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ORIGINAL ARTICLE

Clinico-epidemiology of Washing Powder (‘Prinso’) Poisoning in Sri Lanka

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Abstract

Background: ‘Prinso’ is a fabric stain remover, which is consisted of two sachets, one containing brown crystals (potassium permanganate) and the other white granules (calcium oxalate). The product is made by small scale manufacturers of Sri Lanka and has become popular as a remover of stains. Recently, deliberate self-poisoning with ‘Prinso’ has become common in both rural and urban areas of Southern Province of Sri Lanka.
Methods: A prospective clinical study was conducted in Teaching Hospital Ratnapura, Sri Lanka over 3 years commencing from July 2017. Epidemiological data, clinical features, treatments, and outcomes were collected. The data were analyzed through SPSS software version 21, using descriptive statistics.
Results: There were 274 (9.5%) patients with ‘Prinso’ poisoning out of which 140 (51%) were males and 134 (49%) were females. All were deliberate self-poisonings and 117 (43%) ingested oxalate alone; 31 (11%) ingested only permanganate, and 126 (46%) ingested both chemicals. The age ranged from 13-80 years (mean 31.6 ± 14 years) and the most commonly affected age group was 12-31 years (165;60%). The majority of patients were unemployed (108;39%) and 22 (8%) schooling children. Most patients (n=145;53%) were admitted to hospital within 1-4 hour of the ingestion (median 2 hrs and IQR 2-4 hrs) from Ratnapura (n=74;27%). Most of them (n=78;28.5%) were treated at hospital for 3 days (mean 4.8 days, IQR 2-6 days). Decontamination was done for 88% (n=239). Gastrointestinal symptoms including nausea (n=90;33%), vomiting (n=193;70%) and epigastric pain (n=141;52%) occurred frequently. The main complications among the patients were acute kidney injury [AKI] (110;40%), hypocalcemia (38;14%), hypotension (29;11%), ECG changes (27;10%), and methemoglobinemia (26;9.5%). Hemodialysis was done for 39 (14%) and 20 (7%) were treated at intensive care unit from which 13 (5%) were intubated. Methylene blue (antidote) was given to 25 (9%). Leukocytosis was observed in 118 (43%) patients and 236 (86%) were recovered. Despite medical attempts, 22 (8%) died. Case fatality rate for ‘Prinso’ poisoning was found to be 6.6% (90% CI 2.3-13.4;P < 0.05).
Conclusions: ‘Prinso’ is a deadly poisoning household agent and AKI is the most common clinical manifestation.

Keywords: Oxalate; Acute Kidney Injury; Methemoglobinemia; Methylene Blue; Washing Powder.

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INTRODUCTION

Intentional self-poisoning is a major public health problem in Sri Lanka [1-3]. It is one of the most prevalant methods of suicide in the country [4-6]. Agrochemicals (pesticides and herbicides), poisonous plants, pharmaceuticals, household agents (washing powder), corrosives, and kerosene oil are the main agents used in Sri Lanka for deliberate self-poisoning [4-6]. Among pesticides, organophosphate compounds [6] and among plants, Yellow oleander (*Thevetia peruviana*) and Glory lily (*Gloriosa superb* – ‘Niyagala’) are more frequently used [7]. But, recently, deliberate self-poisoning using household products (washing powder) has recently raised in rural areas of Sri Lanka [8-10] and medicinal drugs in urban areas [11]. As a widely used and easily available fabric stain remover, ‘Prinso’ has been reported to be commonly used in self-poisoning attempts, especially in the Southern regions of the country [8-10]. Deaths have also been reported from this poisoning [9]. It seems that this tendency of ‘Prinso’ poisoning is spreading to other parts of the country due to its being easily available and cheap. It is sold under several trade names such as ‘Prinso’, ‘Pinso’, ‘Ever Light’, ‘Diamond Light’, ‘Leo’, and ‘Sun Shine’. It is available in any small boutique in villages in Sri Lanka. One pack of this powder is around 30-40 Sri Lankan rupees. As a result, this toxic laundry detergent powder is gaining notoriety amongst

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villagers for deliberate self-poisoning, particularly among young people including school children and estate workers.

