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Case Report

Three Cases of Acute Poisoning Caused by Dichloropropanol

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Abstract

This article reports three cases of acute poisoning caused by occupational inhalation of Dichloropropanol (DCP). All patients developed symptoms after exposure in a confined space, mainly presenting with nausea, vomiting, and severe liver damage (marked elevation of transaminases), accompanied by abnormal coagulation function. Laboratory tests showed a sharp increase in Alanine Aminotransferase (ALT) and Aspartate Aminotransferase (AST) levels, and significant abnormalities in coagulation indicators. The treatment included plasma exchange, hemoperfusion, and comprehensive supportive therapies such as liver protection and antioxidation. The patients' liver function and coagulation function significantly improved and they were discharged. This group of cases suggests that the diagnosis of DCP poisoning should be based on exposure history and typical biochemical indicators. Early blood purification and symptomatic support are key to improving prognosis.

Keywords: Dichloropropanol, Acute poisoning, Hepatic insufficiency, Plasma exchange

Introduction

Dichloropropanol (DCP) is a class of halogenated alcohol compounds, mainly including 1, 3-dichloro-2-propanol (1,3-DCP) and 2, 3-dichloro-1-propanol (2,3-DCP). It is widely used as an industrial intermediate in chemical production (such as the synthesis of epichloropropanol) [1]. Its toxicity issue has drawn increasing attention, not only in occupational exposure risks but also in the food processing process or in special scenarios such as the generation of contaminants after heating e-liquid [2]. Acute poisoning can lead to multiple organ dysfunction centered on liver damage, which is closely related to the complex toxicological mechanism of DCP. Studies have shown that 1,3-DCP is mainly activated through the metabolism of cytochrome P450 2E1 (CYP2E1), generating highly active intermediates that consume glutathione (GSH), induce severe oxidative stress and lipid peroxidation, and ultimately lead to hepatocyte necrosis [3]. Furthermore, the latest research reveals that it can also induce ferroptosis by regulating the GPX4 pathway, causing renal cell damage [4]. Therefore, the diagnosis and treatment of DCP poisoning are extremely challenging. In terms of treatment, in addition to conventional supportive therapies, for acute liver injury, blood purification techniques based on the removal of toxins and inflammatory mediators, especially Therapeutic Plasma Exchange (TPE), have been proven to significantly improve the survival rate of patients in acute and acute-on-chronic liver failure [5-7]. As one of the core technologies of in vitro treatment, The principle lies in the rapid removal of pathogenic substances in the plasma [8]. This article reports three cases of acute poisoning accompanied by severe liver injury caused by occupational inhalation of DCP. The aim is to explore their clinical features and diagnostic approaches in combination with the latest literature, and to focus on analyzing the clinical application value of the comprehensive treatment strategy centered on plasma exchange.

Clinical Data

Case 1

A 53-year-old male was admitted on September 19, 2025. Three days prior to admission, the patient developed nausea and vomiting

(three episodes of non-bloody gastric contents) after inhaling a large but unquantifiable amount of dichloropropanol. He reported subjective fever, fatigue, and decreased yellowish urine output, but denied headache, chest tightness, shortness of breath, abdominal pain, distension, or diarrhea. He received plasma exchange and liver-protective therapy prior to admission. His past medical history was unremarkable. Physical examination on admission: T 36.3°C, HR 54 bpm, RR 15 bpm, BP 120/64 mmHg. He was conscious and alert. Lung auscultation revealed clear breath sounds without rales. Heart sounds were normal with no murmurs. The abdomen was soft and non-tender, with a negative Murphy's sign. No edema was present in the lower extremities, and pathological reflexes were absent.

