A Case Report on Organophosphate Poisoning

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Abstract

Organophosphate (OP) compound acute poisoning is a serious worldwide clinical issue that results in thousands of fatalities annually. Most pesticide poisoning cases that result in death happen in underdeveloped nations when the poison is purposefully consumed by the victim. Two regularly consumed OP insecticides are Metacid (Methyl parathion) and Nuvan (Dichlorvos). These OP pesticides are hazardous because they irreversibly inhibit the acetylcholinesterase (AChE) enzyme, which causes acetylcholine to build up and cholinergic receptors to become overactive throughout the body. These individuals initially exhibit delayed polyneuropathy, intermediate syndrome, and cholinergic crisis. The diagnosis is based on the patient's medical history, the pesticides' known toxicity manifestations, and the alleviation of symptoms following atropine administration. The same fundamental management techniques used for any acutely poisoned patient are also applied to the supportive therapy of OP poisoning, namely prompt initial management of breathing, circulation, and airways. Activated charcoal and gastric lavage are common decontamination techniques. The cornerstone of treatment is atropine, which can cure the acute poisoning's potentially fatal symptoms.

Keywords: Organophosphate, poisoning, Gastric Lavage, acute poisoning, insecticide ingestion.

INTRODUCTION

In hospitals and intensive care units in developing nations, organophosphate (OP) poisoning remains a common cause of admission.

[1] The conventional method of analyzing the clinical characteristics of acute OP poisoning has focused on the effects of certain receptors on the central nervous system (CNS), muscarinic, and nicotinic receptors, which produce a variety of symptoms and indicators. Given that atropine reverses muscarinic effects but not nicotinic neuromuscular effects, this traditional classification of clinical characteristics is helpful.

[2] Moreover, medications that can permeate the blood-brain barrier—such as atropine—have a higher propensity than medications that do not reverse symptoms and indicators of the central nervous system. Assessing clinical aspects using the period of symptom onset may be an alternative method, typically, salivation, lacrimation, urination, defecation, gastric cramping, and emesis (SLUDGE) symptoms manifest abruptly within minutes to hours after exposure to OP.

[3] Nevertheless, some patients experience delayed effects following a period of mild or absent clinical features, or following an initial phase of severe cholinergic symptoms and signs.

[4] Patients who experience acute symptoms related to neuromuscular weakness may go on to develop delayed symptoms and signs of various neuronal sub-systems. These symptoms and signs might manifest as a continuum.

[5] The third strategy, which is organ-specific, has concentrated on how OP affects the nervous system, the lungs, or the heart.

PATIENT INFORMATION

An 18 years old male patient, was referred by HH (Himalayan Hospital) to SMIH (Shri Mahant Indresh Hospital) in emergency ICU for ingestion of OP poisoning and was previously diagnosed HBsAg positive. The patient was not alcoholic/ smoker. No family or genetic history of psychological disorders. No history of diabetes, hypertension, bipolar, suicidal attempts, or any abnormal/psychological behavior.The patient

ingested 10-20ml organophosphate poison around 5 PM in the evening, and first episode of vomiting occurred 5 to 10 minutes after the ingestion, which was noticed by patient's uncle, who immediately took him to the nearby hospital i.e. HIMALAYAN HOSPITAL. Where activated charcoal, gastric lavage was done and atropine was given. The patient was admitted there for about 2 hours after which he was referred to SMIH.

CLINICAL FINDINGS

Patient came with chief complaint of vomiting and abdominal pain in umbilical region since ingestion of OPC. Patient was conscious and oriented. Blood pressure – 110/70 mm/Hg. Heart rate was 123bpm. SpO2 – 97% on RA. Respiratory rate was 46/min. Temp – afebrile. Patient was advised for admission in Medicine ICU, and some serological tests were performed, which includes CBC, HBsAg, ANTI HCV, LFT, RFT, ANTIHBe AB, Brain MRI plain, Ultrasound whole abdomen, X-RAY chest, Electrolytes serum, with strict vitals monitoring.

