SIGMOID VOLVULUS: A RARE COMPLICATION OF ATROPINE THERAPY IN A PATIENT WITH ORGANOPHOSPHORUS POISONING: A CASE REPORT

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ABSTRACT

Various complications of atropine therapy in cases of organophosphorus poisoning have been reported. Among them, atropine induced paralytic ileus is a serious gastrointestinal complication with very few cases published worldwide. The tendency of over atropinization in organophosphorus poisoning despite regular monitoring is common and may develop life threatening complications as well. This necessitates knowledge, thorough examination, and an experienced approach to aid in treatment and complications management. We have discussed a case of organophosphorus poisoning, with symptoms of atropine toxicity and acute abdominal complication treated with surgical approach in our setting.

KEYWORDS

Organophosphorus poisoning, Atropine therapy, Sigmoid volvulus

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INTRODUCTION

Poisoning is a major public health problem in agricultural countries like Nepal[1] and organophosphorus (OP) poisoning is the most common entity.[2] OPs are commonly available as insecticides and pesticides. These substances are highly poisonous leading to rapid clinical deterioration on minimal exposure/ingestion.[3] The toxicity is due to irreversible inhibition of acetylcholinesterase (AChE) enzyme leading to accumulation of acetylcholine and subsequent over-activation of cholinergic receptors in the body. The clinical presentation of OP poisoning comprises of acute cholinergic crisis like salivation, lacrimation, urination, defecation, gastric cramps, emesis, intermediate syndrome, or delayed type polyneuropathy.[2] Atropine is the mainstay of therapy and can reverse life threatening features of OP poisoning. However, atropine on itself is also associated with antimuscarinic side effects.[4] Atropine induced paralytic ileus is a major gastrointestinal complication with some case reports documented worldwide.[5]

CASE PRESENTATION

A 35-year-old man with no known premorbid condition, chronic alcohol consumer (Eye opener) was referred to UCMS-TH from peripheral district hospital with the diagnosis of insecticide poisoning (Trade name: Udaan) i.e. Chlorpyriphos 50% and Cypermethrin 5%. He had alleged history of intentional poison ingestion under the influence of alcohol though the exact amount was not known. Following the ingestion, the patient developed nausea, vomiting, frothing from the mouth, restlessness, and shortness of breath. There was no history of seizure like activity. The case was primarily managed at a local hospital with gastric lavage, intravenous fluids (Normal saline), atropine and oximes (Inj. Pralidoxime 2 g i.v. stat). He was atropinized with 400 ml (240 mg) at primary center and referred with maintenance dose of atropine on normal saline drip during transport.

On arrival at our center, patient's GCS was E4V2M5, BP: 110/70 mmHg, heart rate 123 bpm, Respiratory rate 15, SPO2 97% in room air, GRBS 96 mg/dL. Bilateral pupil were fully dilated, axilla was dry, no secretions from mouth. Per abdomen examination showed soft, non-tender abdomen with no organomegaly and normal bowel sounds. Patient was started on maintenance atropine infusion at the rate of 40 ml per hour, kept NPO, IV fluids (DNS II pint and NS III pint over 24 hours), Inj. Thiamine 100 mg IV TDS, Inj. Pralidoxime 1gm iv TDS and shifted to MICU. Routine investigations were done on admission day. Full blood count and coagulation profile were normal. LFT showed mild AST elevation of 59 U/L only. RFT revealed serum creatinine of 1.5 mg/dL, Sodium 142.1, potassium 4.0, urea 53. Patient was periodically evaluated and downward dose titration was done on subsequent days. After 3 hours of infusion, patient started to show psychotic behaviors like inappropriate talking, laughing, crying, and being restless. Patient was restrained with consent of family members and treatment continued with assumption of atropine induced psychosis. Daily renal function was monitored with lab values and urine output charting.

On 5th day, patient's creatinine was normalized to 1.2 mg/dl. Serum sodium level was 144.9 mmol/L however, serum potassium came to be 3.4 mmol/L. Total 60 meq of KCL per day was added on IV fluids over 24 hours. On examination, abdomen was soft with sluggish bowel sounds. Patient has not passed stool since admission. On subsequent days, patients' serum potassium value remained 3.0, 3.3 despite intravenous potassium supplementation.

Patient's abdomen started to distend from day 7 with the complete absence of bowel sounds. On examination, abdomen was distended, non-tender, tympanic on percurssion. Patient didn't have any history of nausea or vomiting. However, patient had developed low grade fever 99.2 F with rise in WBC count of 20,900 (N82 L7 E2 M9 B0). Serum lipase was 15 U/L. X ray abdomen errect and supine was done which revealed grossly dilated large bowel loops with features suggestive of sigmoid volvulus. USG showed mild intra-abdominal collection with prominent bowel loop and reduced peristalsis.

