CASE REPORT

Fatal Acute Arsenic Toxicity Following Consumption Of Ayurvedic Medication Presenting As Rapidly Progressive Encephalopathy With Multiorgan Dysfunction: A Case Report

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ABSTRACT

Background: Traditional Ayurvedic formulations are widely used and often perceived as safe; however, contamination or intentional inclusion of heavy metals such as arsenic poses significant health risks. Acute arsenic toxicity can lead to severe neurological impairment, multi-organ dysfunction, and death. Reports of acute encephalopathy due to such exposure remain rare but clinically important.

Case Presentation: A young female presented in a comatose state following ingestion of an Ayurvedic formulation prescribed for abdominal pain attributed to cholelithiasis. She had consumed approximately 250 mL of a diluted powder solution in divided doses, followed by Ayurvedic oil. Initial symptoms included diarrhea, progressing rapidly to headache, vertigo, and altered sensorium. On examination, she was hypotensive (BP 100/70 mmHg), tachycardic (HR 150 bpm), and unresponsive with a Glasgow Coma Scale of E1V1M1. Neuroimaging revealed diffuse cerebral edema with loss of grey-white matter differentiation and features of subarachnoid hemorrhage. Toxicological analysis of gastric lavage confirmed the presence of arsenic and prednisolone. Despite aggressive management including gastric lavage, mechanical ventilation, and hemodialysis, her condition deteriorated with development of multi-organ dysfunction, including acute kidney injury and metabolic derangements. This case highlights a rare and fatal manifestation of acute arsenic-induced encephalopathy associated with Ayurvedic medication use. It underscores the need for early suspicion of heavy metal toxicity in unexplained neurological deterioration and calls for stringent regulation and safety monitoring of traditional medicinal products.

Keywords: Arsenic toxicity; Ayurvedic medicine; Encephalopathy; Cerebral edema; Heavy metal poisoning; Neurotoxicity; Toxicology; Multi-organ dysfunction

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INTRODUCTION

Traditional systems of medicine, particularly Ayurveda, are widely practiced across India and other developing countries, often coexisting with modern medical care. These formulations are commonly perceived as safe due to their natural origin and long-standing cultural acceptance [1]. However, growing evidence has raised concerns regarding the safety of certain Ayurvedic preparations, particularly those containing heavy metals such as arsenic, lead, and mercury [2]. These metals may be intentionally incorporated as part of traditional herbo-mineral formulations or may contaminate products due to inadequate quality control, improper processing, or environmental exposure during manufacturing. The absence of stringent regulatory oversight and standardized testing further increases the risk of toxicity [3,4].

Arsenic is a well-known environmental and industrial toxin with significant systemic effects. While chronic arsenic exposure, commonly through contaminated groundwater, has been extensively studied, acute arsenic poisoning is relatively rare and often presents a diagnostic challenge due to its nonspecific clinical manifestations [5]. Acute toxicity primarily affects rapidly dividing tissues and high-energy organs by inhibiting cellular respiration through interference with mitochondrial oxidative phosphorylation [6]. Clinically, it typically presents with early gastrointestinal symptoms such as nausea, vomiting, and profuse diarrhea, followed by cardiovascular instability, metabolic acidosis, and neurological dysfunction. In severe cases, rapid progression to encephalopathy, coma, and multiorgan failure may occur, often with a high mortality rate [7,8]. Neurological manifestations of arsenic poisoning,

although less commonly emphasized in acute settings, can be profound. Diffuse cerebral edema and hypoxic brain injury may develop secondary to systemic hypotension, metabolic derangements, and direct neurotoxic effects [9,10]. These complications significantly worsen prognosis and may culminate in irreversible brain damage or brain death. Additionally, acute arsenic toxicity can lead to hematological abnormalities, hepatic injury, renal failure, and immunosuppression, further complicating the clinical course [11].

