International Journal of Medical Toxicology & Legal Medicine **Volume 28, No. 1, 2025**
e-ISSN: 0974-4614
p-ISSN: 0972-0448
https://doi.org/10.47059/ijmtlm/V28I5/037

Lethal Methanol Poisoning: Correlation Between Autopsy, Toxicology, and Vitreous Biochemistry

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Received: 19.01.2025 Revised: 15.02.2025 Accepted: 18.03.2025

ABSTRACT
Background: Methanol remains a frequent cause of fatal poisoning worldwide, especially in low-resource regions where ingestion of cologne or denatured alcohol is common. Postmortem diagnosis can be challenging because of overlapping features with ethanol intoxication and postmortem alcohol production.
Case presentation: We report a fatal case of methanol intoxication in a 42-year-old man who ingested cologne. Autopsy revealed pulmonary and cerebral edema, visceral congestion, and petechial hemorrhages, consistent with a hypoxic mechanism of death. Toxicological analyses performed by direct-injection GC–MS identified methanol at 1.56–1.63 g/L in blood, 2.16 g/L in vitreous humor, and 6.23 g/L in urine, with formic acid concentrations reaching 0.8 g/L in blood and 5.3 g/L in urine. Ethanol was also detected (0.4–1.8 g/L), suggesting partial but insufficient inhibition of methanol metabolism. Thanatobiochemical analysis of vitreous humor using a rapid co-oximeter showed marked lactic acidosis (lactate = 9.68 mmol/L, under limite of detection, Na^+ = 126.9 mmol/L, pH = 7.12), confirming ante-mortem metabolic collapse.
Conclusion: The correlation between autopsy findings, toxicological results, and vitreous biochemistry provided conclusive evidence of fatal methanol poisoning. This case highlights the forensic importance of integrating thanatobiochemistrywith multi-matrix toxicology to confirm methanol-related deaths and elucidate their metabolic mechanisms.

Keywords : Methanol poisoning, Formic acid, Forensic toxicology, Thanatobiochemistry

INTRODUCTION

Methanol poisoning is a major cause of preventable death, often linked to the ingestion of non-beverage alcohols such as industrial solvents, which account for about 10% of cases in some regions(Yayci et al. 2003). Methanol's toxicity results from its hepatic conversion to formic acid, a potent mitochondrial inhibitor responsible for severe metabolic acidosis and optic damage(Pressman et al. 2020; Shabbir et al. 2023). Because methanol levels alone do not determine outcome, measuring formic acid is essential, lethal cases usually show blood formate above 0.5 g/L('The Relationship of Methanol and Formate Concentrations in Fatalities Where Methanol Is Detected - Jones - 2007 - Journal of Forensic Sciences - Wiley Online Library', n.d.). Postmortem diagnosis can be challenging due to ethanol-like odor and decomposition. Vitreous humor analysis offers a stable and isolated matrix, correlating well with blood levels and allowing both toxicological and thanatobiochemical assessment (glucose, lactate, electrolytes) to confirm ante-mortem metabolic derangement('Simultaneous Measurement of Formic Acid, Methanol and Ethanol in Vitreous and Blood Samples of Postmortem by Headspace GC-FID | Journal of Occupational Medicine and Toxicology | Full Text', n.d.).

This case underscores the forensic value of combining GC-MS quantification of methanol/formate with vitreous biochemistry

Case presentation

A 42-year-old man from Algiers, known to be a heavy drinker, chewing-tobacco user, and occasional psychotropic-drug abuser according to relatives, was found dead at home. Single and with no prior medical history, he had suffered for two months from progressive asthenia and precordial pain that left him bedridden in the weeks before death. On the day of the event he suddenly developed severe dyspnea and loss of consciousness, rapidly progressing to coma and death before medical help could be obtained. There were no witnessed seizures. Because of his history of substance abuse and absence of known disease, the circumstances were considered suspicious for poisoning. Interviews with family members revealed that he had been drinking an alcohol-based fragrance(cologne).

Autopsy findings

The forensic autopsy, conducted six hours after death, revealed marked cyanosis of the face and extremities, indicating severe hypoxemia before death. Old linear scars were present on both forearms, consistent with previous self-harm, but no recent injuries were observed. Rigor and livor mortis were in keeping with the estimated postmortem interval.

Internally, there were multiple subgaleal petechial hemorrhages on the scalp and diffuse signs of systemic hypoxia. The left pleural cavity contained dense fibrous adhesions between the lung and chest wall, and on breaking these adhesions a large left pneumothorax was observed. The left lung was collapsed, likely as an agonal or postmortem event related to old pleural pathology rather than trauma. Both lungs were heavy, congested, and exuded abundant frothy fluid on sectioning, findings consistent with acute pulmonary edema.

