

Toxic megacolon as a rare complication following atropine therapy due to organophosphate poisoning: A case report

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Abstract

The main therapeutic basis for a case of organophosphate poisoning is a combination therapy which includes atropine as an anticholinergic drug and pralidoxime. If the poisoning is severe, a high dose of this combination of medicines may be needed, but this may cause serious side effects: paralytic ileus or even megacolon; however, these gastrointestinal events are very rare. Here, we report a case of organophosphate poisoning where atropine therapy was given and led to drug-associated toxic megacolon.

Keywords

Organophosphate, poisoning, atropine, toxic megacolon

Introduction

The treatment of organophosphate poisoning involves a combination therapy that includes atropine as an anticholinergic drug and pralidoxime.¹ In some severe poisoning cases, a high dose of this combination therapy is needed which may cause the patient to suffer serious side effects including a paralytic ileus or even megacolon, and patients with inflammatory bowel conditions are particularly vulnerable.² In other words, severe dilatation of the colon may be reported following the use of certain anticholinergic agents such as atropine³; however, this complication is very rare. We report a case of organophosphate poisoning where treatment that included atropine therapy finally led to drug-associated toxic megacolon.

Case presentation

The patient was a 52-year-old man who was referred to the emergency ward of our hospital with loss of consciousness and a history of consuming a known amount of organophosphate pesticides within the last 24 h. On admission, the patient was not responding to painful stimuli and had no gag reflex. Because of abundant airway and bronchial secretions, the patient was intubated and connected to mechanical ventilation.

His vital signs included systolic/diastolic blood pressure of 90/52 mmHg, with a heart rate of 45 beats/min and arterial oxygen saturation of 67%. On examination, he did not respond to painful stimulus with bilateral miotic pupils and bilateral absent plantar reflexes. Pulmonary examination demonstrated coarse crackles in all lung lobes. The abdomen was soft without distension. In initial echocardiography, the left ventricular ejection was estimated to be 40%. After primary critical supportive measures, a right subclavian vein line was inserted and intravenous atropine 10 mg was ordered via this venous line that led to increase in heart rate (125 beats per minutes) and O₂ saturation (92%) with limiting crackles to lung bases. The findings in initial

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arterial blood gas included PH of 7.59, PCO_2 of 79 mmHg, HCO_3 of 18, and base excess of 10. One litre of normal saline serum was ordered for the patient, and the blood pressure was maintained at 105/70 mmHg. Two hours later, the arterial blood gas parameters changed significantly as a PH of 7.43, PCO_2 of 55 mmHg, HCO_3 of 19, and base excess of -3.1 . First laboratory assessments, recorded: a white blood count of $15,100/mm^3$ with neutrophil percentage of 86%, serum haemoglobin level of 13.8 gr/dl, and platelet count of $130,000/mm^3$. Regarding level of biomarkers, CKMB was measured to be 48 and troponin to be 0.91. The levels of serum magnesium was 1.9 mg/dl, creatinine 0.8 mg/dl, calcium 8.5 mg/dl, sodium 143 mmol/L, potassium 3.7 mmol/L, AST 47 mg/dl, ALT 23 mg/dl, CPK 423 mg/dl, and serum cholinesterase 176 microkat/l. The patient was treated with a combination of atropine (2.5 to 3.5 mg/h, intravenously), pralidoxime (500 mg/h, intravenously), along with bicarbonate 15 meq/day. On day 4 of hospitalisation, there were indications of megacolon in a chest X-ray (Figure 1). Liver ultrasonography showed a fatty liver. In spiral multislice lung CT (computed tomography) scan without contrast (Figure 2), minimal pleural effusion was evident in right hemithorax, and mild pneumothorax was also found on the left side. Atelectasis was revealed at the dependent portion of both lower lobes, and a small consolidation with air bronchogram was evident in the anterior of the right upper lobe suggesting pneumonia. Spiral multislice abdomen and pelvic CT scan (Figure 3) revealed: sigmoid colon dilatation with the diameter up to 110 mm in the left upper quadrant with normal diameters of other portions of colon confirming a final diagnosis of megacolon. No evidence of complete obstruction was noted in the CT scan. No evidence of sigmoid volvulus was found. The imaging of

other organs including kidneys, liver, spleen, pancreas, and adrenals was normal. Maintenance therapy with syrup of lactulose and rectal tube insertion was continued, and a percutaneous tracheostomy was performed on day 10. About 20 days later, Electromyogram (EMG)/Nerve Conduction Velocity (NCV) of quadriceps muscles of the thigh was performed that seemed to be normal. Atropine and pralidoxime were tapered gradually. The level of serum cholinesterase reached 1803 and 3209 microkat/l in the days 26 and 33 after treatment, respectively. The patient was taken off the ventilator on day 30.

