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Colchicine Overdose: Journey to recover. Case Report

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ABSTRACT

Colchicine is an anti-inflammatory alkaloid used for the treatment and prevention of gout, and more recently for the treatment of familial Mediterranean fever (FMF). It has a narrow therapeutic index, with no clear- cut distinction between nontoxic , toxic and lethal doses. Colchicine is rapidly absorbed from the gastrointestinal tract after ingestion, In patients with pre exciting renal and hepatic impairment the possibility of toxicity greatly increases. The clinical course of acute colchicine overdose is divided into three sequential and usually overlapping phases. The first phase is gastrointestinal and the second phase is multi organ failure and death will occur in this phase, the third phase is the recovery stage typically occurs after weeks. We present 38 years old patient who presented to our hospital Accident and Emergency after 8 hours of ingestion of 40 tablets of colchicine. Patient was complaining of nausea and vomiting with repeated watery diarrhea. After 24 hours, Patient was diaphoretic , orthopenic and tachycardic , fluid resuscitation was ongoing , but as patient hemodynamic was unstable requiring vasopressor support patient was shifted to ICU.As the patient had worsening lactic acidosis and acute kidney injury, elective intubation was done and patient was mechanically ventilated. During this stage patient was in multi organ failure (cardiogenic shock, coagulopathy, acute kidney injury, pancytopenia and electrolyte disturbance). After 10 days Patient was tracheostomized due to prolonged intubation , then gradually weaned from mechanically ventilation , pancytopenia resolved with rebound leukocytosis but with delayed in recovery of kidneys so CRRT was continued till renal recovery that was achieved after 3 weeks , Successfully the patient had his tracheostomy tube de-cannulated and discharged after full recovery.

Key words:
Colchicine, over dose, multi organ failure.

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Introduction

Colchicine overdose is uncommon but potentially life threatening due to the narrow therapeutic index. Colchicine is a neutral alkaloid with weak anti- inflammatory activity, it has been used in prevention and treatment of gout and treatment of Familial Mediterranean Fever (FMF).

Colchicine has potent anti- mitotic activity as it binds reversibly and selectively to tubulin, the micro- tubular protein that disrupts the function of the mitotic spindles in those cells capable of dividing and migrating. After ingestion it is rapidly absorbed from the gastrointestinal tract, colchicine is eliminated by the hepatic metabolism by cytochrome P450 3A4 (CYP3A4) and the kidneys have an important role in colchicine clearance, so clearance is reduced in patients with hepatic and renal insufficiency, with higher risk of toxicity in such patients.

The clinical course of acute colchicine overdose is divided into three sequential and usually overlapping phases. The first phase (within 24hours) is gastrointestinal and the second phase (2-7 days) is multi organ failure and death will occur in this phase, the third phase (after 7 days) is the recovery stage which typically occurs after weeks.

Case Report

A 38 years old patient who presented to our hospital Accident and Emergency after 8 hours of ingestion of 40 tablets of colchicine. Patient was complaining of nausea and vomiting with repeated watery diarrhea 10 times. Patient was fully conscious with bilateral reactive pupils, chest was clear and no abnormal murmur over the heart, abdomn was soft with mild epigastric tenderness, ECG was showing sinus tachycardia.

Other Laboratory findings was leukocytosis and impaired renal function.

After 24 hours, Patient was diaphoretic, orthopenic and tachycardic, fluid resuscitation was ongoing as bed side ultra sound revealed collapsed IVC, but patient hemodynamic instability was requiring vasopressor support, so patient was shifted to ICU. ABG showed metabolic acidosis with PH 7.31, PaCo2 26.7 & HCO3 13.3, with lactic acidosis 9.5 mmol/L.

As the patient had worsening lactic acidosis and acute kidney injury, elective intubation done, and patient was mechanically ventilated.