DIAGNOSTIC ASSESMENT

TEST	30/7/23	31/7/23	4/8/23	7/8/23
AMYLASE TEST (U/L)	71 N			
LIPASE TEST (U/L)	99 N			
ANTI HCV	NON-REACTIVE			
BLOOD C & C	NO GROWTH			
DENGU	NEGATIVE			
T. PROTEIN (g/dl)	6.8 N			
ALKALINE PHOSPHATE (U/L)	137 H			
SERUM T. CHOLESTEROL (mg/dl)	137 L			
SERUM BILIRUBIN DELTA (mg/dl)	0.40 H			
SERUM ALBUMIN (g/dl)	3.5 N			
SERUM UREA (mg/dl)	18 L		34 N	
SERUM CRETININE (mg/dl)	0.7 N		0.6 L	
SERUM URIC ACID (mg/dl)	4 N			
S. SODIUM (mmol/L)		133 L	140 N	138 N
S.POT (mmol/L)	5.8 H	4.1 N	5 N	4.8 N
S. CALCIUM (mg/dl)		8.4 N	8.7 N	9.1 N
USG W/A		PANCREAS OBSCURED BY BOWEL		

		GASES. NO ABNORMALITY FOUND		
MRI BRAIN		**THORNWALDT CYST MEASURING 1.09 X 0.8cm IS SEEN IN NASOPHARYNX POSTERIOURLY. NO ABNORMALITY FOUND**		
CHEST X RAY		**NORMAL**		

L* LOW, **N*** NORMAL, **H*** HIGH

VITALS MONITORING

	29/7	**30/7**	**31/7**	**1/8**	**2/8**	**3/8**	**4/8**	**5/8**	**6/8**	**7/8**	**8/8**	**9/8**	**10/8**
BP	110/70	120/70	130/70	110/70	130/70	120/80	110/70	120/80	110/70	110/70	110/70	110/70	120/70
PR	123	100	74	66	88	107	93	80	111	96	90	93	94
RR	46	20	20	18	18	22	18	18	18	18	20	24	16
TM	AFB	FB	AFB	AFB	AFB	AFB	AFB	AFB	AFB	AFB	AFB	AFB	AFB
SpO 2	97	98	98	97	98	98	98	98	98	98	97	96	95

BP* (blood pressure in mm/Hg) **PR*** (pulse rate per min) **RR***(respiratory rate per min) **TM*** (temperature) **SpO2* ON RA** (oxygen saturation), **AFB*** (Afebrile)

NURSING ASSESMENT ON GCS
Day 2 – E3V2M6, Day 4 – E4V2M6, Day 5- E3V2M6, Day 8 – E4V5M6

On day 3 of patient admission, he was referred to PSYCHIATRY for suicidal attempt as a case of OP POISONING WITH HBsAg +. **FINDINGS:** No suicidal tendency was noticed in patient. NO PAST HISTORY OF; abnormal body movement/ head injury, substance use, any self-harm attempt, psychiatry symptoms.

On day 11 patient was shifted to private ward, having blood pressure, pulse rate, respiratory rate, temperature, and oxygen level under control conditions.

THERAPUTIC INTERVENTION

DAY 1 (30/7/23)

DRUG	DOSE	FREQUENCY	ROUTE
INJ. RULPIME SB	3gm	12 hrly	IV
INJ. PAM	500mg	12 hrly	IV
INJ. PANTOP	40mg	12hrly	IV
INJ. SBC	30ml	8 hrly	IV
TAB. ACTIVATED CHARCOAL IN 500 ml LOOZ	4 tbsp	12 hrly	RT
SYP. LACTIHEP	10ml	8 hrly	RT
INJ. ATROPINE	1 amp	8hrly	IV

DAY 2 (31/7/23): CST (CONT. SAME TREATMENT)
DAY 3 (1/8/23)