Regarding its ingredients, 'Prinso' consists of two sachets, one containing deep brown crystals (1.2 g of potassium permanganate-$KMnO_4$) and the other white granules (12.5 g of calcium oxalate-CaC_2O_4) [8]. The product is made by many small scale manufacturers of the country. It has become popular as a remover of stains preferred over the other commercially available products. It has traditionally been used to remove fungus from clothes. The manufacturers recommend that clothes should be soaked in deep brown crystals ($KMnO_4$) for 2 hours and washed with dissolved oxalic acid afterwards. Oxalic acid is a colorless, crystalline, and toxic organic compound used as a reducing agent in photography, bleaching, manufacture of ink, dyes, and dust removal. Potassium permanganate is an antiseptic and astringent agent with powerful oxidizing effects, recommended as a disinfectant and a fixative and stain in microscopy. Diluted solution of $KMnO_4$ is applied to skin lesions such as eczematous dermatitis. The crystalline and concentrated forms are corrosive due to the release of potassium hydroxide when they come in contact with water. Permanganate may also oxidize ferrous (Fe^{2+}) to ferric (Fe^{3+}) of hemoglobin, the resultant methemoglobin is incapable of carrying oxygen effectively, leading to functional anemia and cellular hypoxia.

The quoted oral rat LD_{50} for oxalic acid is 7.5 g/kg [12] and the estimated mean lethal adult dose is about 15-30g [13]. The reported clinical manifestations following oxalic acid poisoning are corrosive effects of upper gastrointestinal tract, acute kidney injury, and hypocalcemia due to formation of calcium oxalate [8-10],[14-15]. The quoted oral rat LD_{50} for $KMnO_4$ is 1090 mg/Kg [16] and the estimated lethal adult dose is about 10g [14]. The complications of $KMnO_4$ poisoning are hemorrhagic pancreatitis [17-18], methemoglobinemia causing respiratory failure [19], disseminated intravascular coagulation, adult respiratory distress syndrome, fulminant hepatic failure, and cardiovascular collapse [20]. The late complications of upper gastrointestinal ulceration include esophageal stricture [21] and pyloric stenosis [22].

Teaching Hospital Ratnapura (THR) is the main tertiary care center in Ratnapura District in Sri Lanka. Recently, it is observed that THR is receiving more patients with 'Prinso' poisoning some of which are fatal. Furthermore, there are lack of information regarding epidemiology and clinical profile of 'Prinso' poisoning in the country and no studies have so far been done in Sabaragamuwa Province in Sri Lanka. At the same time, as this is a new emerging trend of poisoning, there is lack of knowledge concerning its hospital management. Therefore, fatality may be increased particularly due to lack of experience in using antidote, gastric decontamination etc. Consequency, the main objective of this study was to describe clinico-epidemiology of 'Prinso' poisoning in Ratnapura District in Sri Lanka.

METHODS

The current research was a prospective observational hospital-based clinical study done in medical wards in Teaching Hospital Ratnapura, Sri Lanka over 3 years commencing from July 2017. All patients with the history of 'Prinso' poisoning were recruited to the study. On admission and during hospital stay, each patient was examined by the principal investigator (primary data) and then examined once in a day. Data were collected using a pre-tested interviewer-administered questionnaire in which epidemiological data and clinical features were included. Laboratory findings such as electrocardiogram (ECG), full blood count, serum electrolytes, renal function tests (blood urea and serum creatinine), liver function tests (serum glutamic oxaloacetic transaminase-SGOT and serum glutamic pyruvic transaminase-SGPT), prothrombin time (PT), international normalized ratio (INR), activated partial thromboplastin time (aPTT), serum calcium, serum amylase, and random blood sugar level were collected from the routine hospital investigation reports attached to the bed head tickets. Electrocardiographic and hematological changes along with changes in liver and renal functions were assessed using available investigation findings. The data were analyzed by SPSS software version 21, using descriptive statistics. Ethical approval (No. 2016/EC/94) was obtained from the ethics review committee of Faculty of Medicine, University of Peradeniya.

