Original Article

Clinico-epidemiological Profile of Acute Pancreatitis Secondary to Organophosphate Poisoning in Children at a Tertiary Care Centre: Case Series

Manu Prakash S K[1], Kumar S R[2], Harish H N[3], Suma N[4]

[1]Associate Professor, [2,3]Assistant Professor, [4]Postgraduate, Department of Paediatrics, Hassan Institute of Medical Sciences, Karnataka, India.

How to cite this article: Manu Prakash S K, Kumar S R, Harish H N et al. Clinico-epidemiological Profile of Acute Pancreatitis Secondary to Organophosphate Poisoning in Children at a Tertiary Care Centre: Case Series. Indian Journal of Public Health Research & Development 2023;14(4).

Abstract

Organophosphate poisoning is common poisoning seen in developing countries. Accidental poisoning is rare in children but adolescents consume it with suicidal intention. Complications following op compound poisonings are well known but children developing acute pancreatitis is a rare complication.[1-2] Hence we present a case series of children developing acute pancreatitis in op compound poisoning. All five cases were treated conservatively and successfully discharged.

Keywords: organophosphate poisoning, Acute pancreatitis.

Introduction

Organophosphate poisoning is common poisoning seen in developing countries[3], as they are extensively used as pesticides for protection of vegetable and fruit crops. children and women are being the usual victims. Accidental poisoning is rare in children but adolescents consume it with suicidal intention.

Common organophosphate compounds are

Chloroothion, Diazinon, DEP (di-isopropyl fluorophosphate), Malathion, Methyl parathion, OMPA (Octa Methyl pyrophosphoramide), Parathion, TEPP (Tetraethyl pyrophosphate), THIO-TEPP, HETP (Hexaethyl tetraphosphate)[4]

Op compounds irreversibly inhibits cholinesterase causing accumulation of acetylcholine at NM junction resulting in stimulation of autonomic nervous system, central nervous system and skeletal muscle leading to symptoms of organophosphorus compound poisoning. Complications following op compounds are well known however children developing acute pancreatitis due to OP compound is one of the rare complications. Following op compound poisoning there is increase in exocrine secretions of pancreatic fluid in pancreatic duct resulting in increase in pressure causing pancreatitis which usually disappears in a week. Acute pancreatitis as a complication of OP compound poisoning has been infrequently addressed. Early recognition and appropriate therapy for acute pancreatitis may lead to improved outcome[5]. Here we report five rare

Corresponding Author: Suma N, Postgraduate, Department of Paediatrics, Hassan Institute of Medical Sciences, Karnataka, India.

E-mail: sumaprabhu6395gmail.com

Mobile: 7259391271

cases of acute pancreatitis in children induced by op compound poisoning.

Methodology

Five children presenting as acute pancreatitis following organophosphate consumption were included in the study over a period of 2 years (October 2021- February 2023). The data related to age, sex, time of consumption, signs and symptoms were collected. Informed consent was taken. Laboratory parameters such as S.Cholinesterase, S.lipases, S.amylases , blood sugars and imaging such as USG/CT abdomen were obtained. Serial values were monitored and assessed. The data was collected in a predetermined proforma sheet. Data were analysed in a descriptive pattern. Cases were treated as per the standard protocol. Children were treated with the following medications as per the available recommendations.

1. Airway, breathing and circulatory support.
2. gastric lavage
3. Intravenous Atropine at 0.05mg/kg/hr.
4. Intravenous Pralidoxime 50mg/kg/dose

Case 1: A fifteen-year-old female child with history of consumption of op compound (HAMLA – cypermethrin & chlorpyrifos) brought to emergency room presented within 6 hours of consumption. On arrival patient had fasciculations, salivation and bilateral pin point pupils. Stomach wash was given, treated as per the standard protocol. S. cholinesterase level was <455 U/L, blood sugar was 151mg/dl. Serial monitoring of S. cholinesterases and blood sugars were done. Blood sugars were within normal limits. On day eight of consumption, patient developed pain abdomen and vomiting. USG abdomen done was normal, S. lipase (specific for pancreatitis) was raised 112 U/L on day eight, S. amylase was 181 U/L both reached peak level to 960 U/L and 210 U/L respectively on day eighteen. Patient was treated symptomatically, keeping the child nil orally and iv fluids until there was no gastric aspirate. S. lipase levels were reduced to 116 U/Land S. amylase levels to 56 U/L, patient symptomatically improved and was discharged on day22.