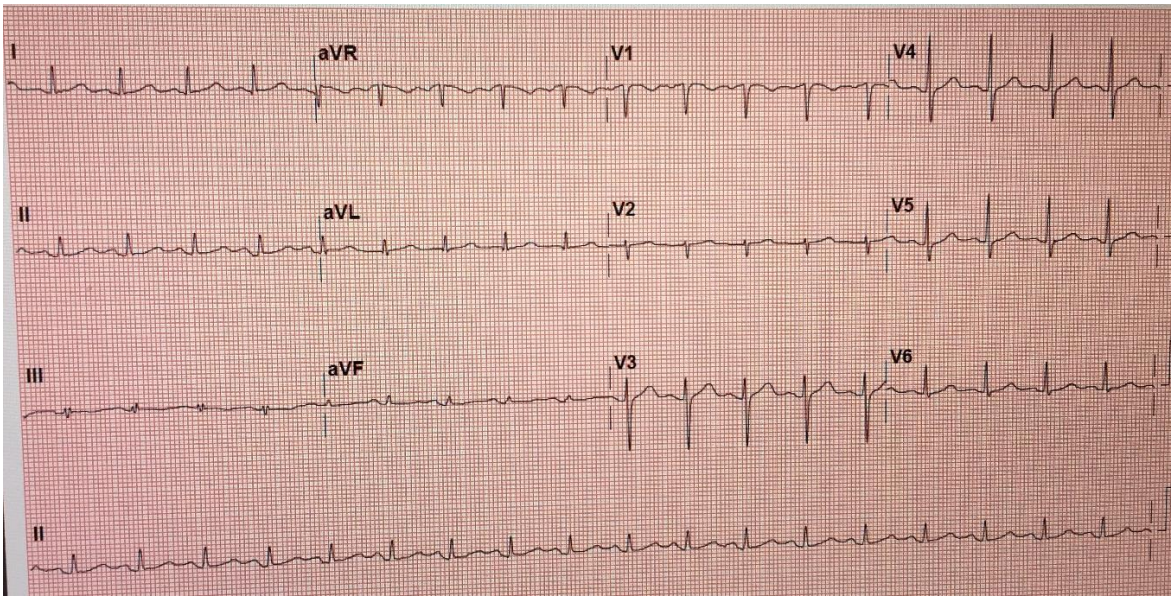
During this stage patient was in multi organ failure (cardiogenic shock requiring inotropic and vasopressor support and coagulopathy requiring frequent blood and blood products transfusion, acute kidney injury with severe uremia and metabolic acidosis so CRRT (Continuous Renal Replacement Therapy initiated), pancytopenia where patient had received broad spectrum antibiotic coverage and G-CSF (Granulocyte-colony stimulating factor), also reverse isolation was initiated. All electrolyte disturbances were perfectly corrected to avoid any cardiac arrhythmia.

CXR showed ARDS picture were lung protective measures continued and patient was tracheostomized after 10 days in view of prolonged intubation then gradually weaned from mechanically ventilation.

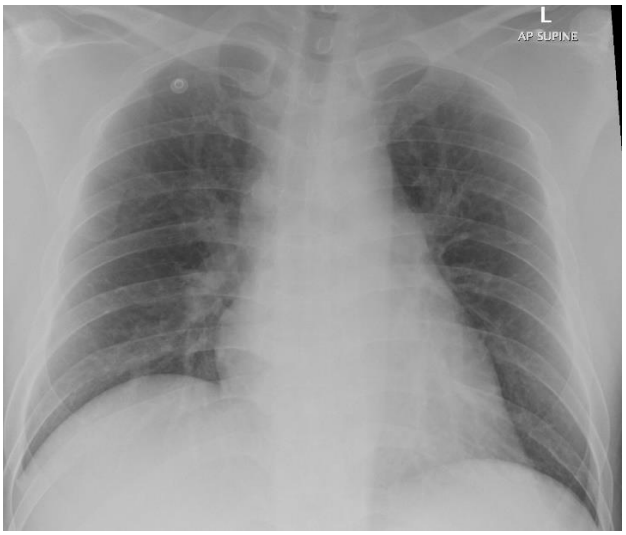
Pancytopenia resolved with rebound leukocytosis and with improvement of his cardiac depression the hemodynamic supports were tapered completely, unfortunately there was delay in recovery of the renal function so CRRT was continued till full recovery of the kidneys after nearly 2 weeks. Successfully the patient had his tracheostomy tube de-cannulated and discharged after full recovery with alopecia after 30 days from hospital presentation.

Discussion

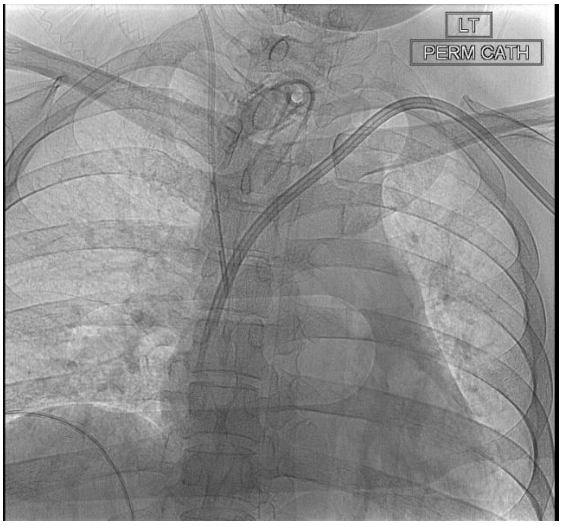
Colchicine is an example of drugs known as a beta- tubulin interactor [(1)]. It is used mainly for the treatment and prevention of gout and for familial Mediterranean fever (FMF). It has a narrow therapeutic index, so must be used with extreme care [(2,3)]. There is no specific antidote for colchicine toxicity, charcoal may be



