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RESEARCH ARTICLE

FATAL MULTI-ORGAN DYSFUNCTION FOLLOWING ACUTE CHLOROFORM INGESTION: AN EMERGENCY MEDICINE PERSPECTIVE

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ABSTRACT

Acute chloroform ingestion is an uncommon but highly lethal toxicological emergency. We report the case of a 23-year-old male who presented to the emergency department following ingestion of approximately 100 mL of chloroform, complicated by prehospital cardiac arrest with return of spontaneous circulation. On arrival, the patient was profoundly hypoxic, hypotensive, and unresponsive, requiring immediate airway control, invasive monitoring, and vasopressor support. Despite aggressive resuscitative efforts including mechanical ventilation and N-acetylcysteine infusion, the patient developed refractory shock and progressive multi-organ dysfunction involving the cardiovascular, hepatic, and renal systems, ultimately resulting in death. This case underscores the critical importance of early emergency department resuscitation, highlights the rapid clinical deterioration associated with chloroform toxicity, and reflects the challenges posed by the absence of a specific antidote.

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INTRODUCTION

Chloroform (trichloromethane) is a halogenated hydrocarbon that was historically used as an anesthetic agent but was abandoned due to its significant toxicity profile. Acute ingestion is now rare, yet it remains associated with high morbidity and mortality. From an emergency medicine perspective, chloroform poisoning presents unique and time-sensitive challenges, including rapid central nervous system depression, myocardial sensitization leading to life-threatening arrhythmias, and early cardiovascular collapse. In addition, delayed hepatotoxicity and renal injury further complicate the clinical course. Due to the rarity of such cases and the lack of a specific antidote, management strategies are largely supportive and rely heavily on early recognition and aggressive resuscitation in the emergency department.

Case Presentation: A 23-year-old previously healthy male was brought to the emergency department with an alleged history of ingestion of approximately 100 mL of chloroform at around 3:30 PM in his hostel room. The patient initially had one episode of vomiting and was conscious; however, during transport, he developed sudden unresponsiveness and was found to be pulseless. Cardiopulmonary resuscitation was initiated, and return of spontaneous circulation was achieved after three cycles.

Primary Survey: On arrival at the emergency department, the patient was critically ill and required immediate assessment and intervention following Advanced Trauma Life Support (ATLS)-based principles. The airway was unprotected, with the patient unresponsive and unable to maintain airway reflexes. Breathing was severely compromised, with a respiratory rate of 13 breaths per minute and oxygen saturation of 33% despite oxygen supplementation via a simple face mask, along with bilaterally reduced air entry. Circulatory assessment revealed profound shock, with a pulse rate of 110 beats per minute and non-recordable blood pressure, indicating severe hemodynamic instability. Disability assessment showed a deeply comatose state with an AVPU score of "U." The patient's temperature was 98°F, and no external injuries were identified on exposure. The overall clinical picture was consistent with post–cardiac arrest state with ongoing severe hypoxia and shock.

Secondary Survey: A focused secondary survey was performed following initial stabilization. Cardiovascular examination revealed audible heart sounds without murmurs.

Respiratory examination confirmed bilaterally decreased air entry without obvious added sounds. The abdomen was soft and non-distended. There were no signs of trauma or focal neurological deficits that could be assessed due to the patient's comatose state. Relevant history obtained from bystanders confirmed chloroform ingestion, with no known prior comorbidities or co-ingestants.

Emergency Department Management: Given the patient's critical presentation, immediate resuscitative measures were initiated using a structured emergency approach. Rapid sequence intubation was performed, and the patient was placed on mechanical ventilation. Central venous and arterial lines were secured for invasive monitoring and management. Aggressive fluid resuscitation was initiated, along with early vasopressor support using noradrenaline infusion to manage refractory hypotension. Empirical broad-spectrum antibiotics were administered in view of possible aspiration and systemic inflammatory response. N-acetylcysteine infusion was initiated for potential hepatoprotection. Continuous hemodynamic and metabolic monitoring was maintained, and the patient was transferred to the intensive care unit after initial stabilization.

Investigations: Initial laboratory investigations revealed leukocytosis with a white blood cell count of 21.74 × 10^3/μL. Liver function tests showed elevated transaminases, with AST of 88 U/L and ALT of 99 U/L. The coagulation profile demonstrated an INR of 1.2. Hemoglobin was 15.1 g/dL, and platelet count was 315 × 10^3/μL. Serial investigations during hospitalization indicated worsening metabolic acidosis, progressive renal dysfunction, and evolving hepatic injury, consistent with the development of multi-organ dysfunction syndrome.

Hospital Course: Despite early and aggressive management, the patient's condition deteriorated progressively. He developed persistent refractory shock requiring escalating vasopressor support. Cardiac complications included arrhythmias and moderate left ventricular dysfunction. Renal function worsened with development of acute kidney injury; however, renal replacement therapy could not be initiated due to severe hemodynamic instability. Hepatic dysfunction progressed, with worsening biochemical parameters and coagulopathy. Multidisciplinary consultations were obtained. Nephrology recommended renal replacement therapy, while gastroenterology advised continuation of N-acetylcysteine infusion and consideration of liver transplantation. However, transfer to a transplant center was not feasible due to unstable hemodynamics and high transport risk. The patient subsequently developed a second cardiac arrest in the form of asystole approximately 24 hours after presentation. Advanced cardiac life support was initiated, but return of spontaneous circulation could not be achieved, and the patient was declared deceased.

