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CASE REPORT

SUBARACHNOID HEMORRHAGE AND RHABDOMYOLYSIS INDUCED ACUTE RENAL FAILURE COMPLICATING ORGANOPHOSPHATE INTOXICATION

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ABSTRACT

Organophosphate is extremely uncommon cause of rhabdomyolysis. This article describe two cases with rhabdomyolysis induced acute renal failure complicating by monocrotophos, an organophosphate compound. First patient had rhabdomyolysis induced acute renal failure and subarachnoid hemorrhage. This is the first reported case of subarachnoid hemorrhage, which may be related with organophosphate intoxication in literature. Second patient described here had rhabdomyolysis induced acute renal failure after organophosphate overdose.

Key Words: Organophosphate; Subarachnoid hemorrhage; Rhabdomyolysis

INTRODUCTION

Acute organophosphate (OP) poisoning is a significant cause of morbidity and mortality in the world.[1] OPs lead to many well defined complication such as cholinergic crisis, intermediate syndrome, besides these complication, they also cause very rare complication such as acute respiratory distress syndrome, acute renal failure.[2,3] We reported here two cases

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with rhabdomyolysis induced acute renal failure complicated by monocrotophos. First case report also had subarachnoid hemorrhage, which may be caused by monocrotophos.

CASE REPORT 1

A 23-year-old male was brought to Emergency room after 2 h of intoxication. On arrival, he stated that he had drunk an agricultural chemical including monocrotophos. His pupils were miotic. Blood pressure was 120/80 mmHg, pulse 60 beats/min. His oral secretions were increased. Within 2 min of arrival, the patient was being bagged with 100% oxygen. Spontaneous respiratory movements were clearly uncoordinated and ineffectual. The patient was intubated and the endotracheal tube immediately filled with frontally sputum and needed to be continually suctioned. Atropin administration was begun 4 mg then 8 mg intravenously. Additional atropin was given while 2 g of pralidoxime were obtained. By 5 min after arrival the patient became unconscious. Computed tomography revealed edema and subarachnoid hemorrhage. A nasogastric tube was placed and drained then 1 g/kg activated charcoal was administered. By 15 min after arrival, 30 mg of atropine had been administered and the patient's bronchorrhea had diminished. His pulse increased to 140 beats/min. One mg/kg day of atropin infusion was initiated. Laboratory examination revealed as follows: white blood cell count (WBC) 18.600/mm^3 and a normal differential, platelets 310.000/mm^3, Glucose 241 mg/dL, BUN 14 mg/dL, creatinine 0.9 mg/dL, sodium 136 mEq/L, potassium 3.5 mEq/L, AST 25 IU/L, ALT 26 IU/L, plasma cholinesterase 0.2 U/mL (normal range 5–12.5).

Two hours after admission, pralidoxime was started as an infusion at a rate of 500 mg/h. Two days after admission the patient's urine output was decreased to 30 mL/h. Central venous pressure was measured as 6 cm H_2O. Laboratory examination revealed as follows: BUN 30 mg/dL, creatinine 4.6 mg/dL, sodium 150 mEq/L, potassium 3.0 mEq/L, pH 7.3, HCO_3^- 14 mEq/L, creatine kinase (CK) 9960 U/L, myoglobin in urine was noted as positive. Three days after admission the patient was anuric. Laboratory examinations were as follows: BUN 32 mg/dL, creatinine 5.8 mg/dL, sodium 160 mEq/L, potassium 7 mEq/L, plasma cholinesterase 1.58 U/mL, CK 9100 U/L. Peritoneal dialysis was performed. One day after peritoneal dialysis cardiac arrest was developed and patient died.