Comments

Some anticholinergic drugs and agents that can reduce intestinal motility or delay intestinal transit time may cause toxic megacolon.⁴ For this reason, Lomotil (a combination of atropine and diphenoxylate) should be used cautiously or even discontinued when

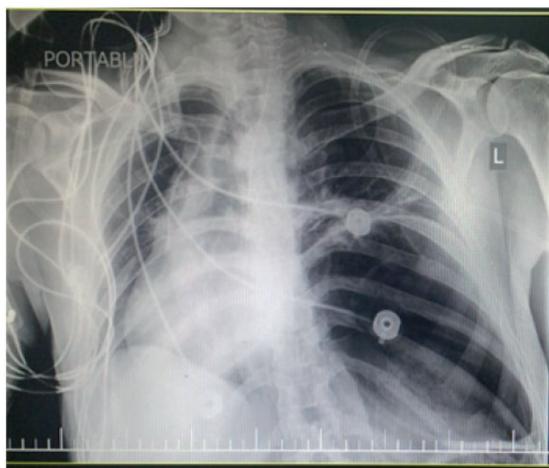


Figure 1. Evidences of megacolon on chest X-ray.

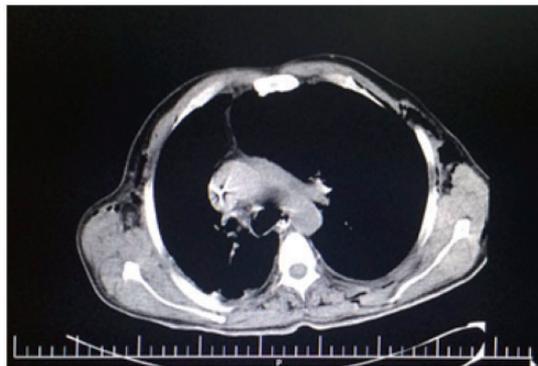


Figure 2. Spiral multislice lung CT scan without contrast indicates minimal pleural effusion with mild pneumothorax and atelectasis.

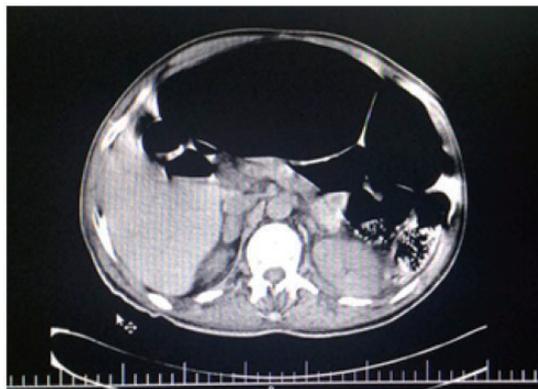


Figure 3. Spiral multislice abdomen and pelvic CT scan indicate colon dilatation with megacolon diagnosis.

abdominal distension occurs or if there is any evidence of toxic megacolon as gastrointestinal symptoms and side effects are predictable with this drug. Adverse effects include inactivity of gastrointestinal peristalsis that may lead to colon paralysis and even megacolon.⁵ The abdomen should therefore be monitored for distension and abdominal pain which should be followed by imaging studies. Some previous studies indicate there is an increased likelihood of megacolon following atropine and as noted by Siegmund,⁶ patients treated with atropine developed megacolon. This highlights the importance of normally functioning ganglion cells being present to prevent this condition. The pathophysiological basis of megacolon following atropine remains unclear; however, it seems to be associated with delaying intestinal motility or the destruction of intestinal ganglion cells.⁷

In our case, soon after atropine was administered, there were clinical and imaging signs of megacolon, but the EMG/NCV findings were normal.

Accordingly, the risk of developing megacolon should be considered when prescribing atropine.

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