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Fatal catecholamine myocarditis in a child with severe scalding injury

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Mortality after severe thermal injury is due principally to multiple organ dysfunction complicating systemic inflammatory response syndrome or sepsis [1]. Cardiac arrest in children is extraordinary and most often due to inadequate fluid resuscitation, hypoxemic respiratory failure, or septic shock [2]. Here we report another unusual and as yet unreported cause of fatal outcome after severe scalding injury, most likely due to an exaggerated stress response with fatal cardiomyopathy.

A 2.5 year-old and previously healthy girl was admitted to our pediatric intensive care unit after severe scalding injury with boiling water. The burn covered about 35% of the total body surface area, and the burn depth was judged to be second and third degree. After initial fluid resuscitation cardiovascular stability was achieved. In the first 3 days of hospitalization the course was uneventful. Analgesia was performed with paracetamol and morphine and judged to be sufficient by our pain score. Antibiotic treatment with cefuroxime was initiated in the early morning of day 4 because of fever (39.5°C), marked increase in C-reactive protein (345 mg/l), leukopenia of 1.4 10^9/l, mild arterial hypotension and slightly diminished urine output. Blood cultures obtained at this time remained sterile. Because of tachycardia volume expansion with crystalloid and colloid solutions was repeated. Several hours later the girl developed slowly progressive dyspnea and hypoxemia, responding to supplemental oxygen. Chest radiography confirmed bilateral perihilar pulmonary edema. Heart size was normal. There was no improvement after administration of furosemide. We decided to proceed with mechanical ventilation. At this time the girl was fully responsive with no signs of neurological deterioration. While preparing for tracheal intubation the girl suffered a sudden generalized seizure and seconds later a cardiopulmonary arrest. Despite immediate cardiopulmonary resuscitation the girl died 2 h later.

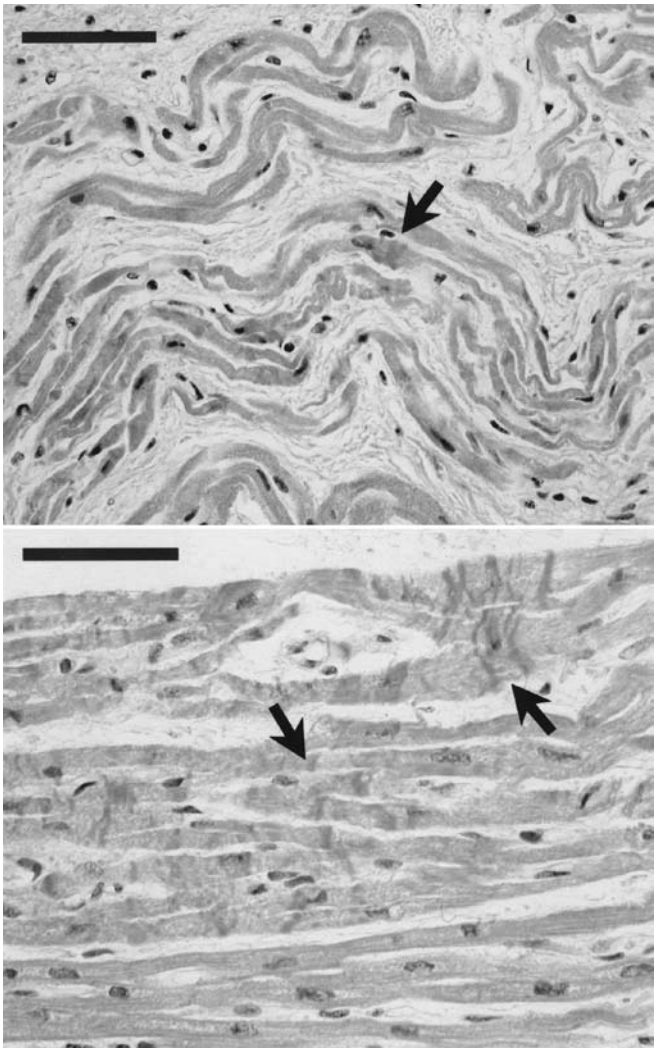

Fig. 1 Catecholamine-induced damage to the myocardium characterized by contraction-band necrosis (*arrows*) and wavy fibers (*upper panel*). *Bar* 0.05 mm

Autopsy showed as main finding a fresh diffuse myocardial infarction (see Fig. 1). No abnormalities were found in the coronary arteries. Further findings were gastric mucosal erosions, marked hemorrhagic atrophy of the adrenal glands with lipid depletion, and severe atrophy of the thymus. It has been shown that burn injuries are related to an enormous endocrine stress response [3]. To reduce secondary stress-related injuries, generous analgesia and sedation is required. In our case the only potential sign for insufficient analgesia was tachycardia, which was thought to be due to systemic inflammatory response syndrome. Surprisingly, the autopsy revealed the typical signs of a "catecholamine myocarditis" [4]. No other cause of circulatory arrest could be found at autopsy. Therefore the assumed sequence of events was initiated by high doses of endogenous catecholamines causing myocardial infarction, cardiogenic shock, cerebral hypoxia (convulsion), and eventually cardiopulmonary arrest. Other autopsy findings (gastric erosions, massive adrenal gland and thymus atrophy) support this theory.

This unusual case of "catecholamine myocarditis" adds another potential cause of fatal outcome after thermal injury. Cardiovascular instability and pulmonary edema should raise clinical suspicion of ischemic cardiomyopathy and indicate further investigations (echocardiography, troponin levels [5], pulmonary artery catheter). Treatment may be more effective if diagnosis is made at an earlier stage than in the presented case.

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