INDEPENDENT FORENSIC CASE ANALYSIS

Critique of Cause-of-Death Attribution | Case W21-1383

Decedent	Matthew J. Eller, 22-year-old male	Case Number	W21-1383
Date of Death	November 10, 2021| 2000 hours	Pathologist	Joseph A. Prahlow, M.D.
Jurisdiction	Muskegon County, Michigan	Stated COD	Toxic effects of mitragynine
Body Habitus	73 in / 138 lbs. | BMI 18.2 (underweight)	Manner	Accident

Section 1: Summary of Critical Findings

This analysis identifies three categories of concern in the Eller autopsy: (1) a documented but unexplained electrolyte abnormality that is independently capable of causing death; (2) anthropometric and neuropathological findings that are inconsistent with the ME narrative; and (3) significant toxicology panel gaps that prevent complete pharmacological characterization of the decedent's exposure.

Finding	Value / Observation	Status
Vitreous sodium	118 mmol/L (NMS ref 135-150), profound hyponatremia	CRITICAL
Cerebral edema	Diffuse swelling with effaced sulci; cerebellar tonsil prominence and effaced hippocampal structures per Lieber neuropathology; swelling described as modest, with no herniation	SUPPORTIVE
BMI 18.2 / emesis in hair	Underweight for age/sex; described as 'well-nourished', inconsistent	GAP
Diuretic screen not in panel	Antidepressants, antipsychotics, and stimulants screened and negative; diuretics not within the documented panel scope	GAP
Mitragynine 2100 ng/mL	No validated lethal threshold in peer-reviewed literature; no tolerance or PMR adjustment documented	NOTE

Section 2: The Hyponatremia Finding

2a. Documented Laboratory Value

Vitreous sodium was measured at 118 mmol/L. The reference range printed on the toxicology report by the issuing laboratory, NMS Labs, is 135 to 150 mmol/L. The measured value of 118 mmol/L is 17 below the laboratory's own lower limit and represents profound hyponatremia. Because vitreous humor is anatomically isolated from postmortem redistribution to a substantially greater degree than blood, this value is considered reliable for estimating antemortem status.

The remaining vitreous chemistries argue against a global concentration artifact as the explanation for the electrolyte picture. A dehydration or hemoconcentration pattern would be expected to raise vitreous urea nitrogen. The urea nitrogen of 17 mg/dL and creatinine of 1.05 mg/dL are both within the laboratory's reference ranges, indicating no azotemia and no dehydration pattern. The sodium is

therefore specifically low against an otherwise normal panel, which supports reading the value as genuine antemortem hyponatremia rather than one feature of a globally deranged postmortem specimen.

2b. The Value Is Not a Collection Artifact

The most common technical objection to a low postmortem sodium value is contamination from the collection tube. NMS Labs raises this caution on its own report. The reference comment for sodium states that quantitative results are affected if the analysis is performed on a gray top tube, because gray top tubes contain sodium fluoride.

The specimen table in the same report identifies the vitreous fluid, specimen 003, as collected in a red top tube. Red top tubes contain no additive. The two femoral blood specimens were drawn in gray top tubes, but the vitreous was not. The caution the laboratory raises therefore does not apply to this specimen. The value of 118 mmol/L was measured in the matrix least affected by postmortem redistribution, collected in the correct additive-free tube, and reported against the laboratory's own reference range. It is not explicable as a collection artifact on anything in this record.

2c. Clinical Significance

Antemortem serum sodium at or below 120 mEq/L is a recognized medical emergency. At this level, osmotic gradients drive water into brain cells, producing cerebral edema. This mechanism is well-characterized in the medical literature and is independent of any co-occurring substance exposure. The clinical presentation of acute severe hyponatremia includes:

- Headache, nausea, vomiting
- Seizure-like activity and loss of consciousness
- Sudden cardiovascular collapse

These are precisely the events described in the circumstances section of this autopsy: the decedent 'stood up after playing a game of cards and then collapsed and demonstrated seizure-like activity.'

