ASIA PACIFIC JOURNAL of MEDICAL TOXICOLOGY
APJMT 15;2 http://apjmt.mums.ac.ir June 2026

CASE REPORT

Hydrogen Cyanamide (Dormex) Toxicity: A Rare Case of Acute Fulminant Hepatitis and Coagulopathy

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Abstract

***Introduction*:** Hydrogen cyanamide (Dormex), a widely used plant growth regulator, poses significant toxic hazards, particularly in low-resource agricultural regions. The ease accessibility of agricultural chemicals facilitates impulsive self-poisoning.
***Case Report*:** We report a case of 16-year-old female who intentionally ingested Dormex in a suicidal attempt. She was initially misdiagnosed as having organophosphate poisoning. The patient developed severe complications, including metabolic acidosis, disseminated intravascular coagulation (DIC), acute fulminant hepatitis, cerebral edema, and multi-organ failure. Aggressive supportive management was initiated, including N-acetylcysteine infusion, fresh frozen plasma, and mechanical ventilation. Her condition gradually improved, and she ultimately made a full recovery.
***Discussion*:** This case report adds to the sparse literature on intentional self-poisoning with hydrogen cyanamide related to occupational context, offering critical insight into its clinical presentation, and multisystem complications. It documents a rare complication of DIC and rapid clinical deterioration following ingestion, yet survival with prompt supportive management.
***Conclusion*:** This case highlights the high toxicity associated with hydrogen cyanamide ingestion and emphasizes the importance of early and accurate diagnosis, in addition to the aggressive supportive management. It also calls attention to the urgent need for regulatory measures, public health education, and safe storage practices in agricultural communities to prevent accidental or intentional exposures.

Keywords: Hydrogen cyanamide, Self-poisoning, Occupational related, Fulminant hepatitis, Coagulopathy

How to cite this article: Ali AM, Fouad MM, Gouda AS. Hydrogen Cyanamide (Dormex) Toxicity: A Rare Case of Acute Fulminant Hepatitis and Coagulopathy. Asia Pac J Med Toxicol. 2026; 15(2):84-89.

INTRODUCTION

Pesticide self-poisoning is one of the most prevalent methods of suicide globally, particularly in low- and middle-income countries where small-scale agriculture is common. According to the World Health Organization [1], pesticide ingestion accounts for nearly one-third of suicides worldwide, highlighting a significant and persistent public health concern. The easy accessibility of these chemicals that are often stored within households for occupational use, facilitates impulsive self-poisoning, especially during periods of acute emotional or psychological distress [2].

Among various toxic agricultural substances, hydrogen cyanamide (commonly marketed under the trade name “Dormex”), a potent plant growth regulator. It is primarily used to break bud dormancy in deciduous fruit trees and has been classified as a restricted-use pesticide. This classification implies it should only be handled by Certified Applicators or individuals under their direct supervision, with usage limited strictly to certified purposes [3]. Despite this regulatory control, Dormex is frequently stored in farmhouses or homes, often without proper labeling or safety measures.

Human toxicity from hydrogen cyanamide is rare but potentially life-threatening. It can produce systemic toxicity resulting in mitochondrial inhibition, metabolic acidosis, cardiorespiratory impairment, and altered mental status in recorded cases [4, 5].

Clinical presentations vary widely, ranging from mild skin reactions such as erythema and dermatitis to severe systemic manifestations like metabolic acidosis, respiratory failure, cerebral edema, and multi-organ dysfunction. In severe cases, fulminant hepatitis and disseminated intravascular coagulation (DIC) can occur [4, 5]. While

dermal absorption of hydrogen cyanamide is relatively slow, gastrointestinal absorption is rapid, with a bioavailability ranging between 45% and 80% depending on the ingested dose [6]. Significant uncertainty exists regarding the whole clinical spectrum, biochemical time course, and appropriate supportive care of acute HCN poisoning in humans, with few studies confirming organ-specific consequences or response to therapies.

Most human studies are sparse case series or brief case reports that focus on acute symptoms but do not provide detailed serial clinical and laboratory data. Although many studies focus on occupational exposure, reports of acute self-poisoning are less common. Nonetheless, intentional ingestion presents a unique and serious clinical scenario with limited documentation in existing literature [7]. We were motivated to present this case because of the paucity of human data regarding the severe systemic consequences of hydrogen cyanamide poisoning and the limited evidence regarding its management. Furthermore, the lack of consensus on targeted therapy highlights the need to fill existing knowledge gaps and guide clinicians dealing with similar exposures.