All internal organs, including the heart, liver, kidneys, and spleen, were markedly congested, indicating polyvisceral stasis. The brain showed moderate cerebral edema with flattened gyri and narrowed sulci, but no focal hemorrhages or basal ganglia lesions were seen. These findings collectively reflected severe terminal hypoxia typical of acute toxic deaths. Because no lethal anatomic cause was identified, comprehensive toxicological investigations were undertaken to determine the cause of death.

Multiple biological specimens, including vitreous humor, femoraland cardiac blood, and urine, were collected six hours after death for toxicological and biochemical analysis. All samples were properly sealed and stored at −20 °C until analysis.

Toxicological analysis

Volatile alcohols were quantified using a validated GC-MS method(Dali Braham. 2025), and Toxicological screening for drugs(Dali Braham 2024) (e.g., psychotropics, other toxins) byimmunoassayRandox evidence multistate and GC-MS and did not reveal any significant substances aside from methanol, although the decedent's history of psychotropic medication abuse could not be corroborated by drug findings.

Formic acid analysis

Because formic acid is the principal toxic metabolite responsible for the severe metabolic acidosis observed in methanol poisoning, its quantification was performed in postmortem biological fluids using a direct injection GC–MS method**.** a direct analytical after centrifugation was applied.A 1.0 mL aliquot of each sample (peripheral blood, cardiac blood, vitreous humor) was acidified with concentrated sulfuric acid, then mixed with diluted acetonitrile (1:5),as the internal standard. The mixture was injected directly into the gas chromatograph operated insplit mode**.** The full chromatographic condition is summarized in the table 1.

The analytical method was validated in accordance with single-case validation criteria recommended for rare analytes, as outlined by Peters et al(Peters, Drummer, and Musshoff 2007). The validation covered both whole blood and vitreous humor. The analytical performance obtained through this validation is summarized in the table 1.

Table 1 : Validation Parameters of the GC–MS Method for Blood and Vitreous Humor Analysisand Validation Parameters of the GC–MS Method for Blood and Vitreous Humor Analysis

Chromatographic Conditions	**Validation Parameters**		
	Parameter	**Result / Range in blood**	**Result / Range in HV**
- Instrument: Agilent GC-MS - Injection Mode: Direct, split 1:20 - Injector Temperature: 220 °C - Carrier Gas: Helium at 0.4 mL/min - Column: DB-WAX capillary column - Oven Program:	Selectivity	No interfering signals	No interfering signals
	Linearity	0.1–1 g/L, $R^2 = 0.998$	0.1–1 g/L, $R^2 = 0.998$
	Accuracy	Low QC (1 µg/mL): –8.0% bias High QC (10 µg/mL): –7.0% bias	Low QC (1 µg/mL): –5.0% bias High QC (10 µg/mL): –9.0% bias

• 65 °C for 2 min • Ramp 30 °C/min to 100 °C, hold 2 min • Ramp 20 °C/min to 110 °C, hold 2 min • Ramp 20 °C/min to 220 °C, hold 2 min	Precision (RSD%)	4.5% (low), 3.2% (high)	3.8% (low), 4.0% (high)
	LOD	0.05 g/L, S/N = 58	0.03 g/L, S/N = 32
	LLOQ	0.1g/L, S/N = 243	0.1 g/L, S/N = 112

Thanatobiochemistry

In parallel with toxicological analyses, postmortem biochemical tests were conducted on vitreous humor and blood to assess the decedent's metabolic status at the time of death. Vitreous humor is a valuable specimen for evaluating certain chemistries (electrolytes, metabolites) since it is less prone to postmortem changes compared to blood('Postmortem Vitreous Analyses: Overview, Vitreous Procurement and Pretreatment, Performable Postmortem Vitreous Analyses', n.d.). Vitreous humor was analyzed for glucose, lactate, electrolytes (Na^+, K^+, Cl^-, Ca^{2+}), pH, and bicarbonates using aco-oximeterRAPIDPoint®500e. This analytical approach has been validated for postmortem applications, notably by Zilg et al. (2021)(Zilg et al. 2022), who demonstrated the accuracy of blood-gasand co-oximetry analyzers for the biochemical evaluation of vitreous humor and cerebrospinal fluid in forensic settings.The obtained values were interpreted according to postmortem reference intervals described by Madea & Rödig (2006)(Madea and Rödig 2006). and further refined in the forensic biochemistry literature, including Boulagnon et al. (2011)(Boulagnon et al. 2011a)and Thierauf, Musshoff & Madea (2009)(Thierauf, Musshoff, and Madea 2009).