2d. Neuropathological Evidence Is Consistent with Hyponatremic Encephalopathy

The following autopsy findings form a coherent, internally consistent picture of hyponatremic cerebral edema as the proximate cause of death:

Autopsy Finding	Significance for Hyponatremic Encephalopathy
Diffuse cerebral swelling (gross)	Explicitly described; sulci effaced; consistent with osmotic water shift into brain cells
Cerebellar tonsil prominence	Documented by Lieber neuropathology; consistent with downward pressure from supratentorial swelling
Effaced hippocampal structures	Documented in Lieber report; a marker of significant brain swelling, not structural pathology
Seizure-like activity preceding collapse	Classic presentation of hyponatremic encephalopathy; sodium of 118 is squarely in the seizure threshold range

Autopsy Finding	Significance for Hyponatremic Encephalopathy
Emesis in hair at autopsy	Vomiting is both a symptom and a potential driver of the hyponatremia; may indicate acute electrolyte derangement
No anatomic COD assigned	ME's own finding, 'Not found: anatomic explanation for death', makes a metabolic mechanism more plausible

2e. The ME's Conclusion Does Not Address This Evidence Chain

Dr. Prahlow's final conclusion attributes death to 'toxic effects of mitragynine' and lists as a co-finding 'not found: anatomic explanation for death.' The vitreous sodium of 118 mEq/L, though documented in the report, receives no narrative analysis, no differential diagnosis, and no explanation. The mechanistic link between mitragynine exposure and the documented cerebral edema is never established in the report.

This represents a significant analytical gap under standard forensic pathology methodology. When a documented laboratory value is independently capable of causing the observed neuropathological findings and the clinical presentation at the time of death, that value must be addressed in the cause-of-death narrative, either attributed, excluded, or designated as a contributing condition.

Section 3: BMI and Nutritional Status

3a. Anthropometric Data

The decedent's recorded height was 73 inches (6'1'') and weight was 138 pounds. Calculated BMI is 18.2, which falls below the threshold of 18.5 used to define underweight status in adults. For a 22-year-old male of this stature, a BMI of 18.2 is mildly underweight, sitting just below the 18.5 threshold.

3b. Internal Inconsistency in the Report

The external examination describes the decedent as 'well-developed, well-nourished.' This descriptor is a standard phrase in autopsy reports but is in tension with a BMI of 18.2 and the presence of emesis in the scalp hair. A clinically accurate characterization of an underweight individual with evidence of recent vomiting would typically prompt notation of nutritional status and consideration of underlying causes.

3c. Clinical Relevance to Hyponatremia

Chronic undernutrition reduces electrolyte reserve and increases vulnerability to electrolyte derangement under physiological stress. Individuals who are chronically underweight may have lower baseline sodium, potassium, and magnesium stores. In this context, an acute physiological stressor such as vomiting, exertion, or excessive fluid intake could precipitate severe hyponatremia more rapidly than in a nutritionally replete individual.

The report does not include any inquiry into the decedent's nutritional history, recent oral intake, or any condition that might explain the low body weight. In the presence of a fatal electrolyte abnormality, this omission limits the completeness of the cause-of-death analysis.

Section 4: Toxicology Panel Gaps

4a. Pharmacological Causes of Hyponatremia: Screened Versus Not in Scope

Several classes of medication are associated with hyponatremia and SIADH, and the toxicology report addresses most of them. The expanded postmortem screen, NMS analysis code 8042B, included a broad LC/TOF-MS panel whose documented scope covers antidepressants, antipsychotic agents, amphetamines, and other CNS stimulants, among other classes. MDMA falls within the amphetamine and stimulant classes, and the urine add-on separately screened amphetamines. None of these returned a positive result. On the face of the report, therefore, antidepressants, antipsychotics, and stimulants including MDMA were within scope and were not detected.

One qualification applies to that conclusion. NMS states that the LC/TOF-MS screen is concentration-dependent and does not include every analyte within each listed class. A negative result at the class level is therefore strong evidence against those agents, but it is not an absolute exclusion of every specific drug within the class.

The genuine gap concerns diuretics. Thiazide diuretics are among the most common pharmacological causes of hyponatremia, yet no diuretic, and no diuretic class, appears anywhere in the documented scope of any panel on this report. There is no reporting limit and no result for this class. Unlike the agents above, diuretics were neither detected nor excluded, because they do not appear to have been tested. Antibiotics, another recognized cause, are likewise absent from the documented scope.

The significance of this is narrower than a claim that the hyponatremia was never investigated pharmacologically, and it is stronger for being precise. The common medication causes that were screened came back negative, which makes the sodium of 118 more conspicuous rather than less, because a leading category of explanation has been removed. What remains uninvestigated is a specific and testable class, diuretics, together with the non-pharmacological causes of severe hyponatremia such as water intoxication and exertional or dilutional mechanisms, none of which were addressed in the report.

