Reversible Myocardial Dysfunction in Septic Shock

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Myocardial dysfunction frequently accompanies severe sepsis and septic shock [1]. Whereas myocardial depression was previously considered a pre-terminal event, it is now clear that cardiac dysfunction, as evidenced by biventricular dilatation and reduced ejection fraction, is frequent in patients with severe sepsis and septic shock [2]. Recent definitions recognize the importance of myocardial depression and include a low cardiac index or echocardiographic evidence of cardiac dysfunction as one of the criteria for the diagnosis of severe sepsis [1]. We report the case of a 65 year old woman with extreme cardiac dysfunction following septic shock with complete reversal of the cardiac dysfunction after recovery from sepsis.

PATIENT DESCRIPTION

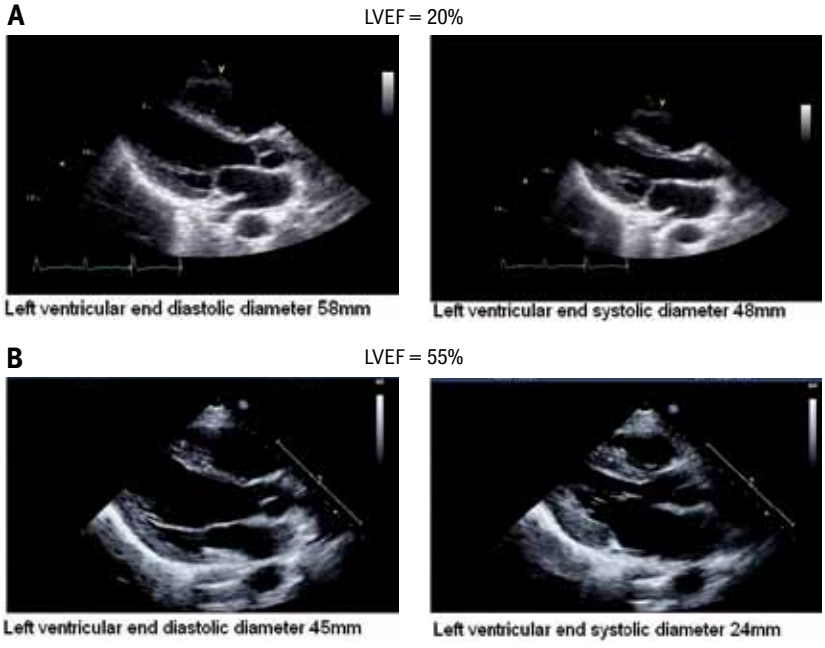
A 65 year old woman of Jewish-Indian origin, unmarried and childless, was admitted to a community general hospital with diffuse abdominal pain and diarrhea. Her past medical history included Bilroth-II resection of the stomach due to peptic disease performed 10 years before the present admission, with recurrent admissions due to abdominal pain and malnutrition. There was no history of heart disease, connective tissue disorders, hypothyroidism or thyrotoxicosis, recent viral illness, drug abuse, exposure to doxorubicin, alcohol or other cardiotoxins.

Two hours after the present admission, the systolic blood pressure dropped to 70 mmHg; the patient developed tachycardia of 130 beats/minute and became hemodynamically unstable despite volume resuscitation. Urgent laparotomy was performed because of clinical and radiological signs of ileus. Surgical abdominal exploration showed closed-loop obstruction due to adhesions with necrosis of the ileum. Resection of the ileum with terminal ilio-ascending colon anastomosis was performed.

During the operation the patient was hemodynamically stable. Two hours post-surgery increasing respiratory distress and shock developed, re-intubation was performed and the patient was admitted to an intensive care unit. Initial laboratory tests showed normal levels of hemoglobin, white blood cells, serum urea nitrogen, creatinine, electrolytes and liver enzymes. Albumin and cholesterol levels were 1.2 g/dl and 56 mg/dl, respectively. Blood cultures were negative. Electrocardiogram showed sinus rhythm of 110 beats/minute and an incomplete left bundle branch block. Chest radiograph revealed an infiltrate in the base of the right lung with no evidence of pleural effusion. Swan-Gans catheter measurements revealed a mean pulmonary capillary wedge pressure of 28 mmHg. Echocardiogram demonstrated [Figure A] mild dilatation of the left ventricle, severe reduction in left ventricular systolic function with global hypokinesis, estimated LV ejection fraction of 20%, mild to moderate mitral regurgitation, mild tricuspid regurgitation, and mild pulmonary hypertension with pulmonary artery pressure of 35 mmHg. Cardiac catheterization demonstrated normal coronary arteries.

