ACCIDENTAL ORGANOPHOSPHATE POISONING: TWO CASE REPORTS

VIKNESWARI S. PARAM PALAM
ZAINAL ABIDIN
GRACIE ONG
T. J. LOH
A. E. DELILKAN

SUMMARY

Accidental organophosphate poisoning may occur in persons coming in close contact with animals being treated with organophosphate pesticides. The poisoning may manifest itself as a severe systemic disorder, but can be diagnosed by an alert physician and confirmed by specific tests of reduced cholinesterase activity in the blood, plasma and red blood cells. Treatment is with intravenous atropine. [1] *Supportive measures may be necessary.* [2]

INTRODUCTION

Careless use of organophosphate pesticides on animals to keep them free from fleas can result in unforeseen danger to their owners. Accidental organophosphate poisoning may occur in such persons and may endanger their lives. Two case reports are presented for discussion.

Case 1

A four-year-old Indian boy was admitted to the Klang District Hospital with complaints of vomiting, diarrhoea and dyspnoea. These symptoms had developed suddenly, two hours after food intake. He had been well until a fortnight before admission, when he developed similar abdominal complaints of diarrhoea and vomiting after intake of food, for which he was also admitted into the Klang Hospital and given a course of antibiotics. Since then he had been having abdominal pains on and off, associated with loose stools.

On examination, the boy was dyspnoeic with excessive salivation, pinpoint pupils, muscle-twitching and abdominal pain. On auscultation, there were coarse crepitations in both lung fields. The heart sounds were dual and regular with a pulse rate of 80. A clinical diagnosis of organophosphate poisoning was made, and he was given a stomach washout, intravenous atropine and pralidoxime. He responded to the treatment, pupils returned to normal size, pulse rate increased to 160, there was cessation of excessive salivation and muscle-twitching, and on ausculation, the lungs revealed clear, air entry.

He was referred to the University Hospital for further management. On admission, he was afebrile, the pulse was 120, the blood pressure was 120/80, the respiration was 24. He was drowsy but well-orientated and responsive to questioning. The pupils were equal and reactive. There was no papilloedema. Muscle tone was increased, and the plantar reflexes upgoing. Examination of the respiratory and cardiovascular systems revealed no abnormality.

Vikneswari S. Param Palam, MBBS
Zainal Abidin, MRCP
Gracie Ong, FFARACS
T. J. Loh, MRCP
A. E. Delilkan, FFARACS
Department of Anaesthesiology
Department of Pediatrics
Department of Medicine, Faculty of Medicine
University of Malaya, Kuala Lumpur

The haemoglobin was 9.8%, the white cell count was 21,800, with 93% neutrophils, 6% lymphocytes, 1% monocytes. Serum sodium was 123 meq/litre, serum potassium was 5.3 meq/litre, blood urea was 30 mg/100 ml, blood sugar was 80 mg/100 ml and prothrombin time was 56%. Serum salicylate was 4.4 mg/100 ml. The serum aspartate aminotransferase (SGOT) was 14 IU/litre. The serum alanine aminotransferase (SGPT) was 8 IU/litre. Arterial blood gas analysis was: PaO_2 103 mm Hg, $PaCO_2$ 34.9 mm Hg, pH 7.35, base excess -5.4 meq/litre. Cerebrospinal fluid analysis showed per microlitre RBC 30 and WBC 2, sugar 90 mg/100 ml and protein 8 mg/100 ml. Blood cholinesterase levels were 37% of normal value, with 37.5% in the red blood cells, and 12.5% in plasma.

He was treated accordingly as a case of organophosphate poisoning in the Intensive Care Unit with continuous intravenous atropine. He became restless the next day; he was flushed, pupils were large. The atropine was discontinued. Cholinesterase level in the red blood cells was 43.8%, and serum cholinesterase level was 25%. He was discharged into the paediatric ward, and sent home the day after.

Case 2

A 47-year-old man was admitted into the University Hospital because of a sudden onset of profuse sweating, giddiness and nausea. He had been well until a week prior to admission, when he had a similar episode which responded to intravenous glucose and stemetil. He was a heavy drinker and smoker, but had abstained from drinking after the first episode.

He was afebrile, pulse rate was 84, the blood pressure was 210/120. He was drowsy but was responsive and orientated. Pupils were pin-point, breathing laboured, and speech slightly slurred. There was profuse sweating and excessive salivation. There was visceral incontinence. On auscultation, the heart sounds were dual and regular. The lung fields were clear. Examination of the abdomen revealed no abnormality. Examination of the central nervous system revealed mild left cerebellar signs with incoordination, pastpointing and tremor. Muscle tone was decreased, reflexes depressed and plantar down-going. There was some sensory loss at T_{12}. Examination of the cranial nerves revealed third and seventh cranial nerve palsy. The gag reflex was questionable.

