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SAPONATED CRESOL POISONING IN CHILDHOOD

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Abstract

A 22-month-old child with 18% burns following the oral and dermal contact of approximately 60mls of a disinfectant containing 50% saponated cresol is described. She developed electrolyte derangement, ocular and haematological manifestations and was treated successfully. The need for public enlightenment on promotion of the safe use of household chemicals and prevention of accidental poisoning in childhood through information and education campaigns is advocated.

Introduction

Exposure to toxic chemicals represents an important public health problem worldwide. According to a WHO estimate, unintentional poisonings led to 300,000 deaths in the year 2000.[1] Over 70000 deaths occurred in children up to 14 years old. Cresol is a known environmental pollutant, toluene metabolite, uraemic toxicant and accidental poisoning product.[2] Cresol is popularly used as a disinfectant and antiseptic.[3] Cresols can be absorbed following inhalation, oral, and dermal exposure by humans and animals. Most of the evidence of absorption in humans is indirect, derived from cases of accidental dermal contact with these substances or accidental or intentional ingestion. Three isomers exist: p-cresol, m-cresol, and o-cresol.[4,5]

KEYWORDS: saponated cresol, accidental poisoning, childhood.

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Mixed cresols are used as disinfectants and wood preservatives. o-Cresol is used as a solvent, disinfectant, and chemical intermediate. m-Cresol is used to produce certain herbicides, as a precursor to the pyrethroid insecticides, to produce antioxidants and to manufacture the explosive, 2,4,6-nitro-m-cresol. p-Cresol is used largely in the formulation of antioxidants and in the fragrance and dye industries.[4] p-Cresol is also an end product of protein breakdown in healthy individuals and an amino acid metabolite of intestinal bacteria.[3]

Saponated cresol is a soap solution of cresol; also known as p-cresol soap solution. It has a strong fungicidal and bactericidal effect. It is a yellow brown to reddish brown viscous liquid with characteristic odour.[5]

Cresol is strongly corrosive to tissues, a strong dermal irritant and causes frequent dermatitis. It is irritating to the skin, eye and respiratory system. It is rapidly absorbed by all routes and can be fatal by any route of exposure. Skin contact is the main exposure route and can result in severe skin burns.[7] Systemic effects may be neurologic, cardiovascular, respiratory, gastrointestinal, hepatic, metabolic, renal and/or haematologic.

Cresol has been a major source of poisoning (accidental and intentional) especially in the developed world. The exact prevalence of cresol poisoning in childhood is unknown. However, the prevalence of accidental childhood poisoning in general is high. For instance, the national estimates of unintentional child poisoning cases treated in the United States hospital emergency departments were an estimated 86,194 child poisoning incidents in 2004, amounting to 429.4 poisonings per 100000 children. Approximately 59.5% of the poisonings involved oral prescription drugs, oral nonprescription drugs, or supplements. Other major product categories resulting in poisonings included cleaning products (13.2%), drugs and ointment preparations intended for external use (4.9%), and personal care products (4.7%).[1] Documented cases of saponated cresol poisoning in our locality both in adult and children is rare. We report a case of accidental saponated cresol poisoning in a child to illustrate the effects of cresol poisoning, demonstrate the need for multidisciplinary intervention and to draw attention to the need for public enlightenment about prevention of poisoning.

Case Report

E.S. is a 22-month-old girl who was brought to the Children Emergency Room (CHER) of the University of Benin Teaching Hospital (UBTH) by her mother with complaints of ingestion of a liquid disinfectant (saponated cresol) three hours prior to presentation and had developed difficulty with breathing an hour before they presented. The child's cry following the ingestion of the poison alerted her mother who was busy with household chores. The disinfectant was purchased by patient's mother for treatment of her fungal nail infection. She claimed that the container was kept on top of a shelf but might have fallen down unnoticed. The content of approximately 60mls was found empty. The child was described as weak, smelled the characteristic odour of the disinfectant and had dark discolouration of her skin extending from the face to the chest and abdomen (delineating the area where the liquid disinfectant poured on the patient). Immediately after the incident, child was given a bath with water and was administered palm oil and palm kernel oil orally. The patient was subsequently taken to a private hospital where she was placed on intravenous fluid, gentamicin, ceftriaxone and prednisolone tablets. She was then orally referred to the UBTH for expert management. The empty container of the disinfectant brought with the child to our children emergency room was labelled as 50% saponated cresol (requiring 1 in 50 dilutions for use as treatment for nail infection and 1 in 100 dilutions for use as disinfectant).