RESULT AND DISCUSSION

Autopsy Finding

The autopsy revealed congested lungs, marked cerebral edema, and petechial hemorrhages, consistent with hypoxic death.Histological sections showed neuronal swelling and necrosisof the lentiform nuclei, findings commonly described in fatal methanol intoxications(Tian et al. 2022; Rahimi et al. 2021).Similar lesions, putaminal hemorrhage, brain edema, andhippocampal herniation—have been reported in both early and delayed deaths from methanolpoisoning, with the severity correlating with survival time(Rahimi et al. 2021).In the present case, thecoexistence of massive pulmonary congestion, visceral cyanosis, and diffuse brain edemaindicatesa terminal metabolic collapse due to tissue hypoxiarather than trauma or othernatural disease.

Toxicological result

Toxicological analyses revealed a consistent multi-matrix profile characteristic of acute methanol poisoning**table 3**. Concentrations of methanol and formic acid measured in peripheral and cardiac blood, vitreous humor, and urine were all within the lethal ranges reported in forensic literature. Methanol levels of 1.5–1.6 g/L in bloodand> 2 g/L in vitreous humor are consistent with fatal outcomes, while urine concentrations exceeding 6 g/L confirm active renal excretion of both the parent compound and its toxic metabolite. Formic acid levels between 0.5 and 0.8 g/L in blood and up to 5 g/L in urine indicate that in-vivo metabolismwas already well advanced before death. These values correspond to those typically observed during the metabolic phase of fatal methanol intoxication.

In this case, the ethanol levels detected (0.4–1.8 g/L in various matrices) fall entirely within this low to moderate range of intoxication, compatible with the euphoric to early excitement stages described by Dubowski(Jones, n.d.). It also has significant diagnostic implications. Ethanol competitively inhibits alcohol dehydrogenase, temporarily slowing methanol oxidation into formaldehyde and formic acid. However, as demonstrated by Ferrari et al. (2003) (Ferrari et al. 2003)**,** low ethanol:methanol ratios (~ 1:4)are insufficient to suppress toxic metabolite accumulation, leading to delayed but ultimately fatal metabolic acidosis. The measured ethanol concentrations therefore suggest a partial protective effect, consistent with an intermediate survival interval of approximately 24–36 hoursbetween ingestion and death.

All concentrations exceeded lethal thresholds (methanol > 0.5 g/L; formic acid > 0.5 g/L).Toxicology screening identified only caffeine and cotinine (nicotine metabolite)

Table 2 : Postmortem Concentrations of Methanol, Formic Acid, and Ethanol in Biological Matrices

Matrix	Methanol (g/L)	Formic acid (g/L)	Ethanol (g/L)
Peripheral blood	1.56	0.51	0.434
Cardiac blood	1.63	0.53	0.432
Vitreous humor	2.16	0.83	0.50
Urine	6.23	5.33	1.82

According to Jones et al. (2007)('The Relationship of Methanol and Formate Concentrations in Fatalities Where Methanol Is Detected - Jones - 2007 - Journal of Forensic Sciences - Wiley Online Library', n.d.), who examined 153 post-mortem cases of methanol exposure, 97 % of confirmed methanol deaths had formate concentrations greater than 0.5 g/L, establishing this value as a reliable threshold for lethality. These findings

are consistent with those of Ferrari et al. (2003) (Ferrari et al. 2003)**,** who proposed that fatal outcomes typically occur once blood methanol exceeds 1 g/L andformate surpasses 0.5 g/L. Together, these studies confirm that concurrent detection of methanol and formate above these limits provides conclusive evidence of fatal methanol intoxication**.**

The relative distribution of analytes across all matrices supports this interpretation. The higher vitreous methanol and formate concentrations compared to blood reflect post-absorptive diffusion typical of delayed deaths. Elevated urinary methanol and formate confirm **r**enal elimination during the agonal phase, verifying ongoing metabolism until shortly before death. These findings align with the toxicokinetic model described by Ferrari (2003) (Ferrari et al. 2003)**.** and expanded by Tian et al. (2022)('Disease-Knowledge-Related Suicide by Cutting Subclavian Catheters in a Woman Undergoing Haemodialysis: Case Report and Review of the Literature - ScienceDirect', n.d.), in which methanol and formate coexist at lethal levels during the metabolic phase leading to death.Moreover, these results are consistent with Rahimi et al. (2021)(Slagter et al. 2021), who observed comparable multi-matrix distributions (blood ≈ 1–2 g/L; vitreous > 2 g/L; urine > 5 g/L) in delayed deaths from methanol outbreaks in SoutheastAsia, where victims survived 24–48 hours after ingestion.Altogether, this integratedtoxicological profile, high methanol and formate in blood and vitreous humor, elevatedurinary concentrations confirming active metabolism, and ethanol at sub-inhibitory levels,provides definitive evidence of acute fatal methanol intoxication during the metabolicphase**,** complicated by formate-induced metabolic acidosisand delayed hypoxic death.