The increasing use of over-the-counter herbal and Ayurvedic medications, often without proper labeling or disclosure of contents, poses a significant public health concern. Patients frequently seek alternative therapies for common conditions, sometimes delaying or avoiding evidence-based treatment [12,13]. This can result in exposure to potentially toxic compounds, as well as delayed recognition of adverse effects. Several case reports and toxicological studies have highlighted the presence of heavy metals in such preparations, underscoring the urgent need for stricter regulation, quality assurance, and public awareness [14,15].

We report a rare and fatal case of acute arsenic poisoning in a young female following ingestion of an Ayurvedic preparation prescribed for cholelithiasis. The patient developed rapidly progressive encephalopathy, diffuse cerebral edema, and multiorgan dysfunction, ultimately leading to death despite intensive supportive care. This case highlights the life-threatening consequences of unregulated traditional medications and emphasizes the importance of early clinical suspicion, prompt toxicological evaluation, and stringent regulatory control to prevent similar outcomes.

CASE PRESENTATION

Patient Information and Background

A 23-year-old female with no known comorbidities presented in an unconscious state following ingestion of an Ayurvedic preparation prescribed for right upper abdominal pain. Prior evaluation had revealed cholelithiasis on ultrasonography, with a gallbladder calculus measuring approximately 6.0 × 4.0 mm, and mild ascites. Surgical intervention had been advised; however, the patient opted for alternative therapy and consumed an Ayurvedic formulation consisting of a powdered preparation diluted in approximately 250 mL of water in multiple doses, followed by ingestion of an Ayurvedic oil preparation reportedly containing olive oil and magnesium sulfate.

History of Present Illness

Soon after ingestion, the patient developed acute gastrointestinal symptoms including 3–4 episodes of profuse watery diarrhea and one episode of vomiting. This was followed by headache, giddiness, and rapidly progressive drowsiness, ultimately culminating in loss of consciousness. She was brought to the emergency department in an unresponsive state. There was no history of seizures, trauma, or prior neurological illness.

Clinical Examination

"On arrival, the patient was deeply comatose with a Glasgow Coma Scale score of E1V1M1. She was afebrile, tachycardic with a pulse rate of 150 beats per minute, hypotensive with a blood pressure of 100/60 mmHg, and tachypneic with a respiratory rate of 26 cycles per minute. Oxygen saturation was 93% on room air, and random blood sugar was 174 mg/dL.

Neurological examination revealed sluggishly reactive pupils, generalized hypotonia, absence of motor response, and absent deep tendon reflexes. Plantar reflexes were mute. Cardiovascular examination showed tachycardia without murmurs, respiratory examination revealed bilateral conducted sounds, and abdominal examination was soft with sluggish bowel sounds".

Investigations

Neuroimaging with CT brain revealed diffuse cerebral edema characterized by effacement of the ventricular system, sylvian fissures, and cortical sulci. Subsequent imaging demonstrated interval worsening of cerebral edema along with subarachnoid hemorrhage involving bilateral fronto-parieto-temporo-occipital regions and perimesencephalic cisterns, consistent with severe global hypoxic brain injury.

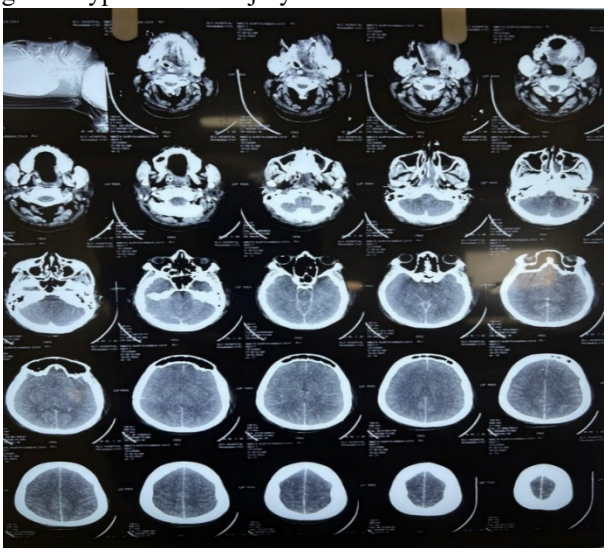

FIG Non-contrast CT Brain Showing Diffuse Cerebral Edema with Loss of Gray–White Matter Differentiation Suggestive of Global Hypoxic Injury

Electroencephalography showed low-voltage indeterminate activity with poorly defined beta activity (<5 μV) and was suggestive of severe bihemispheric

dysfunction.