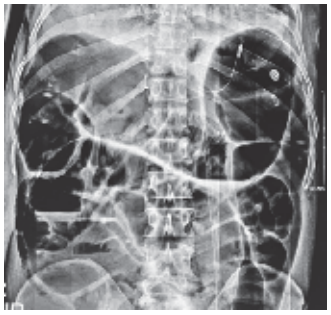

Fig 1. Supine abdominal x-ray

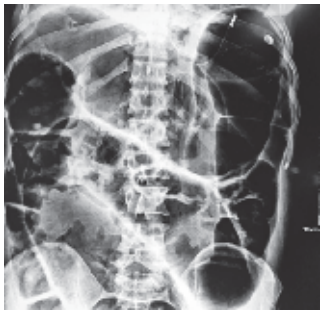

Fig 2. Erect abdominal x-ray

On that day, the patient's atropine was at the rate of 2 ml per hour infusion. The atropine was then stopped, and patient kept on close monitoring. However, no signs of atropine toxicity or rebound symptoms of poisoning recurred. Surgery consultation was done. The patient was kept on free NG drain with drainage of around 100 ml billous fluid over 24 hours. A stimulatory digital rectal examination was performed followed by passage of semisolid yellowish stool along with flatus. Patient was started on Inj. Meropenem and Inj. Metronidazole. No clinical sign of resolution of volvulus was noted with rising abdominal girth in the next 24 hours. Eventually, Hartmann's procedure was done for the definitive management of sigmoid volvulus.

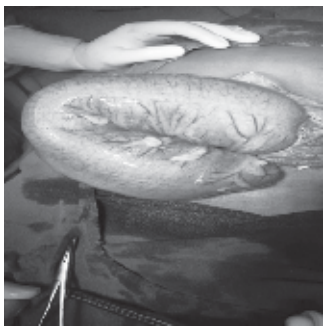

Fig 3. Sigmoid volvulus

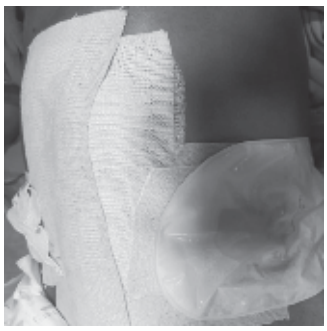

Fig 4. Colostomy

DISCUSSION

Organophosphorus compounds are easily available and widely used agricultural insecticide in Nepal. OP poisoning is a common incident encountered on day-to-day basis at emergency department. It classically presents with signs of cholinergic excess. OP compounds principally inhibit acetylcholinesterase enzyme by phosphorylating it. Some of the phosphorylated cholinesterase can dealkylate leading to aging which is a non-reversible state.[6] The central nervous system (CNS) does contain nicotinic and muscarinic receptors, and the toxic effects on the CNS include central respiratory depression, agitation, seizures, and coma.[7] Specific antidotes include atropine and pralidoxime. Atropine inhibits muscarinic receptors and causes a decrease in acetylcholine-induced cholinergic effects. Pralidoxime in contrast to atropine does not affect any specific receptors; rather it acts to regenerate acetylcholinesterase (AchE), which has been rendered non-functional by the OPs.

Though atropine has been established as a cornerstone on reversal of OP toxicities and survival of patients, few detrimental adverse effects of it can't be overlooked. The most common adverse effects of atropine are related to the drug's antimuscarinic properties, including xerostomia, blurred vision, photophobia, tachycardia, flushing, and hot skin. Constipation, difficulty with urination, and anhidrosis can occur, especially in at-risk populations (most notably, the elderly).[8] In rare cases, delirium or coma may occur.

Atropinic property is an important cause of paralytic ileus. It may be an idiosyncratic phenomenon, as well as dose related or, it may occur when in combination with other drugs predisposing to bowel immobility, such as opiates.[5] Reduction of gastrointestinal muscle tone and gastric secretion are well established effects of atropine. Despite this, paralytic ileus as a complication of atropine therapy or indeed atropine toxicity is extremely rare.[9] The rarity of gastrointestinal side effects reflects the relative insensitivity of gastromotive choline receptors to atropine inhibition.[10] Moreover, variable nature of individual patient sensitivity to atropine cannot be denied.[11] In our case, as the patient was atropinized with high dose and was under high maintenance dose during titration, it might have predisposed to the paralytic ileus. In addition, development of sigmoid volvulus can be due to retaiment of stool for longer duration due to atropine induced ileus. Also, pre-existing predisposing cause to volvulus could be a reason. Persistent hypokalemia in later days of admission might be one of the reason for persistent paralytic ileus despite reduction in dose of atropine.

In sigmoid volvulus (SV), the sigmoid colon wraps around itself and its mesentery. SV accounts for 2% to 50% of all colonic obstructions and has an interesting geographic dispersion.[12] It generally affects adults, and it is more common in males. The etiology of SV is multifactorial and controversial. Our case was also an adult male with higher dose atropine for longer duration of time. Thus, the clinical correlation of sigmoid volvulus in this particular patient can be due to the same reason. The occurrence of sigmoid volvulus in patients of OP poisoning under atropine therapy is very rare and it needs further study to establish the causality.

CONCLUSION

The value of life saving drug atropine for OP poisoning comes with a price of few adverse effects, most of which are mild and reversible. However, the appearance of paralytic ileus with subsequent sigmoid volvulus requiring surgical intervention on patients without known premorbid conditions could alarm us on giving second thought on adverse drug reaction profile of this drug. Along with atropine toxicity various confounding factors might have led to the volvulus. As this is just a single case report, more study is required to establish causal association of atropine with occurrence of sigmoid volvulus.

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CONFLICT OF INTEREST

None

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