This case report describes a rare and severe instance of hydrogen cyanamide poisoning resulting in DIC and fulminant liver failure in a teenage girl. Notably, the initial misdiagnosis as organophosphate poisoning complicated her early treatment. This case aims to highlight new clinical details, including the occurrence of fulminant hepatitis, disseminated intravascular coagulation (DIC) and cerebral edema following hydrogen cyanamide ingestion, the timeline of multi-organ failure and recovery, and the use of early N-acetylcysteine, which have not been systematically described in the literature.

CASE PRESENTATION

A 16-year-old female, weighing approximately 60 kg, was referred to the National Environmental and Clinical Toxicology Research Center (NECTR), Faculty of Medicine, Cairo University, on February 18, 2017. She arrived at 5:30 PM after being transferred from Beni Suef Central Hospital. Earlier that day, at around 9:00 AM, she had intentionally ingested Dormex (hydrogen cyanamide), a chemical that was readily available in her household due to its use on the farm where her father worked. The ingestion followed an episode of acute psychological trauma. The exact ingested dose, concentration and delay time to hospital presentation, despite being crucial for toxicological assessment, were not available because of gaps in patient history and documentation.

At the referring hospital, she received gastric lavage and was administered 30 ampoules of atropine based on a presumed diagnosis of organophosphate poisoning. Upon arrival at NECTR, the patient was drowsy (GCS: 10/15), with acidotic breathing, SpO_2 at 92%, and sinus tachycardia. Blood gas analysis revealed metabolic acidosis (pH 7.12, HCO_3 12.4 mmol/L), with venous SO_2 at 69%. Pseudocholinesterase levels were within the normal reference range (6323 IU/L), effectively ruling out organophosphate toxicity.

Initial labs showed mild leukocytosis, hyperglycemia, hypokalemia, and modestly raised liver enzymes. However, on the third day post-admission, she developed hematemesis, hemoptysis, and hematuria, along with a coagulation profile indicative of DIC (INR 23.4, PT 85). Clinical deterioration continued with worsening renal function and increased leukocytosis. On the fifth day, she became comatose and required mechanical ventilation. A CT brain scan revealed cerebral edema, and liver enzymes rose dramatically, indicating acute fulminant hepatitis. Remarkably, by the sixth day, her condition began to stabilize. Over the next week, she gradually improved, and by day 14, she was weaned off mechanical ventilation and discharged in a fully recovered state. A chronological summary of the clinical and laboratory findings is presented in table 1, 2.

Table 1. Chronological clinical findings summary

Day	Vital Signs / Clinical Status	Labs / Imaging	Treatment / Intervention
1	Comatose, admitted to Toxicology ICU	ABG: pH 7.12, PCO_2 38, HCO_3 12.4, SO_2 69% (venous) ECG: Sinus tachycardia CPK, Troponin: Normal Pseudocholinesterase: 6323 (↑) CBC: WBC 13,300 Na^+ 142, K^+ 3.4 RBS: 240 mg% SGOT : 35 U/L, SGPT: 52 U/L Creatinine: 1.18 mg/dL, Urea: 34 mg/dL CXR: Normal	IV Bicarbonate Oral N-Acetylcysteine (100 mg/kg/8h) Supportive ICU care
2	Vitally stable	K^+ decreased to 3.0	Potassium replacement CVP-guided therapy
3	Vitally stable Hematemesis, hemoptysis, hematuria	PT 85%, INR 23.4 Liver enzymes, lipase, amylase: Normal Abdominal U/S: Fatty liver Na^+ 158, K^+ 2.4, RBS: 83	4 units of FFP Dicynon, Cyklokapron
4	Deteriorating consciousness Vaginal bleeding, hemoptysis Transferred to Medical ICU	INR 12.9 WBC: 15,000 Urea: 70, Creatinine: 1.8 Albumin: 3.3 Na^+ 140, K^+ 3.9 Ca^{2+}: 9.0, Uric Acid: 8.0 FDP: Positive	3 units of FFP
5	Mechanically ventilated	CT Brain: Edema PT: 24, INR: 2.3PTT: 70 SGPT: 4200, SGOT: 4700 CPK: 12,800	Mannitol Hepamerz Gastrobiotic Liver support
6	No active bleeding	Continued high LFTs	Continued ICU care, liver support

