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CASE REPORT

Acute Kidney Injury as a Rare Complication of Prallethrin Poisoning ("All-Out") in a Child

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Abstract: Pyrethroids are widely used as commercial and domestic insecticides due to their high effectiveness and low toxicity in humans and in medicine for the topical treatment of scabies and head lice. Prallethrin is a structural derivative of naturally occurring pyrethrin and active ingredient in liquid mosquito repellents. Acute human poisoning from exposure or ingestion of pyrethroids is rare because of poor dermal absorption and rapid metabolism with little tissue accumulation. Here we present a case of accidental Prallethrin poisoning("ALL –OUT", a liquid mosquito repellent) in a five year old child who had immediate complication as hypovolemic shock with aspiration pneumonia and delayed complications like acute kidney injury, with elevated liver enzymes, requiring renal replacement therapy and had an uneventful recovery.

Keywords: Prallethrin, Poisoning, Insecticide, Paresthesias, Acute kidney injury, Peritoneal dialysis.

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1. INTRODUCTION

Prallethrin – is a Type 1 Pyrethroid compound, commercially available as "ALL –OUT" – a liquid mosquito repellent. Its main effect is on sodium and chloride channels. Pyrethroids modify the gating characters of voltage-sensitive sodium channels to delay their closure. When the channels are kept persistently open, the nerves cannot repolarize and the organism is paralysed. The insects are 2250 times more toxic to the insects than to the humans [1]. Reports of human pyrethroid poisoning are rare. Acute kidney injury is an uncommon presentation as most patients are present with neurologic manifestations. Here we report a case of Prallethrin poisoning due to accidental consumption in a boy, manifesting with acute kidney injury and discuss the relevant literature.

2. CASE REPORT

A five-year-old male child, a known case of Down syndrome was brought to the emergency room with a history of accidental ingestion of "All-Out" mosquito repellent liquid (Prallethrin 1.6% w/w liquid, 35 mL in each bottle, half bottle, approx. 15 ml) and one episode of vomiting. Child was initially managed in a peripheral hospital with gastric lavage and intravenous fluid administration. The child had a global developmental delay with no prior cardiac or renal diseases and was under early stimulation therapy and physiotherapy.

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On admission, he was afebrile, tachycardic with heart rate of 140/min, low pulse volume, prolonged capillary refilling time(3-4 sec), blood pressure of 80/60 mmHg (<5th centile), Respiratory rate of 26/min and SpO_2 of 95% with room air. On examination, the child was drowsy with Glasgow coma Scale(GCS) 12/15 (E3 V4 M5), pupils constricted and bilaterally reacting to light. Oral cavity examination was normal . Systemic examination was found to be within normal limits.

Child was admitted in Pediatric ICU, kept nil per oral and managed with saline boluses, inotropic support (Dopamine), empirical antibiotics and other supportive measures. On evaluation Arterial blood gas analysis showed metabolic acidosis, (pH-7.2, $PaCO_2$-51.2, HCO_3-20.1), normal haemoglobin (11.1 mg/dl) and normal total counts (5700/cumm). CRP was not raised. Renal Function Tests, Liver Function Tests and serum electrolytes were found to be within normal limits. Pseudo-cholinesterase levels were found to be normal. As he improved clinically and became hemodynamically stable, inotropic support was weaned off.

On 6th day of admission, the child had decreased urine output (< 0.3 ml/kg/hr for 10 hours) with associated fever, periorbital oedema and respiratory distress. Further evaluation showed f an impaired renal function test (Blood urea nitrogen: 81mg/dL, Serum Creatinine: 3.92mg/dL). The Urine examination showed no proteinuria and the sediment was bland. ABG and Ultrasound abdomen were within normal limits.

Table 1. Blood investigations and interventions.

Days of Admission	Blood Urea	S. Creatinine (mg/dl)	Urine Output (ml/kg/hr)	K^+	LFT	–	–
Day 8	56	3.88	~1	–	–	–	Peritoneal dialysis started
Day 9	50	4.12	0.89	–	–	–	–
Day 10	44	4.16	0.4	–	–	–	–
Day 11	43	4.01	1.1	–	–	–	–
Day 12	50	4.22	1.5	5.41	–	Yeast like growth in culture	Started inj.Fluconazole
Day 13	57	4.18	1.8	5.94	SGOT-37 SGPT-267	–	–
Day14	53	**4.06**	3.2	**4.66**	–	**Hb: 6.9**	**PRBC** transfusion given
Day15	49	**3.06**	**5.2**	4.44	–	Hb: 14.2	–
Day16	40	**1.75**	**5.4**	3.84	–	–	Peritoneal dialysis stopped
Day17	**13**	**1.5**	–	**4.02**	–	–	**Discharged**

Urinary osmolality was 495.2 and the Fractional excretion of Sodium was elevated (19.6%). On chest x-ray, moderate pleural effusion which was probably secondary to hyper-sensitivity pneumonitis was detected on the right side and was drained using USG guided pig-tail catheter . The pleural fluid sent for analysis was found to be sterile.