Case no: 2 A seventeen-year-old female child presented with history of consumption of combination of Chlorpyriphos & cypermethrin, presented within 4hrs of consumption. On admission, child was disoriented (GCS – E3V2M6), with pin point pupils, excessive salivation and lacrimation. Child was hemodynamically stable. Treatment started as per standard operational Protocol. Lab investigations showed Serum cholinesterase levels < 455U/L and blood sugar of 143mg/dl. Serial monitoring of Cholinesterase and blood sugars were done. Blood sugars were within normal limits. On day 11 of illness, child developed pain abdomen with persistent vomiting, lab investigations revealed raised Lipase level of 102 U/L and amylase level of 134 U/L which peaked to 310 U/L and 180 U/L respectively on day 15.CT abdomen done revealed bulky pancreas without necrotizing lesions. child was kept nil per orally ,treated symptomatically. On day 25 of illness both serum Lipase and Amylase levels were reduced to 80 U/L and 113 U/L respectively. child was clinically improved and discharged on day 29.

Case 3: A seventeen-year-old female child presented with history of consumption of QUINLOPHOS 25% within 4 hours of consumption, on admission vitals were stable with pin point pupils. stomach wash was given and treated as per the protocol. Serum Cholinesterase was <455U/L and blood sugar was 132 mg/dl. Serial monitoring of cholinesterase and blood sugars were done. Blood sugars were within normal limits. On day 5 of illness patient developed nausea, pain abdomen. Serum Lipase and amylase levels were raised to 215 U/L and 130U/L, which peaked to 330 U/L and 150 U/L respectively on day 8. CT abdomen done revealed bulky pancreatitis. child was kept nil oral, treated symptomatically until there was nil nasogastric aspirate. Serum Lipase and Amylase levels started declining by day9 to 113 UL and 94 U/L respectively and patient condition got improved and discharged on day11.

Case 4: A sixteen-year-old male child presented with history of op compound consumption a combination of chlorpyrifos 50% and cypermethrin

5% within 2 hrs. of consumption. On admission, vitals were stable with excessive salivation, fasciculation and pin point pupils. Stomach wash was given, treated as per the protocol. S. cholinesterase was 1080 U/L and blood sugar was 120 mg/dl. Serial monitoring of S. cholinesterase and blood sugars were done. blood sugars were within normal limits. As patient developed vomiting, abdominal pain on day 9, investigations done, Serum Lipase and amylase was raised to 205 U/L and 139 U/L respectively, which reached to peak levels of 340 U/L and 210 U/L respectively by day 14. CECT abdomen revealed minimal ascites. Patient was kept nil per oral treated symptomatically. Serum lipases and Amylases were reduced to 113U/L and 93 U/L. Child was improved clinically and discharged on day 19.

Case 5: A fifteen-year-old male child presented with history of op compound consumption, chlorpyrifos 20% within 3hrs of consumption. On admission, vitals were stable with fasciculations. stomach wash was given, treated as per the standard protocol. S. cholinesterase level was 2120 U/L and blood sugar was 112mg/dl. Serial monitoring of S. cholinesterase and blood sugars were done. Patient developed excessive vomiting with bilious aspirate on day 6, S. lipase and Amylase were raised to 280 U/L and 140 U/L respectively and reached peak levels of 330 U/L and 160U/L by day 9. CT abdomen done revealed bulky pancreas with minimal ascites. child was kept nil oral, treated symptomatically until nil nasogastric aspirate. S. lipase and amylase levels were reduced to 112 U/L and 120 U/L on respectively. Child was improved clinically and was discharged on day 14.