Laboratory tests in the emergency department showed: albumin 30.99 g/L, total bilirubin 61.23 μmol/L, aspartate aminotransferase (AST) 894.27 U/L, alanine aminotransferase (ALT) 1028.60 U/L. After immediate plasma exchange and liver-protective therapy in the emergency room, he was transferred to our department. Follow-up tests showed: white blood cell count 12.16×10^9/L, red blood cell count 4.08×10^{12}/L, platelet count 65.00×10^9/L, C-reactive protein 6.98 mg/L, D-dimer 5290.00 ng/mL, prothrombin time (PT) 20.80 sec, INR 1.85, activated partial thromboplastin time (APTT) >150.00 sec, thrombin time (TT) 35.50 sec, TT ratio 2.09, fibrinogen 1.82 g/L, antithrombin III 54.00%, total bilirubin 44.6 μmol/L, direct bilirubin 18.4 μmol/L, ALT 893.0 U/L, AST 404.6 U/L. Tests for magnesium, lipid profile, uric acid, creatine kinase (CK), and CK-MB were within normal ranges. A blood sample sent for toxicology analysis on September 19, 2025, failed to detect dichloropropanol. The testing agency attributed this negative result to the prolonged time since exposure (leading to low blood concentration) and the fact that the sample was drawn after an initial session of plasma exchange. Preliminary diagnosis: irritant gas poisoning, hepatic insufficiency, and coagulopathy.

Following initial treatment with plasma exchange, liver protection, corticosteroids, and other supportive measures, his coagulation profile improved and transaminase levels decreased. Treatment was deemed effective, and a second session of plasma exchange (1970 ml volume exchanged) was performed. Subsequent liver function tests showed ALT 112.5 U/L and AST 23.5 U/L. The patient reported no discomfort, and ALT levels continued to trend downward. He was discharged after his condition stabilized with continued liver-protective therapy.

Case 2

A 56-year-old male was admitted on September 19, 2025. Four days prior, while working in a confined space, he was exposed to "dichloropropanol," experiencing immediate eye and nasal irritation. This was followed by chest tightness, palpitations, nausea, and multiple episodes of non-coffee-ground emesis lasting until the next morning. After self-administering "strong tea," the vomiting ceased, but nausea, palpitations, and chest tightness persisted. He sought intravenous therapy at a local clinic the next day with unspecified medications and poor response. The following day, he developed dizziness, transient fever and chills (temperature not measured), and generalized joint pain. Self-medication with "Gankang" was ineffective. He presented to our hospital's outpatient clinic the day before admission. Tests revealed significantly elevated transaminases and coagulopathy, leading to treatment with liver protection, gastric protection, antiemetics, fluid resuscitation, and plasma infusion before admission to our department. Past history included hyperlipidemia diagnosed one year prior, treated intermittently with atorvastatin. He denied other chronic illnesses and reported one prior blood transfusion. Physical examination: T 36.6°C, HR 82 bpm, RR 20 bpm, BP 151/82 mmHg. He was conscious and alert. Lung and heart auscultation were normal. Abdomen was soft and non-tender with a negative Murphy's sign. No lower extremity edema or pathological reflexes.

Laboratory tests: WBC 11.05×10^9/L; renal function normal. Liver function: total bilirubin 62.02 μmol/L, AST 3961.19 U/L, ALT 3748.20 U/L. D-dimer: 3840 ng/mL. Coagulation profile: PT 25.5 sec, INR 2.30, antithrombin III 74%. Repeat liver function tests the next day: total bilirubin 53.48 μmol/L, AST 4895.44 U/L, ALT 5731.40 U/L. Preliminary diagnosis: irritant gas poisoning, hepatic insufficiency, and coagulopathy. A blood sample sent for toxicology analysis on September 18, 2025, was negative for dichloropropanol, attributed by the testing agency to low blood concentration from delayed sampling.

After admission, he underwent hemoperfusion and plasma exchange for toxin removal, along with liver protection, anti-inflammatory therapy, gastric protection, and symptomatic support. Transaminase levels improved, although coagulopathy persisted. Follow-up tests showed: WBC 9.71×10^9/L, CRP 10.76 mg/L, ALT 234.8 U/L, AST 49.1 U/L. Albumin level on September 28, 2025, was 38.4 g/L (bromocresol green method). Transaminase levels showed a sustained downward trend, and the patient was discharged after stabilization.

