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Hemodynamic Management of a Sepsis Patient using a Miniaturized TEE Probe (hTEE)*

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Introduction

The surviving sepsis guidelines suggest an initial resuscitation to the following targets: CVP 8-12 mmHg, the use of vasopressors for hypotension (defined as MAP < 65 mmHg), urine output of > 0.5 mL/kg/h, and central venous saturation (ScvO2) > 70% or mixed venous saturation (SvO2) > 65%. If the latter goal is not met initially, additional interventions including additional fluid, transfusion to a hematocrit > 30%, and/or the addition of an inotrope may be considered[1].

Cardiac dysfunction in septic shock is now well known; see Vieillard-Baron's recent review article[2] and references therein[3-5] for an excellent overview. Here is a brief quotation from[2]: "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]." Standard pressure measurements (CVP and PAP) are known to be poor predictors of volume responsiveness[7] since they can be influenced by venous and ventricular compliance, RV dysfunction and valvular pathology. The presence of "persistent preload defect in severe sepsis despite fluid loading"[2] had been previously reported[8]. 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"[9]. RV dysfunction occurs in 18-30% of septic patients[10-12] and LV dysfunction in 31-33% of patients[10]. RV dysfunction is also associated with a higher mortality[12]. We have previously used a miniaturized disposable monoplane transesophageal echocardiography (TEE) probe (the ImaCor ClariTEE®) to perform hemodynamic assessment and monitoring (hTEE™) in our CVICU[13-14].

Here we present a case that demonstrates the utility of hTEE to guide resuscitation in a patient with severe sepsis.

Case Presentation

A 48-year-old gentleman with a 50-pack/year history of tobacco abuse and COPD presents with new onset chest pain. Cardiac catheterization revealed multi-vessel coronary artery disease (CAD): 95% right coronary artery, 95% left anterior descending (LAD, 90% diagonal, 50% circumflex, and 80% obtuse marginal (OM) stenoses. He underwent an uneventful three-vessel off-pump coronary artery bypass grafting on hospital day (HD) #2.

He had a normal ejection fraction (EF) on intraoperative transesophageal echocardiography (TEE). Postoperatively, he received DT prophylaxis per CIWA protocol, but otherwise his CVICU course was uneventful and he was transferred to the floor in stable condition on postoperative day (POD) #1.

On POD #2, he required transfer back to the CVICU with apparent sepsis manifested by hypoxia with an oxygen saturation of 76%, hypotension with a systolic blood pressure (SBP) in the 90s mmHg, and oliguria with a urine output (UOP) of only 200 mL recorded for a 24-hour period. He continued to deteriorate despite early cultures and initiation of broad-spectrum antibiotics. The patient required endotracheal intubation and bi-level mechanical ventilation for support as he progressed to acute respiratory distress syndrome. Invasive hemodynamic monitoring with an arterial line and central venous line (CVL) was initiated, as the patient required substantial, 5/liter fluid resuscitation and multiple vasopressors. Initially the patient was on norepinephrine, but as the dose was escalated > 10 mcg/min, vasopressin 0.04 units/min was added as per our practice protocol. His initial ScvO2 was 47%, his base deficit went as high as -4.1, and lactate as high as 2.5.

To guide resuscitation and monitor cardiac function, a ClariTEE probe was placed at the bedside while a Dobhoff feeding tube was still in. Hemodynamic TEE in this patient demonstrated new RV dysfunction with reduced TAPSE and poor wall thickening toward the apex, felt to be secondary to sepsis. Low dose epinephrine at 2 mcg/min was chosen in the setting of hypotension for inotropic and RV support. The patient was found to have become relatively adrenally insufficient after serial assessments of random serum cortisol levels, so stress dose steroids (hydrocortisone 100 mg q8h) were also added. Over the next 24 hours the patient's hemodynamics improved dramatically and he was weaned off all pressors except low dose epinephrine, which was also stopped a few days later. The patient's ventilator was weaned over the next several days as the patient underwent diuresis and clinical improvement ensued.

Discussion

RV dysfunction in the setting of sepsis may be underappreciated. The current case demonstrates an acute change in a patient's cardiac function over the course of 48 hours that would not have been appreciated if the patient had been monitored with

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a standard central venous line or even pulmonary artery catheter because a rise in filling pressures would reflect an overloaded and poorly functioning RV rather than the adequately filled RV and LV that one would hope for. The information gained from direct assessment of RV function in this case changed management for this individual patient by the addition of epinephrine for RV inotropic support and contributed to a good outcome. Although this is a single case presentation, it demonstrates the dynamic nature of hemodynamic instability in sepsis and the potential of hTEE to guide its management.

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