Case summary of organophosphate poisoning developing acute pancreatitis, Table 1

Cases	**1**	**2**	**3**	**4**	**5**
Age/sex	**15yr/F**	**17yr/F**	**17yr/F**	**16yr/M**	**15yr/M**
Time of presentation following consumption	6 hrs.	4 hrs.	4 hrs.	2hrs	3 hrs.
Compound	HAMLA	Chlorpyrifos and cypermethrin	Quinalphos 25%	IP-L 505	Chlorpyrifos 20%
Symptoms	Pain abdomen, vomiting	Pain abdomen and vomiting	Nausea, pain abdomen	Pain abdomen, vomiting	Excessive vomiting with bilious aspirate
Onset of pancreatitis	Day8	Day 11	Day 5	Day 9	Day 6
S. lipase					
Initial	181 U/L	102U/L	215U/L	205U/L	280U/L
Peak	960 U/L	310U/L	330U/L	340U/L	360U/L
Final	116U/L	80U/L	113U/L	113U/L	112U/L
S. amylase					
Initial	181U/L	134U/L	130U/L	139U/L	140U/L
Peak	210U/L	180U/L	150U/L	210U/L	160U/L
Final	56U/L	113U/L	94U/L	93U/L	120U/L
Blood sugars at admission	151mg/dl	143mg/dl	132mg/dl	120mg/dl	112mg/dl
Peak S. lipase and S. Amylase levels	Day 18	Day 15	Day 8	Day 14	Day 9

Continue.....

Imaging	USG Abdomen – normal	CT Abdomen- bulky pancreas without necrotic lesion	CT Abdomen – bulky pancreas	CECT Abdomen- minimal ascites	CT Abdomen – Bulky pancreas with minimal ascites
Treatment	GL, PPI	GL, PPI	GL, PPI	GL, PPI	GL, PPI
Outcome	Discharged (Day22)	Discharged (Day 29)	Discharged (Day 11)	Discharged (Day 19)	Discharged (Day 14)

GL-gastric lavage; F- female; M-male; PPI – proton pump inhibitors

USG – ultrasonogram; CT – computed tomography; CECT – contrast enhanced computed tomography

