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Rhabdomyolysis and renal failure following defibrillation

Sir,

The effect of electrical cardioversion on total creatine kinase (CK) elevation has been studied by many authors.[1–3] The release of CK after elective cardioversion correlates with the cumulative energy delivered, indicating increased skeletal muscle damage with greater energy. A case of rhabdomyolysis and myoglobinuric renal failure following cardioversion was described by Minor *et al.*[4] A similar recent experience in our hospital is described.

A 54 year old male suffered a severe oppressive chest pain along with profuse sweating, nausea and marked skin pallor. One hour after the onset of pain he arrived at the emergency department of our hospital and almost immediately developed ventricular fibrillation. Despite electrical defibrillation and cardiopulmonary resuscitation (CPR), ventricular fibrillation kept on recurring. The patient received CPR for 65 minutes, 18 electrical countershocks of 360 J each and multiple boluses of epinephrine, bretylium, lidocaine, and sodium bicarbonate. After the 18th attempt at defibrillation, sinus rhythm ensued and an electrocardiogram revealed acute inferior wall myocardial infarction. Thrombolytic therapy was ruled out because of the prolonged CPR.

He was then transferred to the intensive care unit where mechanical ventilation was started. On arrival at the unit he was alert but confused, and agitated. His blood pressure was 80/50 mmHg, pulse rate 110 beats minute, the lungs were clear and cardiac auscultation did not reveal any S_3 gallop, murmurs or pericardial rubs. The urine was dark and qualitative urine myoglobin determination was positive. The level of serum CK peaked 42 hours after onset of chest pain. Serum CK was 64,480 IU/l.

For the first 3 days he had oligo-anuria with progressive worsening of his renal failure. Fifty-four hours after the onset of the myocardial infarction, serum creatinine was 781 μmol/l and serum potassium 6.3 mmol/l.

The patient required peritoneal dialysis for 2 consecutive days. He was extubated on the 11th day. He recovered his normal mental status. His renal function became progressively normal.

The very marked elevation in CK with only 2–3% CK-MB release observed in our case, indicates the importance of the skeletal muscle damage produced.

Rhabdomyolysis and prolonged hypoperfusion during CPR, can be considered the cause of acute renal failure in this case, but it is difficult to say which one contributed more.

A very high serum CK level is predictive of acute renal failure in rhabdomyolysis.[5]

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