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Echocardiography

Pathophysiology of Dynamic Left Ventricular Outflow Tract Obstruction in a Critically Ill Patient

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Left ventricular outflow tract obstruction is not a rare problem in the intensive care units and can precipitate hemodynamic shock unresponsive to catecholamine therapy. The use of echocardiographic examination is extremely important in recognizing this phenomenon and its underlying conditions, finally identifying the most appropriate therapeutic strategy. The simple correction of one or more of these factors can dramatically change patients clinical outcome. We report the clinical case of a 72-year-old man who developed hemodynamic shock in the intensive care unit. Hypovolemia, catecholamine infusion, and mechanical ventilation induced geometric modification of the left ventricle causing a systolic anterior motion of the mitral anterior leaflet and a severe subaortic gradient. Simple restoration of fluids and discontinuation of medical therapy dramatically changed the outcome of the patient. A review of the medical literature has been carried out to deeply investigate pathophysiology of left ventricular outflow tract obstruction in critically ill patients. (Echocardiography 2010;27:E122-E124)

Key words: subaortic gradient, outflow obstruction, mechanical ventilation, hypovolemia

A 72-year-old white man, with a history of chronic obstructive lung disease, was admitted to the emergency department for severe dyspnea, cough, and weakness for a few days. Twelve-lead electrocardiogram showed sinus tachycardia, 115 beats per minute (bpm), without other abnormalities. At clinical examination, blood pressure was 90/50 mmHg, chest mid-basal rales and a 3/6 systolic murmur over the left sternal border were present. Chest x-ray showed right basal pneumonia, congestion of lung bases, normal heart shadow, and a mild dilation of the pulmonary arteries. Blood cell count showed a significant increase in white blood cells (18.5×10^9/L); therefore empirical antibiotic therapy was administered. A bedside transthoracic echocardiographic examination (which was hampered by a very high acoustic impedance) showed a preserved left ventricular function and the absence of significant valvular lesions.

After few hours, due to respiratory failure (blood gases were: pH, 7.21; PaO_2, 56 mmHg; $PaCO_2$, 75 mmHg; base deficit, 3) patient was intubated and assisted with mechanical ventilation. Moreover, despite a positive fluid balance for several hours (100 mL/h), patient developed persisting anuria (serum creatinine was 3.5 mg/dl); hence, intravenous infusion of dopamine (8 mcg/Kg/min) and furosemide (1 mg/h) was initiated. However, soon after medical intervention, a severe hemodynamic deterioration occurred; blood pressure fell to 65/30 mmHg and invasive monitoring showed a marked systolic pressure variation (SPV) ranging from 75 mmHg during inspiration to 55 mmHg during expiration with a gap of 20 mmHg. (Fig. 1) Transesophageal echocardiography (TEE) showed a hyperkinetic left ventricle with mild concentric hypertrophy and a septal wall thickness of 12 mm; end-diastolic diameter was additionally significantly reduced (27 mm), with an increase of wall to cavity ratio. Systolic anterior motion (SAM) of the anterior mitral leaflet causing a significant left ventricular outflow tract obstruction (LVOTO), with a peak gradient of 100 mmHg, was detected. (Fig. 2 and movie clip 1) After discontinuance of dopamine and furosemide infusion, 1.5 L of colloid solution were restored in 30 minutes resulting in a significant reduction of the outflow gradient (from 100 mmHg to 37 mmHg) and a mild increase in blood pressure (90/40 mmHg). The following day, after a positive fluid balance of 3.5 L, due to improved hemodynamic conditions and blood gas analysis, patient was extubated and a medical therapy with atenolol 50 mg q.d. was initiated. Renal function

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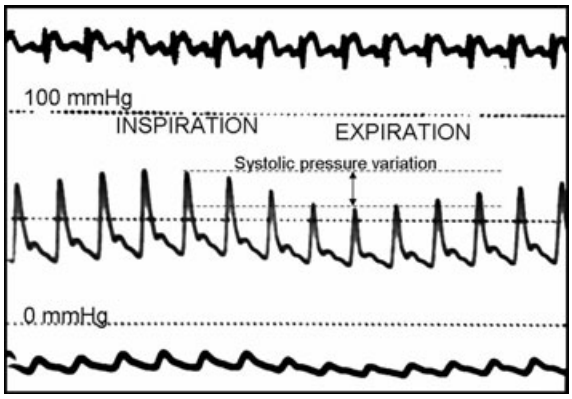

Figure 1. Invasive blood pressure monitoring of our patient in intensive care unit. Systolic blood pressure ranged from 75 mmHg to 55 mmHg with a gap of 20 mmHg. Mechanical ventilation induced modification of left ventricular preload enhanced the left ventricular outflow tract obstruction.

was also restored after few days with a significant reduction in serum creatinine levels. Serial chest x-rays also demonstrated a progressive resolution of the right basal pneumonia. On day 10th after admission, a cardiac magnetic resonance excluded hypertrophic cardiomyopathy (HCM) and showed a structurally normal left ventricle with a mass of 104 g, end-diastolic volume 97 mL and a normal left ventricular outflow tract diameter (21 mm) (Fig. 3).

Discussion:

Dynamic LVOTO may occur in some non-HCM patients and is not a rare problem in the intensive care units.[1] Left ventricular hypertrophy due to hypertension, mitral valve repair, previous aortic valve replacement, abnormalities of the mitral subvalvular apparatus, sigmoid septum and a steep aortic root angle have been recognized as the anatomic substrate in many patients.[2–4] However, these conditions are usually not per se sufficient to determine a subaortic gradient and therefore, LVOTO is the consequence of one or more precipitating factors.