The haemoglobin was 14 g %. Blood urea was 32 mg/100 ml blood glucose was 80 mg/100 ml. Serum sodium was 136 meq/litre, serum potassium was 3.1 meq/litre, and chloride was 101 meq/litre. The serum aspartate aminotransferase (SGOT) was 14 IU/litre, and the serum alamine aminotransferase (SGPT) was 4 IU/litre. Serum creatinine was 1.9 mg/100 ml, uric acid was 10.6 mg/100 ml. A provisional diagnosis of severe hypertension with a brain stem infarction was made. Because of the bizarre neurological picture, blood cholinesterase levels were requested for. The cholinesterase levels were zero, thus raising the possibility of acute organophosphate poisoning. Intravenous atropine was given with good results. Blood pressure came down to 130/90. Pupillary size increased to 3 mm. The palsy improved. He was admitted into the Intensive Care Unit for a few days and then discharged into the general medical ward, where he was found to have gout and hypertension. Serum cholinesterase levels recorded an improvement. A psychiatric assessment found him to have no suicidal tendencies.

DISCUSSION

Organophosphate poisoning is usually associated with a deliberate attempt at suicide. Yet accidental organophosphate poisoning, though not so frequently seen, can occur and the symptoms and signs may be just as dramatic, and the outcome just as tragic, as in the deliberate ingestion of the poison.

The depression of blood cholinesterase levels by the organophosphate can lead to cholinergic and muscarinic over-activity manifested in profuse sweating, diarrhoea and muscle fasciculation. Estimation of blood cholinesterase levels in the University Hospital takes two hours, and active medical treatment should be given, based on a clinical diagnosis, whilst awaiting laboratory results. Cholinergic over-activity can be reversed with atropine, and support of the respiratory system by elective ventilation may be required. [2]

Accidental organophosphate poisoning from contaminated food in children has been reported.[3] Accidental organophosphate poisoning from improper handling of food and organophosphate compound can also happen as our report shows. In the case of the child, there was an element of tragedy in the family as the boy had lost his brother a year ago from somewhat similar, but undiagnosed complaints in a hospital. As a result, he had developed a close attachment to their pet dog. The mother being concerned, had tried to keep the pet dog as clean as possible. The dog was therefore treated regularly by the child's nanny with an organophosphate pesticide. This powder was kept on the dog for a few days at each application before being washed off. The nanny was also responsible for the preparation of the child's food and for feeding the child. The initial complaint of diarrhoea and abdominal cramps after food in our patient had been associated with the delousing of his pet dog, but suspicion of organophosphate poisoning had not been raised.

It was only two weeks later with the second episode of abdominal pain and diarrhoea after food that an alert doctor at Klang District Hospital made the diagnosis. This was confirmed by laboratory estimation of blood cholinesterase levels. Mishandling of the organophosphate compound, accidental contamination of food, absorption through skin through close play with the animal could have resulted in the symptoms arising in the child.

In the case of the adult, he had a race horse on which he had recently started using an organophosphate pesticide regularly. Absorption through the skin, or ingestion of the poison from the cigarettes or food contaminated by careless handling could have happened. There had been an initial episode of giddiness and profuse sweating two prior to his admission, for which he was treated with intravenous glucose and stemetil. The bizarre neurological pattern in the present admission alerted the physician in charge and cholinesterase levels were found to be severely depressed, thus confirming the clinical suspicion of organophosphate poisoning.

CONCLUSION

In conclusion, accidental organophosphate poisoning may be a dramatic event, and systemic involvement may mimic systemic disorders. However a combination of diarrhoea, excessive salivation and muscle fasciculation should alert the medical practitioner to the possibility of an organophosphate poisoning. Direct questioning of exposure to organophosphate compounds for example as an organophosphate pesticide for animals, may prove helpful.

There should be a clinical response to the administration of intravenous atropine and the estimation of blood cholinesterase levels, if facilities are available, could be confirmatory.

REFERENCES

[1]Ganendran A. Organophosphate insecticide poisoning and its management. *Anaesth Intens Care* 1974; 4: 361-368.

[2]Chew LS, Chee KT, Yeo JMS, Jayaratnam FJ. Continuous atrophine in the management of organophosphate insecticide poisoning. *S'pore Med J* 1971; 12: 80-85.

[3]Kanagaratnam K, Wong HB, Tan KH. Parathion poisoning from contaminated barley. *Lancet* 1960; 538-542.