Rhabdomyolysis and Myoglobinuric Renal Failure following Cardioversion and CPR for Acute MI

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A 50-year-old man suffered an MI with VFIB at work, and efforts at resuscitation were initiated immediately. Ninety minutes of CPR and 14 cardioversions were given by trained personnel before VFIB converted to sinus rhythm. Reversible myoglobinuric renal failure ensued, requiring two weeks of hemodialysis. Scanning with technetium-99m pyrophosphate revealed extensive muscle injury in the regions of cardioversion and a large anterolateral MI. Prolonged resuscitative efforts involving repeated cardioversion may predispose to myoglobinuric renal failure.

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VFIB = ventricular fibrillation

Rhabdomyolysis is implicated as the cause of acute renal failure in 5 percent to 7 percent of all cases.1,2 In addition to numerous traumatic etiologies, nontraumatic causes of rhabdomyolysis with myoglobinuria have been increasingly recognized, including alcohol and drug overdoses, hypothermia, sepsis,3 and limb ischemia.4,5 among others. Myocardial infarction6 and cardioversion7 may produce myoglobinuria, although, to our knowledge, neither has been reported to cause myoglobinuric renal failure.

CASE REPORT

A 50-year-old male mechanic suffered an MI at work, and witnesses initiated CPR immediately. An ambulance arrived within seven minutes, finding the patient in VFIB. Intubation, appropriate pharmacologic measures, and ten unsuccessful attempts at cardioversion followed. The CPR was continued, and the patient was transported by helicopter to Iowa Methodist Medical Center, Des Moines. The patient arrived within 90 minutes of his cardiac arrest, in persistent VFIB. After a 14th attempt at cardioversion, sinus tachycardia ensued, and an ECG revealed acute anterolateral MI.

The findings from physical examination were notable for obtundation with blood pressure of 105/50 mm Hg, pulse rate of 110 beats per minute, and respiratory rate of 28/min. The lungs were clear, and an S3 gallop was present. Swan-Ganz catheterization revealed central venous pressure of 14 mm Hg, pulmonary capillary wedge pressure of 20 mm Hg, and cardiac output of 5.0 L/min. Intravenous therapy with nitroglycerin and mechanical ventilation were begun, although thrombolytic agents were not considered because of prolonged CPR.

Laboratory data were remarkable for the following values: WBC, 20,900/cu mm; HCT, 42.7 percent; potassium, 4.4 mEq/L; bicarbonate, 17 mEq/L; BUN, 19 mg/dl; creatinine, 1.6 mg/dl; calcium 8.0 mg/dl (normal, 8.6 to 10.8 mg/dl); and phosphorus, 9.3 mg/L (normal, 2.4 to 4.7 mg/L). The level of CK peaked at 16,820 IU/L, with 5 percent CK-MB and 95 percent CK-MM. Oliguria was noted, and urinalysis revealed specific gravity of 1.032 and pH 5.0, with 12 RBCs and 2 WBCs per high-power field and few hyaline casts. Urine myoglobin was present at 30 mg/dl. A bicarbonate infusion, calcium supplementation, and phosphate binding therapy were given.

Anuria ensued by the third day of hospitalization, and hemodialysis with ultrafiltration was required (Fig 1). The patient recovered full mental status and was able to communicate appropriately by written word. A radionuclide ventriculogram obtained at bedside demonstrated that the left ventricular ejection fraction was reduced to 25 percent. Scanning with Tc pyrophosphate showed large areas of uptake in the regions of paddle placement for cardioversion, in the infarcted myocardium, and in several fractured ribs (Fig 2).

The diuretic phase of acute tubular necrosis ensued, with the BUN level peaking at 116 mg/dl and the creatinine level at 14 mg/dl. Hemodialysis was discontinued, and the patient was extubated. The patient was discharged 22 days after his MI on therapy with diltiazem, isosorbide dinitrate, and buffered aspirin. Six weeks later, an exercise treadmill test was discontinued at 7.5 minutes because of severe fatigue and dyspnea, without ST-segment changes.