At presentation, we found a well-nourished child who was acutely ill, in respiratory distress, with characteristic disinfectant-type odour suggestive of cresol, conjunctiva ejection with exudates and peri-orbital swelling bilaterally, buccal oedema and erythema, obvious hyperpigmentation around the face and trunk (See Figure 1), afebrile (36.8°C), not pale, acyanosed (oxygen saturation ranged between 92-94%), not dehydrated, no pedal oedema. Chest examination revealed tachypnoea (RR 52/min) and dyspnoea, with crepitations on the right middle lung zone. She also had tachycardia (PR 144/min) but normal blood pressure; was conscious but lethargic. An initial assessment of accidental saponated cresol poisoning was made.

Chest radiograph done six hours post-ingestion of poison showed patchy opacities on both lung fields; worse on the right middle lung zone. Serum electrolyte, urea and creatinine revealed hyponatraemia (120 mmol/l),

hypokalaemia (2.5mmol/l) and mild metabolic acidosis (HCO_3^- 18mmol/l). Random blood sugar was 110mg/dl (range; 90-120mg/dl while on admission). She had leukocytosis of 24.4 x $10^3/\mu l$ (granulocytes 84.2%, lymphocytes 10.3%, monocytes 5.5%), haematocrit 36.5% and reduced red blood cell (RBC) indices like mean corpuscular volume (MCV) 72.1fl, mean corpuscular haemoglobin (MCH) 20.3pg, mean corpuscular haemoglobin concentration (MCHC) 28.2g/dl and red cell distribution width (RDW) 17.5%. RBC count (5.06 x $10^6/\mu l$) and platelet count (449 x $10^3/\mu l$) were, however, normal. The liver function test was also normal [alkaline phosphatase 33 IU/l, aspartate aminotransferase (AST) 19 IU/l, alanine aminotransferase (ALT) 6 IU/l, total bilirubin 0.9mg/dl, conjugated bilirubin 0.4mg/dl].

Child was commenced on 120% intravenous fluid maintenance with 5% dextrose saline, had correction for acidosis, hypokalaemia and hyponatraemia and placed on intravenous amoxicillin-clavulanic acid combination and hydrocortisone. Patient was placed on nil per oral and had strict fluid input/output monitoring.

Examination of the integument by the next morning revealed superficial/partial thickness burns of about 18% of her body surface. Also, corneal staining of both eyes with fluorescein dye revealed generalized staining worse on right inferiorly. A diagnosis of chemical pneumonitis, burns, mucocitis, bilateral alkaline ocular injuries with corneal ulceration and conjunctivitis secondary to saponated cresol poisoning was made.

Management of the eyes included saline irrigation of superior and inferior fornices of both eyes, gutt ciprofloxacin six times daily, gutt dexamethasone four times daily for one week, gutt atropine 1% twice daily, gutt timolol 0.5% twice daily, daily glass rodding with copious chloramphenicol eye drops. Chloramphenicol ointment was applied to facial burns and open silver sulfadiazine dressing to the rest of the burns. She was co-managed with the ophthalmologists and plastic surgeons. Other specialties which were invited to see patient included the paediatric nephrology unit in view of possible complication of acute renal failure, the otorhinolaryngology unit and the cardiothoracic unit. They advised that the line of management be continued.

Deranged parameters were corrected within two days on admission and subsequently remained normal. By the eighth day, respiratory signs had resolved, burns healed with residual hypopigmentation (See Figure 1); patient was now tolerating orally but she still had corneal ulceration and was continued on daily glass rodding alongside copious chloramphenicol eye drops. She was discharged home after 16 days on admission at which time the corneal ulceration had healed (no longer stained with fluorescein). Two weeks after discharge, ocular signs had fully resolved. On follow-up a month after discharge, the hypopigmentation of the skin was hardly visualized.

Figure 1: Image showing patient before and after skin denudation.

