Case Report ISSN: 3065-5641

Global Journal of Critical Care and Emergency Medicine

Catastrophic Toxic Alcohol Poisoning from Liquor and Perfume Ingestion: A Case of Rapid Neurological Deterioration and Multi-Organ Dysfunction

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Received: 18 Aug 2025; **Accepted:** 25 Sep 2025; **Published:** 06 Oct 2025

Citation: EL MAWLA Zeinab, MROUEH Fatima, TOFAILI Hawraa, et al. Catastrophic Toxic Alcohol Poisoning from Liquor and Perfume Ingestion: A Case of Rapid Neurological Deterioration and Multi-Organ Dysfunction. Glob J Emerg Crit Care Med. 2025; 2(3); 1-7.

ABSTRACT

Background: *Alcohol intoxication, whether from conventional or unconventional sources, can lead to severe metabolic disturbances and neurological injury. Ethanol-containing consumer products, such as colognes and perfumes, are sometimes intentionally ingested, particularly by individuals with alcohol use disorder, despite containing denatured alcohol. Ingestion of perfume is uncommon in adults but poses a dual risk: ethanol intoxication and toxicity from denaturants and co-solvents like methanol and propylene glycol. This case report highlights the severe consequences of such ingestions.*

Case Presentation: *We present the case of a 28-year-old female with a history of major depressive disorder and chronic alcohol use disorder who ingested a massive quantity of vodka (>750 mL), perfume (≈750 mL), and an overdose of escitalopram. This led to catastrophic toxic alcohol poisoning, resulting in rapid neurological deterioration and multi-organ dysfunction, including refractory mixed metabolic acidosis, central diabetes insipidus, delayed acute kidney injury, and severe thrombocytopenia. Despite aggressive treatment, including hemodialysis, she progressed to brain death within a few hours of presentation.*

Discussion: *This case underscores the critical dangers associated with the ingestion of ethanol-based cosmetics, particularly in large volumes. The combined presence of ethanol, methanol (a common denaturant), and propylene glycol created a complex toxicological scenario. The discussion details the pathophysiology of her presentation, focusing on the interplay of these toxic agents, the evolution of acid-base disturbances, and the mechanisms underlying the observed organ dysfunctions, including delayed acute kidney injury and severe thrombocytopenia. The challenges in managing such refractory conditions and the importance of early intervention are also explored.*

Conclusion: *This case report emphasizes the life-threatening risks of ingesting ethanol-based cosmetics and highlights the rapid and severe progression of multi-organ dysfunction that can ensue. It underscores the importance of a high index of suspicion for toxic alcohol ingestion in patients presenting with severe metabolic acidosis and neurological compromise, even when initial ethanol levels are unavailable. The case also illustrates the complex acid-base dynamics, the development of central diabetes insipidus as a sentinel sign of brain death, and the delayed onset of acute kidney injury and thrombocytopenia due to multiple contributing factors. Early aggressive treatment, including hemodialysis and supportive care, is crucial, but prevention through multidisciplinary efforts remains paramount.*

Keywords
Toxic alcohol poisoning, Perfume ingestion, Methanol, Propylene glycol, Metabolic acidosis, Brain death, Acute kidney injury, Thrombocytopenia, Case report.

Introduction
Alcohol intoxication, whether originating from conventional alcoholic beverages or from less conventional sources, represents a significant public health concern capable of inducing profound metabolic derangements and severe neurological sequelae. A notable, albeit less common, pathway for such intoxication involves the intentional ingestion of ethanol-containing consumer products, such as colognes, perfumes, and mouthwashes. This phenomenon is particularly observed among individuals with alcohol use disorder, who may resort to these substances despite their formulation with denatured alcohol—ethanol rendered unsuitable for potable consumption through the addition of various denaturants, including methanol or other solvents [1,2].

Ingestion of perfume, while infrequent in the adult population, presents a complex toxicological challenge due to its dual risk profile: not only does it lead to ethanol intoxication, but it also introduces the potential for toxicity from its denaturing agents and co-solvents, such as methanol and propylene glycol (PG) [3,4]. Previous studies have documented postmortem blood alcohol concentrations (BACs) ranging from 136 to 608 mg/dL in individuals succumbing to alcohol poisoning, with an average BAC of 400 mg/dL typically associated with severe stupor, respiratory depression, coma, and hypothermia. A BAC exceeding 500 mg/dL is generally considered potentially fatal, primarily due to the suppression of vital brain centers regulating respiration and circulation, ultimately leading to asphyxia [5,6].

Furthermore, the presence of methanol contamination in perfume alcohol is a well-documented concern [3,7]. Methanol poisoning is particularly insidious, as its metabolism yields toxic byproducts that can precipitate high-anion-gap metabolic acidosis, irreversible visual impairment, and severe brain damage [8]. Similarly, propylene glycol, another ubiquitous ingredient in perfumes, undergoes metabolism via alcohol dehydrogenase to lactic acid. In substantial doses, PG can induce severe lactic acidosis, an elevated osmolar gap, and widespread organ dysfunction [9].