RESULTS

Epidemiological Features

During the 3 years of study period, all poisoning patients admitted to the hospital were 2892 from which 'Prinso' poisonings were 274 (9.5%). Demographic features of 'Prinso' poisoning are shown in Table 1. Males outnumbered females and the most affected age group was 12-31 years which was 60% (n=165). The age ranged from 13-80 years (mean 31.6 ± 14 years) and 22 (8%) were in schooling age, ranging 13-19 years. There were elder patients as well including 13 (4.7%) with > 61 years. Also, there were 3 (1%) pregnant mothers with 'Prinso' poisoning in 8, 12, and 18 weeks of period of amenorrhea. Considering education levels, most people were educated between grade 5-11 (n=127; 46%) and there were 20 (7%) patients, who studied up to advanced level examination. Thirty nine percent of people (n=108) were not engaging in any occupation in this cohort of 'Prinso' poisoning; 29 (11%) were manual laborers and 26 (10.5%) were gem miners. All patients with 'Prinso' poisoning were from administrative divisions of Ratnapura district. Accordingly, most of the patients were from Ratnapura (n=74; 27%), Nivithigala (n=59; 21.5%), Kuruvita (n=40; 15%) and Kalawana (n=26; 9.5%).

Considering features related to the poisoning agents (Table 2), all were deliberate (intentional) self-poisonings and 117 (43%) ingested calcium oxalate alone; 31 (11%) ingested potassium permanganate alone and 126 (46%) ingested both chemicals. Most patients (n=145; 53%) were admitted to hospital within 1-4 hour of the ingestion (median 2 hrs and IQR 2-4 hrs). Early admissions (within 8 hrs of the ingestion) were 259 (95.6%) and late admissions (> 8 hrs of ingestion) were 15 (4.4%). There were 11 patients (4%), who sought medical facility after 1 day of poisoning. Most of the patients (n=78; 28.5%) were treated at hospital for 3 days (mean 4.8 days, mode 3 days, SD 3.8 days, IQR 2-6 days). The number of 'Prinso' poisoning patients was annually increasing and it was highest in 2018 (n=87; 27%).

Table 1. Demographic characteristics of 'Prinso' poisoning

Demographic feature	Number (%)
Gender	
Males	140 (51)
Females	134 (49)
Marital state	
Married	161 (59)
Unmarried	91 (33)
Schooling	22 (8)
Age (years)	
12-21	70 (25.5)
22-31	95 (35)
32-41	53 (19)
42-51	26 (9.5)
52-61	17 (6)
62-71	11 (4)
> 71	2 (1)
Level of education	
< Grade 5	40 (15)
Grade 5-11	127 (46)
Up to Ordinary level examination	87 (32)
Up to Advanced level examination	20 (7)
Occupations	
No	109 (40)
Manual laborer	29 (11)
Gem miner	26 (9.5)
Garment worker	21 (8)
Estate worker	16 (6)
Three-wheel driver	11 (4)
Businessman	4 (1.5)
Driver	3 (1)
Mason	3 (1)
Others	26 (9.5)
Previous/current medical illnesses	36 (13)
Asthma	10 (4)
Hypertension	8 (3)
Diabetes	7 (2.5)
Epilepsy	4 (1.5)
Psychiatric illness	4 (1.5)
Others	6 (2)

Table 2. Epidemiological features related to poisoning agent in 'Prinso' poisoning

Epidemiological feature	Number (%)
Time since ingestion to admission	
< 1hr	106 (38.5)
1-3.59 hr	145 (53)
4-7.59 hr	8 (3)
8-11.59 hr	2 (0.7)
12-15.59 hr	1 (0.4)
16-19.59 hr	-
20-23.59 hr	1 (0.4)
> 24 hrs	11 (4)
Duration of hospital stay	
1	22 (8)
2	49 (18)
3	78 (28.5)
4	30 (11)
5	12 (4)
6	13 (5)
7	9 (3)
8	9 (3)
9	11 (4)
10	7 (2.5)
11	7 (2.5)
12	5 (2)
13	4 (1.5)
> 13	18 (6.5)
Co-ingestions	9 (3)
with paracetamol	4 (1.5)
with multiple drugs	2 (0.7)
with chlorpheniramine	1 (0.3)
with brake oil	1 (0.3)
with tile cleaner	1 (0.3)
with organophosphate	1 (03)
Decontamination	
Done	239 (88)
Only gastric lavage	84 (31)
Only administration of activated charcoal	14 (5)
Both methods	141 (51.5)
Not done	35 (13)
Psychiatric referral	
Done	155 (57)
Not done	119 (47)
Transfers from other hospitals	
Yes	115 (42)
No (direct admissions)	159 (58)
Transferred to National Hospital	4 (1.5)
Lost to follow up	11 (4)
Missing from the ward	9 (3)
Left against medical advice	2 (0.7)

