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A Case of Acute Pancreatitis in Organophosphate Poisoning

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Abstract

Organophosphate (OP) poisoning is one of the most harmful and easily accessible pesticides for suicide poisoning. It is linked to high mortality and morbidity. Cholinergic syndrome, intermediate syndrome, and syndrome of delayed polyneuropathy are the three primary syndromes that characterize OP poisoning. Organophosphates are strong toxins that are frequently used as pesticides in agriculture. Patients who have consumed organophosphorus may exhibit subclinical symptoms of acute pancreatitis. Correct biochemical analysis and clinical correlation assist in diagnosis. In the study, we described a 24-year-old female who suffered necrotizing pancreatitis after OP poisoning.

Keywords: Organophosphate Poisoning, Pesticide, Pancreatitis.

Introduction

One of the major public health issues facing the world today is poisoning. In terms of global mortality, it is ranked 45^{th[1]}. One of the most frequent causes of poisoning in developing nations like India is organophosphate (OP) poisoning^[2]. Pesticides have contributed to dramatic increase in quality and quantity in crop yields. Organophosphates (OP) are commonly used as insecticides in agriculture in India and are potent toxicants. Strong cholinesterase inhibition effect. Acute (within minutes to 24 hours), delayed (between 24 hours to 2 weeks), and late (beyond 2 weeks) onset symptoms are the three categories for OP poisoning symptoms. Acute onset

symptoms are caused by the action of nicotinic and muscarinic receptors. It manifests as abdominal pain, diarrhea, vomiting, sweating, hypersalivation, muscle spasm. Acute pancreatitis induced by OP has been reported sporadically, but very few of them belong to severe necrotizing types. Here, we report a 24 years old female who developed necrotizing pancreatitis after OP poisoning.

Case Report

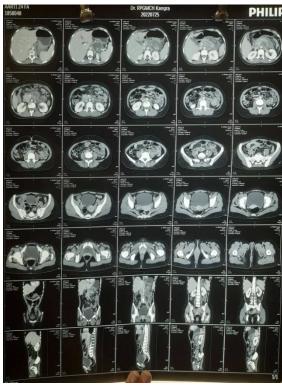
A female presented to Chamba Medical College with vomiting and excessive salivation after ingestion of dichlorvas. She was given atropine for 3 days and referred to Dr RPGMC for pain abdomen from 2 days, radiating to back and increased on taking meals. She did not pass stools and flatus from 2 days

Vitals of patient were

BP 120/60mm Hg , PR 110/min, Spo2 97% , RR 24/min, GCS 15/15, Pupils - mid dilated and equally reacting to light , Per abdomen - soft but tender in epigastric region and Chest was clear.

Table no. 1: Investigations

S. No.	Investigation	Day 1	Day 3
1	Haemoglobin	12.2g/dl	
2	TLC	10,000/microL	
3	Platelet Count	1,68,000/microL	
4	Urea/ Creatinine	18/0.6 mg/dL	
5	AST/ALT/ALP	22/21/102 mmol/L	
6	Amylase/Lipase	133/294	
7	Na+/K+/Ca+2	137.5/3.6/9.2	
8	LDH	622U/L	
9	ESR	70mm/1hr	
10	CRP	156mg/l	47.8mg/l
11	Procalcitnin	3.43ng/ml	1.49ng/ml
12	Triglycerides	148	
13	ABG analysis	pH 7.43 pO2 87.3 pCO2 26.9 HCO318.3	
14	IgM Scrub/ Dengue/ Lepto	Negative	
15	HIV/HBV/HCV	Negative	
16	USG abdomen	Bulky pancreas upto 27.8mm in body region with no peripancreatic fluid	
17	CECT abdomen done on day 7 of pain abdomen	Acute pancreatitis (<30% necrosis) with acute necrotic collection and ascites. Modified CTSI 8/10	



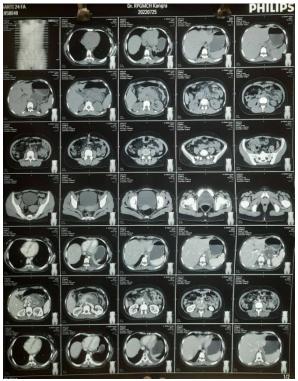


Fig 1 Fig 2

Fig 1 and 2: Showing CECT Abdomen of patient with Acute pancreatitis

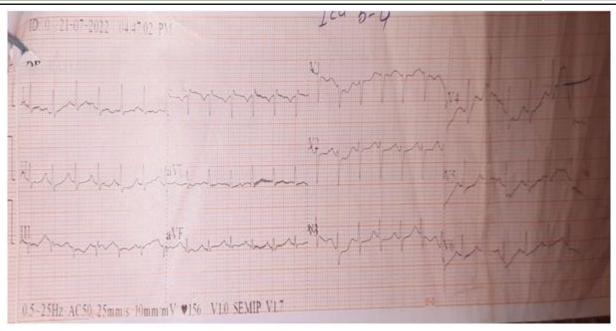


Fig 3: Showing ECG with sinus tachycardia

Diagnosis and Management

Patient was managed as **Acute Pancreatitis**

- Empirically antibiotics Ceftriaxone and Doxycycline were started i/v/o Acute abdomen
- Kept NPO and iv fluids were given
- As the pain settles, liquids allowed followed by semi solid diet
- Discharged on day 12 of poisoning with stable vitals

Discussion

Hyperamylasemia hyperglycemia with and presumptive pancreatic damage have reported but acute pancreatitis is seldom reported in OP poisoning. Hyperamylasemia due to OP also caused by intestinal ischemia, enteritis, and hypersalivation due to the direct action of OP^[3]. Dressel's et al. showed in canine experiments that OP, because an increase in intraductal pressure and excessive pancreatic flow rate that results in extravasation of fluids^[4]. LeBl and and Sergeyeva revealed pancreatic acinar cell vacuolization following cholinergic stimulation by Ach^[5]. Lankisch et al. described two cases of painless acute pancreatitis manifested by paralytic ileus only subsequent to the ingestion of parathion. One of the patients died following a delay in the diagnosis^[6]. Spectrum of clinical findings in acute pancreatitis. Sahin et al. studied 46.95% had elevated serum amylase (>200 S.U) and though ultrasonography was carried out in all the patients, only one showed a swollen pancreas, which was confirmed by CT^[7]. While reports of acute pancreatitis following OP intoxication are infrequent, missed or delayed diagnosis of acute pancreatitis may lead to increased morbidity and even reported mortality, serum pancreatic enzyme estimations and abdominal ultrasonography or CT should be considered in the early assessment of patients with OP intoxication.

Conclusion

Although pancreatitis is a rare complication of organophosphate poisoning, it is necessary to evaluate patients with OP poisoning with serum amylase levels, USG Abdomen and CECT Abdomen in patients with pain abdomen for early detection and management of acute pancreatitis.

Conflicts of Interest

All contributing authors declare no conflicts of interest.

Source of Funding

None.

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