Case 3

A 51-year-old male was admitted on September 19, 2025. Four days prior, he inhaled volatile dichloropropanol in a confined workspace, subsequently developing nausea, recurrent vomiting (gastric contents occasionally tinged dark green), dizziness, generalized joint pain, and a dry cough. He presented urgently to our emergency department. Liver function tests showed: albumin 38.83 g/L, total bilirubin 29.91 μmol/L, AST 2880.16 U/L, ALT 5266.30 U/L. He received glutathione for liver protection, gastric protection, fluid resuscitation, and supportive care. He was admitted to our department with a diagnosis of "irritant gas poisoning."

His past medical history was otherwise unremarkable except for a 9-year history of chronic atrophic gastritis. Surgical history included internal fixation for a left forearm fracture 8 years prior, with good recovery. Physical examination: T 36.5°C, HR 64 bpm, RR 18 bpm, BP 123/71 mmHg. He was conscious and alert. A chemical burn lesion, approximately 3 cm × 5 cm, was noted on the flexor aspect of the distal right forearm. Lung and heart auscultation

were normal. Abdomen was soft and non-tender with a negative Murphy's sign. No lower extremity edema or pathological reflexes.

Ancillary tests: WBC 9.94×10^9/L, D-dimer 3340 ng/mL, PT 23.0 sec, INR 2.06, antithrombin III 81%. A blood sample sent for toxicology analysis on September 18, 2025, to the Shandong First Medical University Institute of Basic Medicine confirmed the presence of dichloropropanol. Preliminary diagnosis: acute volatile solvent poisoning, hepatic insufficiency, coagulopathy, and chronic atrophic gastritis. Treatment included anti-infection, anti-inflammatory, acid suppression, liver protection, fluid resuscitation, and supportive care, along with hemoperfusion, plasma exchange, or hemofiltration. Follow-up tests showed: WBC 14.49×10^9/L, ALT 145.8 U/L. His symptoms improved, and he was discharged.

Discussion

The three cases of acute poisoning caused by occupational inhalation of dichloropropanol (DCP) described in this article share highly consistent clinical features: after a clear history of exposure in a confined space, severe hepatocyte damage (sharp increase in transaminases) and coagulation dysfunction rapidly emerged. This manifestation precisely points to the core target organ toxicity of DCP - the liver. The toxicological basis lies in that DCP (such as 1,3-DCP) after being metabolized by CYP2E1, triggers a chain reaction of GSH depletion, oxidative stress and lipid peroxidation, leading to hepatocyte death [3]. Notably, in addition to traditional industrial production, similar chloropropanol contaminants can also be generated in non-traditional scenarios such as the heating of e-cigarette liquid [2], suggesting the diversity of exposure risks.

In terms of diagnosis, this group of cases highlights a key difficulty in DCP poisoning: the short window period for toxicant detection and its susceptibility to interference from treatment. For instance, the blood toxicant tests in cases 1 and 2 were negative, which might be related to the plasma exchange performed before sample collection or the rapid metabolism of the toxicant in the body [1]. This serves as a reminder that clinical diagnosis should not overly rely on blood drug concentration tests. Instead, a clear exposure history combined with characteristic liver injury and coagulation disorder biochemical profiles should be the core basis for diagnosis. When there is a high clinical suspicion but the toxicant test is negative, more attention should be paid to clinical manifestations and dynamic monitoring.