Table 1 continued. Chronological clinical findings summary

Day	Vital Signs / Clinical Status	Labs / Imaging	Treatment / Intervention
7	Improving	Coagulation profile normalized SGPT: 1200, SGOT: 1400	Supportive
10	Consciousness regained	SGPT: 350, SGOT: 600	Supportive
14	Weaned from mechanical ventilation Started physiotherapy	Continued improvement	Physiotherapy

ECG, electrocardiogram; CPK, creatine phosphokinase; CBC, complete blood count; WBC, white blood cell count; RBS, random blood glucose; CXR, chest X-ray; CVP, central venous pressure; PT, prothrombin time; PTT, partial thromboplastin time; INR, international normalized ratio; U/S, ultrasonography; FFP, fresh frozen plasma; FDP, fibrin degradation products; LFTs, liver function tests; ICU, intensive care unit; SGOT, aspartate aminotransferase (AST); SGPT, alanine aminotransferase (ALT); PCO_2, partial pressure of carbon dioxide; HCO_3^-, bicarbonate; SO_2, oxygen saturation; IV, intravenous; CT, computed tomography.

The initial administration of atropine was inappropriate, as it was based on a misdiagnosis of organophosphate poisoning (a common error in rural clinical settings where labeling and awareness are limited). Once correctly diagnosed, the patient was transferred to the ICU and received comprehensive care. Intravenous sodium bicarbonate was administered to correct metabolic acidosis, and high-dose oral N-acetylcysteine (NAC) was initiated (100 mg/kg every 8 hours for 24 hours) for its hepatoprotective antioxidant properties.

Supportive measures included electrolyte correction, particularly of hypokalemia, and transfusions of fresh frozen plasma (FFP), etamsylate (Dicynone), and tranexamic acid to manage DIC. As her hepatic function worsened, she was treated with mannitol for cerebral edema, Hepa-Merz to assist liver detoxification, and Gastrobiotic to support gastrointestinal integrity. Mechanical ventilation became necessary due to her comatose state and respiratory compromise. With aggressive supportive therapy, her clinical and laboratory parameters improved steadily, leading to full recovery. A comparison of this case with previously published cases is presented in Table 3.

DISCUSSION

Hydrogen cyanamide poisoning is underreported in the medical literature, and most existing documentation relates to occupational exposure rather than intentional ingestion [8]. Compared with other reports of hydrogen cyanamide poisoning, this case is significant for the uncommon combination of fulminant hepatitis, DIC and cerebral edema, as well as the clearly documented temporal course and use of NAC. To our knowledge, this is the first report to describe this combination of complications following hydrogen cyanamide ingestion, thereby expanding the recognized severe toxic phenotype of this rare poisoning. These findings are important because they widen the recognized clinical spectrum of acute ingestion, assist clinicians in anticipating rapid deterioration with hepatic failure and coagulopathy, and provide a practical treatment reference for similar cases.

The incident also raises broader concerns about the storage of agricultural chemicals. A study conducted in rural Zhejiang, China [9], revealed that more than half of households stored pesticides at home, with nearly two-thirds keeping them in easily accessible places. This storage practice doubled the odds of suicidal ideation. The patient in this case consumed a readily available chemical stored in her home, which emphasizes the need for stricter regulation and safer storage.

Hydrogen cyanamide poisoning might be mistaken with other agricultural toxicities, therefore initially go unrecognized. Its symptoms are frequently ambiguous and can overlap with organophosphorus or other pesticide exposures, making it an important differential diagnosis in rural areas. The referring hospital's initial misdiagnosis of organophosphate poisoning was probably caused by overlapping cholinergic syndrome and the high frequency of pesticide exposure in the area. Although detailed records from the referring hospital were unavailable, except for the administration of 30 mg atropine which suggests that clinicians strongly suspected organophosphorous poisoning. Following transfer, the diagnosis was established by excluding organophosphorus poisoning, supported by the family-reported history of Dormex ingestion, the absence of clinical response to atropine, and normal cholinesterase level. The similarity in symptoms, especially those linked to parasympathetic overactivity, can result in inappropriate treatment. Measurement of pseudocholinesterase activity was crucial in confirming the true nature of the poisoning [4].