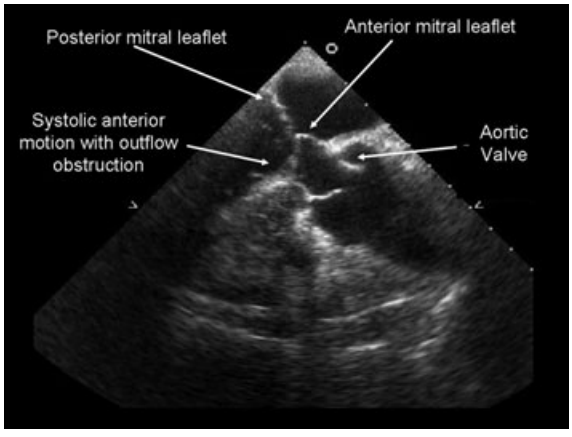

Figure 2. Transesophageal examination performed during hemodynamic shock. Due to hypovolemic conditions and medical therapy, patient had a small and hyperkinetic left ventricular chamber with systolic anterior motion of the anterior mitral leaflet causing subaortic gradient. The magnitude of the obstruction was higher during inspiration due to mechanical ventilation induced modification of left ventricular preload.

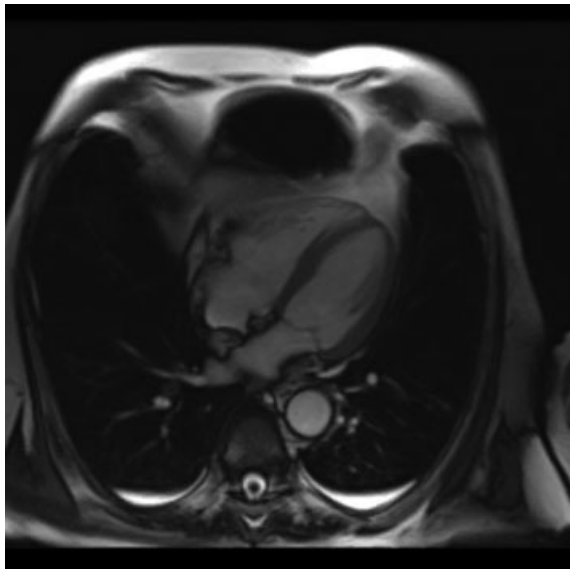

Figure 3. Cardiac magnetic resonance performed during hospitalization showed the absence of left ventricular structural abnormalities.

Drug therapies such as catecholamine infusion or diuretics, which respectively enhance the contractility of the basal segments and reduce the left ventricular cavity, emotional stress (like described in the apical ballooning syndrome), hypovolemia, dehydration, sepsis, and myocardial infarction can all play a determinant role in this setting.[1,5–7] Furthermore, absolute or relative hypovolemia leads to left ventricular under-filling, causing modification in chamber shape and the spatial relations between mitral valve, subvalvular apparatus, left ventricular septum, outflow tract, and aortic root.[1,8] Finally, one of the most interesting features of our clinical case was the role played by mechanical ventilation in the pathogenesis of LVOTO.

The blood pressure curve of a normal subject, breathing spontaneously, is characterized by a mild decrease of systolic pressure during inspiration and a mild increase during expiration. This phenomenon is related to cyclic changes in intrathoracic pressure, venous return, and pulmonary flow during breathing. These modifications are usually mild and the difference between

the maximum systolic pressure during expiration and inspiration usually does not exceed 5 mmHg.[9] A reverse of the cyclic variation of the blood pressure during mechanical ventilation has been described, with an inspiratory increase of blood pressure, followed by a decrease on expiration. This phenomenon has been described as "SPV" and its magnitude is related on the volume status of patients.[9]

During positive pressure inspiration, there is an increase in intrathoracic pressure and lung volume; due to a higher alveolar pressure, the blood in the pulmonary venous bed is squeezed out toward the left atrium, thus increasing left ventricular preload and stroke volume. At the same time, an increase in right ventricular afterload due to high alveolar pressure and a reduced vena cava flow to the right atrium occurs. Moreover, due to increased intrathoracic pressure, a decrease in left ventricular and aortic transmural pressure develops, with the consequence of a reduction of left ventricular afterload.

During expiration instead, a decrease in left ventricular preload, and consequently in stroke volume occurs; this is mainly due to a lower pulmonary venous flow toward the left atrium because of the reduced stroke volume of the right ventricle during the inspiratory phase.[10] In a patient with normal volemia, the SPV is usually less than 10 mmHg; on the contrary, in hypovolemic conditions, an exaggeration of SPV has been described.[9] To understand this phenomenon, the Frank-Starling relation can be used. Under hypovolemic condition a patient has low left atrial and ventricular filling, being positioned on the left side of the curve. In these conditions, even mild modification of left ventricular preload is responsible for a significant increase in stroke volume. In our hypovolemic patient, a significant SPV was recorded (20 mmHg). In particular, reduced pulmonary venous flow during expiratory phase caused a more significant underfilling of the left ventricle with the exaggeration of the dynamic LVOTO.

In conclusion, dynamic LVOTO is a potentially reversible cause of hemodynamic shock in critically ill patients. The use of echocardiographic examination is extremely important in recognizing this phenomenon and its underlying conditions, finally identifying the most appropriate therapeutic strategy. The simple correction of one or more of these factors can dramatically change patients' clinical outcome.

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Supporting Information

Additional Supporting Information may be found in the online version of this article:

Movie clip 1. Transesophageal examination shows a small and hyperkinetic left ventricle with mitral valve SAM causing severe LVOTO. Video clip shows an increased mitral valve SAM during the expiratory phase resulting in increased subaortic obstruction.

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