Case Reports in Clinical Medicine, 2026, 15(3), 75-79
https://www.scirp.org/journal/crcm
ISSN Online: 2325-7083
ISSN Print: 2325-7075

Arsenic Encephalopathy Associated with Excessive Seafood Consumption: A Case Report

Sandra Johnson

School of Medicine, Johns Hopkins Hospital, Baltimore, MD, USA
Email: sjohn334@jh.edu

How to cite this paper: Johnson, S. (2026) Arsenic Encephalopathy Associated with Excessive Seafood Consumption: A Case Report. *Case Reports in Clinical Medicine*, **15**, 75-79.
https://doi.org/10.4236/crcm.2026.153010

Received: January 27, 2026
Accepted: February 25, 2026
Published: February 28, 2026

Abstract

Arsenic toxicity is an uncommon and frequently underrecognized cause of subacute encephalopathy. Diagnosis requires careful integration of exposure history, clinical findings, and laboratory testing, particularly because dietary sources such as seafood can elevate urinary arsenic levels without causing true toxicity. We report the case of a 39-year-old woman presenting with progressive cognitive impairment and functional decline who was found to have markedly elevated urinary arsenic levels in the setting of excessive seaweed consumption. Extensive evaluation for infectious, autoimmune, metabolic, and structural causes of altered mental status was unrevealing. Her cognitive function steadily improved with supportive care and elimination of dietary exposure, without chelation therapy. This case highlights the diagnostic challenges in interpreting elevated urinary arsenic levels, the contribution of seafood to total arsenic measurements, and the critical role of arsenic speciation in clinical interpretation and management.

Keywords

Arsenic Toxicity, Encephalopathy, Seafood, Organic Arsenic, Inorganic Arsenic, Altered Mental Status

1. Introduction

Arsenic is a naturally occurring metalloid that exists in both organic and inorganic forms. Inorganic arsenic is responsible for most of the well-described toxic effects, including gastrointestinal distress, neuropathy, dermatologic manifestations, hematologic abnormalities, and multisystem organ dysfunction, whereas organic arsenic compounds—commonly found in seafood—are generally considered non-toxic and rapidly excreted in urine [1]-[3].

Diagnosis of arsenic toxicity can be challenging for hospitalists, as clinical manifestations are variable and laboratory interpretation may be confounded by dietary exposure. Urinary arsenic testing is the primary biomarker for both acute and chronic exposure; however, total urinary arsenic levels alone cannot reliably distinguish toxic inorganic exposure from benign organic forms [1] [4]. Arsenic speciation is therefore essential to guide management and prevent unnecessary interventions. We present a case of subacute encephalopathy associated with elevated urinary arsenic levels attributable to excessive seafood consumption, highlighting important diagnostic considerations.

2. Case Presentation

A 39-year-old woman with a history of irritable bowel syndrome, gluten and corn intolerance, and no known neurologic disease presented with progressive confusion, dizziness, fatigue, and functional decline over approximately one month. Collateral history from family revealed subtle cognitive changes beginning several months prior, with worsening difficulty managing activities of daily living, impaired reading comprehension, getting lost while driving, and inability to work. She also reported nausea, lightheadedness, and unintentional weight loss.

Approximately one month prior to presentation, she had initiated a glucagon-like peptide-1 receptor agonist for weight loss, which she discontinued after developing fatigue and dizziness. Despite discontinuation, her cognitive symptoms progressed. She denied suicidal ideation, hallucinations, or mood symptoms, though she exhibited marked apathy and limited insight into her condition.

On admission, Montreal Cognitive Assessment (MoCA) score was 18/30, with deficits in visuospatial function, attention, and recall; she was unable to draw a clock. Neurologic examination demonstrated impaired proprioception, coordination difficulties, and a positive Romberg sign. EEG showed diffuse delta and theta slowing with generalized rhythmic delta activity, consistent with a toxic or metabolic encephalopathy. MRI brain with and without contrast was unremarkable.

An extensive evaluation for reversible causes of encephalopathy, including: metabolic testing (thyroid function, ammonia, inflammatory markers), nutritional studies (vitamins A, B1, B2, B6, B12, folate, copper, ceruloplasmin), infectious testing (HIV, syphilis, Lyme disease), toxicology screening (urine drug screen, extended toxicology panel, ethanol, acetaminophen, salicylate levels, and heavy metals), and autoimmune/paraneoplastic evaluation (ANA, GAD65, NMDA receptor antibodies, and serum and cerebrospinal fluid autoimmune encephalopathy panels)—was unrevealing. Neuroimaging (CT head, MRI brain), cerebrospinal fluid analysis, and CT/PET imaging showed no structural, infectious, inflammatory, or neoplastic cause.