This report details the case of a young woman who presented with catastrophic toxic alcohol poisoning following the ingestion of a substantial quantity of vodka (>750 mL), perfume (≈750 mL), and an overdose of escitalopram. Despite the implementation of aggressive therapeutic interventions, including emergent hemodialysis, her clinical course was marked by rapid progression to brain death within a few hours of presentation. This case is particularly noteworthy due to the unusual nature of the ingested substances and the subsequent development of a constellation of severe complications, including refractory mixed metabolic acidosis, central diabetes insipidus, delayed acute kidney injury, and severe thrombocytopenia. We aim to provide a comprehensive discussion of the pathophysiology underlying her presentation, drawing upon relevant literature concerning the toxicity of perfume constituents, the dynamics of acid–base disturbances, and the mechanisms of organ dysfunction observed in similar toxic ingestions.

Case Presentation
The patient was a 28-year-old female with a documented medical history of major depressive disorder, managed with escitalopram (10 mg daily), and chronic alcohol use disorder. Four years prior to presentation, she had undergone sleeve gastrectomy. The acute event was precipitated by the patient's clandestine ingestion of approximately 750 mL of women's perfume, containing denatured alcohol, propylene glycol, glycerin, hydrogenated castor oil, ethylhexyl methoxycinnamate, butyl methoxydibenzoylmethane, limonene, linalool, hexyl cinnamal, citronellol, geraniol, and coumarin. This ingestion occurred following a significant vodka binge, reportedly exceeding one bottle, and was a response to family intervention limiting her access to alcohol. Concurrently, she reportedly ingested ten 10-mg tablets of escitalopram (totaling 100 mg). The onset of symptoms was marked by generalized tonic convulsions, rapidly progressing to a comatose state. No traumatic injuries were observed. The full extent and nature of the ingested substances, particularly the perfume, were not immediately apparent upon hospital admission, with the perfume ingestion only being confirmed several days later by family members who discovered the empty bottles.

Upon arrival at the emergency department, estimated to be several hours post-ingestion, the patient presented in a comatose state with a Glasgow Coma Scale (GCS) score of 7. She exhibited generalized tonic activity, jaw clenching, and hypersalivation. Initial vital signs were stable, with a blood pressure of 120/62 mmHg, heart rate of 84 beats/min, respiratory rate of 8 breaths/min, temperature of 36.7°C, and oxygen saturation of 98% on room air. Bradypnea was noted, consistent with severe alcohol intoxication. Capillary glucose was elevated at 217 mg/dL. Given her neurological status and respiratory compromise, the patient was promptly intubated for airway protection and ventilatory support. Initial management included intravenous administration of diazepam and valproate to control seizures, high-dose multivitamin B cofactors (including thiamine and folate) to empirically address potential toxic alcohol effects, and gastric lavage. Despite these supportive measures, her unresponsiveness persisted, accompanied by severe metabolic disturbances.

Key laboratory findings upon ER admission revealed profound high anion gap metabolic acidosis (HAGMA). Arterial blood gas analysis showed a pH of 6.80, an undetectably low bicarbonate level (approximately 4 mmol/L), and a partial pressure of carbon dioxide (pCO_2) of 16 mmHg, indicating partial respiratory compensation. Serum chemistry results included Na^+ 135 mmol/L, Cl^- 106 mmol/L, and CO_2 approximately 4 mmol/L, resulting in an anion gap (AG) of 25 mmol/L. Serum K^+ was 5.5 mmol/L, and creatinine was 0.93 mg/dL. Due to the unavailability of plasma

osmolality and serum ethanol levels, the osmolar gap and serum ethanol concentration could not be calculated; however, given the reported exposure to multiple alcohols and glycols, both were presumed to be elevated. Urine toxicology and ketone assays were negative. The anion gap acidosis was attributed to toxic alcohol ingestion, potentially including ethanol, methanol, or glycols present in the perfume, compounded by lactic acidosis. The patient also exhibited significant polyuria, producing approximately 400 mL/hour of dilute urine in the emergency department. A non-contrast head computed tomography (CT) scan revealed diffuse supra- and infra-tentorial brain edema, with no evidence of hemorrhage or midline shift. (Figure 1).