Discussion

Accidental poisoning is a potentially preventable situation but represents one of the most common medical emergencies in childhood. As is usual with accidental poisoning, our patient was less than five years old.[8] The young child's explorative instincts as well as desire for oral gratification come into play to ensure the event. Also, she was not within sight of her caregiver, was left to wander about in the home and the poison was kept within the child's reach and sight. Poisoning results from the complex interaction of the agent (poison), the family environment and the child (that is his/her age and mentality).[1] The younger the child, the more care it needs but a mentally retarded child aged seven or eight years is just as vulnerable as a two-year-old toddler of normal intelligence. The poisoning occurred at home as does majority of accidental poisoning.[9] This case had oral, inhalational, dermal as well as ophthalmic exposure to saponated cresol. Since our patient had just buccal oedema and erythema and did not have obvious signs of oesophagitis, one would be drawn to say that she did not swallow the poison. The content on getting in contact with her lips and buccal mucosa probably caused her an unpleasant sensation that prevented her from drinking. She, however, had obvious dermal and ophthalmic involvement.

Serious or fatal poisoning may result if large areas of the skin are wet with cresol and the cresol is not removed. Deaths from dermal exposure to cresols have been documented in an infant and a man who suffered burns on about 7% and 15% of their body surfaces respectively.[4,10] Fortunately, our case had a good outcome. Cresols are irritants and have corrosive properties following exposure to high concentrations by any route of exposure. The poison does this by impairing the stratum corneum and

producing coagulation necrosis by denaturating and precipitating proteins, which explains the toxic effects at the sites of contact (that is, the skin and mucosal surfaces). It is essential to note that the effect of the time of contact of a caustic/corrosive substance on tissue is dependent on its concentration.11 In general, immediate water irrigation is recommended for all chemical burns. Most chemicals can be safely washed off the skin with water.[12] The immediate bath given to the patient was an appropriate response and is commendable. Bathing a victim with dermal exposure to saponated cresol with soap and water would remove some of the poison from as well as reduce the time of contact with the skin and would consequently reduce the degree of burns that the individual would have suffered. The superficial/partial thickness burns observed in the patient would likely have been worse if not for this timely home intervention by her mother. However, the usual cultural practices of forcing the child to drink palm oil or palm kernel oil after accidental poisoning must be discouraged because of the attendant risk of aspiration, lipoid pneumonitis, and resultant respiratory failure which may become fatal. Many children develop respiratory distress as a complication of this home intervention.

The existing information on the health effects of cresols in humans comes almost entirely from case reports of people who accidentally or intentionally swallowed cresol-containing substances or had these substances spilled on them. There are no studies that specifically address the health effects of exposure to cresols in children;[4] therefore, it is unknown whether children differ from adults in their susceptibility to health effects from cresols. There are no paediatric-specific methods to mitigate the effects of exposure to high amounts of cresols. It is thus reasonable to assume that the supportive methods recommended for maintaining vital functions in adults, will also be applicable to children.

Cresols distribute widely among tissues and no specific organ seems to preferentially accumulate the poison. The effects of acute cresol poisoning may be dermal, cardiovascular, gastrointestinal and hepatic, haematological, neurological and renal. Mouth and throat burns, abdominal pain, and vomiting are common gastrointestinal symptoms of cresol poisoning.[13] Buccal mucosa burns was seen in this child. Eye contact produces irritation, redness, corneal burns, keratitis, and possibly, in severe cases, blindness. Apart from blindness, the current case had these features. Inhalation produces coughing, dyspnoea, tachypnoea and may result in respiratory failure.[4,5] Our patient exhibited all these features except respiratory failure. Haematological effects described in our patient are similar to those in some documented cases. For example, as was found in the present case, leukocytosis was also reported in a man who drank 300ml of a 50% saponated solution of cresols.[14] Unlike the normal platelet count recorded in the current case, low platelet count was described in a man who drank an undetermined amount of cresol.[15] We recorded reduced RBC indices. The haematological effects of cresols appear to be due to both an oxidant effect on the cell contents and a direct effect on the red cell membrane.[15] Acute cresol poisoning can result in altered consciousness,[16] profound CNS depression, seizures.[4,5] Hypoactivity and lethargy, excess salivation, dyspnoea,

incoordination, muscle twitches and tremors, convulsions, and coma, have been reported in animals acutely exposed to cresols by gavage.[4] Although our patient was lethargic at presentation, she was conscious all through the period of admission.

