ORGANOPHOSPHORUS POISONING PRESENTING WITH ACUTE CORTICAL NECROSIS: A RARE COMPLICATION OF A COMMON DISORDER IN INDIAN SETTING

Ashwani Kumar¹, Vivek Mohanty¹, Nitin Kumar², Gaurav Karna², Anirudhh Mukherjee³

1Department of General Medicine, All India Institute of Medical Sciences, Rishikesh

2Department of General Medicine, All India Institute of Medical Sciences, Rishikesh

3Department of General Medicine, All India Institute of Medical Sciences, Rishikesh

Corresponding author: Dr Ashwani Kumar

DOI: <https://doi.org/10.32553/ijmsdr.v4i10.687>

Abstract:

Organophosphorus poisoning in the form of insecticides and pesticides acts by inhibition of anticholinesterase activity at the neuromuscular junction leading to overstimulation of the nicotinic and muscarinic receptor. Renal involvement usually manifests as increased urination, urinary incontinence, strangury due to overstimulation of urinary bladder. Acute kidney injury in the form of acute cortical necrosis is an extremely rare complication of OP poisoning. Here we highlight a case 31 year old male who presented to Emergency with Dichlorvos poisoning with anuria due to cortical necrosis requiring hemodialysis with gradually improving renal function thus representing an uncommon complication of Organophosphorus poisoning thus making it as a rare and interesting presentation.

Keywords: Organophosphorus poisoning, Acute cortical necrosis, Granular casts

Introduction:

Organophosphorus poisoning is a very common occurrence in Indian subcontinent because of easy availability in the form of insecticides and pesticides (1). The mechanism of action involves irreversible or reversible inhibition of Anticholinesterase leading to overstimulation of Nicotinic and Muscarinic receptors (2) and manifests as increased salivation, bronchial secretions, sweating, diarrhea, severe bronchoconstriction, pin point pupil, ataxia, convulsions and arrhythmias and is associated with high mortality. Kidney involvement in Organophosphorus poisoning is seen in about 4% of the patients and is a predictor of mortality in these patients (3). The mechanism of Organophosphorus poisoning induced acute cortical necrosis is not known fully but it is believed that Reactive oxygen species produced by the poison may have a role in direct cortical injury and tubular damage (4). Here we highlight a case of 31 year old male who presented with acute intoxication of Dichlorvos and had pre-renal azotemia which was diagnosed due to acute cortical necrosis based on MRI and urinary findings and with Hemodialysis session showed gradual recovery of kidney function.

Case Presentation

31 year male resident presented with Alleged History of ingestion of pesticide following which he was brought to Emergency Department in AIIMS Rishikesh. On arrival patient was unconscious, sweating and salivation excessively with frothing from mouth. Skin was cold. Patient was gasping. He had no history of urination,

diarrhea, vomiting, nausea or any abnormal body movements. On examination, vitals were PR=130/min, BP=70/50, RR=14/min, SpO₂=49% on RA, GCS=E1V1M1 and RBS=125mg/dl. So the patient was intubated for poor GCS and kept on VCV mode of ventilation (FiO₂=100% PEEP=7mmhg, TV=350ml, RR=25/min). On further examination pupils were constricted bilaterally with pupil size around 1mm and plantar reflexes were B/L mute. Clothes were smelling like petroleum but had no external injuries otherwise. For low BP, iv fluid at rate of 100ml/hr was given along with nor-adrenaline infusion at rate (8mg/50ml) 20ml/hr. Later on inquiry the bottle was obtained which contained "Dichlorvas". Based on the clinical finding and examination diagnosis was made as OP Poisoning. Atropinisation was achieved 16mg and so further atropine was infused at rate of 3.2mg/hr. Gastric lavage was done with 1lit NS via RT. foleys catheterization was done. Injection Pralidoxime was given at 2gm stat followed by 500mg/hr infusion. Central line was established on right Internal Jugular Vein. Further investigation showed Acute Kidney injury (Blood Urea – 152 and serum creatinine – 5.62) with had hyperkalemia (k+=7.1) for which hemodialysis was done on. Urine routine microscopy showed granular cast (Figure 1). MRI Abdomen showed low signal intensity on both T1 and T2 weighted sequences affecting the inner renal cortex and swelling of both the kidneys (Figure 2) On the basis of these findings a diagnosis of Acute cortical necrosis was made.

