ISSN: 0975-8585

Research Journal of Pharmaceutical, Biological and Chemical Sciences

Fatal Poisoning With Colchicum Autumnale: A Case Report.

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ABSTRACT

Colchicum autumnale is commonly known as the autumn crocus, wild saffron and naked lady. In local language here in southern Karnataka, it is known as *gowri gedde*. It contains alkaloid colchicine that is antimitotic, blocking the mitosis by preventing DNA synthesis and tubulin polymerization. The clinical manifestation of colchicine poisoning is present in three phases following a latent period of 4-12 hours. The first phase is characterized by peripheral leukocytosis, gastrointestinal symptoms with fluid losses and hypovolemic shock. During 24-72 hours, the second stage of intoxication, life threatening complications occur such as arrhythmias, heart failure, renal failure, hepatic injury, respiratory distress, coagulopathies, bone marrow suppression and neuromuscular involvement. Second phase usually lasts for 5-7 days. Third phase is characterized by leukocytosis and alopecia. Colchicine is rapidly absorbed from the gastrointestinal tract and is primarily metabolized in the liver in a first order process. There is also significant biliary excretion and enterohepatic recirculation. Renal excretion is only responsible for about 20% of unchanged colchicine elimination. We present here a case of fatal poisoning with colchicum autumnale in a middle aged lady who finally succumbed to death due to multi-organ dysfunction.

Keywords: fatal, poisioning, colchicum autumnale.

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ISSN: 0975-8585

Case Report

A 52 year old lady, who is a agriculturist had consumed 3 plants of colchicum autumnale . She had no history of any disease in the past. She had repeated episodes of vomiting and diarrhea after 6 hours of ingestion of the plant. She was brought to emergency department after 14 hours of ingestion. She was treated with gastric lavage and activated charcoal and was shifted to intensive care unit. On examination she was conscious with a GCS of 15 and oriented to time, place and person. Her pulse rate was 116/min and blood pressure of 100/70 mmHg in supine position. She had signs of dehydration. On systemic examination, she had tender abdomen which was more in the epigastric region. The remaining physical examination was unremarkable. Her laboratory results are shown in Table 1.

Table 1

Investigations	Day 1	Day 2	Day 3	Normal value
Hemoglobin(g/dl)	10.4	9.8	9.0	12-15
Total leukocyte Count (x10^9 /l)	20.5	22.5	11.7	4.0-11.0
Platelets (x10^9 /l)	190	95	58	150-400
Creatinine(mg/dl)	1.2	2.6	4.1	0.4-1.4
AST(U/L)	102	960	2165	5-40
ALT(U/L)	65	1185	2750	5-40
International Normalized ratio	1.6	2.7	4.5	1.0
pH of blood	7.28	7.10	6.90	7.35-7.45
Amylase (U/L)	810	1280		23-85
Lipase (U/L)	955	1060		0-160
Colchicine **Serum (mcg/l)** **Urine (mcg/l)**	 10 7	 15 9		
AST, aspartate aminotransferase; ALT, alanine aminotransferase				

Electrocardiogram showed sinus tachycardia and chest X-ray was normal. Ultrasound of the abdomen revealed bulky pancreas with necrosis suggestive of acute pancreatitis. On day 1, she was kept nil per oral and treated with normal saline (4000ml), activated charcoal and broad spectrum antibiotic (piperacillin+ tazobactum). On day 2 , she developed acute kidney injury with severe metabolic acidosis. There was also evidence of hepatic necrosis. She was treated with bicarbonate infusion and dialysis was initiated. On day 3, she developed breathlessness and had a drop in oxygen saturation. Electrocardiogram done showed non-specific ST-T changes and echocardiogram revealed severe LV systolic dysfunction (Ejection fraction-30%). Chest X-ray was suggestive of pulmonary edema. She required assisted mechanical ventilation and noradrenaline infusion had to be started due to hypotension. Dobutamine infusion was considered but could not be given due to persistently low blood pressure. Fresh frozen plasma was transferred due to coagulopathy. On the same, late in the evening she went into asystolic arrest and cardiopulmonary resuscitation was unsuccessful.

DISCUSSION

Poisoning due to ingestion of colchicum autumnale is very rare in this part of India. The colchicine content of colchicum autumnale is 0.1-0.6%. Patients usually present with gastrointestinal symptoms like vomiting and diarrhea which is usually observed at doses less than 0.5mg/kg. Doses greater than 0.8mg/kg are almost fatal. Dehydration in our patient was part of fluid loss due to vomiting and diarrhea which finally led to tissue hypoperfusion resulting in acute kidney injury and hepatic necrosis. Acute cardiac failure was probably the result of direct toxic effect of colchicine on myocardial cells. In tissues without rapid cell turnover, such as the brain, heart, liver, kidney and pancreas, colchicine impairs cell process in which tubulin functions are involved: protein assembly in Golgi apparatus, cell shaping, mobility, endocytosis, transport and exocytosis [1-11] .

ISSN: 0975-8585

CONCLUSION

Colchicine poisoning should be considered in all patients with gastroenterocolitis after a wild plant meal. Diagnosis can be confirmed only by toxicology analysis. Management should include early intensive support measures despite relatively mild clinical picture at presentation.

REFERENCES

[1] Folpini A, Furfori P. J Toxicol Clin Toxicol 1995;33:71-77.
[2] Donovan JW. Nonsteroidal anti-inflammatory drugs and colchicine. In Clinical Management of Poisoning and Drug Overdose, 3rd edition. Edited by Haddad LM, Shannon MW, Winchester JE. Philadelphia, PA: WB Saunders Company; 1999:687-699.
[3] Stapczynski JS, Rothstein RJ, Gaye WA, Niemann JT. Ann Emerg Med 1981;10:364-369.
[4] Sabouraud A, Rochdi M, Urtizberea M, Christen MO, Achtert G, Scherrmann JM. Z Gastroenterol 1992; 30:35-39.
[5] Rudi J, Raedsch R, Gerteis C, Schlenker T, Plachky J, Walter-Sack I, Sabouraud A, Scherrmann JM, Kommerell B. Scand J Gastroenterol 1994;29:346-351.
[6] Bain L, Galloway D, Petrie J, Wood R. Br Med J 1974;1:446-448.
[7] Wallace SL, Omokoku B, Ertel NH. Am J Med 1970;48:443-448.
[8] Danel VC, Wiart JF, Hardy GA, Vincent FH, Houdret NM. J Toxicol Clin Toxicol 2001;39:409-411.
[9] Bismuth C, Baud F, Dally S. J Toxicol Clin Exp 1986;6:33-38.
[10] Mery P, Riou B, Chemla D, Lecarpentier Y. Intensive Care Med 1994;20:119-123.
[11] Borisy GG, Taylor EW. J Cell Biol 1967;34: 525–533