Case study

Unintended Inhalational Organophosphate Poisoning Presenting As Angioedema: A Rare Case Report

Abstract:

Organophosphorus compounds are chemical agents in widespread use throughout the world, mainly in agriculture. In developing countries organophosphorus (OP) poisoning is a commonly encountered problem. Here we present a rare case of OP poisoning with angioedema as the presenting feature. Prompt treatment of these cases will help in reducing the morbidity and mortality.

Keywords:

organophosphate poisoning, angioedema, atropine

Introduction:

In Indian population, especially, the rural areas, one of the major public health concerns is suicidal poisoning by organophosphorus pesticide,due to its cheap and easy accessibility. Since organophosphorus poisoning is associated with high mortality and morbidity it is of prime importance to the clinicians[(1)]. Early diagnosis should be made to initiate prompt treatment in order to save the patient's life. Sometimes the presentation is atypical and onset of symptoms are delayed[(2)]. In such cases, even when the symptoms are mild in initial stages , still the patients should be observed for longer periods of time.There have been no previous case reports of angioedema seen in organophosphate poisoning patient.

Case Report:

A 22-year-old male (weight 56 kg) was admitted in our hospital because of acute organophosphate (OP) poisoning. He had history of inhalational poisoning with monocrotophos and profenophos while spraying insecticide in a farm. He

had multiple episodes of vomiting the following day and was brought to the hospital by his mother. He had swelling of both eyes and upper lip since one day. He had difficulty in deglutition suggestive of laryngeal edema and ryle's tube was inserted. At the time of hospitalization, he was conscious and oriented. He had no history of diabetes mellitus, hypertension and psychological problems. On admission, his vital signs revealed a pulse rate of 68/min, blood pressure of 110/70 mmHg, respiratory rate of 18/min, afebrile, and O2 saturation of 97%. Neurological examination was done andGlasgow Coma Scale was 15/15.On local examination he had edema around both eyelids and swelling of the upper lip (Figure 1).Pupils were myotic on examination. On respiratory examination bilateral crepitations were present. Gastric lavage via nasogastric tube was done in the casualty.

Laboratory investigations revealed hemoglobin as 14.9g/dL, total leukocyte count 10,400/cumm and platelet count 2,32,000/cumm. Peripheral smear showed predominantly normocytic, normochromic RBC's, PT INR was 1.07, PT was 12.8 (PT control was 11.9) and APTT was 30.6 (APTT control was 29.5). His serum sodium was 143mg/dL, potassium was 4.6 mg/dL. Liver enzymes was normal (Aspartate aminotransferase of 30mg/dL and Alanine aminotransferase of 27mg/dl, total bilirubin 0.9mg/dL, conjugated bilirubin 0.2mg/dl, unconjugated bilirubin 0.7mg/dL, albumin 4.4g/dL, globulin 3.3g/dL). Serum creatinine was 0.6mg/dL, urea was 13mg/dL. Electrocardiogram, and chest X-ray were normal.There was decreased serum cholinesterase in our patient of level 0.2 mg/dL.Fluid therapy was started, and atropine therapy was administered. He did not have hypotension or respiratory distress. In view of angioedema of face IV hydrocortisone 100 mg and chlorpheniramine 10 mg were given. The general condition of the patient improved after management. After he became conscious oralcetirizine was also given. To improve the clinical manifestations of OP poisoning, pralidoxime infusion was also started for the patient. After 5 days of hospitalization, he was discharged from the hospital in stable condition.

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Comment [RW3]: must mention type of crepts . also describe CVS.

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Comment [RW6]: Must mention route and dose of Atropine- important

Comment [RW7]: When did he loose consciousness. His GCS was 15/15

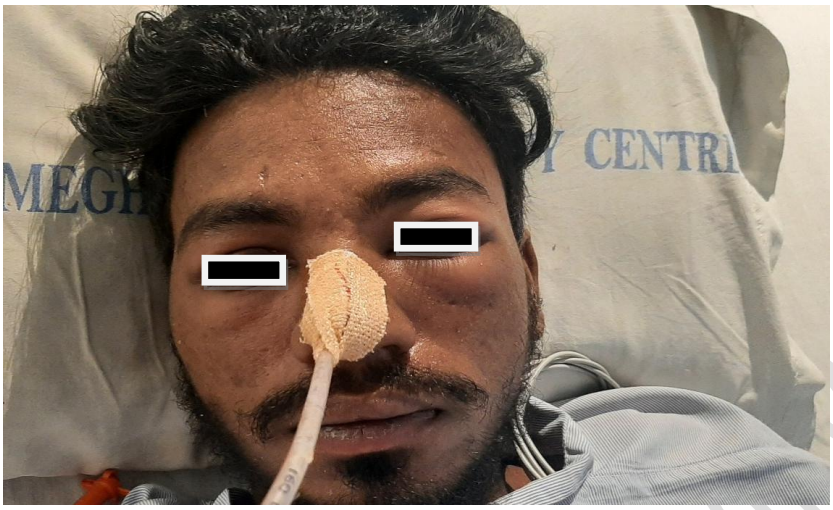

Figure 1: Angioedema around eyelids and swelling of upper lip

DISCUSSION:

In organophosphorus poisoning the symptom onset depends on the route of exposure. The common routes are oral, inhalation, occasionally parenteral and through skin. The onset of symptoms is most rapid in inhalational poisoning occurring over a few minutes. In farmers, while spraying insecticides, the direction of wind may result in accidental inhalation exposure. The duration of symptom onset in exposure through skin depends on few factors like the skin intactness, solubility and amount of organophosphoruscompound[3,4]. In organophosphorus poisoning after oral ingestion, the time lag period for symptom onset would depend on the amount consumed and the absorption characteristics of the OP compound. Usually the symptoms occur within few minutes to hours. In India OP pesticides are commonly used for suicide purposes particularly in the rural areas. Another important mode of poisoning is occupational exposure. The outcome of OP poisoning depends on the dose, route of exposure and the time interval before starting treatment[5].The clinical features can be broadly classified as secondary to (i) muscarinic effects, (ii) nicotinic effects and (iii) central receptor stimulation. Three different types of paralysis are recognized largely based on the time of occurrence and their differing pathophysiology (i) Type I paralysis or acute paralysis, (ii) Type II paralysis or Intermediate syndrome and (iii) Type III paralysis or Organophosphate induced delayed neuropathy[6]. Cholinesterase estimation is

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the only useful biochemical tool for confirming exposure to op. In ingestional poisoning gastric lavage should be given and the sample collected must be sent for analysis and medico legal purposes. Atropine is the specific antidote for muscarinic effects and has no effect on nicotinic symptoms. It helps in reversing life threatening complications that may result in death such as central respiratory depression, bronchospasm, severe bradycardia and hypotension[7]. Current guidelines recommend use of incremental dose regimen followed by setting up an infusion to follow these end points. Oximes are also administered and their mechanism of action is by reactivating acetylcholinesterase that has been bound to the op molecule[8].

Conclusion:

This case highlights the unusual presentation of OP poisoning. Eliciting a proper and detailed history and other clues found on examination will help in making an early diagnosis. The physicians have to be aware of the various intoxication syndromes caused by OP so that prompt and appropriate treatment can be provided. The various OP compounds have different outcomes due to its unique characteristics. All this should be kept in mind when approaching a patient with OP poisoning.

COMPETING INTERESTS DISCLAIMER:

Authors have declared that no competing interests exist. The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

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