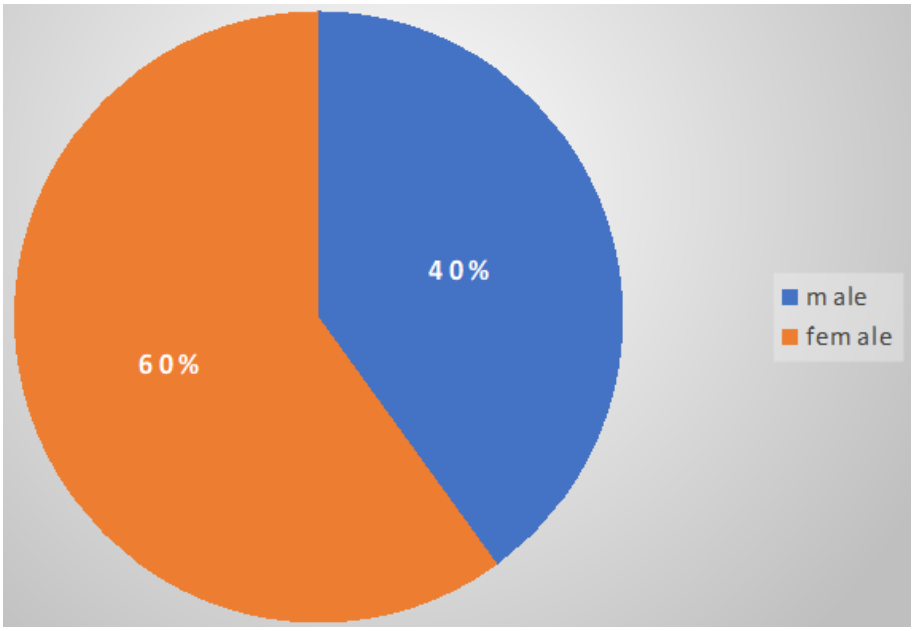

Figure 1: Gender distribution

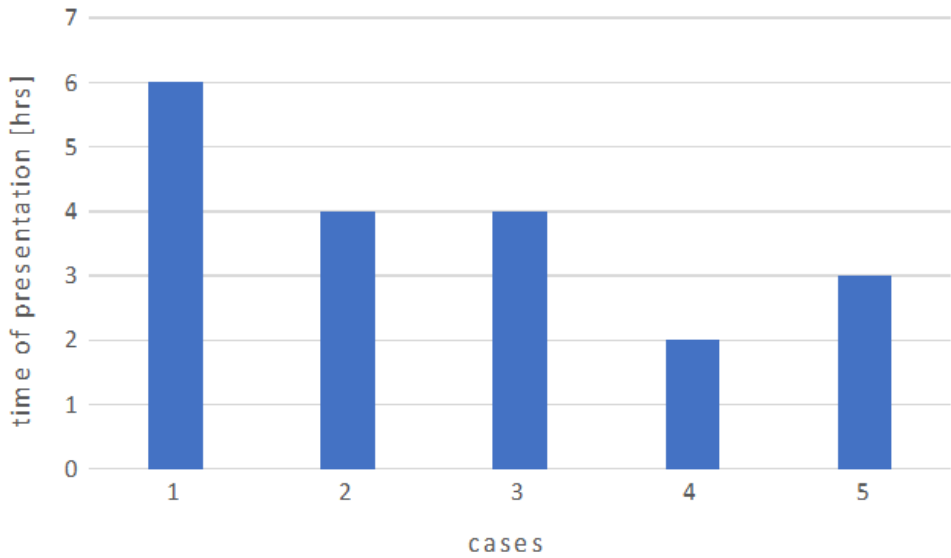

Figure 2: Time of presentation following op compound consumption

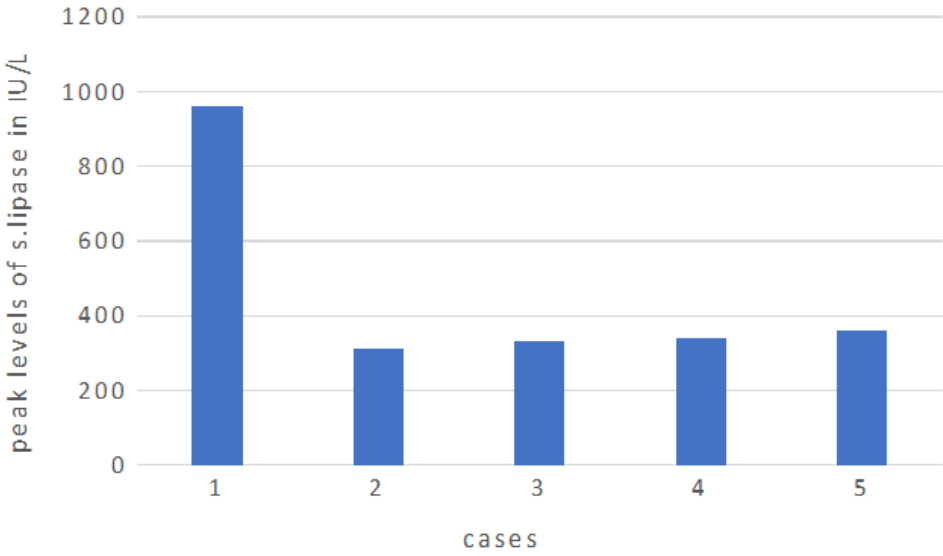

Figure 3: peak levels of s. lipase in IU/L

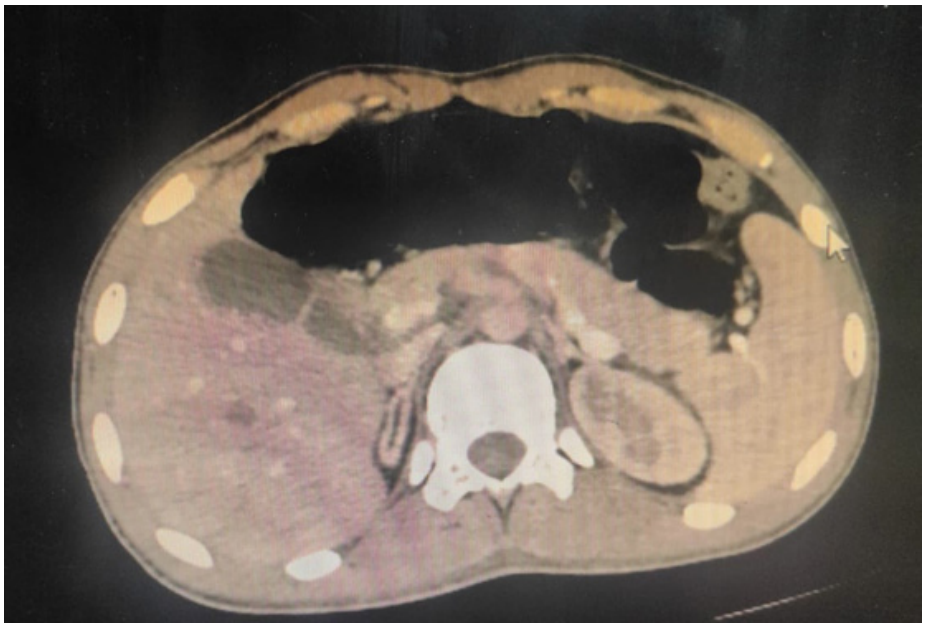

Figure 4: CT abdomen showing bulky pancreas with minimal ascites

Discussion

Organophosphate poisoning is a common poisoning seen in developing countries. Children developing acute pancreatitis following op compound poisoning is a rare complication[6-7]. case reports on children with organophosphate poisoning developing acute pancreatitis are not available. Hence, we are reporting these five cases who developed acute pancreatitis. Literature suggests that possible pathological mechanism for pancreatic insult is due to excessive cholinergic stimulation of the pancreas and ductal hypertension[8]

In our study common age of organophosphate consumption presenting as acute pancreatitis are between the age of 15-17 years, 40% were male and 60% were female child [figure 1]. Mean time of onset of presentation as pancreatitis following op compound consumption was on eighth day [figure 2]. All five children developed severe pain abdomen, vomiting and nausea as a common presentation which is similar to study done by Manjunath goud, Bhavna Nayal et al[9]. serial monitoring of S. cholinesterase, S. amylases, S. lipase were done. Mean peak levels of S. lipase and Amylase reached on thirteenth day, peak

levels of S. lipases were 960IU/L,310IU/L,330IU/L,340IU/L,360IU/L in our cases 1,2,3,4,5 respectively [figure 3]

All children underwent imaging, CT abdomen of three children showed features of pancreatitis with bulky pancreas, one had minimal ascites [figure 4] and another child had normal USG reports.