Clinical Profile

Clinical manifestations (Figure 1 and 2), complications, and treatments of 'Prinso' poisoning are shown in Table 3. Gastrointestinal symptoms including nausea and vomiting occurred predominantly and they were 33% (n=90) and 70 % (n=193) respectively. In addition, epigastric pain (n=141; 52%), hematemesis (n=25;9%), diarrhea (n=23;8%), and dysphagia (n=18;7%) were observed as gastrointestinal disturbances. Difficulty in breathing was found in 26 (9.5%) patients. Acute kidney injury was the main complication (n=110; 40%), which occurred patients with oxalate poisoning. Of them, oliguria was found in 98 (35%), anuria in 3 (1%) and normal urine output in 9 (3%). Twenty-two (8%) patients died due to the poisoning from which, 2 (0.7%) ingested $KMnO_4$ alone, 2 (0.7%) ingested oxalate alone and 18 (6.6%) ingested both sachets. Two hundred thirty-six patients (86%) completely recovered. Percent case fatality for poisoning of both permanganate and oxalate was 6.6% (90% CI 2.3-13.4; $P < 0.05$). Death rate for the whole cohort was 8% (n=22).

Laboratory Findings

Laboratory findings of 'Prinso'poisoning are shown in Table 4. Of hematological findings, leucocytosis was detected in most of the patients (n=118;43%). Reduction of lymphocyte counts were seen in 134 (49%) patients. Out of biochemical parameters, elevated levels of serum creatinine and hypocalcemia were observed in 110 (40%) and 38 (14%) patients, respectively.

Treatments

Gastric decontamination was the mainstay of treatment, which was done within 4 hours of the ingestion for stable patients. For oxalate poisoning, gastric lavage was done by dissolving 10 g of calcium lactate in lavage fluid. Single dose activated charcoal was then administered. Thus, decontamination was done for 88% (n=239) of patients from

