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Case Report Open Access

Early Onset Cardiogenic Shock in Acute Colchicine Overdose

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Background

Colchicine is an alkaloid from the family of the spindle poisons. It is mainly used as treatment and prevention for gout or other forms of microcrystalline arthritis, for familial Mediterranean fever, Behçet disease and for pericarditis. It has a narrow therapeutic index and colchicine overdose is associated with high mortality rate [1]. As for the clinical course of colchicine poisoning the first phase is dominated by gastro-intestinal symptoms (0-24 hours); the second phase is characterized by multi-organ dysfunction, metabolic derangements and bone marrow suppression (1-7 days). Surviving patients will enter the third phase (7-21 days) by recovery of bone marrow depression and resolution of organ failure [2]. Death from acute colchicine poisoning is usually due to hemodynamic collapse and cardiac arrhythmias (typically 24-36 hours after ingestion) but early cardiogenic shock has not been previously reported [3,4].

Case History

We report the case of a 50 year old woman (65 kg), who was admitted to the ICU for an acute drug overdose. Her past history was significant by hypothyroidism and suspected mild Behçet's disease, treated by colchicine. She declared having ingested 50 mg of Colchimax® (colchicine 1 mg, opium 12.5 mg and tiemonium methylsulfate 5 mg), 100 mg of furosemide, 75 mg of hydroxyzine, 2 g of pristinamycin and 120 mg of propranolol.

At her admission in ICU, 7 hours after ingestion, she had vomiting and diarrhea. Physical examination was unremarkable other than abdominal tenderness. Vital signs were normal on admission. Initial laboratory tests were normal except troponine I (0.18 ng/ml) and serum level of colchicine at 24.7 µg/l (therapeutic range 0.3 to 2.5 µg/l, toxicity rate >5 µg/l). ECG found an increased QTc (502 ms) and a diffuse ST depression. Echocardiography revealed severe dysfunction of left (LVF=25%, cardiac output=2 l/min) and right ventricles.

Thirteen hours after ingestion, laboratory exams showed a deterioration of renal and liver functions, troponine I at 3.52 ng/ml and metabolic acidosis (pH=7.31, HCO3-=18.5 mmol/l, lactate=4.77 mmol/l). At the same time, hemodynamic status deteriorated and the patient became anuric. Treatment with dobutamine was started followed by epinephrine. But hemodynamic status continued to deteriorate. Therefore an extra-corporeal life support (ECLS) was initiated 18 hours after ingestion. The multi-organ failure persisted despite the ECLS with hemorrhagic shock treated with massive transfusions and at day 2, she presented a bilateral mydriasis with suspicion of an intracranial hemorrhage. We decided to withdraw life sustaining therapies and the patient died within a few hours.

Discussion

Cardiac toxicity of colchicine has been well described in experimental studies with structural and functional damages. Administration of colchicine in adult rats markedly impaired intrinsic myocardial contractility, isotonic relaxation and load dependence of relaxation. Colchicine also accelerated isometric relaxation [5]. Diffuse ST depression on ECG, initial elevation of troponin I and severe ventricular dysfunction found on echocardiography are consistent with myocarditis.

The earliness and severity of cardiogenic shock was surprising. Several hypotheses can be advanced to explain the high serum level of colchicine we observed. Co-ingested opioid and anticholinergic compounds may have altered colchicine absorption and gastrointestinal transit leading to impaired elimination [6]. Moreover colchicine is partially metabolized by the liver cytochrome P450 3A4 inhibited by pristinamycin, which can then potentiate colchicine toxicity [2]. Last the co-intoxication with a beta-blocker (propranolol), eventhough oral intake was low and heart rate was normal, probably increased the hemodynamic collapse. We do not use activated charcoal in this observation because of the presence of vomiting.

As compared to the immunotherapy used in digitalis toxicity, the efficiency of immunotherapy by colchicine-specific antibodies has been assessed in a patient who had ingested 60 mg of colchicines [7]. Unfortunately colchicine-specific Fab fragments are no more commercially available in Europe. Without specific treatment, supportive care is the only available care with aggressive resuscitative measures. ECLS is indicated in acute heart failure caused by toxic myocarditis, including colchicine, when patients are not responding to conventional treatments. ECLS remains an invasive technique, not lacking in potential risks but should be considered early in the management of such patients [8].

Conclusion

Cardiogenic shock is a possible early dramatic complication in acute colchicine poisoning especially when associated with co-ingested compounds and drugs which can potentiate toxicity. Due to lack of specific Fab, ECLS may be considered as soon as possible.

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