Serum arsenic was undetectable, consistent with the rapid clearance of arsenic from the bloodstream [1] [5]. However, urinary arsenic was markedly elevated at 144 μg/L. Further dietary history revealed near-daily consumption of seaweed snacks (approximately 2 - 3 servings daily), with frequent seafood intake (approximately 4 - 5 meals per week), raising concern for substantial dietary arsenic exposure. She also reported use of multiple over-the-counter supplements at the time of admission, including hyoscyamine (0.125 mg as needed), vitamin K, omega-3 fatty acid formulations (pro-omega, EFA/DHA), a multivitamin preparation without iron, elderberry supplements, digestive enzyme preparations, fruit-based nutritional powders, and a urinary health supplement containing pumpkin seed extract and D-mannose, representing additional potential sources of trace element exposure. Poison Control was consulted and advised against chelation therapy, citing the absence of clinical features consistent with inorganic arsenic poisoning, including gastrointestinal distress, autonomic instability, dermatologic changes, anemia, or transaminitis [2] [6]. Arsenic speciation was recommended to distinguish organic from inorganic forms, though management was unlikely to change. The patient was advised to strictly avoid seafood, seaweed, and supplements.

Over the course of hospitalization, her cognitive function steadily improved. MoCA scores increased from 18/30 on admission to 23/30 by hospital day 8, 26/30 the following week, and 29/30 at discharge. Her clinical improvement correlated with dietary avoidance and declining urinary arsenic levels. At short-term follow-up, urinary arsenic levels decreased to 55 μg/L (7/2) and were undetectable by 7/15, with sustained cognitive and functional recovery. No formal clinical or laboratory follow-up was obtained after hospital discharge, representing a limitation of this report.

Written informed consent for publication of this case report was obtained from the patient.

3. Discussion

This case highlights several important diagnostic principles relevant to hospital-based practice.

First, arsenic toxicity may present with predominantly neurocognitive symptoms and minimal systemic findings, particularly in cases of low-level or dietary exposure. Although classic inorganic arsenic poisoning is associated with gastrointestinal distress, dermatologic changes, peripheral neuropathy, and hematologic abnormalities, isolated encephalopathy and cognitive dysfunction have been reported and may lead to misattribution to psychiatric or functional disorders [2]-[4] [7]. Prior reports of arsenic-associated neurocognitive presentations most commonly describe peripheral neuropathy or multisystem toxicity, whereas isolated or predominant cognitive impairment without systemic manifestations appears less frequently reported, making diagnostic interpretation particularly challenging [1]-[4].

Second, interpretation of arsenic testing requires integration of clinical context and exposure history. Urinary arsenic measurement is the gold standard for assessing arsenic exposure and reflects recent exposure over the preceding 1 - 3 days

[1] [8]. Spot urine testing is practical and correlates well with 24-hour collections, though creatinine adjustment may be useful when comparing serial measurements [1]. Importantly, total urinary arsenic levels may be significantly elevated following seafood consumption, as seafood contains organic arsenic compounds that are non-toxic but rapidly excreted [1] [8] [9].

Third, arsenic speciation is critical for distinguishing toxic inorganic arsenic and its methylated metabolites from benign organic forms such as arsenobetaine [1] [4]. This distinction is particularly important because some seafood-derived compounds contribute to elevated urinary dimethylarsinic acid (DMA), which may not reflect inorganic arsenic metabolism. Some experts suggest that inorganic arsenic plus monomethylarsonic acid (MMA) may be more reliable indicators of toxic exposure than DMA alone [8] [9]. When speciation is unavailable or inconclusive, repeat testing after 1 - 2 weeks of seafood abstinence can help clarify the significance of elevated levels [1].

Other biomarkers have limited utility in acute clinical decision-making. Blood arsenic testing is not recommended due to rapid clearance and poor correlation with chronic exposure [1] [5]. Hair and nail analysis may identify longer-term exposure but are susceptible to external contamination and should be interpreted cautiously [1] [6] [10] [11].

