Case Report

Recurrent Hypoglycaemia in Organophosphorus Compound Poisoning – An Unusual Complication

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ABSTRACT

Organophosphorus compound poisoning commonly presents with muscarinic, nicotinic and central nervous system manifestations. Endocrinal complications of various organophosphorus compounds are rare and cannot be explained by commonly known mechanisms. We report a clinically proven case of organophosphorus compound poisoning in a young male, in whom at least 22 episodes of hypoglycaemia (blood sugar levels in the range of 47-80 mg %) were observed over 2 weeks during his hospitalization. Hypoglycaemic episodes completely disappeared upon recovery from poisoning. The exact mechanism for such recurrent hypoglycaemia requires detailed evaluation.

This report emphasizes the need to observe blood sugar levels in patients with organophosphorus compound poisoning, so as to detect and promptly treat this rare yet important and correctable complication.

Key Words: Organophosphorus compound; Hypoglycaemia

Introduction

Organophosphorus (OP) compound poisoning commonly presents with muscarinic, nicotinic and central nervous system effects. Endocrinal effects are rarely described. Here we report a case of OP poisoning in whom we observed frequent episodes of hypoglycaemia.

The Case: A 24 year old male farmer was brought to the hospital with alleged history of consumption of unknown substance, possibly an insecticide according to his father. He displayed classical muscarinic, nicotinic and central nervous system features of OP poisoning. His serum cholinesterase level was 153 U/L (normal value: 5320-12920 U/L). He was managed along standard lines of therapy with atropine, pralidoxime, ventilation and supportive care.

After initial rapid improvement, his clinical condition began to deteriorate with development of intermediate syndrome and respiratory infection. Broad spectrum antibiotics were started and assisted ventilation was continued. After 14 days his condition started improving and gradually he was weaned off from the ventilator. However he was detected to have vocal cord palsy with secondary aspiration, which only partially responded to therapy. He was discharged against medical advice after total hospital stay of 25 days.

During the period of his ICU admission in the hospital, we observed frequent episodes of hypoglycaemia. His random blood sugar on admission, checked by glucometer was 156 mg/dl. After 24 hours of routine screening, it dropped to 47 mg/dl. This was cross-checked in the laboratory also by standard methods. Since then frequent monitoring of blood sugar was done and regular parenteral glucose administration was given followed later on by administration through NG tube. We observed a total of 22 episodes of hypoglycaemia (blood sugar below 80 mg/dl) over a period of 2 weeks, out of which on 13 occasions the levels hovered between 61 and 70 mg/dl, and on 6 occasions they were even lower (less than 60 mg/dl).

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Upon recovery from muscarinic and nicotinic effects, the hypoglycaemic episodes also subsided. An attempt was made to find out the cause of recurrent hypoglycaemia. Hepatic and renal function tests (repeated twice) were normal. Investigations for pancreatic involvement and specialized tests including serum insulin level, C peptide level, and tests for adrenal insufficiency were not done.

Discussion

Common causes of hypoglycaemia in any critically ill patient include liver disease, sepsis, drug-induced, renal failure and chronic malnutrition.[1] They were ruled out in this case by appropriate clinical and laboratory evaluation. Lack of temporal co-relation of hypoglycaemic episodes with sepsis did not favour sepsis as the sole cause for hypoglycaemia, although it could have contributed to it.

As the hypoglycaemic episodes disappeared upon recovery from muscarinic and nicotinic effects of OP poisoning, chronic underlying endocrinal causes such as islet cell and non-islet cell tumours were also unlikely.

This indicates the possibility of direct endocrinal effect of OP compound. Autonomic nervous system, which is modified by organophosphorus compounds, plays an important role in the regulation of insulin secretion. Stimulation of the vagus nerve or the administration of parasympathomimetic agents both are known to stimulate insulin secretion.[2,3] In one experimental study by Permutt, cholinergic blockade caused decrease in serum insulin level resulting in hyperglycaemia.[4] Two observational studies in OP poisoning showed hyperglycemia more often than hypoglycaemia, although possible mechanisms were not mentioned in either of the studies.[5,6]

The effect of OP compounds on pancreas is mentioned in literature. These compounds by their effect on cholinergic innervation of pancreas, may cause increase in the secretion from acinar pancreatic glands and cause pancreatitis with hyperglycemia.[7,8] One case report suggested acute pancreatitis could possibly be caused by OP poisoning.[9] The rodenticide "vacor" has been described as toxic to the pancreatic beta cells, causing massive release of stored insulin with profound hypoglycaemia, followed later by diabetes.[7]

In our patient, detailed endocrinal and toxicological evaluation for finding out the exact substance as well as mechanism of hypoglycaemia could not be done. Nevertheless this case emphasizes the need to monitor blood sugar constantly in OP compound poisoning.

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