CASE REPORT

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Rhabdomyolysis Following Violent Behavior and Coma

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ABSTRACT: An individual suspected of being under the influence of phencyclidine (PCP) exhibited acute psychotic and violent behavior which was followed by cardiac arrest, coma, and renal failure. Sections of the damaged muscle showed rhabdomyolysis, and sections of the kidneys showed myoglonin casts positive for immunoperoxidase stain. Extensive toxicology studies for narcotics, PCP, and cocaine were negative. Therefore, a correlation between PCP and rhabdomyolysis associated with acute psychotic and violent behavior could not be made with certainty.

The etiology and pathogenesis of rhabdomyolysis are discussed in depth.

KEYWORDS: pathology and biology, rhabdomyolysis, phencyclidine

Rhabdomyolysis is an increasingly recognized clinical and pathological entity characterized by muscle cell injury and release of cellular content into the plasma. Any disease or condition that results in the destruction of striated muscle can lead to the disorder. The integrity of the cell membrane is damaged allowing the contents of the cell to escape into the extracellular fluid [*1*].

The cellular contents released include enzymes and electrolytes but can also include myoglobin and other cellular proteins, which in some cases can appear in the urine and be associated with renal failure [*2*].

Rhabdomyolysis is known to occur in a wide variety of conditions which can include excessive muscular activity in healthy physically fit individuals [*3*], prolonged coma [*4*], seizure activity [*1*], phencyclidine (PCP) intoxication [*5,6*], strychnine poisoning [*7*], narcotic abuse [*8*], and viral myocytosis [*9*] as well as the more generally recognized crush injury syndrome.

The following is a case report of an individual suspected of being under the influence of PCP who exhibited violent and psychotic behavior which was followed by cardiac arrest, coma, and eventual hospitalization.

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Case Report

This case involves a 30-year-old black male who developed bizarre and abnormal behavior and had no known serious illnesses. He began talking and acting irrationally. His family took him to the hospital for evaluation of a possible mental disorder. He was seen, evaluated, and given a prescription for thorazine and sent home.

On the following day, about noon, he was taken back to the hospital by his family, five members of whom were required to restrain him because of his violent and bizarre behavior and his superhuman strength. By the time he reached the hospital, he was subdued and quiet, appeared rational, and cooperated with the medical personnel. However, he told the examining physician at the time that he believed he was going to become violent. He was again evaluated, placed on thorazine, and sent home.

Later that evening, while at home, he became violent again and began to overturn and break the furniture. He was restrained by family members once more. The paramedics were called, but when they arrived they refused to treat the subject or to transport him to a hospital because they believed he was under the influence of PCP. The paramedics called the police who arrived shortly thereafter.

With the help of the family, the police were able to restrain the subject, place handcuffs on his wrists, and apply cord-cuff leg restraints to his ankles. The police applied the restraints for the subject's own safety and well being; he was not placed under arrest. He was escorted to the police car, placed in the back seat, and transported to the hospital for medical treatment. Just before the car reached the hospital, the subject was noted to become unresponsive and it was noted that his breathing had stopped.

He was rushed into the emergency room where cardiopulmonary resuscitation (CPR) was initiated and later taken to the Intensive Care Unit. His respiration and heart beat were restored, but he became comatose and unresponsive and remained so until his death six days later.

A toxicology screen was performed during the second hospital day. Blood for alcohol, barbiturates, PCP, and hypnotics were negative. Urine for codeine, heroin and phenothiazine were negative.

Abnormal results of laboratory studies performed in the hospital included creatinine phosphokinase (CPK) more than 60 000 units/L, lactic dehydrogenase (LDH) 2910 units/L, serum glutamic-phosphopyruric transaminase (SGPT) 550 units/L, serum glutamic-oxaloacetic transaminase (SGOT) 1505 units/L, creatinine 9 mg/dL, blood urea nitrogen (BUN) 68 mg/L, and potassium 5.7 mg/L.

At autopsy, old needle tracks were found in the left antecubital fossa associated with subcutaneous scarring, thickening of the vein walls, and thrombus formation. Numerous petechial hemorrhages were found in the bulbar conjunctiva and the eyelids.

Numerous partially healed abrasions and contusions were noted of the hairline, chin, neck, shoulder, elbows, forearms, hands, back, thighs, and legs. A circumferential abrasion was seen of the right wrist consistent with handcuffs. No abrasion was seen on the left. Circumferential abrasions were seen around both ankles consistent with ankle restraints. Healing crusted abrasions were seen of the right side of the neck. Contusion was present in the underlying sternocleidomastoid muscle. Subgaleal hemorrhage was noted in the frontal region; a contusion was found in the mesentery.

