A case of clinically suspected acute myocarditis associated with influenza b and managed with veeeco and high dose steroids.

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Topic(s):
Acute Cardiac Care – Cardiogenic Shock

Citation:
We describe a 29y woman with no significant medical history, presented to our ED for recurrent syncope after 5 days of influenza-like symptoms. BP was 90/60 mmHg and HR was 125 bpm. She was afebrile, with cool extremities, T3, and jugular venous distension. ECG showed minimal diffuse ST segment elevation. Blood tests revealed increased lactate (4.3 mEq/L, URL 1.7), normal leucocytes, raised CRP (0.61 mg/dl), negative PCTI, and raised troponin I (11.49 ng/mL, URL 0.04).

A TTE showed non-dilated left ventricle (LV) with 10% EF due to global hypokinesia, LV thickness of 14 mm, and a modest impairment of right ventricular systolic function. ScvO2 was 34%. For haemodynamic instability despite inotropic support, IABP was inserted. Given clinical suspicion of acute fulminant myocarditis, endomyocardial biopsy (EMB) with right heart catheterization were performed: CI was 1.01 l/kg/min2 with a PCWPm of 16 mmHg and a RAPm of 12 mmHg. For a further increase in lactate (10.7 mEq/L), VA-ECMO support was implanted using a femoro-femoral approach. Within 24 hours, 164700 copies/ml of influenza B were detected in nasal swab. All the remaining serological test were negative.

EMB was negative for myocarditis according to the Dallas criteria, but showed interstitial fibrosis and oedema with contraction-band necrosis, and a few platelet microthrombi in intramural small vessels. Both EMB and blood sample were negative for viral RNA. Peak troponin I was 17.52 ng/ml on day 1.

Suspecting a virus-induced immune-mediated acute myocardial inflammation, high-dose steroids in addition to oseltamivir were started on day 3.

Rapid improvement of cardiac function and LV wall thickness were observed, allowing weaning from ECMO and IABP on day 8. At day 14 CMR confirmed complete biventricular recovery but multiple areas of myocardial edema consistent with acute myocarditis (figure) with minimum late gadolinium enhancement in the distal septum. Steroids were stopped after 1 month. Almost complete resolution of edema occurred within 2 months.

Conclusions: myocarditis are important diagnostic and therapeutic challenges. Our patient’s history along with the complete myocardial contractile recovery and normalized wall thickness, support the clinical diagnosis of acute myocarditis even if EMB was negative. While EMB is still the gold standard for diagnosis, it has potential limitations. Myocarditis is usually a focal process that require adequate sampling of the myocardium. Moreover, influenza B associated myocardial injury seems to be immune-mediated without direct viral cytotoxic mechanisms, preventing the detection of the virus in myocardial samples even by immunohistochemical stain. This last observation provided a strong rationale for high-dose steroids treatment. Finally, although cellular infiltrate is the histologic hallmark of myocarditis, other mechanisms can lead to myocyte necrosis (ie intra-
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