INJ. RULPIME SB	3gm	12 hrly	IV
INJ. PAM	500mg	12hrly	IV
INJ. PANTOP	40mg	12hrly	IV
INJ. ATROPINE	1amp	8hrly	IV
SYP. LACTIHEP	6tbsp	8hrly	RT

DAY 4 (2/8/23)

INJ. RULPIME SB	3gm	12 hrly	IV
INJ. PAM	500mg	12hrly	IV
INJ. PANTOP	40mg	12hrly	IV
INJ. ATROPINE	1amp	8hrly	IV
SYP. LACTIHEP	6tbsp	8hrly	RT
INJ. LEVIPIL	1gm	12hrly	IV
TAB. CHARCOAL	2tbsp	8hrly	RT

DAY 5 (3/8/23): CST
DAY 6 (4/8/23)

INJ. RULPIME SB	**3gm**	**12 hrly**	**IV**
INJ. PAM	500mg	12hrly	IV
INJ. PANTOP	40mg	12hrly	IV
SYP. LACTIHEP	6tbsp	8hrly	RT
TAB. CHARCOAL	2tbsp	8hrly	RT
INJ. LEVIPIL	500mg	12hrly	IV

INJ* (INJECTION), **SYP***(SYRUP), **TAB***(TABLET), **AMP***(AMPOULE), IV***(INTRA VENOUS).

DAY 7 (5/8/23) TO DAY 13 (11/8/23): CST
The patient was managed conservatively on IV and oral medications. Patient was stable on day 14 and was discharged with follow up in medicine OPD after 15 days. Advise on discharge INJ. LEVIPIL 500mg BD, TAB CEFTUM 500mg BD, TAB PRAZO DSR 40 OD, CAP KALVO OD.

DISCUSSION

About two-thirds of people with OP poisoning have cardiac symptoms. [7] Typical ECG observations include T wave irregularities, QTc prolongation, and ST-T segment alterations. Additional cardiac symptoms include ventricular premature complexes, hypo- or hypertension, sinus bradycardia or tachycardia, supraventricular and ventricular arrhythmias, and noncardiogenic pulmonary edema. [8] Conversely, OP poisoning frequently results in respiratory problems. The muscarinic effects of rhinorrhea, bronchorrhea, bronchospasm, and salivation led to hypoxemia and increased respiratory effort. [9] Nicotinic effects lead to paralysis and weakening of the muscles and increase the risk of hypercapnic respiratory failure [10]. The central causes of seizures, agitation, and restlessness worsen respiratory function. [11] Early in OP poisoning, there are digestive symptoms that can be quickly resolved with atropine therapy.There are worries that atropine increases the duration of OP toxicity and slows down intestinal transit time [12]. In one instance, the OP was shown to remain in the stomach for ten days following poisoning.[13] In individuals who have been exposed to OP, atropine therapy may also prevent early enteral feeding.[14] However, only 6.9% of patients receiving enteral feeds in a pilot study experienced gastric stasis following the early introduction of hypocaloric feeds (by 48 hours).[15] In 12.8% of cases of OP

poisoning, pancreatitis is documented [16]. Additionally discussed are metabolic problems such as hyperglycemia, glycosuria, and OP poisoning that manifest as diabetic ketoacidosis. [17]

CONCLUSION

Diverse perspectives on the indications and symptoms of OP poisoning could enhance our comprehension of the fundamental mechanism, so aiding in the treatment of individuals who are acutely contaminated. [18] According to recent studies, the majority of patients were young, and there were more female patients than male patients. [19] Suicidal poisoning was more common than inadvertent poisoning [20]. The most frequent symptoms that patients reported were nausea and vomiting, but the most frequent symptom that the research team's treating physicians saw was miosis. [21]

REFFERENCES

[1] Aman, Sakib, Shrebash Paul, and Fazle Rabbi Chowdhury. "Management of organophosphorus poisoning: standard treatment and beyond." *Critical Care Clinics* 37.3 (2021): 673-686.