Figure 1: Urine microscopy showing granular casts

Figure 2: MRI Abdomen suggestive of Bilateral cortical necrosis

Table 1: Blood Investigations

	1 st Day	2 nd Day	4 th Day	6 th Day	8 th Day	10 th Day	14 th Day
Hb (gm/dl)	12.01		11.13	11.2	9.7	9.2	
TLC (*1000/cumm)	6.82		8.187	7650	4385	9080	
D. Count (N/L/M/E/B)	83/3.64		72/11/13/1.75/0.9	83/8/3/3/0.4	89.29/4.3/5.4/0.4	84.9/7.4/6.8/0.6	
PLT (*lac/cumm)	9.4		8.0	1.35	2.92	4.43	
PT (sec) /INR /aPTT (sec)	14.2/1.05		13.2/0.98				
TB/Direct (mg/dl)	1.8/0.17		1.02/0.51				1.56/0.93
SGOT/SGPT (U/L)	93/98		55/177.8				46/21
ALP (U/L)	165		203.7				456
TP/Albumin (gm/dl)	3.85/2.57		4.20/2.37				5.05/1.78
B. urea/ S. creat (mg/dl)	145/5.86	152.6/5.57	181.7/6.3		85/3.06	79.9/3.10	70/2.94
Serum Na+/ K+/Ca+/P- (meq/l)	150/5.5/7.8	145.4/5.3/9.16	148.2/5.6/8.12		129.8/6.2/8.25/7.7	128.3/6.1/8.86/9.13	127/5.8/7.2/6.3
PT/INR				11/1.06			

Treatment and Follow up

The patient had persistent anuria for which alternate day Hemodialysis was done for 1 week. After 2 week the patient urine output increased to > 400m/day with decreasing trends of Blood urea and serum creatinine suggestive of recovering cortical necrosis. Also he had reversal of toxicity due to Organophosphorus poisoning and was successfully extubated. He was discharged without any further need for hemodialysis and asked for periodic follow up with Kidney Function Test report.

Discussion:

Organophosphorus induced Acute kidney injury is a extremely rare occurrence whose mechanism is still not clear (5). One cohort study found that patients with OP poisoning had a 6.17-fold higher risk of AKI compared with the comparison cohort (3).

Specific antidotes include atropine and pralidoxime. Atropine inhibits muscarinic receptors and causes a decrease in acetylcholine-induced cholinergic effects. Pralidoxime in contrast to atropine does not affect any specific receptors; rather it acts to regenerate acetylcholinesterase (AChE), which has been rendered non-functional by the OPs. Due to anti-inflammatory effects of IL-10, it has been used in the management of OP poisoning involving organs such as the kidney, liver, and lungs (6).

Our patient has toxin induced cortical necrosis which was confirmed based on presence of granular casts in the urine and MRI abdomen. Acute kidney injury is a early predictor of mortality in Organophosphorus poisoning (7). Like previous cases (8,9) our patient too was successfully managed with Hemodialysis. The damage is usually reversible with the removal of toxins from the blood as evident in our case where at the end of 2 weeks the patient showed gradual improvement in renal functions.

Still there are no clear cut guidelines for the management of these patients and they should be treated on case to case basis

Conclusion

OP poisoning usually presents with typical signs and symptoms of cholinergic excess and rarely can cause acute renal failure. Timely diagnosis and management of this complication confer a favorable prognosis to the patient but if unattended, can lead to a progressively grave clinical course. Hemodialysis role as a treatment option needs to be explored further.

Acknowledgments: Department of medicine and Radiology, AIIMS Rishikesh

Funding source: This research did not receive any specific grant from funding agencies in the public, commercial, or not-for-profit sectors.

Competing Interest: authors have no competing interests to declare

INTELLECTUAL PROPERTY RIGHTS ASSIGNMENT OR LICENSE STATEMENT

References

1. Bawaskar HS, Joshi SR. Organophosphorus poisoning in agricultural India—status in 2005. JAPI. 2005 May;53:422-24.
2. Fukuto TR. Mechanism of action of organophosphorus and carbamate insecticides. Environmental health perspectives. 1990 Jul;87:245-54.
3. Lee FY, Chen WK, Lin CL, Lai CY, Wu YS, Lin IC, Kao CH. Organophosphate poisoning and subsequent acute kidney injury risk: a nationwide population-based cohort study. Medicine. 2015 Nov;94(47).
4. Poovala VS, Huang H, Salahudeen AK. Role of reactive oxygen metabolites in organophosphate-bidrin-induced renal tubular cytotoxicity. Journal of the American Society of Nephrology. 1999 Aug 1;10(8):1746-52.
5. Cavari Y, Landau D, Sofer S, Leibson T, Lazar I. Organophosphate poisoning-induced acute renal failure. Pediatric emergency care. 2013 May 1;29 (5): 646-7.
6. Yurumez Y, Ikizceli I, Sozuer EM, Soyuer I, Yavuz Y, Avsarogullari L, Durukan P. Effect of interleukin-10 on tissue damage caused by organophosphate poisoning. Basic & clinical pharmacology & toxicology. 2007 May;100(5):323-7.
7. Banday TH, Tathineni B, Desai MS, Naik V. Predictors of morbidity and mortality in organophosphorus poisoning: a case study in rural hospital in Karnataka, India. North American journal of medical sciences. 2015 Jun;7(6):259.
8. Agostini M, Bianchin A. Acute renal failure from organophosphate poisoning: a case of success with haemofiltration. Human & experimental toxicology. 2003 Mar;22(3):165-7.
9. Rubio CR, Felipe Fernández C, Manzanedo Bueno R, del Pozo BA, García JM. Acute renal failure due to the inhalation of organophosphates: successful treatment with haemodialysis. Nephrology Dialysis Transplantation Plus. 2012 Dec 1;5(6):582-3.