Toxicological analysis of gastric lavage confirmed the presence of arsenic compounds on color testing and prednisolone on thin-layer chromatography.

Laboratory investigations revealed marked leukocytosis with white blood cell counts ranging from 15.8 to 44.1 $\times 10^3/\mu L$ with neutrophilic predominance up to 96% and toxic granules present. Hemoglobin levels ranged from 5.5 to 11.8 g/dL, indicating normocytic normochromic anemia. Platelet counts were markedly reduced, ranging from 9,000 to 57,000/µL, suggestive of thrombocytopenia.

Renal function tests showed significantly elevated urea levels ranging from 194 to 238 mg/dL and creatinine levels between 3.55 and 4.22 mg/dL, indicating acute kidney injury. Electrolyte abnormalities included sodium levels up to 161 mEq/L, potassium levels ranging from 3.83 to 6.46 mEq/L, and bicarbonate levels between 10.9 and 15.2 mEq/L, consistent with metabolic acidosis.

Liver function tests revealed elevated AST (up to 200 U/L) and ALT (up to 158 U/L), with total bilirubin levels reaching 4.38 mg/dL and direct bilirubin up to 3.75 mg/dL. Serum albumin levels were low, ranging from 2.3 to 2.5 g/dL. Inflammatory markers were markedly elevated, with C-reactive protein of 61.5 mg/L and procalcitonin of 2.31 ng/mL.

Coagulation profile showed prolonged activated partial thromboplastin time (APTT up to 47.3 seconds) and INR up to 1.52. Peripheral smear findings were consistent with normocytic normochromic anemia with neutrophilic leukocytosis and thrombocytopenia.

Screening for infectious etiologies including dengue NS1 antigen and IgM/IgG antibodies was negative, and leptospira IgM was also negative.

Management and Clinical Course

The patient was immediately intubated and mechanically ventilated. Gastric lavage was performed, and she was managed with intravenous fluids, broad-spectrum antibiotics including colistin, augmentin, clindamycin, and doxycycline, along with corticosteroids, proton pump inhibitors, and supportive care.

Due to worsening renal function and metabolic acidosis, the patient underwent hemodialysis followed by sustained low-efficiency dialysis. Persistent hypotension required multiple inotropes including adrenaline, noradrenaline, dopamine, and vasopressin.

She developed ischemic hepatitis secondary to prolonged hypotension and acute kidney injury. Progressive neurological deterioration was noted with non-reactive pupils and absent brainstem reflexes, consistent with brain death.

During the hospital course, she developed ventilator-associated pneumonia, severe sepsis, and disseminated intravascular coagulopathy. She required multiple transfusions including packed red blood cells, platelets, and fresh frozen plasma.

Outcome

Despite aggressive intensive care management, the patient progressed to refractory septic shock with multiorgan dysfunction syndrome. She subsequently developed cardiac arrest and could not be revived despite resuscitative efforts. The final diagnosis was fatal acute arsenic poisoning following ingestion of an Ayurvedic preparation, complicated by severe encephalopathy, diffuse cerebral edema, hypoxic brain injury, and multiorgan failure.

DISCUSSION

The present case highlights a rare yet catastrophic manifestation of acute arsenic toxicity following ingestion of an Ayurvedic formulation, culminating in severe encephalopathy, multi-organ dysfunction, and death. Similar concerns regarding heavy metal contamination in traditional medicines have been well documented in literature. Pandey et al. (2013) emphasized that although Ayurveda is widely regarded as safe, certain formulations may contain heavy metals either intentionally or as contaminants, raising significant safety concerns [1]. Supporting this, Mikulski et al. (2018) demonstrated through a public health investigation that Ayurvedic preparations can contain toxic levels of metals such as arsenic and lead, leading to clinically significant poisoning [2]. This aligns with the present case where toxicological analysis confirmed arsenic exposure.