The successful treatment of this group of cases hinges on the early and proactive adoption of a blood purification and combined support therapy regimen centered on Therapeutic Plasma Exchange (TPE). Recently, multiple high-quality pieces of evidence have strongly supported this strategy: a randomized controlled trial confirmed that for patients with Acute Liver Failure (ALF) accompanied by cerebral edema, standard-volume plasma exchange can significantly increase the 21-day survival rate without transplantation and effectively reduce blood ammonia levels and improve systemic inflammatory response syndrome [7]. Subsequent systematic reviews and meta-analyses further clarified that TPE is significantly associated with improved 30-day and overall survival rates in ALF patients [6]. More recent meta-analyses have even extended the survival benefits to patients with acute-on-chronic liver failure (ACLF), showing that TPE can reduce their 30-day, 90-day, and even one-year mortality rates [9]. As a mature extracorporeal treatment technology, the therapeutic value of TPE lies in its ability to non-selectively remove inflammatory cytokines, endotoxins, damage-associated molecular patterns, and some protein-bound toxins or their metabolites [8], which precisely targets the pathophysiological core of the "cytokine storm" and liver failure caused by DCP poisoning [7]. In this group of cases, TPE was combined with hemoperfusion, liver protection, and supplementation of coagulation factors, among other comprehensive measures. It was observed that the patients' transaminase levels rapidly decreased and their coagulation function improved, which is consistent with the mechanisms and therapeutic effects reported in the above-mentioned literature.

In conclusion, for acute DCP poisoning, clinicians should remain highly vigilant. Diagnosis should be closely based on the patient's history and characteristic liver damage manifestations. In terms of treatment, therapeutic plasma exchange should be incorporated into the comprehensive treatment plan as early as possible to remove toxins, stabilize the internal environment, and gain time for liver cell regeneration. At the same time, given its mechanism of depleting GSH, the combined use of antioxidants such as N-acetylcysteine is a reasonable symptomatic support strategy. In the future, in addition to optimizing clinical treatment plans, strengthening industrial protection from the source, exploring bioremediation technologies [4], and developing specific antidotes targeting the toxicity mechanism are the fundamental directions for preventing and controlling such poisoning.

Acknowledgement

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Conflict of Interest

All authors declare that there is no conflict of interest.

References

1. Lee BQ, Wan Mohamed Radzi CW, Khor SM (2016) A simultaneous derivatization of 3-monochloropropanediol and 1,3-dichloropropane with hexamethyldisilazane-trimethylsilyl trifluoromethanesulfonate at room temperature for efficient analysis of food sample analysis. J Chromatogr A 1432: 101-110.
2. El-Hage R, El Hellani A, Haddad C, Rola Salman, Soha Talih, et al. (2019) Toxic emissions resulting from sucralose added to electronic cigarette liquids. Aerosol Sci Technol 53(10): 1197-1203.
3. Hammond AH, Garle MJ, Fry JR (1996) Toxicity of dichloropropanols in rat hepatocyte cultures. Environ Toxicol Pharmacol 1(1): 39-43.
4. Huang Y, Wen L, Zhang L, Jijun Xu, Weiwei Wang, et al. (2022) Community-integrated multi-omics facilitates screening and isolation of the organohalide dehalogenation microorganism. Innovation (Camb) 4(1): 100355.
5. Kim JD (2023) [Acute Liver Failure: Current Updates and Management]. Korean J Gastroenterol 81(1): 17-28.

6. Beran A, Mohamed MFH, Shaear M, Tarek Nayfeh, Mohammed Mhanna, et al. (2024) Plasma exchange for acute and acute-on-chronic liver failure: A systematic review and meta-analysis. Liver Transpl 30(2): 127-141.

7. Maiwall R, Bajpai M, Singh A, Tanvi Agarwal, Guresh Kumar, et al. (2022) Standard-Volume Plasma Exchange Improves Outcomes in Patients with Acute Liver Failure: A Randomized Controlled Trial. Clin Gastroenterol Hepatol 20(4): e831-e854.

8. Cervantes CE, Bloch EM, Sperati CJ (2023) Therapeutic Plasma Exchange: Core Curriculum 2023. Am J Kidney Dis 81(4): 475-492.

9. Kumar SE, Sithamparapillai K, Choudhury AK, P Krishna Bharadwaj, Jonel Trebicka, et al. (2025) Therapeutic Plasma Exchange in Patients with Acute-On-Chronic Liver Failure Improves Survival-An Updated Meta-Analysis. Liver Int 45(5): e70018.