CASE REPORT

Fatal colchicine intoxication

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ABSTRACT

Colchicine is an alkaloid extracted from autumnal Colchicum plant which is used primarily for its anti-inflammatory therapy effect. Acute intoxication with colchicine is uncommon but often severe and results in multiple visceral organ dysfunctions. The intoxication severity and mortality are directly depending on the ingested dose. The treatment is manly symptomatic. However, the development of specific anti-colchicine immunotherapy would offer a new therapeutic perspective.

Authors report a case of a young patient that ingested 40 tablets colchicine, which caused a multiple organ failure and with fatal outcome.

Key words: *Acute intoxication, colchicine, multiple organ failure, shock*

INTRODUCTION

Colchicine is Autumnal-Colchicum alkaloid extract used for anti-inflammatory purposes. Acute colchicine-intoxication (CI) is rare leading to multi-visceral failure. CI severity depends of ingested doses. However, low-doses (5-10 mg) are fatal.[1] We discuss a-16-year-old-patient that ingested colchicine for suicidal purpose.

CASE REPORT

The patient was admitted in the emergency department after 6-hours of voluntary ingestion of 1 mg × 40 colchicine pill for suicidal purpose. This caused diffuse abdominal pains, vomiting, myalgia, and arthralgia. At-admission, the patient was awake with normochromic conjunctivas, the systolic and diastolic blood pressures were 90 mmHg of 65 mmHg, respectively, the cardiac frequency was 95 beats/minutes, and eupneic.

Gastric wash was achieved. Two-hours later, the patient presented shock state, frissons and profuse sweating. The systolic/diastolic blood pressures were 60/22 mmHg. The patient was administrated salty isotonic serum. The oxygen-therapy was carried with 10 μg/kg/minute dobutamine.

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The hemoglobin was 12.8 g/dl, white-blood-cells-count was 9000 elements/mm^3 and normal platelets. The renal, hepatic functions, and gasometry were normal.

A transient clinical improvement was followed by aggravation after 24-h of admission. The patient presented vomiting with respiratory distress and low blood-pressure requiring intubation, mechanical-ventilation and vasoactive drugs increase. The biological assessment of control showed hemoglobin at 11.2 g/dl, hyperleucocytose at 27000 elements/mm^3, platelets-rate of 43 3000 elements/mm^3. The renal function was slightly disturbed with urea rate at 0.66 g/l, the creatinine-coefficient was 17 mg/l, the transaminase was twice the normal, and the prothrombin rate (factor II) was 25% with activated-cephalin-time in 48 s versus 30 s. The troponin was 1.07.

The X-ray revealed acute lung edema. The electrocardiogram demonstrated diffuse repolarization disorders; the evolution was fatal with multi-visceral failure despite the considered reanimation measures.

DISCUSSION

The colchicine is an alkaloid extracted from colchicum and Gloriosa-superb plants belonging to spindle poisons

family. It is used for anti-inflammatory purposes.[2] The CI has a narrow therapeutic window and high potential of intoxication risk and might provoke a multi-visceral failure.[3] The colchicine consists fixated on tubulin in a reversible and prolonged way. This stops the polymerization into microtubules.[4] This property is responsible for a cytostatic effect with cellular functions inhibition which is connected to inflammation mechanisms.

The CI overdose expressed own toxicity on fast regeneration cells. This originates multi-visceral failure after a free-interval of 6-12 h and appearance of digestive, respiratory, metabolic, hematologic, neurological, and cardiovascular disorders.[5] The hemodynamic instability, rhythm disorders, and appearance of infectious or hemorrhagic complications lead to fatal. Signs severity and mortality are associated with ingested doses although mortal cases are reported for lower doses of 5-10 mg.[5]

The CI has strong morbidity for doses higher than 0.5 mg/kg. The symptoms are digestive-disorders and decreased coagulation factors. Doses between 0.5-0.8 mg/kg demonstrated a death risk of 10% with additional medullar aplasia. Doses higher to 0.8 mg/kg are fatal with major refractory circulatory deficiency in 72-h.[3] Our patient ingested 40 mg which demonstrated hemodynamic failure in the first hour of admission and finally multi-visceral failure after 24 h of admission.

There is not any specific colchicine antidote, the CI therapy is symptomatic. This consists of gastric wash; ingestion of active-coal which that absorbs colchicine; hydroelectrolytics and proteins loss compensation, shock state correction and/or catecholamines administration.[5] The mortality remains high despite the advanced medical care.[1] Considering colchicine distribution in tissue, exsanguino-transfusion, the hemoperfusion, and assisted-ventilation are not effective.[2,5] The immunotherapy using anti-colchicine polyclonal antibodies allows modifying the prognosis. Antibodies in a patient that ingested colchicine 60 mg with fatal prognosis were reported. Indeed the immunotherapy allows good evolution with fast correction of the biological/physiological disorders.[2,5] However, these polyclonal anti-colchicine-antibodies are in preclinical study stage.

CONCLUSION

The CI originates high mortality with acute complications. The treatment is symptomatic. Early and suitable care would improve the prognosis. The immunotherapy using (45 kD) anti-colchicine allows improving critical form's prognosis.

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