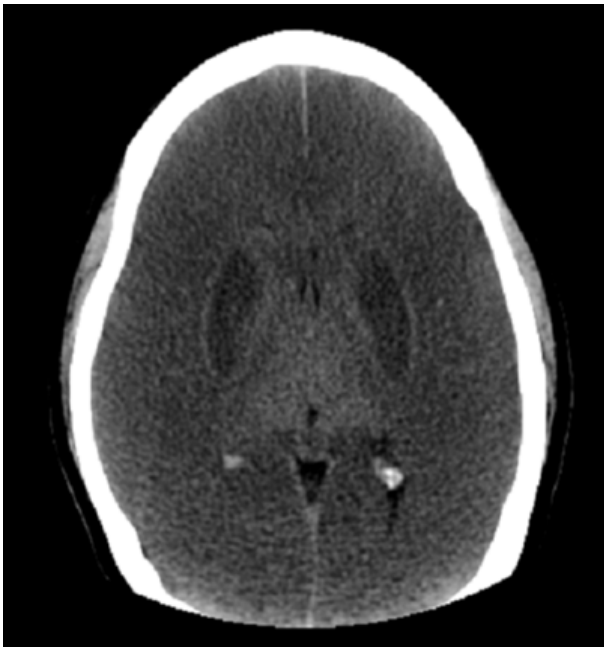

Figure 1: A non-contrast CT scan revealed diffuse brain edema.

Initial resuscitation efforts focused on correcting the severe acidosis and facilitating the removal of suspected toxins. The patient was sedated and mechanically ventilated to ensure airway protection and adequate ventilation. Repeated boluses of intravenous sodium bicarbonate, totaling approximately 200 mEq, were administered to buffer the severe acidemia. Seizure control was achieved with diazepam (10 mg IV total) and valproate (1 g IV), followed by continuous sedation. Cofactor therapy, including high-dose B vitamins (folate, thiamine, pyridoxine), was empirically initiated to support alcohol metabolism. Given the refractory acidosis (pH persistently below 7.0 despite bicarbonate administration) and strong suspicion of toxic alcohol poisoning, emergent hemodialysis was initiated approximately 4 hours after hospital arrival.

Over the first hospital day, the patient remained comatose. Hemodialysis and buffering strategies led to a partial improvement in acidosis. Approximately 6 hours into admission, her arterial pH had risen to 7.27, with bicarbonate levels around 8 mmol/L, though the anion gap remained elevated at approximately 23 mmol/L. By 12 hours post-admission, the pH further improved to 7.34, with bicarbonate at approximately 13 mmol/L. However, her neurological status showed no signs of recovery. Following the cessation of sedation, her GCS remained at 3, and she exhibited absent brainstem reflexes, including unreactive pupils, absent corneal reflexes, no oculocephalic or oculovestibular responses, and absent gag and cough reflexes, in addition to a positive apnea test. Neuroimaging, specifically MRI and MR angiography performed approximately 12 hours post-admission, demonstrated global cerebral edema with absent intracranial blood flow, consistent with diffuse anoxic injury and impending herniation. The absence of flow in intracranial arteries on MRA and diffuse cytotoxic edema on diffusion-weighted imaging were indicative of irreversible loss of cerebral perfusion, suggesting brain death. (Figure 2) Formal declaration of brain death was deferred pending further correction of metabolic confounders.

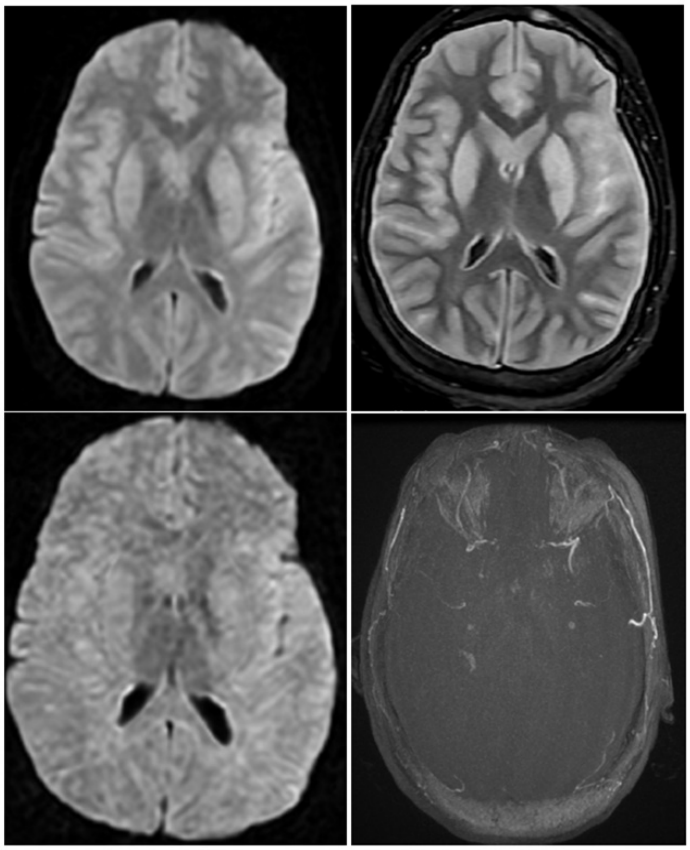

Figure 2: MR showed global cerebral edema with absent intracranial blood flow, consistent with diffuse anoxic injury and impending herniation.

