Dichloromethane Injection: A Case Report

THUNYAPORN TANGTRONGCHITR¹, NAT KRAIROJANANAN², KITISAK SANPRASERT²,*

¹Ramathibodi Poison Center, Ramathibodi Hospital, Bangkok, Thailand
²Department of Trauma and Emergency Medicine, Phramongkutklao Hospital, Bangkok, Thailand

Abstract

**Background:** Dichloromethane (a chlorinated hydrocarbon) is normally used as a solvent. Dichloromethane poisoning has been reported from occupational exposure and the common routes of dichloromethane poisoning are ingestion and inhalation.

**Case presentation:** We described a case of 51-year-old man who received subcutaneous injection of dichloromethane and presented with local wound necrosis at his forearm, but carboxyhemoglobin levels were normal.

**Discussion:** The corrosive property of dichloromethane result in venous thrombosis formation at his wound that might prevent systemic absorption of dichloromethane leads to reduced hepatic converted dichloromethane to the carbon monoxide.

**Conclusion:** Symptomatic treatment and monitoring of CO production remain the mainstay in the treatment of patients with subcutaneous injection of dichloromethane.

**Keywords:** Carbon Monoxide; Methylene Chloride; Subcutaneous Injection

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**INTRODUCTION**

Dichloromethane (a chlorinated hydrocarbon) is normally used as a solvent. Dichloromethane poisoning has been reported from occupational exposure and the common routes of dichloromethane poisoning are ingestion and inhalation. Dichloromethane (CH₂Cl₂) or Methylene Chloride is a halogenated aliphatic hydrocarbon compound. It is a colorless and flammable liquid with a mild sweet odor.

Dichloromethane is used as raw material in many products such as household cleaner products, paint removers, and deodorants. The Liver is the primary site of dichloromethane metabolism when metabolized, producing Carbon monoxide (CO) via cytochrome P-450 2E1 and Glutathione-S Transferase (1).

**CASE PRESENTATION**

A 51-year-old man without significant medical history presented with his local wounded forearm at the hospital. His is a craftsman and usually uses dichloromethane as a solvent for inlaying plastic frame. He had several pressures and stresses from his family and workplace, so he decided to harm himself by self-injected dichloromethane (2 mL) into his forearm. At hospital, we found an erythematous lesion (10 cm) with severe pain at his left forearm (Figure 1). His vital signs showed he had hypertension (BP = 168/104 mm Hg, PR = 67/min, RR = 20/min, BT = 37 °C), and oxygen saturation by pulse oximeter was normal (99% on room air).

We did local debridement and found several venous thromboses occurring in his wound (Figure 2). After completion of debridement, his pain was significantly reduced.

We used pulse CO-oximeter (MASIMO Rad - 57 Pulse CO-Oximeter) for monitoring carboxyhemoglobin production and found carboxyhemoglobin levels shown in Table 1. Patient was discharged from the hospital without any clinical findings of CO poisoning during hospital admission.

**DISCUSSION**

Dichloromethane (CH₂Cl₂) or Methylene Chloride is a halogenated aliphatic hydrocarbon compound. It is a colorless and flammable liquid with a mild sweet odor. Dichloromethane is used as raw material in many products such as household cleaner products, paint removers, and deodorants. The Liver is the primary site of dichloromethane metabolism when metabolized, producing Carbon monoxide (CO) via cytochrome P-450 2E1 and Glutathione-S Transferase (1).

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**Table 1. Carboxyhemoglobin level**

<table>
<thead>
<tr>
<th>Post dichloromethane injection (hr.)</th>
<th>2</th>
<th>6</th>
<th>10</th>
<th>48</th>
<th>72</th>
</tr>
</thead>
<tbody>
<tr>
<td>Carboxyhemoglobin level</td>
<td>4.7%</td>
<td>5.5%</td>
<td>1.6%</td>
<td>4%</td>
<td>3%</td>
</tr>
</tbody>
</table>

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metabolism when metabolized, producing Carbon monoxide (CO) via cytochrome P-450 2E1 and glutathione-S-transferase (1) (Figure 3). From an animal model, hepatic conversion of dichloromethane into CO was a saturable metabolic rate. (2)

The elimination half-life of dichloromethane is 13 hr at room air (3) and it decreased by oxygen administration. During exposure to dichloromethane, carboxyhemoglobin (COHb) derived from liver metabolism might be responsible for the clinical manifestations of dichloromethane poisoning. The earliest symptoms of CO poisoning are often non-specific. Central nervous system bore the predominant symptoms of CO poisoning. Delayed neuropsychiatric symptoms such as amnesia, psychosis, cognitive impairment, seizure, and coma have been described in CO poisoning patients. (4) The pathophysiology of this delayed symptoms remains controversial. Hypoxia alone is insufficient to explain this symptom.

The dichloromethane poisoning treatment includes removal from exposure, supplemental of oxygen, supportive treatment and CO production monitoring. Dichloromethane poisoning can cause many manifestations, including acute kidney injury (5), hepatotoxicity (6) and CO poisoning. In this case, we monitored COHb level for 5 days and did not find the rising of COHb level. Venous thrombosis formation in his wound might play a role to prevent systemic absorption of dichloromethane.

CONCLUSION

The corrosive property of dichloromethane result in venous thrombosis formation at his wound that might prevent systemic absorption of dichloromethane leads to reduced hepatic converted dichloromethane to the carbon monoxide. Symptomatic treatment and monitoring of CO production remain the mainstay in the treatment of patients with subcutaneous injection of dichloromethane.

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REFERENCES