients in septic shock. The present patient's normal pre-op EF of 59% suggested that hemodynamic instability, despite high-dose pressors, would be best addressed with aggressive fluid resuscitation. However, direct visualization of LV and RV depression led the physician to consider an alternative possible cause for hemodynamic instability, namely inadequate contractility, which might be addressed by dobutamine infusion. Improved hemodynamics following this infusion confirmed this diagnosis. Note that the presence of "persistent preload defect in severe sepsis despite fluid loading" had been previously reported[8].

In summary, hTEE revealed the underlying cause of this patient's hemodynamic instability and helped guide a successful change in management. Additional fluid administration and changing from pressors to inotropes led to improved LV and RV function in this complicated septic patient.

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