Upon referral to the University of Iowa Hospitals, Iowa City, for further evaluation, the patient’s BUN level was 21 mg/dl, and the creatinine level was 1.5 mg/dl. Cardiac catheterization revealed a 100 percent proximal occlusion of the LAD without evidence of other coronary disease. The RCA had an anomalous origin from the left sinus of Valsalva. Ventriculography showed marked anteroapical dyskinesia consistent with an aneurysm. Right heart catheterization revealed normal hemodynamics.

DISCUSSION

Acute renal failure has been described following electroconvulsive therapy,8 although injury due to lightning has been reported to cause myoglobinuria without renal complications.9 In the case presented herein, several factors

FIGURE 1. Course of myoglobinuric renal failure. Arrows represent days when hemodialysis and ultrafiltration were performed.
may have contributed to the total renal burden of myoglobin, including repeated cardioversion, prolonged CPR, and extensive MI; however, the scan with 99mTc pyrophosphate demonstrated the most marked uptake at the sites of cardioversion, suggesting rhabdomyolysis of chest wall muscle was responsible for the majority of myoglobin released. In myoglobinuric renal failure, 99mTc compounds have been previously utilized to visualize the site and extent of localized muscle injury not evident clinically.12

Acute tubular necrosis following rhabdomyolysis may be due to myoglobin's direct toxic effect on distal tubular cells or its obstruction of the lumen of distal tubules.5,13 Vasconstrictive mediators released concomitantly may cause renal ischemia. Dehydration, acidosis, or a decrease in renal perfusion predispose to myoglobinuric renal failure. Serum CK levels greater than 6,000 IU/L may also be predictive.13

Myoglobinuric renal failure is often accompanied by an unusually rapid rise in the serum level of creatinine and characteristic derangements in serum calcium and phosphate levels (Fig 1). Hypocalcemia during the oliguric phase has been attributed to the release of phosphate compounds from damaged muscles, leading to hyperphosphatemia, with deposition of calcium phosphate salts in injured tissues.12 Hypercalcemia during the diuretic phase may be due to increases in serum levels of 1, 25-dihydroxycholecalciferol,14 although the remobilization of calcium from soft tissues to the extracellular space serves an important role.

Anomalous origin of the RCA from the left sinus of Valsalva occurs in less than 0.2 percent of the patients undergoing selective coronary arteriography.15-18 Previously thought to represent a benign anomaly, it has recently been associated with MI,19 syncope and nonfatal VFIB,20 and sudden death.21 In the case presented herein, sustained VFIB was most likely secondary to thrombotic occlusion of the LAD and not directly related to the anomalous RCA.

In summary, the patient described suffered both myocardial and skeletal muscle damage leading to rhabdomyolysis and renal failure. The findings of the scan with 99mTc pyrophosphate suggested that muscle injury from repeated cardioversion was primarily responsible. Multiple cardioversions during prolonged resuscitative attempts may predispose patients to the development of myoglobinuric renal failure.

REFERENCES
3 Cabow PA, Kaehny WD, Kelleher SP. The spectrum of rhabdomyolysis. Medicine 1982; 61:141-52
6 Haimovici H. Muscular, renal, and metabolic complications of acute arterial occlusions: myonephropathic-metabolic syndrome. Surgery 1979; 85:461-68
10 Selzer ML, Reinhart MJ, Deeney JM. Acute renal failure following EST. Ann J Psychiat 1963; 120:602-03
11 Yost JW, Holmes FP. Myoglobinuria following lightning stroke. JAMA 1974; 228:1147-48
12 Patel R, Mishkin FS. Technetium-99m pyrophosphate imaging in acute renal failure associated with nontraumatic rhabdomyolysis. AJR 1986; 147:815-17
18 Roberts WC, Siegel RJ, Zipes DP. Origin of the right coronary artery from the left sinus of Valsalva and its functional consequences: analysis of ten necropsy patients. Am J Cardiol 1982; 49:963-68