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Echocardiography in Hemodynamic Monitoring

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Echocardiography in Hemodynamic Monitoring

To the Editor:

Although interest in ultrasound-based techniques has emerged during the last few years, echocardiography still remains largely not used, or under used, in many ICUs. Basic echocardiography can indeed identify critical echocardiographic patterns in critically ill patients.[1] Early diagnoses of acute pulmonary embolism, myocardial infarction with accompanying complications (cardiac tamponade, wall rupture, valvular regurgitation), or aortic injuries are possible examples. In patients with shock, echocardiography should then be used to guide therapy. Evaluating cardiac output, assessing preload responsiveness and right and left ventricular systolic functions, and detecting a patent foramen ovale should be considered as the standard of care in patients with shock.

Because echocardiography is available at bedside, this easy-to-use, easy-to-learn, noninvasive instrument offers a quick and timely assessment of hemodynamic status.[2,3] Moreover, follow-up measures can be obtained after any intervention (eg, fluids, vasoactive drugs, or thrombolysis).

For example, in our ICU, we recently had a case that perfectly illustrates the value of bedside echocardiography in the management of patients with shock. A 59-year-old woman with a medical history of multiple sclerosis with paraplegia and depression was admitted in a psychiatric center after trying to commit suicide. Five days after admission, fever led to the diagnosis of urinary tract infection with left-sided back pain, a high leukocyte count, and *Escherichia coli* in a urine culture. She was then transferred to the ICU with a diagnosis of shock (eg, marbling, mean arterial pressure of 44 mm Hg, heart rate of 150/min). Multiple organ failure rapidly developed, with the need for mechanical ventilation, fluid loading, norepinephrine infusion (1.5μ/kg/m), and renal replacement therapy. Laboratory examination showed a high leukocyte count (21,000/mm^3), lactic acidosis (pH 7.10, arterial lactate 6.4 mmol/L), acute renal failure (anuria), and mild hypoxemia ($Pao_2/Fio_2 = 300$). Hepatic and pancreatic tests were normal. An abdominal CT scan showed diffuse colitis with no sign of perforation and lack of kidney abnormality. The diagnosis of septic shock was suggested, and wide spectrum antibiotics were initiated. Upon hemodynamic optimization, transesophageal echocardiography was performed to assess cardiac function because of the patient's unstable state, with persistent lactacidemia despite fluids and vasopressors. An unexpected image of acute core pulmonale was found, with right ventricular dilation, a paradoxical interventricular septal motion pattern, and no respiratory variation of the superior vena cava diameter with a massive mobile thrombus in the right atrium (Fig 1 and online video supplement), confirming the diagnosis of massive pulmonary embolism. Extension of the thrombus to the right pulmonary artery was seen. Thrombolysis was subsequently implemented, leading to a decrease in vasopressors and the disappearance of thrombus from the right atrium on the follow-up by echocardiography.

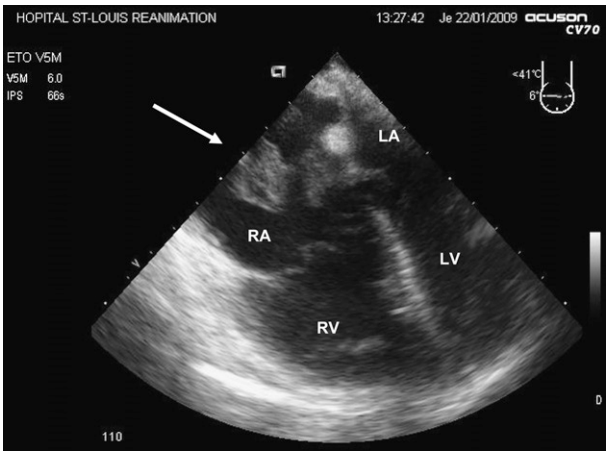

FIGURE 1. Four cavities view with transesophageal echocardiography showing a massive thrombus in the right atrium (arrow). RA = right atrium; RV = right ventricle; LA = left atrium; LV = left ventricle. See online video supplement.

This case report illustrates why echocardiography should be considered as a major tool in the early hemodynamic assessment of patients with shock. Along this line, basic and advanced training programs for ICU clinicians to provide skills, master competence, and develop professionalism in echocardiography should be encouraged.[4]

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The Role of Aldosterone in Pulmonary Venous Hypertension

To the Editor:

We read with interest the recent article in *CHEST* (July 2009) by Robbins et al[1] demonstrating an association between the metabolic syndrome and the existence of pulmonary venous hypertension. Recent research has also demonstrated an association between serum aldosterone, a known cause of left ventricular dysfunction and remodeling, and the metabolic syndrome.[2-4] Thus, it is possible that increased circulating aldosterone might be independently associated with the risk for pulmonary venous hypertension. Do the authors have data examining aldosterone or renin activity in their subjects?

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Response

To the Editor:

We thank Drs Farber, Walkey, and Alikhan for bringing to our attention the importance of aldosterone in the development of left ventricular (LV) diastolic dysfunction. The studies noted in their letter highlight the importance of metabolic derangements in the development of LV diastolic dysfunction, which can occur even in the absence of systemic hypertension or LV hypertrophy.[1-3] We did not measure aldosterone levels in our study[4] but plan to do so in future studies.

There is increasing evidence that features of the metabolic syndrome are likely to contribute to the development of pulmonary hypertension in susceptible patients.[5] This is an area that requires further investigation, and we were intrigued by the recent publications of Dr Farber and his colleagues about the potential role of adiponectin deficiency in the development of pulmonary hypertension.[6]

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A Comparative Study of Two Different Metered-Dose Inhaler-Valved Holding Chambers in the Administration of Salbutamol

To the Editor:

Rapid-acting, inhaled β-2 agonists are frequently delivered from a pressurized, metered-dose inhaler (MDI) used with a valved holding chamber (VHC). Several studies have shown that VHCs enhance the efficacy of short-acting β-2 agonists in patients who have poor MDI technique and in children. However, different VHCs are available for inhaled therapy without information in the summaries of product characteristics and patient information leaflets on studies of efficacy and compatibility between drugs and