Elevation of serum AST and ALT concentrations after ingestion of cresol has been documented by authors[15,17] with associated hepatic dysfunction. As in our case, Okamoto et al[16] recorded normal AST (20 IU/L) in a 43-year-old woman who presented with second degree chemical burns to 9% of the total body surface area due to cutaneous contact with cresol. The serum ALT was increased (61 IU/L) but the patient did not have evidence of hepatic dysfunction possibly dus to a low cresol concentration in her portal vein and liver. The explanation proffered by the authors[16] for the difference in hepatic involvement between oral and dermal poisoning is that it is possible that almost all of the gastrointestinal absorbed cresol enter the liver via the portal vein, while cutaneously absorbed cresol may enter the systemic circulation without passing through the liver. Cresols have severe permeability and protein degeneration effect similar to phenol. First-pass effect defined with absorption route is most important even if the final concentration is highest in the liver tissue. This may explain why kidney dysfunction is relatively more severe than hepatic dysfunction in percutaneous cresol intoxication.[16]

The excretion of cresol involves two steps: the initial step is cresol glucuronidation during biliary excretion, followed by excretion in the urine; and renal damage is possible.[18,19] Markedly elevated blood urea nitrogen (BUN) and serum creatinine have been documented,[14] our patient had normal serum creatinine. While no explicit mention of adverse metabolic effects was made in some reports of ingestion of cresols, marked metabolic acidosis was reported by Hayakawa[15] and Kamijo et al.[17] The metabolic acidosis found in our patient was mild. In cases of severe toxicity, electrolyte imbalance, including a sudden rise in potassium, may occur. However, we demonstrated hyponatraemia and hypokalaemia.

The successful management of cresol poisoning necessitates multidisciplinary intervention. Depending on the degree of chemical burns and the percentage of the total body surface area involved due to cutaneous contact with cresol, the plastic surgeons may be required. Ophthalmic injury which may result in blindness if not appropriately handled is the duty of the ophthalmologist. Oesophageal erosion as a result of ingestion of the corrosive substance needs to be assessed and managed by the cardiothoracic team. The nephrologist is needed for the associated acute oliguric kidney injury requiring haemodialysis. Massive acute intravascular (Heinz-body) haemolytic anaemia which may occur several hours after cresol ingestion can successfully be treated with immediate erythrocytapheresis (by the haematologist) followed by blood transfusions and forced diuresis.[20] The use of a cell separator allow easy and safe exchange of nearly half the patient's red cell mass over a short period of time.[21] In severe cases, there is need to control early respiratory failure with artificial ventilation (usually by the Paediatric Intensivist or Anaesthetist), but the patient remains at risk from toxic damage to the myocardium and lungs. With intensive supportive care

and appropriate multidisciplinary approach to management, the patient may recover and be discharged with no sequelae.[18]

Blood levels and urine concentration of poisons generally ought to be analyzed in the course of management of patients with poisoning. Although, the facility to do so is not available at our centre, it is important to note that no biomarkers that uniquely implicate exposure to cresols have been identified in humans or animals.[4,5] Cresols are formed from the commonly found amino acid tyrosine, and occur naturally in human and animal tissues, fluids, and urine. Cresols are also formed as minor metabolites of toluene, and an increased presence of o-cresol in the body could be due to exposure to this substance, although toluene or hippuric acid in the urine seem to be more reliable indicators of occupational exposure to toluene than o-cresol.[21] The use of cresols as a biomarker of exposure to cresol would require a considerable elevation to exceed biological background levels and potential confounding from conversion of other environmental agents. Research into the development of biomarkers of exposure for cresols would be valuable for both adults and children.

Early awareness of poisoning and appropriate therapeutic measures taken usually would be efficacious and prevent mortality.[12,18] Awareness and education about the potential toxicity of commonly used household substances may help in reducing the burden of acute poisoning in children. As part of management of a child with accidental poisoning, health education of mothers or caregivers is mandatory. The content of the health education would include among others proper storage of household chemicals which should not only be out of reach but also out of sight of children, storing chemicals in their original containers and not in beverage or soft drink containers, the child should not be left to wander about in the home but should be under strict observation by a vigilant adult and not left in the care of another child. The use of child-resistant containers for household products and drugs is an important intervention in the reduction of the incidence of childhood poisoning.[22] The interaction between the agent (or poison), the child and the environment that aided the accidental poisoning episode in this case must be understood and dealt with. In case of oral poisoning, the caregiver must avoid induction of emesis, give water or milk and seek medical attention. Furthermore, public enlightenment (via print and electronic mass media) on promotion of the safe use of household chemicals and prevention of accidental poisoning in childhood through information and education campaigns is advocated.

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