As in the present case, previous reports have linked hydrogen cyanamide poisoning with altered consciousness, tachycardia, and metabolic acidosis. A 22-year-old male with similar findings was described but had a fatal outcome [10]. The systemic stress response, elevated catecholamines, and mitochondrial dysfunction all contribute to laboratory findings such as leukocytosis, hyperglycemia, and liver enzyme abnormalities [5, 11].

By day three, this patient developed clinical signs of coagulopathy and progressed to overt DIC, a severe and rarely reported complication of hydrogen cyanamide poisoning. It has been suggested that hydrogen cyanamide causes oxidative stress and endothelial injury, leading to tissue factor exposure and activation of the coagulation cascade [12]. While hematuria and hematemesis have been reported previously [7, 13], this case appears unique in demonstrating overt DIC associated with multi-organ failure.

Table 2. Laboratory findings in details

Day	SGOT	SGPT	K^+	Na^+	Creatinine	Urea	WBC count	INR	Other
Day 1	35	52	3.4	142	1.18	34	13,300	Normal	ECG: Sinus Tachycardia CXR: Normal VBG: pH 7.12, PCO_2 38, HCO_3 12.4, SO_2 69% CPK: Normal Troponin: Normal Pseudocholinesterase: 6323
Day 2	-	-	3	-	-	-	-	-	
Day 3	37	55	2.4	158	-	-	-	23.4	Abdominal U/S: Fatty liver Amylase: Normal Lipase: Normal
Day 4	-	-	3.9	140	1.8	70	15,000	12.9	FDP: +ve Uric acid: 8 Calcium: 9 Albumin: 3.3
Day 5	4700	4200	-	-	-	-	-	2.3	CT Brain: Edema CPK: 12,800
Day 7	1400	1200	-	-	-	-	-	Normal	
Day 10	600	350	-	-	-	-	-	-	

SGOT, aspartate aminotransferase (AST); SGPT, alanine aminotransferase (ALT); WBC, white blood cell count; INR, international normalized ratio; ECG, electrocardiogram; CXR, chest X-ray; VBG, venous blood gas; PCO_2, partial pressure of carbon dioxide; HCO_3^-, bicarbonate; SO_2, oxygen saturation; CPK, creatine phosphokinase; U/S, ultrasonography; FDP, fibrin degradation products; UA, uric acid; Ca, calcium; CT, computed tomography.

Table 3. Case summary compared to some cases published

Study	Patient demographics	Exposure details	Main clinical features	Complications	Treatment	Outcome
Bernasconi et al., 2023 (Case 1)	45-year-old man	Accidental ingestion of approximately 50 mL of the undiluted product	Nausea, vomiting, coma	Severe central nervous system (CNS) depression requiring intensive care	Supportive care, including intubation and intensive care unit (ICU) admission	Full recovery
Bernasconi et al., 2023 (Case 10)	74-year-old man	Accidental ingestion of approximately 15 mL	Nausea, vomiting, confusion, agitation, buccal ulcers	No significant caustic injury on endoscopy	Supportive care	Full recovery
Bernasconi et al., 2023 (Case 11)	74-year-old man	Accidental ingestion of approximately 50 mL of the undiluted product	Agitation, confusion, coma, hemiplegia	Acute pancreatitis, elevated amylase and lipase levels, prolonged ICU stay (27 days)	Intubation, ICU admission, supportive care	Complete recovery
Bernasconi et al., 2023 (Case 12)	42-year-old man	Accidental ingestion of approximately 50 mL of the undiluted product	Nausea, vomiting, confusion, agitation, coma, hallucinations	Hepatic necrosis, acute pancreatitis, corrosive esophagitis, gastritis, aspiration pneumonia	Intubation, supportive care, endoscopic evaluation	Clinical improvement; long-term follow-up unavailable
Sheshadri et al., 2011	Young adult (demographic details not fully reported)	Acute hydrogen cyanamide poisoning	Severe acute poisoning	Insufficient information available	Not fully reported	Outcome not fully reported
The current case	16-year old female	Intentional ingestion of dormex of unknown amount	Drowsiness, sinus tachycardia, metabolic acidosis and modest elevated liver enzymes	DIC, fulminant hepatitis and cerebral edema	Supportive treatment added to N acetyl cysteine	Full recovery

Liver injury in this case was marked and consistent with previous case reports and experimental findings. Hydrogen cyanamide generates reactive oxygen species that deplete hepatic antioxidants, resulting in hepatocyte necrosis [14]. Elevated transaminases (AST and ALT) indicate hepatocellular injury, and reduced thrombopoietin levels

resulting from hepatic dysfunction contributes to thrombocytopenia, further increases bleeding risk [15].