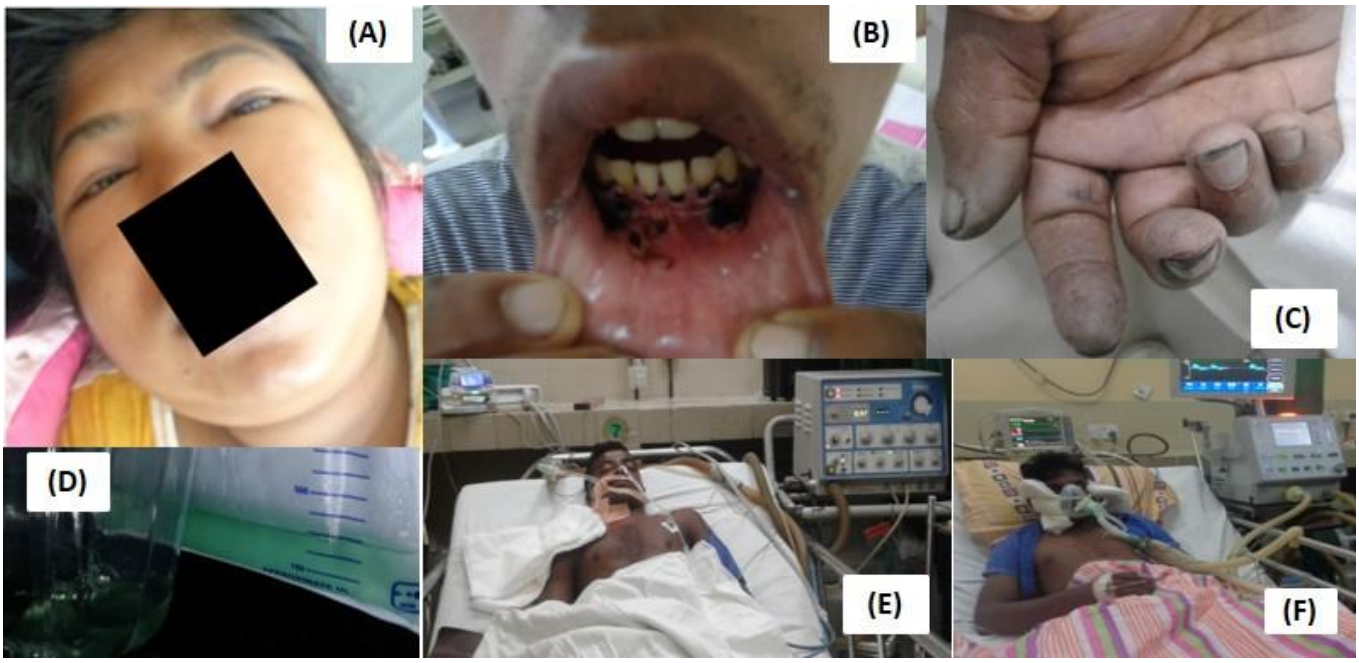
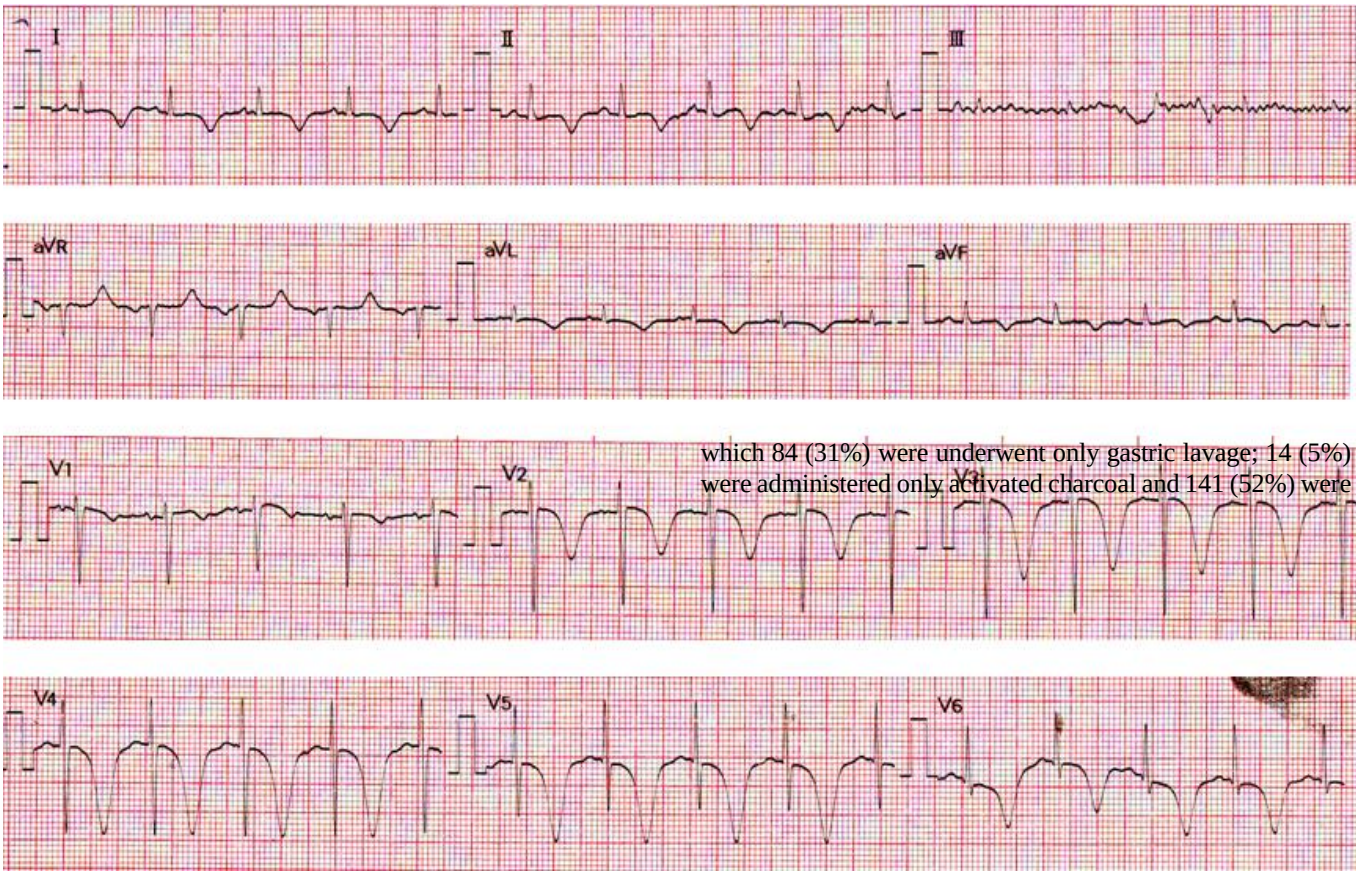


