Heart failure and acute pulmonary edema linked to sepsis: a case report and a short review of literature

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Summary. Heart failure is a most dangerous and insidious complication of the septic state. We describe the case of a patient admitted to our department for weakness of all four limbs and fever. During hospitalization the patient developed a septic state that has manifested itself through the development of heart failure with acute pulmonary edema. (www.actabiomedica.it)

Key words: sepsis, heart, acute pulmonary edema, fever, dyspnea

Case report

The case

We describe the case of 54 years old woman admitted to our department for weakness of all four limbs and fever. During hospitalization to investigate the genesis of fever was required the urine culture resulting positive for Enterococcus Faecium and Klebsiella Pneumoniae carbapenemase (KPC). The antibiotic therapy was started without an efficient fever control. After some days the lady developed improvise dyspnea. Systolic blood pressure was 90 mmHg with diastolic blood pressure undetectable, oxygen saturation was about 70%, heart rate was about of 100 bpm. Troponinemia and CK-MB serum concentration were only little increasing (Troponinemia 0.59 ng/ml; CK-MB 8.2 ng/ml). A thorax X-ray shows evident signs of acute pulmonary edema and an echocardiographic exam shows a not previously present global depression of the left ventricle (ejection fraction about 20%). Electrocardiogram was substantially comparable with the previous and doesn’t shows ischemic signs.

The patient was therefore transferred in intensive care Unit. Coronary angiography was performed that shows no pathologic/stenotic lesions. After several days of antibiotic therapy and hemodynamic support the ejection fraction of the left ventricle has improved progressively (EF 25%-EF 40%-EF 50%) and the fever has resolved. This data confirms the diagnosis of heart failure related to a septic state.

Definition

Heart failure during sepsis is characterized by the presence of myocardial systolic and diastolic dysfunction that develops suddenly, without the occurrence of coronary ischemic event and it is potentially reversible with the resolution of the infection state. Heart failure in sepsis is related to high mortality for its difficult response to fluids infusion and inotropic drugs. May occur during several diseases characterized by tissue inflammation such as head trauma, stroke, anaphylaxis, organ transplantation, severe pancreatitis, ARDS.

Pathogenesis

Several mechanisms have been proposed to explain the cardiac complications during sepsis. One of the most accepted hypothesis considers that the pro-
inflammatory cytokines released during septicemia are involved in myocardial depression. Pro-inflammatory cytokines such as IL1, IL2, IL4, IL6, IL8, IL10, TNF alpha, IFN gamma, and C5a may depress myocardial function. In particular, IL6 is considered a predictor of septic state (1). The pro-inflammatory cytokines are able to alter the concentrations of two important mediators implicate in the pathogenesis of myocardial dysfunction: nitrogen monoxide (NO) and prostanooids.

During sepsis are produced a large quantity of prostanooids (in particular thromboxanes and prostacyclins) that alter the autoregulation and coronary endothelial function (2).

NO in low concentration increasing ventricular pump function, while in high concentration is associated with depression of myocardial function. Septic state causes the expression of inducible myocardium NO synthase (iNOS) (3) that increase NO concentration determining heart failure through several mechanisms including cytotoxic peroxynitrite and superoxide production.

**Diagnosis**

Sepsis state can manifest itself through electrocardiographic and echocardiographic atypical alteration. In some cases is possible to notice increase in BNP or troponin serum concentration.

Electrocardiographic exam can shows an ischemic pattern such as ST segment depression, BBSx, T wave specific alteration.

The most frequent echocardiographic alteration is the depression of left ventricular function identify trough Ejection fraction (EF%) evaluation. In some cases, however, EF is normal. This is due to the low peripheral vessels resistance that can mask a possible hypokinetic of left ventricular wall. However the most sensible echocardiographic approach is the tissue doppler pattern. The most important parameters is the peak systolic velocity of the mitral wall of the left ventricle: this parameter is related to mortality in patients with sepsis state (4). Experimental studies have shows that the tissue doppler systolic velocity is a better related to the true ejection function of the left ventricular chamber respect the ejection fraction that is sometimes normal in this patients (5).

**Therapy**

At the moment doesn’t exist a specific therapy for heart failure in sepsis and the fundamental measure are control of infectious process trough antibiotics drugs and hemodynamic stabilization (hydration, inotropic drugs, vasoconstrictor drugs) (6).

The first step is the volemic expansion trough hydration. After, when systemic hypotension is present, the use of Norepinephrine represent often the first choice. Norepinephrine and Dobutamine is a valid choice in association when the patient are unresponsive at the only Dobutamine. Dopamine is often the last choice for its tachycardia and arrhythmogenic effect.

Dobutamin another valid option.

Levosimendan represent a valid choice in patients with heart failure unresponsive to beta-adrenergic drugs. This is possible because Levosimendan has an adrenergic independent activity and determinate diastolic and systolic improvement without increase of intracellular calcium intake (7). However Levosimendan has a peripheral vasodilatation activities due to opening of potassium channel (8) that determinate improvement of coronary (9) and splanchnic flow without hypotension systemic effect. It has also an anti-inflammatory (10) and anti-apoptotic effect (11). Levosimendan myocardial positive effect persists some days after suspension.

Beta-Blockers is another possibility in sepsis heart failure. Its activity is usefulness for reduction of oxygen consumption, heart rate and intake stroke volume. Their use is however controversial due to negative inotropic effect.

Finally some studies suggest a possible benefit effect of statins for their anti-inflammatory and pleiotropic effect.

**Discussion**

Heart failure is a possible complication of the septic state. Heart failure represent a marker of severe sepsis and this condition is inscribed in the Systemic
Inflammatory Response Syndrome (SIRS) that is possible to observe during the severe septic state. However, echocardiographic EF can’t be able to define heart failure and in this circumstances Tissue Doppler valuation is more specific. In this particular case anyhow EF was just depressed and not other echocardiographic investigation are needed.

References


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