CASE SERIES

Reversible myocardial ischaemia in septic shock: Case series

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We present the case of a young (34 years) previously fit and well woman who was admitted to our ICU for the management of severe septic shock following faecal peritonitis. She did not have any traditional cardiovascular risk factors and performed regular strenuous exercises at the local gymnasium until immediately prior to her illness. Forty eight hours after admission she developed an inferior ST-elevation myocardial infarction (STEMI). Troponin-T levels were elevated at 2.1 ng ml⁻¹ 1 h after first complaining of chest pain and 4.12 ng ml⁻¹ 12 h later. The corresponding EKG at the time of the chest pain is shown in Fig. 1. ECHO cardiogram showed the presence of regional wall motion abnormalities and an ejection fraction of 45%. She was managed conservatively and made a complete recovery. Coronary angiogram performed 5 weeks later was normal (Fig. 2A and B). This case history raises the question as to why a young woman with a normal coronary circulation developed STEMI during a septic illness.

In this context, we report a series of 7 patients with septic shock and impaired cardiac ejection fraction (EF) who underwent myocardial perfusion scans during the acute stage at the in-house Nuclear Cardiology Facility, Critical Care Unit, University of Cairo, Egypt. Standard Tc-99 perfusion scans were performed in all 7 patients on admission to ICU and repeated 5–7 days later. All 7 patients had reversible perfusion defects either within a single well defined coronary territory or with a more patchy distribution that did not correlate to a single coronary territory. The perfusion defects were reversible to varying degrees in all 7 patients (Using an objective 20 segment scoring systems the mean ± SD perfusion defects were 17.7 ± 5.7 Vs 7 ± 5.9) with two patients showing complete reversibility (Fig. 3). At the time of the repeat perfusion scans ventricular function too had recovered in all but one patient (Mean EF: 46 ± 2 Vs 56 ± 7%).

Myocardial dysfunction seen in patients with severe sepsis may be attributed to a variety of factors including ischaemia, pro-inflammatory cytokines (systemic or local), NO, oxidative stress or acidosis [1]. Elevation of Troponin I – used in the definition of septic cardiomyopathy by some authors [2,3], is in fact a biomarker of myocardial damage and supports the view that ischemia may indeed be an important aetiological factor. Ischaemia in these subjects could be relative due to a myocardial oxygen supply–demand imbalance brought about by sinus tachycardia and hypotension – particularly in subjects with pre-existing fixed coronary narrowing. Cunnion et al. [4] and Dhaianaut et al. [5] have demonstrated that coronary sinus blood flow was normal or increased in patients with septic shock. Furthermore, net myocardial lactate extraction and myocardial oxygen availability were by and large normal in sepsis. These observations have been used to support the view that ischaemia was an unlikely cause of septic septic cardiomyopathy. In this context, our present case report demonstrating STEMI – in the presence of a normal coronary circulation, and reversible perfusion defects in patients with septic shock raises the possibility of transient coronary occlusion in patients with septic shock. It
is well recognised that intravascular coagulation and microvascular thrombosis are important components of severe sepsis [6]. Microvascular thrombosis may involve the coronary circulation too and account for some of the key manifestations associated with septic cardiomyopathy. The underlying ischaemia may be large/overt (as in the Manchester patient) when one

Figure 1  ECG: time of chest pain showing inferior STEMI.

Figure 2  Normal coronary angiogram performed 5 weeks after an inferior STEMI.

Figure 3  Myocardial perfusion defects (arrows) on admission (left panel) and – hours after admission (right panel) in 2 patients with septic shock. Partial (A) or complete (B) reversibility of perfusion defects is clearly evident (the stress score refers to the admission scans and the rest score refers to the repeat scans).
of the major arteries is involved or silent (the Cairo series) if only the smaller peripheral tributaries are occluded.

The possibility of microcirculatory abnormalities within the coronary circulation has been suggested by previous authors too [1]. The perfusion scans provided in this report provide the first direct evidence to this phenomenon. The role of circulating and local humoral factors [7–9] in causing global contractile dysfunction is now well established in sepsis. Defining the role of ischaemia in septic cardiomyopathy is important as the common treatment strategy based on aggressive volume resuscitation and large doses of vasoconstrictor agents such as norepinephrine may be particularly counterproductive in the presence of overt or silent myocardial ischaemia.

References