Renal injury, as evidenced by rising creatinine levels, is often a poor prognostic indicator in hydrogen cyanamide poisoning [7]. The cerebral edema observed on CT imaging parallels findings in previous reports, [4, 16], and may reflect systemic inflammatory response and capillary leakage.

Hydrogen cyanamide most likely caused the presented severe toxicity by inhibiting mitochondrial oxidative metabolism, resulting in cellular hypoxia, ATP depletion, lactic acidosis, and oxidative hepatocellular injury, which may have led to acute liver failure, coagulopathy, cerebral edema and multi-organ dysfunction [5].

Therapeutically, the use of NAC to counteract oxidative liver injury was considered important. It was administered empirically because acute liver injury had developed, as NAC is frequently used as supportive therapy in toxic hepatic injury and non-acetaminophen acute liver failure [17]. There is no documented specific antidote for hydrogen cyanamide poisoning, thus NAC in our case should be regarded as an empiric hepatoprotective intervention rather than an evidence-based standard treatment for hydrogen cyanamide poisoning.

The use of mannitol in reducing intracranial pressure and various hemostatic agents in controlling bleeding were also essential in management. Other authors have reported success using similar approach [4, 5]. In contrast, a fatal outcome was reported despite supportive care, highlighting the importance of early recognition and intervention [10].

According to the 2023 Pavia Poison Control Center series, ingestion cases were generally more severe than inhalational exposures and were occasionally complicated by coma, pancreatitis, or hepatic necrosis [8]. Although this series aids in defining the broader serious spectrum of toxicity, it does not explain the particular combination of DIC, severe fulminant hepatitis and cerebral edema observed in our patient. More recently, an Egyptian occupational exposure case demonstrated altered mental status, visual hallucinations, and skin lesions following Dormex exposure, emphasizing the clinical spectrum's variability and the lack of a precise antidote [18].

The 2011 fatal ICU case report demonstrated refractory cardiogenic shock despite extensive supportive treatment due to hydrogen cyanamide consumption [10]. While it confirmed the severity of this poisoning, it did not report the same pattern of fulminant hepatic, DIC, and cerebral edema of our case. Taken together, these findings demonstrate that our case adds a new and more severe clinical phenotype to the existing literature.

The key lesson from this case is the importance of considering hydrogen cyanamide toxicity whenever exposure to agricultural chemicals is suspected. Because the agent may be misidentified or mislabeled, diagnosis relies heavily on clinical suspicion. Early recognition is crucial because confirmatory testing is often unavailable. Rapid neurological deterioration, metabolic acidosis, and progressive organ dysfunction may indicate severe poisoning and help identify patients at high risk of deterioration. Despite the severity of the illness, this patient survived, presumably because of early intensive care, airway protection, and sustained supportive therapy throughout the critical phase.

CONCLUSION

This case underscores that hydrogen cyanamide can cause rare and life-threatening complications, including DIC, fulminant liver failure, and cerebral edema, while still allowing complete recovery with aggressive supportive care. Clinicians in agricultural regions should consider this toxicant in relevant exposure scenarios, recognize systemic manifestations early, differentiate it from other pesticide toxicities, and consider NAC as an adjunct to supportive management.

There is an urgent need for public health interventions to improve the safe storage and regulation of hydrogen cyanamide. Increased awareness among healthcare professionals, especially in rural areas, may help prevent misdiagnosis and improve outcomes. Finally, further research into the pathophysiology, epidemiology, and potential antidotes for hydrogen cyanamide toxicity is warranted.

Contribution:

Nermin H Zawilla (Professor of Occupational and Environmental Medicine, Faculty of Medicine, Cairo University, Cairo, Egypt; Head of National Environmental and Clinical Toxicological Research Centre (NECTR), Cairo University, Egypt) as a contributor for supervising and reviewing the final draft of manuscript.

ACKNOWLEDGMENTS

Not applicable.

Conflict of interest: The authors declare no conflict of interest.

Funding and Support: The authors received no financial support.

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