Aggressive supportive care continued over the first 24–36 hours. Osmotherapy with intravenous mannitol was initiated to manage intracranial pressure. Broad-spectrum antibiotics (vancomycin and ceftriaxone) were empirically started due to a rising white blood cell count (20×10^3/μL with left shift), raising suspicion for meningitis. Diabetes insipidus (DI) became evident, characterized by massive polyuria (approximately 5.7 L in the first 24 hours)

and a rapidly rising serum sodium level, peaking at 165 mmol/L. Despite aggressive fluid replacement, she developed hypernatremia with low urine specific gravity. A trial of desmopressin resulted in a dramatic reduction in urine output, confirming central DI, likely secondary to hypothalamic damage. Brain death was formally confirmed on hospital day 4 based on neurological examination (GCS 3, absent brainstem reflexes off sedation with a positive apnea test) and prior imaging evidence of absent cerebral blood flow.

Throughout the first week in the intensive care unit, the patient's metabolic parameters demonstrated a notable evolution. The HAGMA gradually resolved following dialysis, subsequently transitioning to a normal-gap (hyperchloremic) acidosis. The initially extremely high anion gap normalized by days 2–3 after toxin clearance, and in some instances, even dropped below normal (0–6 mmol/L) as chloride levels increased. Concurrently, acute kidney injury (AKI) manifested later in the course; her creatinine, initially within normal limits, began to rise on day 5, peaking at 8.1 mg/dL by day 8, necessitating another session of dialysis. Furthermore, the patient experienced marked thrombocytopenia; after an initial stress-related elevation (platelets 421×10^3/µL on admission), her platelet count declined to a nadir of approximately 58×10^3/µL by day 5. This thrombocytopenia subsequently reversed towards normal.

The timeline of major events and laboratory trends unfolded as follows: During hours 0–12, the patient presented with severe anion gap metabolic acidosis (pH nadir 6.80, AG 25), accompanied by seizures and coma. Hemodialysis was initiated, and neuroimaging revealed global cerebral edema. By days 1–2, the coma persisted with areflexia, and brain death was confirmed. The anion gap began to trend downwards, reaching approximately 8 by 24 hours. Diabetes insipidus, likely present since admission given the patient's polyuria in the emergency room, was managed with desmopressin. Between days 3–5, the anion gap further declined (AG 1–3 by days 4–5), accompanied by hyperchloremia, and electrolytes were managed accordingly. Fever and leukocytosis were empirically treated as possible sepsis, and antibiotics were continued despite negative cultures. A high vancomycin trough (60 µg/mL) was noted, and the dose was held. By day 5, creatinine had risen from approximately 0.9 to 3.4 mg/dL, marking the onset of acute kidney injury, and platelet count reached a nadir of around 58×10^3/µL. On days 6–8, renal failure worsened, with creatinine rising from 6 to 8 mg/dL by day 7 despite adequate hydration, and the patient required dialysis again on day 8 while remaining polyuric. Platelets began to recover, reaching 123×10^3/µL by day 9. From day 9 onward, supportive care continued while awaiting family decisions regarding care withdrawal. The patient's neurological status remained consistent with brain death, and she ultimately passed away after 2 weeks.

Discussion

The patient's critical presentation was primarily driven by the ingestion of a significant quantity of ethanol and perfume, the latter containing denatured alcohol (methanol) and propylene glycol (PG). The estimated ethanol intake, calculated at approximately 710.1 grams, translates to a dose of 8.9 g/kg for an 80 kg individual, a level critically high and far exceeding the fatal serum concentration of 500 mg/dL [6]. Pharmacokinetic back-calculation suggested an estimated serum ethanol concentration of approximately 1,483 mg/dL, consistent with the observed severe central nervous system (CNS) depression, including coma and respiratory depression, which are hallmarks of severe ethanol poisoning. The rapid onset of these symptoms underscored the profound impact of such a massive ethanol load on the patient's physiological functions.