LV = left ventricular

Echocardiographic image of left ventricle (parasternal long-axis view) during sepsis **[A]** and after recovery **[B]**. LVEF = left ventricular ejection fraction



Treatment in the ICU included mechanical ventilation, infusion of broad-spectrum antibiotics, fluid resuscitation and inotropic support. The next day the patient was extubated, and as the post-operative course was uneventful she was transferred one week later to the sub-acute geriatric department. There was no finding on the physical examination reflecting the severity of left ventricular dysfunction, nor was there orthopnea or peripheral edema. The jugular venous pulse was not elevated. The results of precordial palpation were normal, and there were neither a third heart sound nor systolic murmurs on the auscultation. Repeat echocardiography revealed the following findings [Figure B]: normal LV chamber size with normal systolic function and normal region wall motion, mild mitral regurgitation and trace tricuspid regurgitation. After 2 weeks of rehabilitation in the sub-acute department the patient was discharged. There were no signs of myocardial dysfunction during the 1 year follow-up.

COMMENT

A broad array of conditions associated with tissue inflammation and metabolic stress may be associated with reversible myocardial dysfunction. Such conditions include massive neurological injury (stroke and cranial trauma), severe acute respiratory failure, anaphylaxis, trauma, organ transplantation, severe pancreatitis, post-cardiac arrest, and a variety of other severe illnesses. Myocardial depression is less obvious to the clinician than the decrease in vascular tone [2]. The most frequent cardiovascular abnormality in sepsis is vascular dysfunction, which induces hypotension, compromises tissue perfusion and is responsible for most sepsis-related patient deaths. Myocardial dysfunction may occur in up to 64% of cases of sepsis or septic shock [3]. Human septic myocardial depression is characterized by reversible biventricular dilatation, decreased ejection fraction, and decreased response to fluid resuscitation and catecholamine stimulation in the presence of overall hyperdynamic circulation. Myocardial dysfunction is considered to be associated with poor outcome. Previously it was found that patients with myocardial dysfunction have significantly higher mortality compared to septic patients without cardiovascular impairment [2]. In a prospective study of 106 patients with severe sepsis and septic shock, myocardial dysfunction was not associated with increased 30 day or 1 year mortality [3].

Some mechanisms are suggested for myocardial dysfunction in sepsis. An infectious stimulus (e.g., endotoxin or another microbiological element) induces the release of local and systemic inflammatory mediators, especially the cytokines tumor necrosis factor-alpha and interleukin-1β, from monocytes/macrophages and other cells. These cytokines stimulate polymorphonuclear leukocytes, macrophages and endothelial cells to release a number of downstream inflammatory mediators, including platelet activating factor and nitric oxide, further amplifying the inflammatory response [3]. Several anti-inflammatory mediators are also released as part of this amplification cascade, namely IL-10, transforming growth factor-beta and IL-1 receptor antagonist.

The relative contribution of these cytokines determines the severity of the septic episode. If the inflammatory reaction is particularly intense, homeostasis of the cardiovascular system will be disrupted, leading to septic shock. Attempts to block individual cytokines such as TNFα and IL-1β, as well as attempts to block nitric oxide production have not been shown to be effective [2].

The patient's blood cultures were negative. However, most intraabdominal infections are due to inflammation of the intestinal wall. The microorganisms that cause these infections come from the gastrointestinal flora, which cause polymicrobial infections mixed with a predominance of anaerobic bacteria.

Low serum albumin is an inflammatory and biochemical marker of poor survival in sepsis-induced myocardial depression. Hypoalbuminemia, which seems to reflect a combination of low nutritional status and complex disease process (expressed as a negative acute-phase protein) [4], may contribute to the extreme myocardial depression and the dramatic clinical deterioration in our patient.

The course of the disease in our patient progressed according to that described in other studies. Reduction in ejection fraction occurs within 24 hours after the onset of sepsis and involves both right and left ventricles. In patients who survive, myocardial function recovers within 7 to 10 days after the onset of sepsis [5].

In summary, our case presentation displays severe reversible myocardial dysfunction with cardiogenic shock in a 65 year old woman with septic shock. After recovery from sepsis all the cardiac manifestations completely resolved. While the current therapy in patients with septic shock is still directed towards reestablishing organ and tissue perfusion and oxygen delivery by fluid resuscitation and inotropic support, ongoing research aimed at understanding the cellular mechanisms of cardiac dysfunction may lead to new therapeutic options.

ICU = intensive care unit

IL = interleukin
TNFα = tumor necrosis factor-alpha

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