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Methylene Chloride Burns: A Case Report and Review of Literature

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Abstract

Chemical burns account for a small percentage of burns but contribute to significant number of burn-related mortalities. The major challenge posed by chemical burns is difficulty in correct depth estimation, as the damage continues to progress until they are effectively neutralized. Besides the most common etiology of chemical burns by alkalis and acids, there are many other unique causes of chemical burns. The author describes a case of chemical burns by heavy duty paint remover, the main composition of which is methylene chloride. Although several studies about methylene chloride poisoning are available in literature, only one case report of burns due to methylene chloride exists in literature. These burns are unique in their presentation. The author describes the presentation and challenges in management of this burn.

Clinical trial registration Not applicable.

Keywords
- chemical burns
- dichloromethane burns
- methylene chloride burns

Introduction

Chemical burns account for 3% of all burns but constitute 30% of burn deaths[1,2]. Sixty percent of these burns are workplace-related. Methylene chloride is a colorless, volatile, liquid organic solvent with a sweet, chloroform like odor and is commonly used in paint removers/strippers, metal cleaner/degreaser, and pharmaceutical manufacturing. Its most common routes of poisoning are via inhalation in closed spaces and through skin absorption, although occasional poisoning via oral ingestion is also reported.[3] This poisoning, although rare, has very high fatality rates.[4,5] Early recognition of the condition and prompt initiation of treatment are the only measures to reduce fatality. Only one other case of methylene chloride burns is reported in medical literature.[2] The authors are sharing their experience of a case of methylene chloride burns and reviewing literature of this form of poisoning in this article.

Case Presentation

A 55-year-old male went to clean the paint on the inner walls of his overhead water storage tank. When he did not return back even after an hour, his family found him lying unconscious inside the tank. He presented to our emergency department (ED) unconscious and burns over the face, neck, abdomen and left lower limb. There were no associated comorbidities or any other relevant contributory or past history. He was pulseless and had hemodynamically unstable ventricular tachycardia on arrival and was started on advanced cardiovascular life support (ACLS). Electrical cardioversion was done with 200 Joules of energy, and he was given 150 mg of injection amiodarone intravenous (IV) bolus stat to achieve normal sinus rhythm. Along with this, he was given 100 mEq of injection sodium bicarbonate, 3 mg of injection midazolam, and started on 100 mL/hour of normal saline infusion in ED. Since he was having irregular

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Table 1

Drug	Dose and frequency	Route of administration
Injection amiodarone	300 mg	Intravenous infusion
Injection cefoperazone/ sulbactum	3 g stat followed by 1.5 g twice a day for 5 days	Intravenous
Injection clindamycin	600 mg thrice a day for 5 days	Intravenous
Normal saline	100 mL/hour for 2 days then tapered and stopped	Intravenous
Levosalbutamol sulfate 1.25 mg + ipratropium bromide 150 µcg in 2.5 mL	2.5 ml respule four times a day for 1 week	Nebulization
Budesonide 0.5 mg	2 mL respule four times a day for 1 week	Nebulization
Injection midazolam	Started at 1 mg/hour; titrated over 48 hours as per patient's behavioral pain scale score and then stopped	Intravenous
Injection fentanyl	Started at 100micrograms/ hour; titrated over 48 hours as per patient's behavioral pain scale score and then stopped	Intravenous
Injection noradrenalin	Started at 40 nanograms/kg body weight/min; titrated as per patient's mean arterial blood pressure and stopped after 48 hours.	Intravenous
Ventilation	Patient put on IPPV mode; then, shifted to SIMV mode, CPAP mode, and finally put on T-piece. Initially, FiO2 was kept at 70% and then reduced to 50% after 4 hours as he started maintaining SPO2 of 100% with lesser oxygen requirement. Thereafter, it was further weaned off.	
Supportive measures	Ryle's tube in situ for 72 hours after which it was removed. Foley's catheter in situ for 5 days. DVT prevention stockings.	

Abbreviations: CPAP, continuous positive airway pressure; DVT, deep vein thrombosis; IPPV, intermittent positive pressure ventilation; SIMV, synchronized intermittent mandatory ventilation.

breathing, severe respiratory acidosis, and was unconscious even after ACLS, he was intubated and shifted to medical intensive care unit (MICU). The chemical bottle brought was labeled as "heavy duty paint remover," with its main composition being methylene chloride. Serial hemogram, liver and kidney function tests were done and were normal throughout the course of his treatment. Initial arterial blood gas analysis revealed severe acidosis, which progressively improved over the course of treatment. However, carboxyhemoglobin levels could not be measured as the hospital did not have a co-oximeter. In the MICU, he received the treatment, as shown in ►**Table 1**.