The presence of methanol as a denaturant in the perfume introduced a particularly dangerous dimension to the intoxication. Methanol is metabolized to formic acid, a highly toxic compound responsible for metabolic acidosis, ocular toxicity, and potential basal ganglia damage [1]. While direct measurement of serum methanol was unavailable, the clinical picture, including refractory acidosis, seizures, and progression to brain death, aligned with severe methanol poisoning. Cases from various countries document methanol poisoning outbreaks from colognes and perfumes used as surrogate alcohol, highlighting the insidious nature of this exposure [1]. The patient's presentation with coma, respiratory depression, and severe metabolic acidosis, particularly an arterial pH <7 on admission, are all indicators of a poor prognosis in methanol poisoning [11]. Propylene glycol (PG), another constituent of the ingested perfume, further compounded the patient's metabolic derangements. PG is metabolized by alcohol dehydrogenase into lactate, pyruvate, and other substrates [9]. Excessive PG load can lead to lactic acidosis and an elevated osmolar gap [12]. In this case, PG likely contributed to the patient's acidosis by elevating serum lactic acid levels, thereby exacerbating the overall metabolic insult from ethanol and methanol. The combined presence of ethanol, methanol, and PG created a synergistic toxicological effect, leading to a perfect storm of metabolic poisons, overwhelming the patient's homeostatic mechanisms.

Upon admission, the patient presented with a severe high anion gap metabolic acidosis (HAGMA), characterized by a pH of 6.8 and an anion gap (AG) of 25. This biochemical derangement signified the accumulation of unmeasured anions, which in this context, included lactate, formate, ketones, and potentially glycolate from the ingested toxins. The patient's respiratory compensation was appropriate, as indicated by a PCO2 of 16 mmHg, consistent with Winter's formula, and suggesting the absence of a coexisting primary respiratory acid-base disorder. However, a more detailed analysis using the delta ratio ($\Delta AG/\Delta HCO_3^-$) yielded a value of 0.65 (<1), which is indicative of a mixed metabolic acidosis. This suggested the presence of a normal anion gap metabolic acidosis (NAGMA) occurring concurrently with the HAGMA. The underlying mechanism for this mixed picture pointed towards an increase in bicarbonate loss from the kidneys with concomitant chloride retention. In typical scenarios of NAGMA, hyperchloremia is a common finding. However, in this patient, the chloride level

was paradoxically normal (Cl^- = 106 mmol/L). This seemingly contradictory finding can be elucidated by considering the specific context of toxic alcohol ingestion. The intake of propylene glycol and the subsequent initiation of mannitol therapy, administered for cerebral edema, likely induced significant osmotic diuresis. This diuresis would have led to substantial losses of sodium bicarbonate, contributing to the NAGMA component. Furthermore, the chloride level, while numerically normal, was disproportionately elevated relative to sodium ($Cl^-/Na^+ \approx 0.79$), exceeding the normal ratio of 0.75, thereby suggesting a state of relative hyperchloremia [14]. This nuanced interpretation clarified the acid-base dynamics in the presence of multiple confounding factors. Following dialysis, a critical intervention aimed at removing circulating toxins and correcting metabolic imbalances, the unmeasured anions such as lactate and ketones were effectively cleared. This therapeutic success was reflected in the restoration of the anion gap towards normal limits. However, the patient subsequently developed a low bicarbonate and hyperchloremic metabolic acidosis, consistent with the resolution of the HAGMA and the persistence of the NAGMA component. On day 3, a transient rebound in the anion gap to 14 was observed. This phenomenon could be attributed to the dilution of serum chloride, which decreased from 130 to 120 mmol/L, following the administration of free water for the management of diabetes insipidus. Moreover, the ongoing acidosis at this stage suggested the presence of residual unmeasured anions or a rebound lactic acidosis, potentially secondary to tissue ischemia or sepsis, a suspicion supported by the upward trend in C-reactive protein (CRP) and the patient's requirement for vasopressor support. By days 4 to 5, the patient's albumin level exhibited a slight decrease to approximately 3.0 g/dL, while chloride levels remained elevated (approximately 113 to 117 mmol/L). This combination resulted in a measured anion gap that fell below the normal range (1 to 3 mmol/L). A low or negative anion gap is an uncommon finding and can be indicative of the accumulation of unmeasured cations, hyperchloremia, or hypoalbuminemia [15]. Given that albumin is a negatively charged protein, a reduction in its concentration can significantly impact the calculated anion gap. Specifically, the anion gap decreases by approximately 2.5 mmol/L for every 1 g/dL drop in albumin. Therefore, an albumin level of 3 g/dL would reduce the "normal" AG by 3 to 4 points, partly explaining the observed AG of 1 to 3. In summary, the acid-base course in this case was dynamic and multiphasic: initially, a very high anion gap metabolic acidosis primarily due to lactate and toxic alcohols; followed by a normal anion gap metabolic acidosis with hyperchloremia post-dialysis; a transient mild high anion gap metabolic acidosis attributable to chloride dilution and potential accumulation of minor acids; and finally, a low anion gap due to hypoalbuminemia and persistent hyperchloremia. This complex acid-base trajectory underscored the critical importance of serial acid-base analysis, coupled with the delta ratio, for identifying mixed disorders and monitoring concealed chloride levels [14].