Figure 1. Clinical manifestations of 'Prinso' poisoning: (A) periorbital swelling on day 2 of oxalate poisoning in SN 68 patient (B) mouth ulcers on day 2 following both oxalate and permanganate poisoning (C) Peripheral cyanosis of fingers on day 1 following permanganate poisoning in SN 51 patient (D) Greenish color urine produced with the administration of methylene blue (antidote for $KMnO_4$) in SN 211 patient (E) Mechanical ventilation (SIMV mode) for respiratory failure (SN 211 patient) on day 1 following both oxalate and permanganate poisoning (F) Mechanical ventilation (C-PAP mode) for respiratory failure (SN 150 patient) on day 1 following both oxalate and permanganate poisoning



which 84 (31%) were underwent only gastric lavage; 14 (5%) were administered only activated charcoal and 141 (52%) were

Figure 2. Extensive T wave inversions on ECG (lead L_I,L_{II},aVL,aVF,V_1-V_6) on day 2 of 'Prinso' poisoning (both permanganate and oxalate) in SN 23 patient

given both methods. Thirty-nine patients (14%) needed hemodialysis 1-7 cycles (median 4 cycles, IQR 2-6 cycles). Methemoglobinemia was detected using methemoglobin color charts in 26 (9.5%) patients from which 13 (4.7%) needed intubation and mechanical ventilation. Methylene blue (antidote-0.1mL/Kg) was administered for 25 (9%) patients and 20 (7%) were given intensive care treatments.

Table 3. Clinical profile and treatments of 'Prinso' poisoning

Clinical feature	Number (%)
Vomiting	193 (70)
Epigastric pain	141 (51.5)
Nausea	90 (33)
Faintness	36 (13)
Headache	29 (11)
Difficulty in breathing	26 (9.5)
Hematemesis	25 (9)
Diarrhea	23 (8)
Dysphagia	18 (7)
Fever	16 (6)
Confusion	10 (4)
Periorbital swelling	9 (3)
Backache	6 (2)
Mouth ulcer	5 (1.8)
Convulsions	4 (1.5)
Chest pain	3 (1)
Throat pain	3 (1)
Laryngeal edema	2 (0.7)
Angioedema	2 (0.7)
Malena	2 (0.7)
Oral bleeding	2 (0.7)
Vaginal bleeding	1 (0.4)
Hematuria	1 (0.4)
Restlessness	1 (0.4)
Vertigo	1 (0.4)
Dizziness	1 (0.4)
Complications	
Acute kidney injury	110 (40)
Hypocalcaemia	38 (14)
Hypotension	29 (11)
ECG changes	27 (10)
Sinus bradycardia	9 (3)
Ischemic changes	3 (1)
Sinus tachycardia	2 (0.7)
Ventricular tachycardia	2 (0.7)
Supraventricular tachycardia	1 (0.4)
Right bundle branch block	1 (0.4)
Left bundle branch block	1 (0.4)
Sinus arrhythmias	1 (0.4)
Methemoglobinemia	26 (9.5)
Respiratory failure	13 (5)
Hypertension	2 (0.7)
Pancreatitis	2 (0.7)
Acute respiratory distress syndrome	2 (0.7)
Urinary tract infection	2 (0.7)
Heart failure	1 (0.4)
Treatments	
Gastric decontamination	239 (88)
Hemodialysis	39 (14)
Administration of methylene blue (antidote)	25 (9)
Admission to intensive care unit	20 (7)
Outcomes	
Recovered	236 (86)
Followed up in nephrology clinic	6 (2)
Died	22 (8)
Lost to follow up	11 (4)