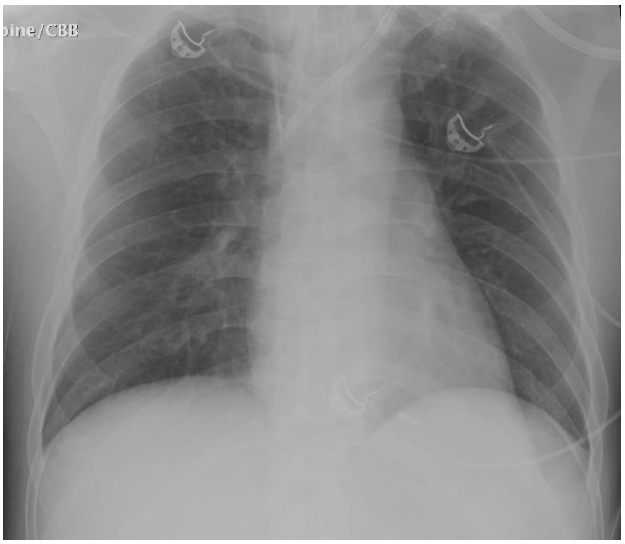
ECG on admission



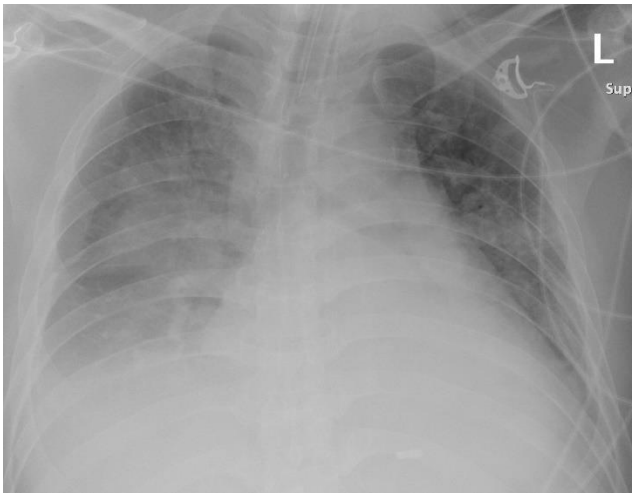
On Admission



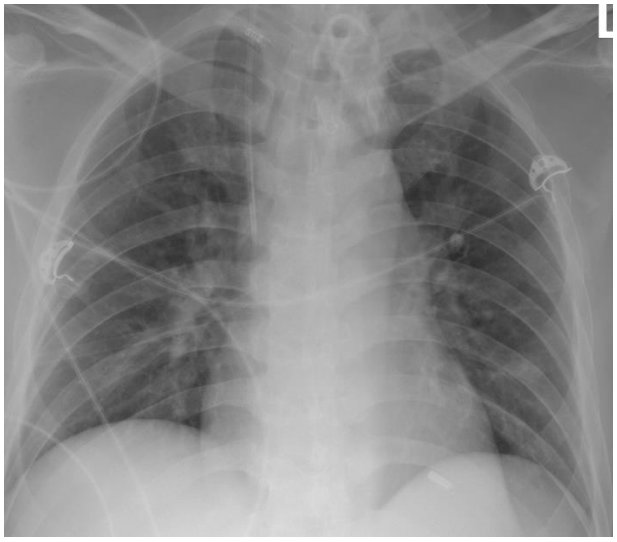
After Perma Cath Insertion for CRRT



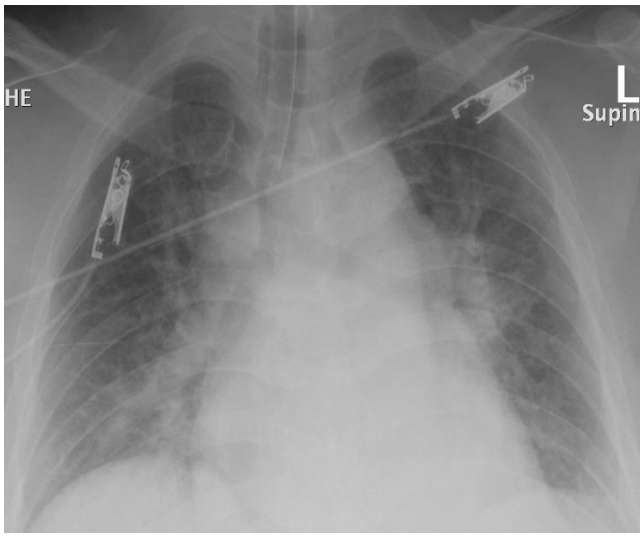
After Weaning From Mechanical Ventilation