Shortly after admission, the patient developed polyuria and hypernatremia, clinical manifestations highly suggestive of diabetes insipidus (DI). This condition is a recognized complication in brain-dead organ donors, with studies reporting an incidence of up to 80% [16]. Central DI arises from a deficiency in antidiuretic hormone (ADH) due to injury to the hypothalamus or pituitary gland. The posterior pituitary, in particular, is exquisitely sensitive to increased intracranial pressure, and ADH production can cease following brainstem herniation or infarcts within the hypothalamic tissue. In the absence of ADH, the renal tubules are unable to concentrate urine, leading to copious dilute urine output and a subsequent rise in serum osmolarity and sodium concentration. In the intensive care unit (ICU) setting, DI is typically diagnosed when urine output exceeds a specified threshold per hour, accompanied by dilute urine (specific gravity <1.005) and elevated serum sodium levels. Consistent with this, our patient exhibited a urine output of up to 6 liters per day, with serum sodium levels escalating to 165 mmol/L despite aggressive fluid replacement strategies. The administration of desmopressin, a synthetic ADH analog, resulted in a significant reduction in urine output to 20 ml per hour and a gradual normalization of serum sodium concentration (Na^+ = 150 mmol/L), thereby confirming the diagnosis of central diabetes insipidus. This case highlighted DI as a sentinel complication of brain death, and its emergence in comatose patients should prompt a thorough evaluation for severe cranial injury or impending brain death [17].

One of the notable features in this clinical scenario was the delayed onset of acute kidney injury (AKI). Despite severe shock and acidosis on admission, the patient's creatinine remained normal (~0.9 mg/dL) for the initial 3 to 4 days. However, on day 5, creatinine acutely increased to 3.4 mg/dL, further rising to 8.1 mg/dL over the subsequent 2–3 days. Initially non-oliguric, the patient eventually became oliguric with increasing potassium levels, necessitating dialysis on day 8. This delayed presentation suggested a subacute kidney injury rather than an immediate ischemic acute tubular necrosis (ATN) resulting from the initial insult. Several mechanisms likely contributed to this delayed AKI. Firstly, the patient had significant exposure to propylene glycol (PG) from the ingested perfume. High serum PG levels have been associated with acute tubular necrosis in some studies [18]. Secondly, both PG and mannitol, administered for cerebral edema, can induce osmotic nephrosis. This phenomenon involves the uptake of high-osmolar substances by proximal tubular cells, leading to vacuolar degeneration [19]. The patient received a moderate dose of mannitol (approximately 60 g in total over 24 hours), which, combined with PG (and potentially glycerol from the perfume), imposed a substantial osmotic load on the kidneys. This osmotic burden initially accentuated the polyuria caused by DI and subsequently contributed to subacute tubular injury. Furthermore, osmotic diuresis can mask early ATN, as evidenced by the patient's very low blood urea nitrogen (BUN) (4 mg/dL) during early ICU days due to hyperhydration, reflecting a state of high-flow diuresis. The accumulation of BUN only commenced when AKI became apparent. Pathologically, osmotic nephrosis often manifests a few days after the initial insult, aligning with the patient's clinical timeline [20]. Thirdly, the patient received empirical vancomycin for suspected pneumonia or sepsis. Vancomycin is a well-known

nephrotoxin, particularly at high doses or in combination with other nephrotoxic agents. On day 5, the patient's vancomycin level was found to be 60 µg/mL, significantly above the usual therapeutic range of 10–20 µg/mL. This accumulation was likely due to the patient's deteriorating renal function. Vancomycin-associated AKI typically presents as tubular necrosis or interstitial nephritis, occurring 4 to 8 days after antibiotic initiation, with a greater than 50% rise in creatinine. In this case, the initial rise in creatinine from 0.9 to 3.4 mg/dL coincided with 3–4 days of vancomycin therapy, prompting its discontinuation. However, stopping vancomycin did not immediately reverse the kidney injury, which progressed to stage 3 AKI, suggesting that the damage (ATN) was already established or that additional factors were at play. A review indicated that 85–90% of reported vancomycin-induced thrombocytopenia cases also exhibited nephrotoxicity, suggesting a systemic hypersensitivity or combined toxicity effect [21]. Finally, brain death itself can contribute to AKI. The catecholamine surge associated with brain death can lead to vasodilation and impaired renal perfusion [22]. Although the patient's blood pressure was maintained, microscopic ischemic insults cannot be entirely excluded. In analyzing the etiology of AKI, a multifactorial scenario is most probable: initial subclinical ATN from lactic acidosis and hypotension, exacerbated by the nephrotoxic effects of vancomycin, and direct osmolar injury from mannitol and PG. This combination resulted in a delayed but severe AKI. This pattern is consistent with cases of propylene glycol toxicity where AKI develops after the acidosis, not immediately [23]. This case underscored the importance of close monitoring of renal function, as the absence of early AKI does not guarantee subsequent renal stability. Once creatinine increased, further nephrotoxins were avoided, all medication doses were adjusted, and dialysis was initiated on day 8 due to metabolic indications (severe acidosis and hyperkalemia). Although the patient's creatinine slightly improved to ~6.8 mg/dL after dialysis, long-term renal recovery could not be assessed due to the progression to brain death.