On internal examination, the brain was edematous and soft showing the generalized changes characteristic of a respirator brain but showing no focal alterations. The lungs were heavy and consolidated and showed aspiration pneumonia. Focal subepicardial hemorrhages were seen in the left ventricle of the heart. The liver, spleen, and kidneys showed congestion.

Toxicological studies were performed and blood for alcohol, barbiturates, neutrals, bases, phenothiazines, morphine, codeine, and cocaine were all negative. Blood, liver, and kidney

were tested for PCP and all were negative. Hemoglobin electrophoresis was performed and only hemoglobin A was found.

The damaged muscle appeared grossly edematous and firm; pallor with focal areas of hemorrhage was also noted. Microscopically, the muscle cells showed edema, hyaline degeneration, and loss of cross striations. No interstitial inflammation was observed. The kidney sections showed hyaline casts in the renal tubules which on immunoperoxidase stain was positive for myoglobin.

Discussion

Clinically, this patient demonstrated a typical picture of rhabdomyolysis and renal failure. His skeletal muscle enzymes were elevated including creatinine phosphokinase, lactic dehydrogenase, glutamic-oxaloacetic transaminase, and glutamic-phosphopyruvic transaminase. Although no myoglobin was detected in the serum, the BUN and creatinine were elevated. An elevated potassium was also noted.

The appearance of the muscle tissue both grossly and microscopically was typical of rhabdomyolysis. Formalin-fixed sections of kidney were stained with the immunoperoxidase technique [*10*] and were positive for myoglobin.

Interest has been raised recently about a possible connection between phencyclidine (PCP) intoxication and rhabdomyolysis [*5,6*]. In a recent study, rhabdomyolysis was found in 25 of 1000 patients (2.5%) who were admitted to a hospital for PCP intoxication; 10 (1%) of these patients also showed renal failure [*6*].

In the present case, PCP intoxication was suspected by the paramedics and by the police based on the subject's behavior at the time of the incident. The subject had no known history of PCP abuse according to his family. He did have a history of intravenous drug abuse, however, as was manifest by needle track scars on his forearm at the time of autopsy. His behavior during the incident is consistent with PCP intoxication, particularly the bouts of abnormal and violent behavior interspersed with episodes of apparent calm which he displayed while being examined in the emergency room. He also had no known history of mental or behavioral problems not associated with drugs. Toxicological studies performed in the hospital two days after the onset of symptoms were negative for PCP and other drugs. Not unexpectedly, toxicological studies performed on autopsy material were also negative for drugs. Because toxicological studies were not performed until two days after the incident, it is not known what the drug levels were when the subject collapsed.

Because of the possible association of rhabdomyolysis and PCP intoxication, striated muscle tissue and kidney were examined for evidence of rhabdomyolysis to see if this could be useful retrospectively in identifying PCP. Such a relationship, if it exists, could be helpful in a case such as this where the toxicology was performed two days too late.

The mechanism of rhabdomyolysis in PCP intoxication is not clear. It has been postulated that it may be the result of the increased muscular activity and heightened isometric tension which accompanies PCP use. Hyperpyrexia, another common finding in PCP use, may be a factor or it may result from the direct toxic action of PCP on the muscle tissue. A single cause has not been identified, and the mechanism is thought to be multifactorial [*6*].

Another common cause of rhabdomyolysis is prolonged coma which this patient exhibited. The subject had a flat electroencephalogram (EEG) and was totally unresponsive for six days. The pathophysiology is secondary to local pressure necrosis caused by compression of the muscle tissue by the weight of the patient's own body. The compression causes focal areas of ischemia and myonecrosis leading to edema and elevated compartmental pressure with resultant muscle infarction. The mechanism is a form of crush syndrome.

The rhabdomyolysis of PCP intoxication in some cases may be due to coma and not to PCP itself. In the present case, the rhabdomyolysis is more likely a result of the coma and consequent muscle compression rather than by PCP or other drugs. Because of the uncertain

etiology of rhabdomyolysis in PCP abuse combined with the multiplicity of causes, we determined that rhabdomyolysis cannot be used as a marker for PCP intoxication.

Conclusion

A case of rhabdomyolysis with acute renal failure is presented in a patient who was in coma for six days following an episode of violent and psychotic behavior which was believed to be caused by PCP intoxication. An attempt to correlate the rhabdomyolysis with PCP abuse could not be made.

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