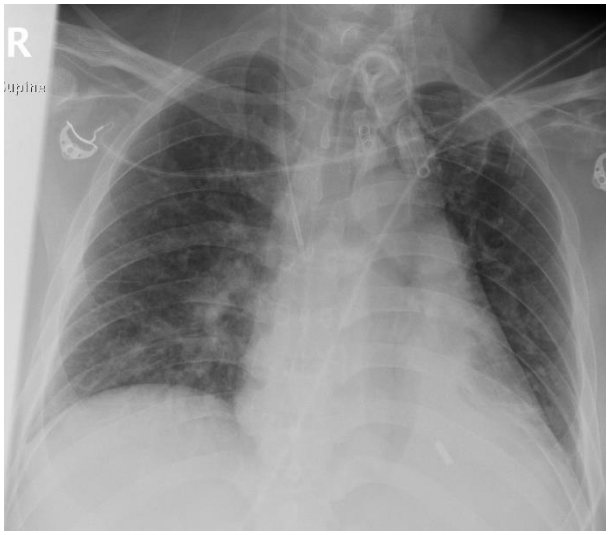
Bilateral Infilterates and Bilateral Effusion (Most Worse)



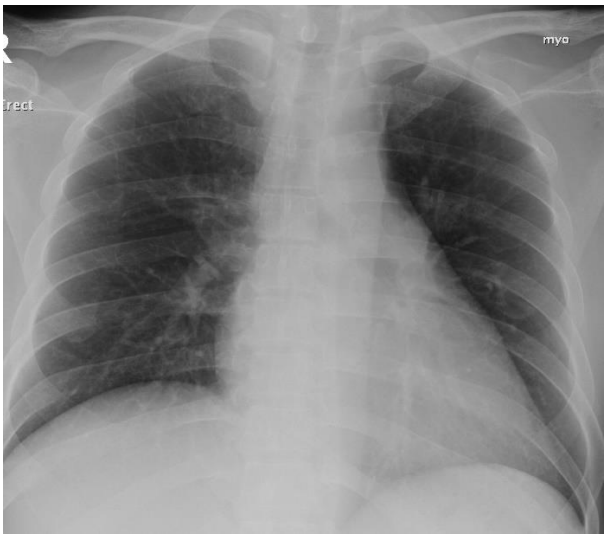
Improvement of CXR After Tracheostomy



Post Intubation



Post Trach



ICU Discharge

(After Decanulation of Tracheostomy , Removal of Permacath)

considered, but treatment is mainly supportive [3]. After ingestion, colchicine is rapidly absorbed from the GI tract. It has a large volume of distribution and binds significantly to plasma proteins and has rapid distribution. Those who present with a delay after ingestion, and those with pre-existing hepatic or renal insufficiency are at higher risk of toxicity [4].

Our case presented in stage I of colchicine toxicity with GIT symptoms and progressed rapidly to stage II with multiple organ failure with the following typical symptoms confusion and altered level of consciousness, cardiovascular collapse and shock, respiratory failure, bone marrow suppression with profound leucopenia and oliguria and acute renal failure with metabolic acidaemia [4,5,6].

Vikram et al reported the death of colchicine overdose on the third day after ingestion of 40 tablets [4].

Maxwell et al reported death of a colchicine toxicity case even more rapidly after 11 hours of ingestion of 53 tablets [7].

Percutaneous tracheostomy facilitated the progress of our case weaning from mechanical ventilation, Bone marrow suppression was managed with G-CSF (Granulocyte-colony stimulating factor) and reverse isolation [8]. Peake et al stated that G-CSF is reportedly beneficial in colchicine – induced pancytopenia [9,10]. Broad spectrum antibiotics should also be given to prevent development of septicemia [11,12].

Experimental treatment with colchicine – specific Fab fragment antibodies was reported as the high affinity of the Fab fragments antibodies to colchicine prevents the drug from binding to other peripheral sites, but this treatment is not yet commercially available [13,14].

Conclusion

This case illustrated the classical presentation of Colchicine overdose with GIT symptoms and progression to multi organ failure with prolonged course and fortunately ended with full recovery.

Conflict of Interest

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Acknowledgement

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