The patient experienced a significant decline in platelet count, from an initial 421 × 10^3/µL on admission to 58 × 10^3/µL by day 5. Several mechanisms and triggers could explain this notable drop. Initially, the very high platelet count on admission could represent a reactive response to stress, an epinephrine surge, or hemoconcentration secondary to dehydration. Within 12 to 24 hours, following fluid resuscitation, the platelet count decreased to approximately 181 × 10^3/µL and then to 130 × 10^3/µL. Some literature suggests that severe head injury or brain death can activate the coagulation cascade, sometimes referred to as "neurogenic disseminated intravascular coagulation (DIC)," due to the release of brain thromboplastin. However, in this case, coagulation tests were largely normal, making overt DIC unlikely. Nevertheless, a mild consumptive coagulopathy resulting from brain tissue necrosis could have contributed to the platelet reduction. Among the medications administered, vancomycin is a well-documented cause of immune-mediated thrombocytopenia (VIT). Vancomycin can act as a hapten, triggering the formation of antibodies that lead to platelet destruction upon re-exposure. Typically, VIT manifests after 5–7 days of therapy and can be quite severe [24]. In this patient, vancomycin was initiated on day 1, and by day 5, platelets had plummeted to 58 × 10^3/µL, representing an approximately 86% drop from baseline. This rapid and severe thrombocytopenia strongly suggested an immune-mediated mechanism. Furthermore, as previously noted, the patient's vancomycin level was toxic (60 µg/mL), which might have exacerbated this reaction, although even therapeutic levels can trigger VIT. Conversely, heparin-induced thrombocytopenia (HIT) was considered less likely, as the patient was on a prophylactic dose of heparin, and the timing of thrombocytopenia was too early for HIT (which typically occurs 5 to 10 days after therapy), with no evidence of thrombosis. Therefore, vancomycin was the most probable cause for the patient's platelet nadir. Vancomycin was discontinued after day 5, and consistent with the natural course of VIT, the platelet count increased over the subsequent 3 to 4 days, reaching 123 × 10^3/µL by day 9. This rebound after drug cessation strongly supported an immune drug-induced mechanism, as vancomycin-induced thrombocytopenia has been shown to resolve within one week after drug cessation [24]. In summary, the most likely etiology of the severe thrombocytopenia was vancomycin immune-mediated thrombocytopenia, potentially compounded by the underlying critical illness. The resolution of thrombocytopenia following drug cessation and the absence of signs of DIC further corroborated this diagnosis. This case underscored the importance of meticulously reviewing medication lists when a patient in the ICU experiences a decline in platelet count. Vancomycin-induced thrombocytopenia has been increasingly reported and carries the risk of life-threatening bleeding. Clinicians should consider testing for drug-dependent platelet antibodies in unclear cases; however, in clinical practice, discontinuing the offending drug often confirms the diagnosis when platelet counts recover.

Conclusion

This case highlights the dangers of ingesting ethanol-based cosmetics, like perfume, which can cause toxic alcohol poisoning. Large volumes contain ethanol, leading to respiratory failure, and denaturing agents like methanol and propylene glycol can damage organs, causing metabolic acidosis, seizures, or brain injury. Early treatment with dialysis, fomepizole, and cofactors is crucial, especially for severe acidosis (pH < 6.9), which may require hemodialysis but risks cerebral hypoxia or edema. Serial acid–base analysis, including the anion gap and delta ratio, helps identify mixed acidosis; correcting hyperchloremia can restore balance. Brain-dead patients may develop central diabetes insipidus (DI), with polyuria and hypernatremia responding to desmopressin, vital for stability and organ donation. Delayed nephrotoxicity may occur days after poisoning due to osmotic nephropathy from high osmolar loads like propylene glycol, glycerol, or mannitol, with nephrotoxic drugs like vancomycin compounding injury; frequent dose reevaluation is necessary. Thrombocytopenia in ICU patients can stem from immune reactions to vancomycin, causing about 90% platelet reduction after a week. Discontinuing the drug generally leads to recovery, preventing unnecessary transfusions and facilitating antibiotic changes.

This case highlights the risky behavior of alcohol-dependent patients ingesting perfume in distress. Multi-disciplinary efforts, including mental health support and restricting product access, should work together to prevent such incidents. From a critical care view, it provided valuable data for understanding the pathophysiology of complex toxic cases. Future cases may benefit from early antidotal therapy (like fomepizole, if available) and prompt dialysis to improve survival and neurological recovery. Despite optimal treatment, brain death was unavoidable due to severe poisoning, underscoring the danger of ingesting concentrated ethanol from non-beverage sources.

