Strychnine Intoxication in a Child

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The use of strychnine is restricted to veterinary preparations, such as rodenticides. The tablets are a bright pink, making them attractive to children. Strychnine is rapidly absorbed following ingestion. General muscles spasm and seizures can occur a short time after intoxication and in severe cases can be fatal. We report a case of a child poisoned accidentally through ingestion of this material, who successfully responded to a muscle relaxant combined with mechanical ventilation.

Patient Description

A 6 year old otherwise healthy Bedouin boy was admitted to the Pediatric Intensive Care Unit due to severe muscle cramps. An hour before his admission he had ingested a pink tablet of unknown substance, and after approximately 30 minutes post-ingestion he began to vomit and shiver and exhibited severe, general muscle spasms. He had found the tablets on a street near his house in a Bedouin village. These tablets are used by exterminators to rid areas of unwanted rodents and stray dogs and cats. An hour after ingestion, his parents brought the rest of the pink tablets to the PICU and the substance was identified by the exterminators as strychnine, which they had scattered a few hours before the child had found them.

At the local primary care clinic the child had been treated with repeated doses of diazepam 0.5 mg/kg/dose intravenously without any response. On admission to the PICU he was conscious but disoriented. His vital signs were: blood pressure 101/49 mmHg, heart rate 125 beats/min, temperature 37.4°C, and respiratory rate 15/min. Physical examination revealed severe, general muscle tetania and dilated pupils.

Upon admission, tests of arterial blood gases (with oxygen) showed pH 7.07, $PaCO_2$ 33 mmHg and PaO_2 272 mmHg, with base excess of -20 and HCO_3- 9.6 mEq/L. Blood examinations revealed hemoglobin 10.5 g/dl, white blood cell count 12,400 cells/mm^3 with normal differential count, and platelet count 341,000/mm^3. Urine or blood strychnine level was not measured because this test is not available in our hospital and the toxic substance had been identified.

Blood analyses showed glucose 228 mg/dl and calcium 9.1 mg/dl. Renal and hepatic function were within the normal range; creatine phosphokinase was 309 U/L and after 24 hours 3,193 U/L; CPK-MB was 18% and myoglobin 750 ng/ml. The chest X-ray was normal and electrocardiogram showed only sinus tachycardia. The boy was intubated, mechanically ventilated, and treated at that time with a continuous drip of vecuronium bromide i.v. in a dose of 0.1 mg/kg/hour for 20 hours.

The child developed rhabdomyolysis with a peak CPK of 4,992 U/L, high serum myoglobin of 750 ng/ml, and mild renal dysfunction (creatinine 1.3 mg/dl). On the second day of hospitalization the patient developed fever and had a white blood cell count of 13,400 cells/mm^3. A chest X-ray revealed an infiltrate on the left lower lobe. The sputum culture grew *Streptococcus pneumoniae* and he was treated with cefuroxime i.v. The boy was discharged from the PICU after 3 days in good condition; blood gases, CPK and myoglobin had returned to normal.

PICU = pediatric intensive care unit

CPK = creatine phosphokinase

Comment

Strychnine was first used in Germany as a poison for rats and other animals in the 16th century. A potentially lethal dose in a child is 5–10 mg, but survival can follow ingestion of large doses [1]. The diagnosis of strychnine poisoning is based on identification of the ingested material and measurement of the strychnine level in the blood or urine. In the past, strychnine has been used for the treatment of non-ketotic hyperglycinemia [2] and sleep apnea, and as a laxative. Today strychnine is used mainly in rodenticides and in exterminating rabies-infected dogs. An unusual source of poisoning occurs among drug abusers who have snorted or injected strychnine unintentionally, when it was used as a drug adulterant or as a substitute for heroin and cocaine.

Strychnine blocks action of inhibitory neurotransmitter glycine in the brainstem and on the motor neuron receptor of the ventral and dorsal horns of the spinal cord. It interferes with the normal functioning of glycine, resulting in excessive motor neuron activity, exaggerating responses to auditory, visual and tactile stimulation, and heightening perception of pain due to muscle spasms [3]. Therefore, in cases of strychnine poisoning, avoidance of external stimuli tends to alleviate pain. Strychnine also appears to block the depressant affects of GABA, another neurotransmitter that inhibits motor neurons [4]. Strychnine has first-order kinetics, primarily by enzymes involving the liver microsomal system, and the half-life is about 10 hours [5].

Generalized muscle spasms can occur within 5 minutes of intranasal or intravenous use and within 15 minutes of ingestion; therefore, induction of vomiting for gastrointenstinal decontamination is contraindicated. Our patient had ingested a tablet of 140 mg strychnine and after about 30 minutes he started vomiting, accompanied by severe aching muscle cramps. In contrast to epileptic seizures, strychnine-induced seizures are characterized by tonic-tetanic, rather than tonic-clonic motor activity. The early benzodiazepine treatment in our patient did not affect the severe muscle spams because he was experiencing severe muscle tetania and not convulsions. Benzodiazepine as a muscle relaxant is effective only when there are mild muscles contractions. Thus, in severe cases of muscle tetania, benzodiazepine is not adequate, but morphine and a muscle relaxant, such as vecuronium bromide, is indicated.

Secondary to the intense muscle spasms, rhabdomyolysis and myoglobinuria may occur [1], as it did in our patient despite the fact that he was treated with vecuronium bromide very soon after ingestion. Since leukocytosis and hyperthermia [1] are also common in patients with seizures or muscle hyperactivity, one might assume that the findings in our patient might be related to the strychnine intoxication. However, since there was a positive sputum culture and a lung infiltrate upon admission, we can only presume that the patient developed aspiration pneumonia during the acute tetania.

In severe cases, acidosis, respiratory paralysis (due to chest muscle and diaphragm spasms), and acute respiratory distress syndrome could develop. Most probably the development of metabolic acidosis in our patient was caused by lactic acidemia secondary to neuromuscular hyperactivity and hypoxia [1]. However, blood lactic acid was not measured.

In conclusion, strychnine ingestion should be considered in all cases of general muscle spasms or tetania. Early treatment of drug-induced neuromuscular paralysis can inhibit further complications, such as rhabdomyolysis, myoglobinuria and renal failure.

References

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