Fluid challenge followed by Furosemide infusion was given in view of low urine output, but there was no response and the renal function was further declined. Peritoneal dialysis was started and serum creatinine, blood urea and electrolytes were frequently monitored. Antibiotics were changed from Inj Clindamycin to Inj Cefotaxime in view of the raised CRP (126). On day 9 of hospital admission, the liver function test done was deranged (SGOT: 144U/L, SGPT: 1228U/L, Total bilirubin- 0.53mg/dL, Albumin- 2.3g/L, LDH- 554U/L) and he was started on N-Acetyl Cysteine and Albumin infusion. Liver enzymes were followed up and responded well to the treatment and reached the baseline in 3-4 days

Over a period of four days, no improvement was noted in the renal function, the serum creatinine level remained high (4.01/mg/dL) and the child was continued on peritoneal dialysis. The patient's renal function gradually improved after 9 days of peritoneal dialysis as evidenced by increased urine output, decreased blood urea and serum creatinine levels. Peritoneal dialysis was stopped after 10 days. The patient was discharged after 17 days of hospital stay in a clinically stable condition.

3. DISCUSSION

Prallethrin – a Type 1 Pyrethroid compound, is an active compound in many insecticides. Its main effects are on sodium and chloride channels. Pyrethroids modify the gating characters of voltage-sensitive sodium channels to delay their closure. A protracted sodium influx (referred to as sodium tail current) results which, if it is sufficiently large or long, lowers the action potential threshold and causes repetitive tingling effect, which may be the mechanism of paresthesia. At relatively high concentrations, pyrethroids can also act on GABA-gated chloride channels, which may be responsible for the seizures [1]. Type 11 Pyrethroids (Deltamethrin & Fenvalerate) causes nerve membrane depolarisation and blockade leading to paralysis [2]. Pyrethroids may have an effect on Calcium channels which are important in neuronal functioning, for neurotransmitter release, gene expression and electrical excitability in nervous system [3]. Cardiac conduction disturbance due to prallethrin poisoning has been reported, which can probably occur due to its effect on sodium channels in the heart [4].

Acute human poisoning from exposure to pyrethroids is rarely reported. The outbreak of acute Deltamethrin poisoning in spray men of China in 1982 was the first case report of pyrethroid poisoning. After that, few reports of pyrethroid poisoning have been reported, mostly as occupational overexposure [2]. Occupationally, the main route of pyrethroid absorption is through the skin. Inhalation is much less important but increases when pyrethroids are used in confined spaces. The main adverse effect of dermal exposure is paraesthesias which may resolve in 12-24 hours. Within minutes, pyrethroid ingestion can cause sore throat, nausea, vomiting and abdominal pain. There may be mouth ulceration, increased secretions and/or dysphagia. Systemic effects occur 4-48 hours after exposure. The principal life threatening events are coma, convulsions and severe hypersensitivity reactions [1]. Convulsions generally occur with consumption of doses above 500mg. The frequency of convulsions can be 10-30 times a day. Severe cases may be associated with pulmonary complications including aspiration pneumonitis and pulmonary oedema, some of which may be attributable to organic solvents in the formulation. Fatal doses of these compounds are not known exactly and as there is no specific antidote known, treatment is entirely supportive and symptomatic [5 - 7].

About 40% patients are present with atypical presentations [8]. In a retrospective study conducted among 59 pyrethroid poisoning cases, atypical presentations were seen in 22 patients (39.3%). In a retrospective study of 59 patients,, atypical presentations were seen in 22 patients and included low Glasgow Coma Scale (19 patients), respiratory failure requiring ventilator care (10 patients), hypotension (6 patients), acute kidney injury (6 patients), pneumonia (4 patients), seizure (2 patients) and death (2 patients) [8]. Our case had transient elevation of liver enzymes which can be attributed to the generation of reactive oxygen species causing damage to

various membranous components of the hepatocytes [9]. In this case, there were no features suggestive of corrosive injury or mediastinitis that may later contribute to acute renal failure.

The first reported case of pyrethroid-induced toxic Acute Tubular Necrosis (ATN) was in a 66-year-old healthy woman receiving no prior nephrotoxic medications presented with extreme weakness, decreased urine output, and acute kidney injury following prolonged exposure to Permethrin spray in 2013 [7]. In our case, the AKI may not be just due to prallethrin and may well be due to hypotension/sepsis and this has to be kept in mind.

The signs and symptoms of Pyrethroid poisoning can be easily misdiagnosed as organophosphate or organochlorine poisoning. Atropine may be given to decrease secretions in cases with salivation and pulmonary edema. Low doses of atropine (0.5-10/mg) are generally sufficient as there is no inhibition of plasma cholinesterase [2]. As there is no specific antidote, early diagnosis and aggressive supportive therapies are the only remedies to prevent mortality.

CONCLUSION

There is limited data on features of severe prallethrin poisoning and its manifestations, although the mechanisms of toxicity have been well described. Most cases of prallethrin poisoning present with neurological and gastrointestinal manifestations, while acute kidney injury is rarely reported. We report a rare case of severe prallethrin poisoning with acute kidney injury, that reversed completely following peritoneal dialysis.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

Not applicable.

HUMAN AND ANIMAL RIGHTS

Not applicable.

CONSENT FOR PUBLICATION

Informed consent was obtained from the patient.

CONFLICT OF INTEREST

The authors declare no conflict of interest, financial or otherwise.

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Declared None.

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