Table 4. Laboratory findings of 'Prinso'poisoning

Laboratory finding	Number (%)
Hematological parameters	
Leucocytosis	118 (43)
Neutrophilia (Neutrophil leukocytosis)	145 (53)
Neutropenia	8 (3)
Lymphocytosis	12 (4)
Lymphopenia	134 (49)
Thrombocytosis	7 (2.6)
Thrombocytopenia	8 (3)
Decreased hemoglobin level	33 (12)
Biochemical parameters	
Increased serum creatinine	110 (40)
Increased SGOT/AST	73 (26.6)
Increased SGPT/ALT	45 (16)
Hypocalcaemia	38 (14)
Increased blood urea	30 (11)
Hyperkalemia	40 (14.6)
Hypokalemia	23 (8.4)
Hypernatremia	15 (5.5)
Hyponatremia	17 (6)
Clotting profile	
Elevated prothrombin time (PT)	33 (12)
Elevated international normalized ratio (INR)	33 (12)
Elevated partial thromboplastin time (aPTT)	21 (8)

DISCUSSION

This study brings to light an emerging epidemic of self-poisoning of 'Prinso' in Ratnapura District in Sri Lanka, which is freely available over the counter (OTC) washing powder in the country. The results showed that 'Prinso' poisoning is spreading throughout the Ratnapura region involving in both urban and rural areas. Also, the number of patients is annually increasing and the accessibility to the poisoning agent is also increasing as it is made by many small-scale manufacturers. On the other hand, it is evidenced, as 'Prinso' is cheap and is available in any boutique in the country, self-poisoning is increasing. Both males and females were almost equally affected; the frequency of each was 51% and 49%, respectively. But, a study done in Southern Province of Sri Lanka showed male predominance of 62.7% [10]. It is noteworthy that in addition to adults, schooling age group was also affected by 8% of the current study. This is not a good trend and early interventions should be considered. All poisonings in the current study were intentional and majority of patients (n=148; 54%) were in working age group and young people. On the other hand, 40% of people in the study group were not engaged in any occupation. This implies that the productivity of the country, which depends on the working age group is somewhat affected. However, the education level of people in the current study was satisfactory and 32% had educated up to ordinary level examination.

Considering the clinical profile, the most important manifestation was AKI as observed in 40 % of patients. It was caused only with oxalate poisoning. In previous studies, AKI was detected in 14% [8], 28% [9] and 66.6% [10]. Acute kidney injury is defined as an increase in serum creatinine level of 0.3 mg/dL (26.5µmol/L) or more within 48 hrs or a urine volume less than 0.5ml/kg/hr for 6 hrs [23]. In oxalic

acid ('Prinso') poisoning, AKI occurs due to deposition of oxalate in renal tubules causing acute tubular necrosis [15],[24]. This is reversible and has complete recovery with supportive treatments including fluid therapy and hemodialysis. Even though the cost of one packet of 'Prinso' is around 40 Sri Lankan rupees, the treatment of its poisoning is very costly because these patients need several cycles of hemodialysis and sometimes, they may also need intensive care treatments. In our cohort of AKI following 'Prinso' poisoning, only 14% needed hemodialysis, 1-7 cycles depending on the severity of clinical condition of the patient. One study on 'Prinso' poisoning concludes that the median serum creatinine was 1.7 mg/dL (IQR 0.91 - 4.4, normal range:0.5-1.3mg/dL) on day 2 of the poisoning [9]. Most patients developed symptoms of the gastrointestinal tract (nausea and vomiting) within the first 24 hours of the poisoning and early vomiting may have beneficial effects.

Methemoglobinemia is a fatal complication of 'Prinso' poisoning. It is caused only with $KMnO_4$ ingestion and results respiratory failure. Thus, in the current study, methemoglobinemia was detected in 9.5% of patients from which respiratory failure was observed in 4.7% and they were given intensive care. It occurs when red blood cells contain methemoglobin (MetHb) at levels higher than 1%. The formation of methemoglobin occurs from the presence of iron in the ferric form, instead of the usual ferrous form in hemoglobin. This results in a decreased availability of oxygen to the tissues. However, the ferrous iron has an increased affinity for bound oxygen. The binding of oxygen to MetHb results in an increased affinity of oxygen to the three other heme sites within the same tetrameric hemoglobin unit. This leads to an overall reduced ability of the red blood cell to release oxygen to tissues. Arterial blood with elevated MetHb levels has a characteristic chocolate-brown color as compared to normal bright red oxygen-containing arterial blood [25]. If methemoglobinemia is suspected, an arterial blood gas and co-oximetry panel should be obtained. Pulse oximetry is typically less accurate than co-oximetry in the setting of methemoglobinemia. Normal MetHb level is about 1% (range, 0-3%). The antidote for methemoglobinemia is methylene blue. In the present study, antidote was given for 9% of patients. However, there is no facility in Sri Lanka to measure blood MetHb level and it can be roughly quantified using standard color charts [25] and the antidote is administered accordingly. This method is used in medical facilities in order to detect MetHb. Methylene blue turns urine into greenish color because blue pigments of it combine with urochrome in urine and form greenish pigments (Figure 1D).