DISCUSSION

Chloroform toxicity, though rare in contemporary clinical practice, remains a fulminant and often fatal toxicological emergency with complex, multisystem involvement. The clinical trajectory observed in this case—marked by early cardiac arrest, refractory shock, and rapid progression to multi-organ dysfunction—is well described in prior reports and reinforces the aggressive nature of this toxin. From an emergency medicine standpoint, the initial hours following exposure are critical, as early physiological collapse often determines overall prognosis despite optimal resuscitative efforts. Cardiovascular toxicity is a dominant and early manifestation of chloroform exposure. Chloroform is known to sensitize the myocardium to circulating catecholamines, predisposing to malignant arrhythmias and sudden cardiac arrest. This phenomenon has been consistently reported across case series, including the review by Lionte, where rapid cardiovascular collapse and fatal arrhythmias were central features in lethal exposures. Similarly, Jagadeesh *et al*. described abrupt deterioration with cardiovascular instability following ingestion, emphasizing that even initially stable patients may rapidly decompensate. In the present case, prehospital cardiac arrest with subsequent refractory shock reflects this profound cardiotoxic effect, highlighting the need for early anticipation of peri-arrest states and aggressive hemodynamic support in the emergency department.

Beyond direct cardiotoxicity, chloroform poisoning has been associated with a significant systemic inflammatory response, which may contribute to vasodilatory shock and worsening organ dysfunction. Dettling *et al*. reported that chloroform intoxication can precipitate a systemic inflammatory response syndrome (SIRS), characterized by leukocytosis and hemodynamic instability, even in the absence of infection. This inflammatory component likely compounded the refractory shock observed in our patient, where escalating vasopressor requirements failed to achieve hemodynamic stability. For emergency physicians, this overlap between toxicological insult and inflammatory physiology presents a diagnostic and therapeutic challenge, often necessitating empiric broad-spectrum antimicrobial therapy during early resuscitation. Hepatotoxicity is another critical component of chloroform poisoning and is primarily mediated by hepatic cytochrome P450 metabolism, generating phosgene, a highly reactive metabolite that induces centrilobular necrosis. Jayaweera *et al*. demonstrated that chloroform ingestion can result in significant hepatocellular injury, often accompanied by gastrointestinal mucosal damage and systemic toxicity, with plasma chloroform levels correlating with severity. Lionte's review further emphasizes that hepatic injury may evolve over time, progressing to acute liver failure even after initial stabilization. In the present case, early transaminase elevation progressed to hepatic dysfunction with coagulopathy, consistent with this well-described pattern. Although N-acetylcysteine is frequently administered for its hepatoprotective properties, its efficacy in chloroform toxicity remains uncertain and is largely extrapolated from its use in other toxic liver injuries.

Renal injury, typically manifesting as acute tubular necrosis, is frequently observed in severe chloroform poisoning and contributes to the progression of multi-organ dysfunction. In several reported cases, including those summarized by Lionte, renal failure has been a significant contributor to mortality. Our patient developed acute kidney injury; however, initiation of renal replacement therapy was not feasible due to persistent hemodynamic instability. This limitation is commonly encountered in critically ill toxicology patients and underscores the importance of early stabilization to enable advanced organ support. The spectrum of chloroform toxicity also includes direct tissue injury beyond hepatic and renal systems. Jayaweera *et al*. reported severe gastrointestinal injury and chemical dermatitis following ingestion, indicating

both local corrosive effects and systemic toxicity. Additionally, prolonged exposure, particularly via inhalation, has been associated with fatal outcomes, as illustrated by Milić *et al*., who described a pediatric case of lethal chloroform inhalation resulting in progressive organ failure. These findings highlight that toxicity varies not only with dose but also with route and duration of exposure, further complicating clinical assessment. Delayed clinical deterioration is another important feature of chloroform poisoning. Jagadeesh *et al*. noted that patients may exhibit transient initial stability followed by rapid decline due to evolving organ dysfunction, particularly hepatic failure. This underscores the need for prolonged observation and monitoring even in patients who appear stable at presentation. In contrast, the present case followed a hyperacute course, with early cardiac arrest and rapid progression to refractory shock and death, reflecting a high-dose exposure and severe toxicity.

From a systems perspective, several challenges complicate the management of chloroform poisoning in the emergency setting. Early diagnosis is often difficult due to nonspecific clinical features and limited availability of rapid toxicological assays. While plasma chloroform concentrations can confirm exposure, as demonstrated by Jayaweera *et al*., such testing is rarely available in real time. Furthermore, the absence of a specific antidote necessitates reliance on aggressive supportive care, placing emphasis on early airway control, hemodynamic stabilization, and timely escalation to intensive care. Advanced therapies such as extracorporeal membrane oxygenation or liver transplantation may theoretically offer benefit in selected cases; however, their application is frequently limited by hemodynamic instability and logistical constraints, as seen in this case. In summary, the present case aligns closely with existing literature in demonstrating that chloroform poisoning is characterized by rapid cardiovascular collapse, systemic inflammatory activation, and progressive multi-organ dysfunction. It reinforces the importance of early, aggressive, and protocol-driven resuscitation in the emergency department while highlighting the significant limitations in definitive management. The consistently poor outcomes reported across studies emphasize the need for heightened awareness, early recognition, and further research into targeted therapies for this rare but devastating toxic exposure.

CONCLUSION

Acute chloroform ingestion is a rare but devastating toxicological emergency associated with rapid clinical deterioration and high mortality. From an emergency medicine standpoint, early recognition, prompt airway management, aggressive hemodynamic resuscitation, and anticipation of complications are essential. Despite optimal supportive care, outcomes remain poor in patients presenting with cardiac arrest and refractory shock. This case emphasizes the importance of protocol-driven emergency care and early multidisciplinary involvement in managing such critically ill patients.

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