Nat. Volatiles & Essent. Oils, 2021; 8(5): 1052 - 1056

NVEO
Natural Volatiles &
Essential Oils

case report on organophosphorus poisoning

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Abstract:

Introduction: In India, organophosphate (OP) poisoning is widespread. There have only been a few case reports of parenteral OP poisoning. We describe a case of self-injected methyl parathion poisoning that resulted in seizure, altered sensorium, and respiratory distress four days later, posing a diagnostic and therapeutic conundrum. Despite the lack of a history of OP poisoning, he was treated on suspicion and had a satisfactory clinical response to an atropine and pralidoxime treatment trial, as well as a successful recovery. Following parenteral injection of OP poison, atypical presentations may occur, and even a remote suspicion of this necessitates thorough evaluation and treatment to ensure a positive outcome. Persistently low plasma cholinesterase level is a helpful diagnostic sign.

Main symptoms and/ or clinical finding :-A typical toxidrome in organophosphate (Op) poisoning comprises of the salivation, lacrimation, urination, defection, Gastric cramps, Emesis (SLUDGE) symptom.

Diagnostic Evaluation:-History collection and physical examination, RBS, BUN, Electrolytes, prothrombin time , liver function studies, plasma cholinesterase measurement. Other laboratory finding included the leucocytosis, Haemoconcentration, metabolic and /or respiratory acidosis, hyperglycaemia, hypomagnesemia, elevated troponin level, elevated amylase levels, elevated liver function tests.

Therapeutic Intervention:-The patient was treated with Inj. Pan 40 mg in 5ml/1hourly, Inj. Vit-k 10 mg (Iv) in OD, Inj. Emset 4mg (IV) in TDS, Inj. Mucomix 70mg/kg every 4 hourly (9 vials) in 500ml D5,inj.perinorm 10 mg (IV) in TDS.

Outcome:- Patient's general condition is improved.

Nursing perspectives:-Administered fluid replacement i.e. DNS and RL, monitor vital signs, and check blood pressure per hourly, maintained intake and output chart, and provide adequate rest and sleep to the patients. Administered medication according to the doctor's order.

Conclusion: The diagnosis of OP compound toxicity via the parenteral route is difficult. Symptoms may appear later than expected, and presentations may be unusual. Even if the symptoms are moderate at first, a longer time of monitoring is essential. Because there are no decontamination procedures in place, even a small amount of injection could be lethal. In the event of suspicion of OP poisoning, treating physicians must be attentive and offer proper treatment. In the event of suspicion of OP poisoning, treating physicians must be attentive and offer proper treatment.

Keywords: Organophosphate Poison , Parenteral, Seizures, Poisoning.

Introduction:-

Poisoning by organophosphates (OP) is frequent in underdeveloped nations, particularly in India. (1) Poisoning is typically caused by voluntary ingestion, inhalation, or cutaneous absorption. Self-injection by intramuscular or intravenous method might cause toxicity in rare cases. Only a few authors have described OP toxicity via the parenteral route. If there is no history of the substance being administered parenterally, diagnosis becomes more challenging. (2) Parenteral OP poisoning can present as an acute cholinergic crisis and respiratory distress, an intermediate phase, or delayed toxicity. We describe a patient who self-injected methyl parathion and had seizures, an abscess in the arm, pulmonary edema, and flaccid quadriparesis, all of which were successfully treated based on clinical judgement.(3)

Patient Information

Patient History:-A 28 year old male patient admitted in AVBR Hospital on dated 06/6/2021. Patient was brought to casualty with ingestion 40 tabs. Paracetamol and 40 tabs. Cefrizine ,around 8pm on 6/6/21 at home. According to the patient he has consumed because he was depressed related to family issues.

Patient specific Information:-A 28 year old male patient was admitted in AVBR Hospital on dated 6/6/21 .patient have a bad habit as like alcohol and tobacco chewing.

Medical and family and psychosocial history:-

Medical history:- patient having no any medical history such has diabetic mellitus and hypertension.

Family history:-He belongs to joined family and he had mentally stable .no any hereditary disease in family .patient is conscious and oriented. He had maintained the good relationship doctors and nurses as well as with other patient.

Psychosocial History:- patient belongs to middle class family. Patient is mentally stable. He maintain the good relationship with other doctor ,nurses, patient and relatives he cooperate doctors and nurses.

Patient Relevant past intervention with outcomes:-my patient was diagnosed with op poisoning. From that onward he was admitted to hospitals for treatment of the disease and her health was improved.