Three out of five children discharged within 20 days and two children after 20 days. All five children survived and discharged successfully.

Conclusion

Acute pancreatitis following organophosphate poisoning is rare complication in children. Diagnosis of acute pancreatitis should be given more importance and timely appropriate management can reduce the hospital stay and can be lifesaving in children.

Funding: No Funding Sources

Conflict of interest: None Declared

Ethical approval: the study was approved by the Institutional Ethics Committee, HIMS, HASSAN, Karnataka, Approval date : 26/04/2023, Approval number: Ref: IEC/HIMS/ RR 440/26-04-2023 Date: 26/04/2023

References

1. Moore PG, James OF. Acute pancreatitis induced by organophosphate poisoning. Postgrad Med J 1981;57:660-62
2. Tafur AJ ,Gonzalez L et al, unusual complication of an organophosphate intoxication. Emerg Med J 2005;22:531-531
3. Jayaraj S. Intensive use of agricultural chemicals in India and their ecological and environmental impacts. Proc Third Agri Sci Cong. Ludhiana: Punjab Agricultural University;1997. p. 115-30.
4. Utpal Kant Singh and FC Layland et al, textbook of poisoning in children, insecticide poisoning, page number 109, 3rd edition, jayapee publishers (p) ltd new Delhi.
5. Hsiao CT, Yang CC et al, Acute pancreatitis following organophosphate intoxication. J Toxicol Clin Toxicol 1996;34;343-47.
6. Dressel TD, Goodale RLJr, et al, pancreatitis as a complication of anticholinesterase insecticide intoxication. Ann Surg 1979;189;199-204
7. Dagli AJ, Moos JS et al, Acute pancreatitis as a complication of diazinon poisoning. A case report J Assoc Physicians Ind 1981; 29:794-5.
8. Dr Kumar Pankaj Shah, Dr. M Rajkumar Md (Gen Med) et al, A Study on Serum Amylase Level and Prevalence of Acute Pancreatitis in Organophosphate Poisoning.
9. Manjunath goud, Bhavna Nayal et al, A case of acute pancreatitis with occupational exposure to organophosphorus compound.