[2] Eddleston, Michael, and Fazle Rabbi Chowdhury. "Organophosphorus poisoning: the wet opioid toxidrome." *The Lancet* 397.10270 (2021): 175-177.

[3] Chen, Junyi, et al. "Synergistic enhancement of the emergency treatment effect of organophosphate poisoning by a supramolecular strategy." *Chemical Science* 12.14 (2021): 5202-5208.

[4] Dong, Ning, et al. "Development of a practical prediction scoring system for severe acute organophosphate poisoning." *Journal of applied toxicology* 40.7 (2020): 889-896.

[5] Paudyal, Buddhi Prasad. "Organophosphorus poisoning." *JNMA; journal of the Nepal Medical Association* 47.172 (2008): 251-258.

[6] Marrs, Timothy C. "Organophosphate poisoning." *Pharmacology & therapeutics* 58.1 (1993): 51-66.

[7] Kumar, Subhas Vijay, et al. "Current review on organophosphorus poisoning." *Archives of applied science research* 2.4 (2010): 199-215.

[8] Peter, John Victor, Thomas Isiah Sudarsan, and John L. Moran. "Clinical features of organophosphate poisoning: A review of different classification systems and approaches." *Indian journal of critical care medicine: peer-reviewed, official publication ofIndian Society of Critical Care Medicine* 18.11 (2014): 735.

[9] Thiermann, H., et al. "Modern strategies in therapy of organophosphate poisoning." *Toxicology letters* 107.1-3 (1999): 233-239.

[10] Minton, Neil A., and Virginia SG Murray. "A review of organophosphate poisoning." *Medical toxicology and adverse drug experience* 3 (1988): 350-375.

[11] Sungur, Murat, and Muhammed Güven. "Intensive care management of organophosphate insecticide poisoning." *Critical care* 5 (2001): 1-5.

[12] Worek, Franz, et al. "Diagnostic aspects of organophosphate poisoning." *Toxicology* 214.3 (2005): 182-189.

[13] Banerjee, Indranil, S. K. Tripathi, and A. Sinha Roy. "Clinico-epidemiological characteristics of patients presenting with organophosphorus poisoning." *North American journal of medical sciences* 4.3 (2012): 147.

[14] Chowdhary, Sheemona, Rajasri Bhattacharyya, and Dibyajyoti Banerjee. "Acute organophosphorus poisoning." *Clinicachimica acta* 431 (2014): 66-76.

[15] Karki, P., et al. "Cardiac and electrocardiographical manifestations of acute organophosphate poisoning." *Singapore medical journal* 45.8 (2004): 385-389.

[16] Singh, Gagandeep, and Dheeraj Khurana. "Neurology of acute organophosphate poisoning." *Neurology India* 57.2 (2009): 119.

[17] Samantasinghar, Pusparaj. "Clinical patterns and outcomes of acute poisoning cases in a tertiary care hospital in Bhubaneswar, India."

[18] Thirunavukkarasu, S., S. Radhakrishnan, and R. Shankar. "Serum Amylase A Prognostic Indicator in Acute Organophosphorous Compound Poisoning." *International journal of health sciences* 6.S8: 5028-5035.

[19] Parate, Tilottama Ramesh, Shweta Vasant Madavi, and Ramesh Parate. "Study of Levels of Creatine Phosphokinase and Lactate Dehydrogenase as Prognostic Markers in Acute Organophosphorus Poisoning." *Vidarbha Journal of Internal Medicine* 33.2 (2024): 57-62.
[20] Hosen, M. A., et al. "Clinical Profile and Outcome of Organophosphorus Poisoning in a Tertiary Hospital." *Mymensingh Medical Journal: MMJ* 33.2 (2024): 509-515.
[21] Siddique, Mohammad Abu Naser, Imran Uddin Robel, and UmmayFatema Khatun. "Pattern of Pesticide and Herbicide Poisoning Among People Admitted to Medicine Wards of a Tertiary Care Hospital." *Medicine Today* 36.1 (2024): 36-40.