Heavy metal contamination in herbal medicines is not restricted to a single region. Luo et al. (2021) conducted a comprehensive risk assessment and reported widespread contamination of herbal products with arsenic and other toxic metals, reinforcing the global relevance of this issue [3]. Similarly, Kim et al. (2014) described adverse clinical outcomes associated with metal-contaminated traditional medicines, including neurological and systemic toxicity, which parallels the rapid neurological deterioration observed in the present patient [4].

The pathophysiology of arsenic toxicity explains the severity of clinical manifestations. Ganie et al. (2023) described arsenic-induced oxidative stress, mitochondrial dysfunction, and cellular apoptosis as key mechanisms leading to multi-organ injury [5]. These mechanisms are consistent with the diffuse cerebral edema and organ dysfunction observed in this case. Additionally, Lee and Hüttemann (2014) highlighted that disruption of oxidative phosphorylation plays a central role in acute inflammatory states and sepsis, which may explain the progression to septic complications and systemic deterioration [6].

Neurological involvement is a critical feature of arsenic

toxicity. Tyler and Allan (2014) and Mochizuki (2019) both reported that arsenic exposure can result in profound neurotoxicity, including encephalopathy, cognitive dysfunction, and structural brain injury [9,10]. The diffuse cerebral edema and loss of grey-white matter differentiation seen in this case are consistent with these findings. Furthermore, Kumar et al. (2017) discussed the interplay between metabolic derangements and cerebral edema, which may have contributed to the rapid neurological decline [8].

Multi-organ involvement, particularly hepatic and renal dysfunction, is another hallmark of arsenic poisoning. Fontana (2008) described acute liver failure as a consequence of toxic insults, while Concessao and Prakash (2025) detailed mechanisms of arsenic-induced nephrotoxicity, including tubular injury and oxidative damage [7]. These findings correlate with the development of acute kidney injury and metabolic derangements in the present patient.

The increasing use of herbal medicines without stringent regulatory oversight further compounds the risk. Hassen et al. (2022) and Ekor (2014) both highlighted the challenges in monitoring the safety of herbal supplements and the potential for serious adverse effects due to contamination [12,13]. Jaishankar et al. (2014) also emphasized the systemic toxicity of heavy metals, affecting neurological, renal, and hepatic systems simultaneously, as observed in this case [14].

Recent advancements in detection and risk prediction have been explored by Khatoon et al. (2026), who underscored the importance of improved monitoring and predictive models to identify heavy metal toxicity early [15]. However, the present case demonstrates that despite available knowledge, delayed recognition and exposure to unregulated formulations can still result in fatal outcomes.

Overall, this case is consistent with existing literature but underscores a more acute and fulminant presentation of arsenic-induced encephalopathy, highlighting the urgent need for stricter regulation, increased awareness, and early diagnostic vigilance in patients consuming traditional herbal medications.

CONCLUSION

This case underscores a rare but fatal presentation of acute arsenic toxicity following ingestion of an Ayurvedic formulation, leading to rapidly progressive encephalopathy and multi-organ dysfunction. The clinical course, supported by radiological findings of diffuse cerebral edema and toxicological confirmation of arsenic exposure, highlights the severe neurotoxic and systemic effects of heavy metal poisoning. In comparison with existing literature, this case demonstrates a more fulminant and rapidly deteriorating clinical trajectory, emphasizing the unpredictability of such exposures.

The findings reinforce the growing concern regarding the safety of unregulated herbal medications, which may contain toxic heavy metals either intentionally or as contaminants. Early recognition, prompt toxicological evaluation, and aggressive supportive management are crucial to improving outcomes. This case also highlights the urgent need for stricter quality control, regulatory oversight, and increased public and clinical awareness regarding the potential life-threatening complications associated with traditional medicine use.

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