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Acute Kidney Injury in Organophosphorous Poisoning, How Frequently You See?

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Abstract: A 24 year old male with alleged of history of ingestion of unknown quantity of Quinal phos admitted with GCS E1VTM1, Pupils B/L5MM reacting, Blood pressure- 100/70mmHg, heart rate at 170bpm, SPO_2 was 100, ABG- mixed acidosis and vomiting, involuntary bowel and bladder. Investigations revealed Serum Creatinine 3.13, Blood urea nitrogen 90mg/dl, K+ 4.7 with compensated metabolic acidosis in arterial blood gas, Ultra sound abdomen reveals grade one renal parenchymal changes. There was a progressive rise of creatinine to 7.14 for which hemodialysis was initiated alternate day, urea & creatinine values came down with each dialysis. Patient showed improvement leucocytosis came down, electrolytes, liver function test, lactate levels corrected.
Keywords: Organophosphorous poisoning, Nephrotoxicity, Hemodialysis, Tracheotomy

INTRODUCTION

Poisoning with pesticides is a global public health problem and accounts for as many as 300 000 deaths worldwide every year, majority is due to consumption of organo phosphorous compounds due to various reasons. In organophosphorous poisoning, acetylcholinesterase is inhibited and there is increase in acetylcholine in the synaptic junctions, causing an overstimulation and disturbance of neurotransmission in the central and peripheral nervous systems. [1]. The pathogenesis of renal injury is unknown, there is not much experimental data demonstrating direct nephrotoxicity from organophosphates. Some proposed mechanisms for AKI are increased intratubular organophosphate, rhabdomyolysis, and prerenal azotemia from hypovolemia. These findings were transient and reversible after 3-8 day.

CASE REPORT

A 24 year old male presented to emergency room with alleged of history of ingestion of unknown quantity of Quinal phos (Organ phosphorus compound) on 19/11/13 at 9:30 pm. Patient was initially treated in private hospital (intubated in view of respiratory failure) and referred here for further management. On arrival to emergency room, GCS: E1VTM1, Pupils: B/L 5MM reacting, BP: 100/70mmHg, heart rate @ 170bpm, SPO_2:100, ABG- mixed acidosis. Stomach wash done, Activated Charcoal was given shifted to the ICU after stabilization in ER. H/o vomiting, involuntary bowel and bladder present, no h/o seizure, No h/o of DM, HTN, BA, Epilepsy, no h/o previous suicidal tendencies. Patient was treated with Inj atropine infusion, Inj. PAM was given for 48 hours, Antibiotics, PPI and other supportive medicines were continued. Investigations revealed Serum amylase, coagulation profile and were normal. Serum Creatinine 3.13, Blood urea nitrogen 90mg/dl, K+ 4.7 with compensated metabolic acidosis in arterial blood gas, Ultra sound abdomen reveals grade one renal parenchymal changes (Table 1). Enteral nutrition started on 3rd day of admission. There was a progressive rise of creatinine to 7.14 for which hemodialysis was initiated, after three sessions of hemodialysis his urine output improved to 60 ml/hr. Patient was weaned of from ventilator on 5th day of admission in ICU. On day 7, patient aspirated and was reintubated in view of hypoxemia, and tracheotomy was done on day 12 of ICU admission in view of prolonged ventilator support. Atropine was tapered off on 12thday of admission since the patient has been successfully atropinized. BAL was performed cultures shows Klebsiella species treated accordingly to antibiotic sensitivity, urine and blood cultures show no bacterial growth. Patient underwent dialysis alternate day as per nephrologists' advice, urea & creatinine values came down with each dialysis. Patient showed improvement leucocytosis came down, electrolytes, liver function test, lactate levels corrected. Patient was weaned off from ventilator and was on T oxygenator on day 26 of ICU stay. Due to financial constraints patient was discharged against medical advice on day 28th of ICU stay.

Table 1: Investigation details

	HB	TC	SE	RFT		LFT			Lact	Mg	Ca	CRP
			Na/K	Urea	Creatinine	SGOT	SGPT	Alb				
Day 1	11	22,000	135/4.7	90	3.13	60	36	3.7	12	1.5	7	120
Day 4	9.5	20,100	140/5.6	155	7.14	nd	nd	nd	nd	nd	7.2	nd
Day 8	8.5	18,100	139/7	330	10.64	nd	nd	nd	nd	1.8	nd	nd
Day 12	10	11,000	142/5.8	134	6.7	nd	nd	nd	nd		nd	nd
Day 16	10	17,000	140/5.2	124	4.2	55	18	2.7	14	1.7	7.9	56
Day 20	11	15,300	137/5.6	197	3.09	nd	nd	nd	nd	nd	7.2	nd
Day 24	11.2	12,000	138/4.7	220	2.68	45	20	3.1	6	1.8	8	35
Day 28	11	9,000	136/3.9	150	1.89	nd	nd	nd	nd	nd	nd	nd

HB: Haemoglobin, TC: Total lymphocyte count, SE: Serum electrolytes, RFT: Renal function test, LFT: Liver function Test, SGOT: Serum glutamic oxaloacetic transaminase, SGPT: Serum glutamic pyruvic transaminase, Alb: Albumin, nd: not done

DISCUSSION

Organo phosphorous pesticides are the most commonly available over-the-counter insecticides in India for agricultural and household use and their consumption is responsible for a large number of deaths [2, 3]. Their toxicity is attributed due to the direct effect, oxidative stress and acetyl choline excess. All the organ systems are affected like the immune, reproductive and endocrine systems, liver, pancreas, lungs; very few suffer from rhabdomyolysis [4, 5]. Organo phosphorous compounds are a rare cause of renal failure and only <10 cases have been described [6], which is more frequent in severe poisoning, not attributable to the degree of acetylcholinesterase inhibition. There have been reports of oliguric and non-oliguric acute renal failure, acute tubular necrosis and proteinuria, with unknown pathogenesis due to lack of experimental data, mostly in animals which suggested that it could be due to the effect of the toxin on the tubule, as the experiments revealed an increase in the urine with low osmolarity, so several mechanisms have been proposed: direct damage to the distal convoluted tubule, rise in oxidative stress, rhabdomyolysis and due to dehydration [7]. Determination of serum acetylcholinesterase activity is useful for diagnostic purposes, prognosis cannot be defined, nor the toxicity of the offending agent. Treatment consists of supportive measures, with a few requiring ventilator support, gastro intestinal decontamination, antidote administration-Atropine, PAM; although specific treatments with antidotes do not significantly reduce morbidity or mortality. Immediate administration of atropine blocks the muscarinic effects and pralidoxime reactivates the acetylcholinesterase inhibited by the toxic agent.

AKI in the setting of OP poisoning is fatal as renal replacement therapies proved to be futile, which could be due to the offending agents' toxicokinetics. This could be due to the particular toxikinetics of the agents making it unfavourable for renal replacement therapies. As the AKI is attributed due to oxidative stress, the role of IL -10 has been promising as it is potent anti-inflammatory, as well as cytoprotective agent in reducing the multi organ damage [8].

CONCLUSION

Pesticide poisoning is a common cause of mortality in India. Pesticide poisoning with AKI is a rare entity. Very few cases are reported in the context of Pesticide poisoning with AKI, about <10 and their recovery by dialysis is even rare with one report of recovery by hemodialysis, one with hemofiltration. Due to the rarity of presentation we present this case in which our patient had oliguric renal failure metabolic acidosis, rhabdomyolysis and hypertension attributed to organophosphorous poisoning, uremia which decreased following conventional hemodialysis.

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