The scalp, face, neck, right shoulder, left side of abdomen and left lower limb were involved in 15% burns ►**Fig 1**. Fluid requirement was revised accordingly. Wounds were thoroughly washed with saline. Facial burns had a glossy dark brown appearance, while debridement of abdominal blisters revealed yellowish green staining of the underlying dermis. Burnt surface areas were neutral to red and blue litmus papers. Facial and neck burns were treated with 2% mupirocin ointment application thrice a day, while nanocrystalline silver dressings were used for abdomen and left lower limb. The patient was on ventilatory support for initial 48 hours after which he was extubated. After a week, the patient was discharged on oral antibiotics and painkillers and followed-up on outpatient basis for burns dressings. Facial burns healed in 10 days, while left lower limb and abdominal wounds healed in 3 weeks under dressings ►**Fig 2**.

Discussion

Methylene chloride poisoning typically occurs due to its volatile nature in a typical setting of closed space. In the case report by Wells GG, the victim was cleaning a 3 feet × 3

Fig. 1 Burns right side of face. Patient on ventilatory support after cardiopulmonary resuscitation (day 1 of admission).

Fig. 2 Burns front of Face. (2 months later).

feet × 6 inch vessel when toxic fumes built inside the vessel rendered the patient unconscious, resulting in his fall and subsequently developing burns to areas with direct contact with methylene chloride.[2] Its toxicity is due to solvent induced narcosis and carbon monoxide generation, which leads to carboxyhemoglobinemia.[6] It metabolizes via two pathways:

1) At low exposure via oxidative CYP2E1 pathway, formyl chloride forms, most of which gets metabolized to carbon mono-oxide (CO).
2) At higher exposures via glutathione S-transferase pathway, methylene chloride conjugates to glutathione.

The important points learnt from our case and reviewing literature are as follows:

1) Methylene chloride poisoning, although rare, carries a high fatality rate.[4,7-9]
2) The main organ affected is the central nervous system (CNS), with presenting symptoms varying from stupor, irritability, unconsciousness, decreased motor activity, changes in response to sensory stimuli, acute neurobehavioral deficits, memory loss, and fatty liver. Besides acute toxic effects, it is known to cause brain, liver, hepatobiliary, and hematopoietic malignancies.[5] Early diagnosis and prompt initiation of treatment is the only way to prevent fatality. Burn surgeons should be aware of this presentation to initiate appropriate prompt treatment.
3) Burns are usually second or third degree.[7] Both in the case reported by Wells[2] and that being reported by the author, they healed under appropriate dressings without any surgical intervention. Burn treatment is usually of secondary importance here, as systemic symptoms are severe and need attention on a priority basis to save life. This is different from acid or alkali burns where with a similar percentage of burns, the main presenting complaints are local burn-related symptoms.
4) These burns may mimic livor mortis, which sets in 1 to 2 hours after cardiac arrest. Mimicry of methylene chloride burns appearance with liver mortis may lead to a false impression to the treating physician that patient may have suffered cardiac arrest hours back rather than few minutes back, leading to an error of judgement, translating to pursuance of the cardiopulmonary resuscitation with less enthusiasm and causing death, which can be avoided if the burn surgeon is well versed with above facts.[7]
5) Rescuers and caregivers must wear proper gloves, gowns, and goggles before handling these patients, else they may become victims, as the mode of this poisoning is via inhalation and skin absorption. They must also have self-contained positive-pressure breathing apparatus with a full-face mask for self-protection, as they are prone to suffering inhalation injuries during rescue/treatment.[7]
6) Prompt recognition and early resuscitation is the only approach of preventing fatality in methylene chloride poisoning and burns.[4,7-9]

Conclusion

Methylene chloride burns are a rare entity with dramatic systemic presentation. Burns are usually second or third degree and can be managed conservatively as per author's experience and the only case report by GG Wells in 1984, however lack of knowledge of the presentation of these burns may lead to misdiagnosis, delay in treatment, and death. The only hope for survival in these burns is prompt recognition of the condition and initiating treatment at the earliest.

Earlier Presentation
None.

Role of Author
Entire idea and write-up.

Statement of Institutional Review Board Approval
Not applicable.

Financial Disclosures
None.

Conflicts of Interest
None declared.

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