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Paralytic Ileus after Prolonged Transient Hypocholinesterasemia Induced by Fenitrothion Poisoning

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Abstract The patient was a 62-year-old woman who was found in an unconscious. She had paroxysmal atrial fibrillation, dyslipidemia and anxiety neurosis. On arrival, she was in a state of deep coma with hypotension and hypoxia. She underwent tracheal intubation and continuous infusion of noradrenalin. Her serum cholinesterase (butylcholinesterase) level was 2 IU/L. Organophosphate poisoning was suspected. On day three, she regained clear consciousness and accepted that she had intentionally ingested a bottle of fenitrothion with suicidal intent. On the 3rd day, her serum cholinesterase level decreased to 0 U/L, and remained at this level until the 7th day. Prolonged artificial ventilation was required due to weak respiration. On the 14th day, she presented shock, a deterioration of consciousness, a distended abdomen with the peritoneal stimulation sign. She was diagnosed with paralytic ileus with septic shock. Her general condition gradually improved with supportive therapy. At the end of treatment in our hospital, she barely could walk and feed for herself and was transferred to another hospital for rehabilitation. To our knowledge, this is the first reported case of delayed paralytic ileus after prolonged transient hypocholinesterasemia induced by fenitrothion poisoning, in which atropine was not used as treatment. Physicians should pay attention to the complication of paralytic ileus when a patient with organophosphate poisoning shows prolonged hypocholinesterasemia.

Keywords: *fenitrothion, ileus, delay, peritonitis*

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1. Introduction

Organophosphates and carbamates have a wide variety of applications, most commonly as pesticides used to eradicate agricultural pests or control populations of disease-carrying vectors. [1] Both classes inhibit acetylcholinesterase, leading to the excessive accumulation of acetylcholine at nerve terminals. In the setting of toxicity, clinical syndromes result from excessive nicotinic and muscarinic neurostimulation. In the acute phase, the most common clinical signs of poisoning were miosis, bronchorrhea, vomiting, diarrhea, hypotension; in rare cases, patients may develop acute respiratory insufficiency and acute cardiocirculatory failure. [2] Ileus has been reported as a complication after the administration of atropine for organophosphate poisoning. [3,4] We herein report a rare case of paralytic ileus after prolonged transient acholinesterasemia induced by fenitrothion poisoning, even though the patient was not treated with atropine.

2. Case History

A 62-year-old woman visited the department of neurosurgery in our hospital with complaints of sleepless, dysarthria and a general loss of strength. However, no neurological condition was detected and a head computed tomography (CT) study was negative; thus, she returned home. On the next day, she was found in an unconsciousness state by her husband at noon. She was transported to our hospital. She had paroxysmal atrial fibrillation, dyslipidemia and anxiety neurosis, and was prescribed eszopiclone, setiptiline maleate and bisoprolol fumarate. On arrival, she had a Glasgow Coma Scale of 3, a systolic blood pressure of 60 mmHg, a heart rate of 60 beats per minute, a respiratory rate of 22 breaths per minute, and an SPO2 value of 70% on room air. Her pupils were isocoric and her light reflex was dull. She underwent tracheal intubation and continuous infusion of noradrenalin. When the nasogastric tube was inserted, the gastric juice appeared an artificial color with an off-flavor;

and drug intoxication was therefore suspected. A urinary drug test was positive for barbiturate and tricyclic antidepressant. A second whole body CT study was negative. The main results of a blood analysis were as follows: white blood cell count, 12,600/μL; hemoglobin, 16.1 g/dL, Glucose 253 mg/dL; serum cholinesterase (butylcholinesterase), 2 IU/L. Organophosphate poisoning was suspected based on the clinical symptoms and her low cholinesterase level. During an investigation by her family, an empty bottle of fenitrothion was found in her home. On day three, her consciousness became clear and she accepted that she had ingested a bottle of fenitrothion with suicidal intent. On the 3rd day, her serum cholinesterase level decreased to 0 U/L until the 7th day and prolonged artificial ventilation was required for weak respiration. Thus, tracheostomy was performed on the 5th day (Figure 1). However, on the 14th day, she displayed shock, a deterioration of consciousness, distended abdomen with peritoneal stimulation sign. Enhanced CT showed swelling of the whole bowel with mild ascites without free air (Figure 2). Macroscopic melena was not observed; however, a fecal occult blood test was positive. She was diagnosed with paralytic ileus with septic shock, and was treated with massive fluid resuscitation, cardiopressor and antibiotics. An ascites culture later revealed *Enterobacter cloacae*; thus, she was diagnosed with paralytic ileus with idiopathic bacterial peritonitis, probably due to bacterial translocation from the swollen bowel. [5] Her general condition gradually improved in response to the increase in her serum cholinesterase level. Muscle weakness, which predominantly affected the lower extremities, developed as a complication, probably due to critical illness neuro-myopathy or delayed neurotoxicity due to organophosphate poisoning. [6] At the end of treatment, she could barely walk or feed herself, and she was transferred to another hospital for rehabilitation.