Thanatobiochemistry

Vitreous humor analysis performed at a short postmortem interval of six hours revealed lactate 9.68 mmol/L, glucose indetectable, Na^+ 126.9 mmol/L, Cl^- 117 mmol/L, pH 7.12, low bicarbonate, and O_2 saturation 44.9%was in blood. Considering this brief delay after death, these biochemical alterations mainly reflect ante-mortem metabolic failure rather than postmortem change. The combination of marked hyperlactatemia, near-total glucose depletion, and low pH indicates terminal anaerobic metabolism caused by formate-induced mitochondrial inhibition. The associated hyponatremia and decreased bicarbonate confirm severe metabolic acidosis and circulatory collapse before death. Overall, this biochemical pattern corresponds to the metabolic phase of methanol intoxication and is consistent with formate-mediated cellular hypoxia, as described in similar fatal cases (Madea and Rödig 2006; Thierauf, Musshoff, and Madea 2009; Boulagnon et al. 2011b).

Table 3 : Thanatobiochemical Parameters Measured in Vitreous Humor and TheirInterpretation

Parameter	Value VH	Range of quantification	Reference post-mortem HV(9)	Interpretation
pH	7.12	6.500–7.800	7.0–7.4	borderline acidosis
Lactate	9.68 mmol/L	0.18–30.00	< 4 mmol/L	severe lactic acidosis
Glucose	Indetectable mmol/L		> 1.5 mmol/L	hypoglycemia
Na^+	126.9 mmol/L	100.0–200.0	140–155	hyponatremia
Cl^-	117 mmol/L	65.0–140.0	105–125	normal
Ca^{2+}	1.2 mmol/L	0.20–5.00	> 1.1	low-normal
O_2 Sat	44.9 % (Blood)	1.33–93.32	-	hypoxia

Correlation: autopsy, toxicology, thanatobiochemistry

Autopsyrevealedpulmonary and cerebral edema**,** visceral cyanosis**,** andpetechial hemorrhagesa classichypoxic death phenotype**.** Where examined**,** basal ganglia/putaminal injuryis the neuropathological signature frequently associated with methanol fatalities (Tian et al. 2022; Rahimi et al. 2021).Toxicologydemonstratedmethanolat1.56–1.63 g/L (blood)and2.16 g/L (vitreous) withformic acid0.51–0.53 g/L (blood)and 0.83 g/L (vitreous)**;** urine contained6.23 g/L methanoland 5.33 g/L formate**.** Ethanolwas present **(**0.43–0.50 g/L blood/vitreous; 1.82 g/L urine**),** implyingpartial but insufficient ADH competition**.** Literature thresholds indicate that formate > 0.5 g/L strongly associates with fatality and differentiates genuine ingestion from artifact ('The Relationship of Methanol and Formate Concentrations in Fatalities Where Methanol Is Detected - Jones - 2007 - Journal of Forensic Sciences - Wiley Online Library', n.d.); methanol > 1 g/L withformate > 0.5 g/Ltypifies lethal cases (Ferrari et al. 2003)**.**

Thanatobiochemistry(very high lactate, vanishing glucose, low pH/low HCO_3^-, hyponatremia) proves apre-mortem, high-anion-gap metabolic acidosisand tissue hypoxia—the physiologic bridgethat links thetoxicology(formate present at lethal levels) to themorphology **(**edema, petechiae, basal ganglia injury). Thevitreous > bloodmethanol pattern andvery high urinary methanol/formateare typical of anintermediate survival window (~24–36 h)**:** full systemic absorption, partial metabolism to formate, and ongoing renal elimination at the time of deathexactly the "metabolic phase" described in fatal series (Tian et al. 2022; Rahimi et al. 2021; Ferrari et al. 2003).

Integrated conclusion:The triad of (i)toxicologyshowing co-existent lethal methanol and formate(with sub-protective ethanol**),** (ii)thanatobiochemistrydocumentingtrue ante-mortem lactic acidosis and hypoxia**,** and (iii)autopsydemonstrating edema/petechiae ± basal ganglia damage**,** provides convergent, mutually reinforcing evidence foracute fatal methanol intoxicationwithformate-mediated mitochondrial failureas the mechanism of death.

CONCLUSION

The combined toxicological, thanatobiochemical, and autopsy findings conclusively demonstrate a fatal methanol intoxication characterized by active metabolism before death, leading to severe lactic acidosis and formate-induced mitochondrial failure. The coexistence of lethal methanol and formate levels, biochemical evidence of metabolic collapse, and autopsy features of hypoxic injury (polyvisceral congestion, pulmonary and cerebral edema, petechial hemorrhages) underscores the diagnostic value of integrating methanol/formate quantification with vitreous biochemistry. This multidisciplinary approach highlights the essential role of forensic thanatobiochemistry in confirming ante-mortem metabolic failure and clarifying the mechanism of death in suspected methanol poisoning.

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