References

1. Nekoukar Z, Zakariaei Z, Taghizadeh F, et al. Methanol poisoning as a new world challenge: A review. Ann Med Surg. 2021; 66: 102445.
2. Bekatorou A. Alcohol: Properties and determination. Encyclopedia of Food and Health. 2016.
3. Brahmi N, Blel Y, Abidi N, et al. Methanol poisoning in Tunisia: report of 16 cases. Clin Toxicol (Phila). 2007; 45: 717-20.
4. Exner DV, Brien WF, Murphy MJ. Superwarfarin ingestion. CMAJ: Canadian Medical Association journal = journal de l'Association medicale canadienne. 1992; 146: 34-35.
5. Celik S, Karapirli M, Kandemir E, et al. Fatal ethyl and methyl alcohol-related poisoning in Ankara: A retrospective analysis of 10,720 cases between 2001 and 2011. J Forensic Leg Med. 2013; 20:151-4.
6. Dasgupta, A. Alcohol: Use, abuse, and issues with blood alcohol level. In A. Dasgupta (Ed.), Alcohol and its biomarkers (pp. 1–36). Elsevier. 2015. https://www.sciencedirect.com/science/article/pii/B9780128003398000018
7. Kalkan S, Cevik AA, Cavdar C, et al. Acute methanol poisonings reported to the Drug and Poison Information Center in Izmir, Turkey. Vet Hum Toxicol. 2003; 45: 334-7.
8. Kruse JA. Methanol and ethylene glycol intoxication. Critical care clinics. 2012; 28: 661-711.
9. Zosel A, Egelhoff E, Heard K. Severe lactic acidosis after an iatrogenic propylene glycol overdose. Pharmacotherapy. 2010; 30: 219.
10. Glowala J, Richards JB. Methanol Poisoning Leading to Brain Death: A Case Report. J Crit Care Med (Targu Mures). 2021; 8:66-70.
11. Liu JJ, Daya MR, Carrasquillo O, et al. Prognostic factors in patients with methanol poisoning. J Toxicol Clin Toxicol. 1998; 36: 175-81.
12. Downey K, Keklik R, Morrissey B, et al. Propylene glycol toxicity in an adolescent secondary to chronic cornstarch ingestion. J Am Coll Emerg Physicians Open. 2024; 5: e13188.
13. Khan M, Vartanyan A, Scalzo A, et al. Propylene glycol neurotoxicity due to sodium citrate therapy in an infant with renal tubular acidosis. Clin Nephrol Case Stud. 2020; 8: 33-36.
14. Durward A, Skellett S, Mayer A, et al. The value of the chloride: sodium ratio in differentiating the aetiology of metabolic acidosis. Intensive Care Med. 2001; 27: 828-35.
15. Lee S, Kang KP, Kang SK. Clinical usefulness of the serum anion gap. Electrolyte & blood pressure: E & BP. 2006; 4: 44-46.
16. Weiß M, Rücker F, Thieme V, et al. Incidence and Treatment of Arginine Vasopressin Deficiency (Central Diabetes Insipidus) in the Setting of Brain Death and Associations with Renal Function and Hemodynamics in Organ Donors. J Clin Med. 2024; 13: 7073.
17. Greer DM, Shemie SD, Lewis A, et al. Determination of Brain Death/Death by Neurologic Criteria: The World Brain Death Project. JAMA. 2020; 324: 1078-1097.
18. Zosel A, Egelhoff E, Heard K. Severe lactic acidosis after an iatrogenic propylene glycol overdose. Pharmacotherapy. 2010; 30: 219.
19. Perazella MA, Juncos LA. Drug-Induced Osmotic Nephropathy: Add SGLT2-Inhibitors to the List?. Kidney360. 2021; 3: 550-553.
20. Nomani AZ, Nabi Z, Rashid H, et al. Osmotic nephrosis with mannitol: review article. Ren Fail. 2014; 36: 1169-76.
21. Shah RA, Musthaq A, Khardori N. Vancomycin-induced thrombocytopenia in a 60-year-old man: A case report. Journal of Medical Case Reports. 2009; 3: 7290.
22. Custódio G, Schwarz P, Crispim D, et al. Association between vitamin D levels and inflammatory activity in brain death: A prospective study. Transpl Immunol. 2018: 48: 65-69.
23. Mohammadi M, Jahangard-Rafsanjani Z, Sarayani A, et al. Vancomycin-Induced Thrombocytopenia: A Narrative Review. Drug Saf. 2017; 40: 49-59.
24. Mohammadi M, Jahangard-Rafsanjani Z, Sarayani, A, et al. Vancomycin-Induced Thrombocytopenia: A Narrative Review. Drug Saf. 2017; 40: 49-59.