In this patient, elevated urinary arsenic levels in the setting of excessive seafood consumption, absence of classic systemic toxicity, and steady clinical improvement with dietary avoidance strongly supported organic arsenic exposure rather than toxic inorganic arsenic poisoning. Consistent with current guidelines, chelation therapy was not indicated [1] [2] [10] [11]. Importantly, a causal relationship between organic arsenic exposure and the patient's encephalopathy cannot be definitively established; rather, this case primarily illustrates the challenges of interpreting elevated urinary arsenic levels and distinguishing clinically significant toxic exposure from benign dietary sources. Additionally, the absence of post-discharge clinical and laboratory follow-up limits assessment of the durability of improvement and the long-term relationship between arsenic levels and neurologic recovery.

4. Conclusion

Arsenic exposure should be considered in patients presenting with unexplained subacute encephalopathy, even in the absence of classic features of toxicity. Elevated urinary arsenic levels must be interpreted in the context of dietary history, particularly seafood and seaweed consumption. Arsenic speciation plays a crucial role in distinguishing toxic inorganic exposure from benign organic forms and in guiding appropriate management. Increased awareness of these principles may help hospitalists avoid misdiagnosis and unnecessary treatment.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

References

[1] Baker, B.A., Cassano, V.A. and Murray, C. (2018) Arsenic Exposure, Assessment, Toxicity, Diagnosis, and Management. *Journal of Occupational & Environmental Medicine*, **60**, e634-e639. https://doi.org/10.1097/jom.0000000000001485

[2] Ratnaike, R.N. (2003) Acute and Chronic Arsenic Toxicity. *Postgraduate Medical Journal*, **79**, 391-396. https://doi.org/10.1136/pmj.79.933.391

[3] Rahaman, M.S., Rahman, M.M., Mise, N., Sikder, M.T., Ichihara, G., Uddin, M.K., *et al*. (2021) Environmental Arsenic Exposure and Its Contribution to Human Diseases, Toxicity Mechanism and Management. *Environmental Pollution*, **289**, Article 117940. https://doi.org/10.1016/j.envpol.2021.117940

[4] Mochizuki, H. (2019) Arsenic Neurotoxicity in Humans. *International Journal of Molecular Sciences*, **20**, Article 3418. https://doi.org/10.3390/ijms20143418

[5] Hughes, M.F. (2006) Biomarkers of Exposure: A Case Study with Inorganic Arsenic. *Environmental Health Perspectives*, **114**, 1790-1796. https://doi.org/10.1289/ehp.9058

[6] Hall, A.H. (2002) Chronic Arsenic Poisoning. *Toxicology Letters*, **128**, 69-72. https://doi.org/10.1016/s0378-4274(01)00534-3

[7] Martínez-Castillo, M., García-Montalvo, E.A., Arellano-Mendoza, M.G., Sánchez-Peña, L.D.C., Soria Jasso, L.E., Izquierdo-Vega, J.A., *et al*. (2021) Arsenic Exposure and Non-Carcinogenic Health Effects. *Human & Experimental Toxicology*, **40**, S826-S850. https://doi.org/10.1177/09603271211045955

[8] Marchiset-Ferlay, N., Savanovitch, C. and Sauvant-Rochat, M. (2012) What Is the Best Biomarker to Assess Arsenic Exposure via Drinking Water? *Environment International*, **39**, 150-171. https://doi.org/10.1016/j.envint.2011.07.015

[9] Doerge, D.R., Twaddle, N.C., Churchwell, M.I. and Beland, F.A. (2020) Reduction By, Ligand Exchange Among, and Covalent Binding to Glutathione and Cellular Thiols Link Metabolism and Disposition of Dietary Arsenic Species with Toxicity. *Environment International*, **144**, Article 106086. https://doi.org/10.1016/j.envint.2020.106086

[10] Nurchi, V.M., Buha Djordjevic, A., Crisponi, G., Alexander, J., Bjørklund, G. and Aaseth, J. (2020) Arsenic Toxicity: Molecular Targets and Therapeutic Agents. *Biomolecules*, **10**, Article 235. https://doi.org/10.3390/biom10020235

[11] Frumkin, H. and Thun, M.J. (2001) Arsenic. *CA*: *A Cancer Journal for Clinicians*, **51**, 254-262. https://doi.org/10.3322/canjclin.51.4.254