Initially, the cardiotoxic effects of 'Prinso' poisoning were widely evaluated the result of which led to the identification of patients with acute ischemic changes (Figure 2), hypotension, bradycardia, tachycardia, arrhythmias, hypertension, and heart failure. It was caused by both oxalate and $KMnO_4$. Hypotension, ventricular tachycardia, and atrial fibrillation have previously been reported following 'Prinso' poisoning [9]. Nevertheless, heart failure is a new finding in this study. Furthermore, the current study highlights that 'Prinso' is a fatal self-poisoning agent and the percent case fatality for ingestion of both chemicals was 6.6% (90% CI 2.3-13.4; $P < 0.05$). The death rate for the whole group was 8%. Thus, the ingestion of both sachets was associated with a significantly higher risk of death. This is compatible with previous studies that concluded that the ingestion of both $KMnO_4$ and oxalic acid is associated with a case fatality rate of 9.8%. The ingestion of more than one sachet was associated with a significantly higher risk of death [9]. A fatal case of $KMnO_4$ poisoning has previously been reported due to disseminated intravascular coagulation, hepatic necrosis, adult respiratory distress syndrome (ARDS), and renal failure 6 days after the ingestion [20]. Another fatal case was documented following ingestion of 20 g of $KMnO_4$ with ARDS, cardiovascular collapse, and hemorrhagic pancreatitis [17]. In the present study also, there was a fatal case of 'Prinso' poisoning ingesting both oxalate and $KMnO_4$ having AKI, ARDS, hemorrhagic pancreatitis [18]. Furthermore, permanganate causes severe laryngeal edema that leads to acute death in 'Prinso' poisoning. We observed 2 patients (0.7%) with severe laryngeal edema in the present study. In $KMnO_4$ poisoning, airway management may be difficult due to laryngeal edema [26]. Percent case fatalities of some commonly used poisoning agents in Sri Lanka are glyphosate- 2.4 (90% CI 1.5-3.6), carbamates - 5.1 (90% CI 3.8-6.7) and organophosphate - 11.2 (90% CI 10.1-12.3) [27]. Thus, this newly emerging poisoning agent 'Prinso' has somewhat high percent case fatality (6.6%) which needs our early attention.

Hypocalcemia is one of main complications in 'Prinso' poisoning and in the current study, it was observed in 14%. It may cause convulsions, QT prolongation on ECG, muscle twitching and depression of central nervous system. It is caused by calcium oxalate which is a mitochondrial poison in experimental models [28]. Therefore, it could be assumed that oxalic acid can induce mitochondrial toxicity in other organs that may, in turn, lead to death. Postmortem findings (macroscopy) of deaths following 'Prinso' poisoning have previously been documented and found esophageal erosions, congested lungs, cerebral edema, pale and swollen kidneys [9]. However, microscopic findings (histology) were not described in previous studies. The laboratory findings of 'Prinso' poisoning were widely evaluated in this study and found leukocytosis, hypokalemia, hyperkalemia, hyponatremia, hypernatremia and hypocalcemia. But the significant findings were the elevated serum creatinine and blood urea levels associated with AKI.

LIMITATIONS

We were unable to measure serum oxalate and permanganate levels in these patients, as there were no laboratory facilities in the hospital. But, if they were available, they could have been useful in order to understand clinical profile of 'Prinso' poisoning comparable with its serum levels.

CONCLUSION

'Prinso' is a deadly poisoning household agent. Acute kidney injury is the most common clinical manifestation of its poisoning, which needs early interventions. As number of 'Prinso' poisoning has gradually increased within 3 years of

study period, there should be regulations of manufacture and sale of this product in order to prevent further deaths. There are regulations in the usage of pesticides in Sri Lanka. But, this level of regulatory control does not exist for household goods in the country. Therefore, measures should be taken and implemented to ban or control the manufacture of this laundry detergent. As an improving method of curative side, proper hospital management guidelines of this emerging poisoning should be prepared using previous research experiences.

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