CASE REPORT 2

A comatose 45-year-old male was brought to emergency room after 4 h of ingestion of monocrotophos. His wife stated that the patient had drunk

about 100 mL of agricultural product involving monocrotophos. On arrival the patient was comatose, his pupils were miotic. There was fasciculation on face and chest. His oral secretions were increased. Blood pressure was 110/70 mmHg, pulse 62 beats/min. Spontaneous respiratory movements were uncoordinated. The endotracheal tube was inserted for ventilatory support. Atropin was administered 4 mg then 8 mg while 2 g of pralidoxime was initiated. A nasogastric tube was placed and drained then 1 g/kg activated charcoal was administered. By 20 min after arrival 50 mg of atropin had been administered and the patient's bronchial secretions had diminished. His pulse increased 130 beats/min. One mg/kg day of atropin infusion was initiated. Laboratory examinations were as follows: WBC 19.000/mm^3, and a normal differential, Hb 15.4 g/dL, platelets 320.000/mm^3, BUN 16 mg/dL, creatinine 1.0 mg/dL, glucose 133 mg/dL, sodium 136 mEq/L, potassium 3.5 mEq/L, plasma cholinesterase 0.1 U/mL, amylase 80 U/L. Two hours after admission, pralidoxime was started as an infusion at a rate of 500 mg/h. Three days after admission urine output of patient was decreased to 30 mL/h while central venous pressure was noted as 7 cm H_2O. Laboratory examinations were as follows: BUN 40 mg/dL, creatinine 5.4 mg/dL, sodium 158 mEq/L, potassium 3.1 mEq/L, HCO_3^- 12 mEq/L, pH 7.2, plasma cholinesterase 2.1 U/mL, CK 10.800 U/L, myoglobin in urine was noted as positive. Bicarbonate and 0.45% Isotonic solution infusion was started. Four days after admission laboratory examination were as follows: BUN 50 mg/dL, creatinine 5.4 mg/dL, sodium 165 mEq/L, potassium 5.8 mEq/L. Urine output was noted 40 mL/h. Peritoneal dialysis was performed. Two days after peritoneal dialysis the patient's urine output was increased to 1500 mL/day, laboratory examination revealed: BUN 25 mg/dL, creatinine 2.5 mg/dL, sodium 140 mEq/L, potassium 4.5 mEq/L, CK 8700 U/L, myoglobin in urine was noted as positive. Peritoneal dialysis treatment was stopped: The patient was still intubated for ventilatory support. Twenty days after admission the patient was extubated. Twenty-five days after arrival the patient had acute myocardial infarction and then ventricular fibrillation. Despite cardiopulmonary resuscitation the patient died.

DISCUSSION

OPs not only affect the acetylcholinesterase but also may alter the liver, kidney and endocrine glands functions.[3,4,5] OPs are an extremely uncommon cause of rhabdomyolysis. Rhabdomyolysis is a syndrome characterized by injury to skeletal muscle with subsequent release of intracellular contents such as myoglobin, CK, aldolase, LDH, and potassium. The common terminal event of rhabdomyolysis is the disruption of the Na^+, K^+-ATPase pump and calcium transport, resulting in increased intracellular calcium and subsequent muscle cell necrosis. Drugs of abuse that have commonly been

implicated in acute rhabdomyolysis include cocaine, amphetamines, LSD, heroin, and phencyclidine.[6] There is only a few reports about OP induced rhabdomyolysis and acute renal failure. De Wilde et al.[7] first reported a case with rhabdomyolysis induced acute renal failure due to fenthion intoxication. They showed rhabdomyolysis by biopsy of muscle. Abend et al.[8] reported second case with acute renal failure complicating by OP intoxication. Betrosian et al.[3] reported a case with adult respiratory distress syndrome and acute tubular necrosis induced by organophosphate intoxication.

Our first case described here is the first reported case with subarachnoid hemorrhage and rhabdomyolysis related with OP intoxication. We did not hypothesized the mechanism of subarachnoid hemorrhage in this case. The presence of subarachnoid hemorrhage may or may not be causally linked to the monocrotophos toxicity. We propose branin CT in patient with OP intoxication who became unconscious suddenly. This patient also had rhabdomyolysis induced acute renal failure. Our second case described here had rhabdomyolysis induced acute renal failure complicated by monocrotophos intoxication.

In conclusion we suggest that brain computed tomography may be used in every patient with OP intoxication who became unconscious suddenly. We also suggest that CK and myoglobin in urine should be monitored in patient with OP intoxication.

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