Acute carbolic acid poisoning: A report of four cases

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Abstract
Phenol (carbolic acid) is one of the oldest antiseptic agents. Apart from being used in many commercially available products, in rural India, it is often used in the household to prevent snake infestation. We hereby present four cases of acute carbolic acid poisoning which we saw over the last monsoon. The cases highlight the multiple routes of absorption of carbolic acid as well as its multi-organ complications which often necessitate intensive care of local therapy decreases systemic phenol absorption from cutaneous exposure and may help in reducing severity.

Keywords: Acute kidney injury, carbolic acid, phenol, poisoning

Introduction
Phenol (carbolic acid) is a flammable, highly corrosive chemical which is well absorbed by all routes exposure including inhalation, cutaneous, or oral. Phenols denature and precipitate cellular proteins and results in tissue injury.[1] In comparison to adults, children are more vulnerable to toxicants absorbed through the skin because of their relatively large surface area to body ratio. We hereby present, to the best of our knowledge, the first such case series of children with phenol toxicity.[2]

Case Reports

Case 1 [Table 1]
A 2-year-old girl was admitted within 2 h of accidental ingestion of carbolic acid with burn injury of about 8% of body surface area (BSA). Lips, tongue as well as part of the face, and chest were involved due to spillage, but pharynx and airway were spared. She was irritable and had drooling of saliva but no other systemic complications or airway-related issues. Glycerine was applied over the exposed skin as local therapy to prevent further absorption of carbolic acid. Initially, kept nil orally for 2 days and then started taking liquids followed by solids without any difficulty.

Case 2 [Table 1]
A 14-month-old girl was admitted 8 h postaccidental spillage of carbolic acid. On admission, she had acidotic breathing with a Glasgow Coma Scale of 9/15, convulsion, and blackish-blue discoloration of the skin of left thigh, trunk, and a portion of left hand involving a total of 25% of BSA. Topical glycerine was applied on the exposed parts. After 24 h of admission, she became oliguric and started passing dark brownish urine, and creatinine progressively increased from 0.11 to 3.18 mg/dl, along with the evidence of intravascular hemolysis [Table 1]. Hemodialysis was initiated on day 3 of admission, but fortunately could be discontinued after four cycles as renal parameter started improving. At last 9-month follow-up, she had normal renal parameters with healing skin lesions.

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**Case 3 [Table 1]**

A 2-year, 2-month-old boy was admitted at an interval of 10 h with burns of 40% BSA (trunk, abdomen, legs, and hands) [Figure 1]. On admission, he was in gasping condition and was immediately intubated and ventilated. He had ventricular tachycardia, very poor perfusion, severe metabolic acidosis, and refractory hypotension. Postresuscitation, he was commenced on multiple inotropes. Similar to Case 2, he developed intravascular hemolysis with hemoglobinuria. Hemodialysis was initiated on D2, and because of hemodynamic instability, sustained low-efficiency dialysis was chosen as the modality. His liver function also worsened and needed N-acetylcysteine infusion with supports of various blood products. He also started having refractory convulsions which required multiple antiepileptic drugs. Both liver and renal function improved slowly. He was extubated at 9 days of mechanical ventilation, hemodialysis discontinued after 12 cycles, and ultimately discharged after 24 days of hospital stay. At 10-month follow-up, he is doing well with normal renal function, healing skin lesion, and mild neurological sequelae.

**Case 4 [Table 1]**

A 1-year, 6-month-old boy was presented to us 8 h postexposure with burn of 20% BSA involvement including the lower part of the face and anterior chest wall. After initial resuscitation including intubation and ventilation, he was hyperhydrated assuming the possibility of acute kidney injury (AKI) secondary to pigment nephropathy, but in spite of this, his urine output started falling from day 2 of admission, and hemoglobinuria became obvious along with rising creatinine from 0.28 to 0.98 mg/dl. Furosemide infusion was started to maintain urine output >1.5 ml/kg/h, and slowly with judicious fluid support and furosemide infusion, urine started clearing from 4th day of admission and creatinine normalized. Evaluation of the gastrointestinal tract by endoscopy revealed esophagus Grade 2b and stomach Grade 2a burn injury. He was extubated by day 6 of mechanical ventilation. He was initially kept on total parenteral nutrition for 7 days and thereafter started on clear liquids which were gradually built up to semi-solid. Solids were initiated after 6 weeks, once repeat endoscopy revealed normal esophageal and gastric mucosa. At present, he is doing well without any long-term complication.

According to the parents/onlookers, none of the children ingested significant amount of carbolic acid as they spat it out immediately, and the majority of the carbolic acid was splattered on their various body parts.