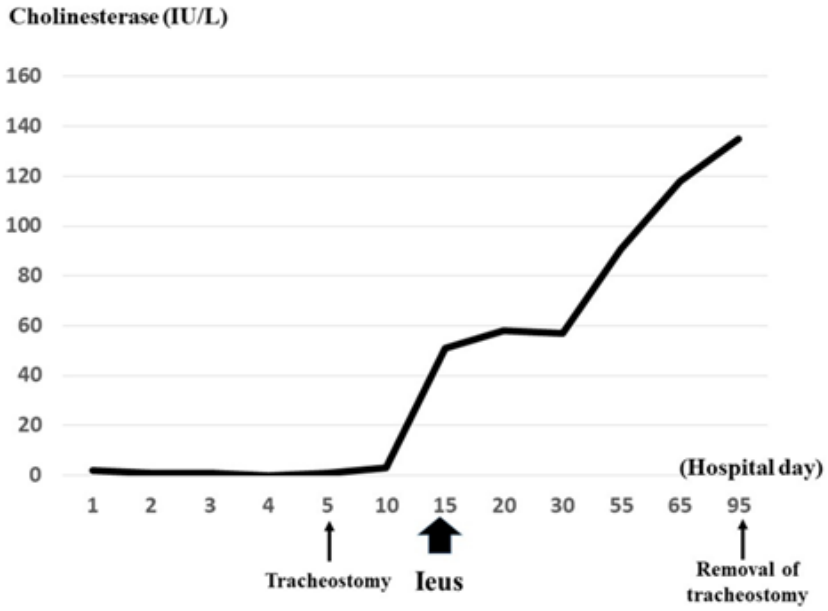

Figure 1. The time course of the serum cholinesterase level From hospital day3 to 7, the serum cholinesterase level was 0 IU/L.

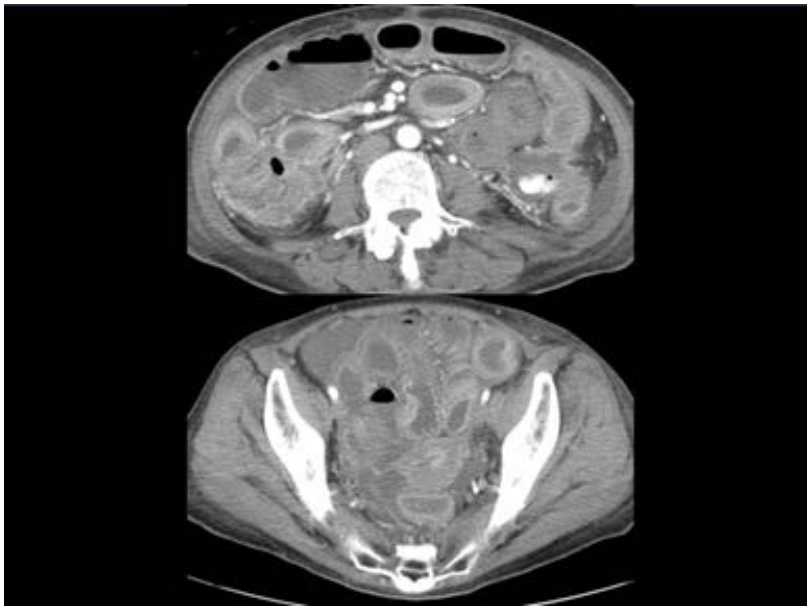

Figure 2. Enhanced computed tomography (CT) after deterioration Enhanced CT showed swelling of the whole bowel with mild ascites without free air.

3. Discussion

To our knowledge, this is the first case report of delayed paralytic ileus after prolonged transient acholinesterasemia and hypocholinesterasemia induced by fenitrothion poisoning in which atropine was not used as a treatment.

There are two hypotheses regarding the formation of paralytic ileus after fenitrothion poisoning without atropine use. One is that paralytic ileus is induced by direct injury of the bowel by fenitrothion. Organophosphate poisoning is typically associated with significant gastrointestinal manifestations that include vomiting, diarrhea, cramps and increased salivation in the acute phase. [7] In addition, organophosphate poisoning directly induces gastrointestinal complications, like mucosal erosion, gastric ulcers with perforation and peritonitis, duodenal ulcer and multiple small intestine perforations with peritonitis in the acute and subacute phases. [7] However, the present case did not show melena or free air suggesting perforation, and abdominal manifestation occurred on the 14th hospital day, suggesting that the possibility of direct injury to the bowel by fenitrothion was low.

The other hypothesis is that paralytic ileus was induced by prolonged acholinesterasemia or hypocholinesterasemia. Prolonged serum hypocholinesterasemia suggested that the level of acetyl-cholinesterase in the bowel wall was low. [8] The reflexive activities of the gastrointestinal tract are regulated, in part, by precise interactions between the neurons and glia in the enteric nervous system (ENS). [9] Enteric glial acetylcholine in the ENS and glial activation by acetylcholine is a physiological mechanism that contributes to the functional regulation of the intestinal reflexes. Accordingly, the dysfunction of the ENS induced by an abnormal level of acetylcholine may have induced the paralytic ileus in the present case. This may be a kind of delayed neurotoxicity in the bowel due to the inhibition of neurotoxic esterase, such as lower extremity motor paralysis and nervous erethism.

Tanabe et al. reported a case in which delayed severe lower intestinal hemorrhage requiring a total of 30 units of transfusion occurred 2 weeks after the ingestion of an organophosphorus pesticide. [10] Their case was complicated by severe stenosis and passage disturbance in the colon, which required partial colectomy. They hypothesized that the inclusion of a spreading agent, as an additive, may have caused delayed gastrointestinal damage. The pesticide in the present case contained xylene and ethylbenzene, in addition to fenitrothion; however, there have been no reports of ileus induced by xylene or ethylbenzene.

4. Conclusion

To our knowledge, this is the first reported case of delayed paralytic ileus after prolonged transient hypocholinesterasemia induced by fenitrothion poisoning in which atropine was not used as a treatment. Physicians should pay attention to the complication of paralytic ileus when a patient with organophosphate poisoning shows prolonged hypocholinesterasemia.

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