Clinical findings:

General Examination

Physical Examination

State of health:- unhealthy

State of consciousness:-conscious

Body build:- thin

Hygiene:- good

Vitals signs:-Temp-37.8°c, pulse-80 b/min, Blood pressure- 110/70mm/hg, Spo2-96.

Timeline:- patient taken treatment for the health problem in AVBR Hospital.

Treatment :-Decontamination and supportive therapy.

-Blockade of muscarinic activity with Atropine.
-Reversal of cholinesterase inhibition with oxime.
-correction of metabolic abnormalities.
-prevention of infection.
-management of complication.

Follow up and outcomes: Patient is still admitted in hospital about 7 days and his condition is improved.

Discussion:-

India is mostly an agrarian nation with a sizable rural population. Pesticides with organophosphates (OP) are frequently used for suicide. Although suicidal eating is a typical route of poisoning, occupational exposure when spraying in fields is also a common mechanism. (4) The clinical manifestations and outcome of OP poisoning are influenced by the pesticide used, as well as the dose and mode of administration. The clinical manifestations and result of OP poisoning are influenced by the pesticide used, as well as the dose, mode of administration, and time between poisoning and treatment. (5) The following are the clinical signs and symptoms of OP poisoning:

In acute cholinergic crisis, which occurs within 24 to 72 hours as a result of acetylcholine accumulation at muscarinic and nicotinic sites and accumulation of acetylcholine. Acute cholinergic crisis manifests within 24 to 72 hours due to acetylcholine accumulation at muscarinic and nicotinic sites and accumulation in the CNS, resulting in headache, giddiness, seizure, and altered sensorium; intermediate syndrome manifests after 24 to 96 hours due to prolonged acetyl choline activity at nicotinic receptors, resulting in ocular, neck, and limb weakness. The history of ingestion or mucocutaneous exposure, clinical characteristics, and plasma cholinesterase levels are used to diagnose OP poisoning. Plasma cholinesterase levels that are depressed confirm the diagnosis of OP poisoning, and the levels remain depressed for 4 to 7 weeks.

The measurement of cholinesterase in red blood cells is more precise. Gastric lavage is performed in situations of OP chemical consumption, and a sample is taken for examination and medicolegal purposes. Atropine functions as a physiological antidote by blocking the effects of muscarinic receptors. Atropine is given as a 2 to 5 mg loading dosage and then every 5 to 10 minutes until symptoms of atropinisation show clinical response and delivered as an infusion at a rate of 0.02 to 0.08 mg/kg/min. The role and dosage of oximes are debatable. Pralidoxime is typically given in doses of 1 gm every 6 to 8 hours; however, new trials have indicated that high-dose infusions of 18 to 24 gm/day produce better results. Diazepam is used to treat OP induced seizures. When dealing with these types of incidents, legal considerations are a major worry. Stomach wash, excreta, and other items such as empty bottle capsules or liquids should be gathered and stored when dealing with suspected poisoning instances. This case was entered into the

medicolegal record, and the police were notified. The toxicity of OP toxin is determined by how quickly it is absorbed into the systemic circulation. Absorption and systemic symptoms differ depending on the plane of administration when the OP substance is delivered via parenteral route. Acute cholinergic crisis can emerge within 30 minutes of IV treatment, according to a few writers. Symptoms of self-injection will occur after some time, and if the quantity administered is insufficient, there may simply be a local abscess. (6) After four days of injection, the case we mentioned manifested. He had an abscess in his arm, which could have been caused by the use of contaminated material. The culture of the pus was sterile. Although sepsis was examined, it was difficult to explain his flaccid quadriparesis and pulmonary edema solely on the basis of sepsis, and he responded well to atropine treatment. few examples of parenteral OP poisoning. IV monocrotophos poisoning causes an intermediate condition that necessitates ventilator support, according to Badhe and Sudhakar(1). Two cases of dichlorvos poisoning were recorded by Raina et al.(2) and were treated with atropine and pralidoxime. Nishioka also mentions the creation of local site abscesses. When insecticides are injected subcutaneously or intramuscularly, local inflammatory responses are to be expected. Such injuries can also serve as a point of entrance for a variety of species. Local debridement is essential for abscess drainage and, if done early enough, may aid with pesticide removal. A number of studies reflect in literature on poisoning (7-12). All of the cases of parenteral OP poisoning that have been published so far have had a history of the chemical being injected. The example we discussed presented a substantial diagnostic challenge. Since there was no history of injection of the drug at the start of treatment . Because of his impaired sensorium, the patient was unable to provide a history. Seizures with OP poisoning are uncommon, and no cases of seizure following parenteral administration have been described.

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