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**Figure 1:** Extensive burns due to spillage of carbolic acid in the body

**Table 1: Clinical details of the cases**

|                | Case 1  | Case 2  | Case 3  | Case 4  |
|----------------|---------|---------|---------|---------|
| Age            | 2 years | 14 months | 2 years | 1 year 6 months |
| Sex            | Girl    | Girl    | Boy     | Boy     |
| Time to presentation (h) | 2       | 8       | 10      | 8       |
| Whether ingested | Yes (20 ml) | No | No | Yes (30 ml) |
| Skin exposure percentage (%) | 8       | 25      | 40      | 20      |
| Depressed sensorium | No      | Yes     | Yes     | Yes     |
| Seizures       | No      | Yes     | Yes     | No      |
| Stridor        | No      | No      | No      | Yes     |
| Pneumonitis/pulmonary edema | No | No | No | No |
| Hypotension    | No      | Yes (transient) | Yes | No |
| Arrhythmia     | No      | No      | Yes     | No      |
| Hemolysis      | No      | Yes     | Yes     | Yes     |
| Acute liver failure | No | No | Yes | No |
| Acute kidney injury | No | Yes | Yes | Yes |
| Coagulopathy   | No      | Yes (mild and transient) | Yes | Yes (esophagus Grade 2b and stomach Grade 2a burn injury) |
| Gastrointestinal tract burn injury | Yes, restricted to oropharynx only | No | No | |
| Hemoglobin     | 10.8    | 7.2     | 6.4     | 8.1     |
**Discussion**

Phenol (carbolic acid) is used in many commercially available products, but in rural India, another popular use of phenol is in the household to prevent snake infestation. We hereby presented our experience of four cases of acute carbolic acid poisoning over a span of 2 months during 2015 monsoon.

Our children had skin exposure ranging from 8% to 40% BSA, and the severity of the illness seems to correlate with skin exposure rather than oral ingestion [Table 1]. There are few case reports among adults mentioning skin exposure with phenols leading to systemic side effects.[34] Pediatric case reports are fewer with hardly any mention of correlation if any between percentage of skin exposure and degree of systemic side effects.[2] In children, as the ingestion is usually accidental, majority of the exposure is likely secondary to spitting or spilling over various body parts. Lin and Yang mentioned that 40% BSA exposure in adults can lead to systemic intoxication and multiple organ failure (MOF), whereas in our case series, exposure >20% was found to be grievous.[6] This likely reflects the greater BSA to weight ratio in children. Although the incidence of AKI in children with phenol toxicity is not properly defined,[3] majority of our children did have significant AKI. Various mechanisms have been proposed for phenol-induced AKI ranging from excretion of unconjugated phenol damaging the glomeruli and renal tubules, renal ischemia, formation of casts due to hemoglobin precipitation, and depleted glutathione levels.[34]

Similar to our case series, intravascular hemolysis is a well-known complication of phenol poisoning as it is CNS depression.[5,7] Similar to our Case 3, cardiac arrhythmia have also been reported.[2]

Decontamination must begin as soon as possible to minimize phenol absorption and includes the removal of contaminated clothing and either irrigation or wiping of exposed areas with low-molecular-weight polyethylene glycol or glycerine. If these are not available in high-flow jet can be used but low-flow jet is not recommended as phenol has a tendency to thicken and become difficult to remove thereafter.[1,8] Most of our cases arrived late to us and had not received any local decontamination underlying the need for improving the awareness not only among doctors but also general public.

**Conclusion**

- Our pediatric case series suggest that even dermal exposure may rapidly progress to MOF
- Severity and MOF seem to vary with degree of skin exposure and time interval to intervention
- Intravascular hemolysis resulting in AKI seems to be an important contributing factor toward morbidity.

**Declaration of patient consent**

The authors certify that they have obtained all appropriate patient consent forms. In the form the patient(s) has/have given his/her/their consent for his/her/their images and other clinical information to be reported in the journal. The patients understand that their names and initials will not be published and due efforts will be made to conceal their identity, but anonymity cannot be guaranteed.

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**Conflicts of interest**

There are no conflicts of interest.

**References**

1. Agency for Toxic Substances and Disease Registry (ATSDR). Toxicological Profile for Phenol. Atlanta, US: US Department of Health and Human Services; 1998.
2. Unlü RE, Alagöz MS, Uysal AC, Orbay H, Kılıç H, Tekin F, et al. Phenol intoxication in a child. J Craniofac Surg 2004;15:1010-3.
3. Seak CK, Lin CC, Seak CJ, Hsu TY, Chang CC. A case of black urine and dark skin – Cresol poisoning. Clin Toxicol (Phila) 2010;48:959-60.
4. Foxall PJ, Bending MR, Garthland KP, Nicholson JK. Acute renal failure following accidental cutaneous absorption of phenol: Application of NMR urinalysis to monitor the disease process. Hum Toxicol 1989;8:491-6.
5. Ghosh S, Ahlawat A, Rai KK, Arora A. An unusual cause of status epilepticus. Indian J Crit Care Med 2009;13:106-7.
6. Lin CH, Yang YJ. Chemical burn with cresol intoxication and multiple organ failure. Burns 1992;18:162-6.
7. Hassan AB, Seligmann H, Bassan HM. Intravascular haemolysis induced by pentaehlorophenol. Br Med J (Clin Res Ed) 1985;291:21-2.
8. Bruce RM, Santodonato J, Neal MW. Summary review of the health effects associated with phenol. Toxicol Ind Health 1987;3:535-68.