4b. Mitragynine Concentration in Context

The femoral blood mitragynine concentration of 2100 ng/mL is at the higher end of concentrations reported in postmortem cases attributed to kratom in the forensic literature. However, several contextual factors limit the interpretive weight of this figure:

- No validated lethal concentration threshold for mitragynine has been established in peer-reviewed literature. The most frequently cited postmortem case series (Mata & Andera, Forensic Chemistry, 2019) reported central blood concentrations ranging from 10 to 4,310 ng/mL across 20 cases, with a median of 123 ng/mL. Three of those cases listed mitragynine as the sole cause of death, at central blood concentrations of 1,250 to 4,310 ng/mL. Eller's femoral value of 2,100 ng/mL falls within that range. However, the authors themselves noted that in no case was mitragynine the only drug detected, including the three sole-attribution cases. This observation, drawn from the paper most often cited in support of mitragynine lethality, reinforces that polypharmacy and incomplete toxicological screening remain material interpretive concerns even when mitragynine concentrations are elevated.
- Femoral blood is preferred over central blood for postmortem redistribution (PMR) concerns, but PMR can still occur, particularly in cachectic or nutritionally compromised individuals.

- No information regarding tolerance is documented. Regular kratom users may accumulate higher circulating concentrations without proportionate pharmacological effect due to tolerance.
- No product analysis or stomach content alkaloid screening is reported. The 250 mL of gastric contents noted at autopsy was not analyzed for alkaloid composition.

Section 5: Additional Anatomic Findings Not Developed in the Report

The autopsy documents two cardiac findings that were noted on internal examination but were not analyzed, excluded, or reconciled with the final finding that no anatomic explanation for death was present.

First, the report describes tunneling of the proximal left anterior descending coronary artery. Tunneling, also called myocardial bridging, is a structural coronary variant in which a segment of the artery runs through the heart muscle rather than over its surface. It is a recognized, though debated, substrate for transient myocardial ischemia, arrhythmia, and sudden collapse in young people, particularly during or immediately after exertion. The decedent collapsed on standing. The report notes the finding but does not develop it.

Second, the report notes slight biventricular dilatation, right greater than left. Standing alone this is a nonspecific observation, but in combination with the coronary variant it is the kind of finding that ordinarily prompts either further cardiac workup or an explicit statement of why it was set aside.

Neither finding is offered here as the cause of death. The point is one of analytical completeness. The final report states that no anatomic explanation for death was found, while the body of the same report documents structural cardiac findings that were neither developed nor excluded. As with the vitreous sodium, a documented finding capable of bearing on the mechanism of a sudden collapse was left unaddressed in the cause-of-death analysis.

Section 6: The Seizure Claim

Public characterizations of this case have described the death as caused by a ‘seizure from kratom.’ The autopsy record does not support this framing. The following analysis applies:

Claim	What the Record Shows
‘Kratom caused the seizure’	The report documents seizure-like activity as a witnessed event. The report does not establish a mechanism by which mitragynine caused seizure activity. A sodium of 118 is low enough to cause seizures on its own. That finding was documented in the autopsy and never explained.
‘Kratom was the cause of death’	The autopsy found brain swelling. Severely low sodium is a known cause of brain swelling. The report never connected those two findings.
‘No other explanation’	The ME’s own co-finding states ‘not found: anatomic explanation for death.’ A sodium of 118 mEq/L in vitreous is a metabolic, not anatomic, explanation, and one that was neither addressed nor excluded in the report.

Section 7: Summary of Analytical Gaps Requiring Further Inquiry

The following questions are raised by the data in the autopsy record and are not answered within it:

1. What caused the vitreous sodium of 118 mEq/L? This value is independently life-threatening and no etiology is identified in the report.
2. Diuretics, a leading pharmacological cause of hyponatremia, do not appear within the documented scope of any panel on the toxicology report. Were they tested?
3. What was the decedent's dietary and fluid intake history in the 24 to 48 hours prior to death? Vomiting, excessive water intake, or fluid restriction can precipitate or worsen hyponatremia.
4. Nutritional status**:** Although a BMI of 18.2 is only mildly underweight and not inherently abnormal, nutritional status can influence electrolyte reserve and susceptibility to acute hyponatremia. In the context of a documented sodium of 118 mEq/L, noting recent dietary and fluid intake would have been clinically informative.
5. Was the gastric contents sample (250 mL documented) submitted for alkaloid analysis? Product characterization from stomach contents can inform both dose estimation and product type.
6. What mechanism connects mitragynine to the documented cerebral edema? No direct mitragynine-to-edema pathway is established in the report or in the peer-reviewed literature.
