



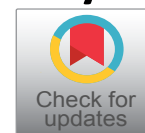
CASE REPORT

Elevated Inorganic Arsenic Levels in a Nonagenarian Resident of a Personal Care Facility: A Case Study and Reflective Analysis

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Abstract

Background: Arsenic exposure is a recognized cause of peripheral neuropathy; however, data regarding arsenic toxicity in geriatric populations-particularly among residents of personal care and long-term care facilities-remain sparse.

Case Presentation: We describe a 96-year-old female residing in a personal care facility who presented with progressive lower-extremity neuropathy and was found to have markedly elevated inorganic arsenic levels in blood and urine despite no identifiable shared environmental source.

Discussion: A comprehensive investigation revealed no facility-wide exposure. Dietary modification and nutritional supplementation resulted in normalization of arsenic levels, suggesting impaired arsenic metabolism potentially related to AS3MT genetic polymorphism. This case raised critical operational and ethical questions regarding institutional response to isolated toxicologic findings.

Conclusion: This case highlights the complexity of arsenic toxicity in advanced age and underscores the need for structured guidance for long-term care facilities when elevated arsenic levels are identified in individual residents.

Introduction

Peripheral neuropathy is common in elderly patients and is frequently attributed to diabetes, nutritional deficiencies, or idiopathic etiologies. Toxic neuropathies, including those caused by heavy metals, are well documented but often overlooked in institutionalized populations presumed to be environmentally protected [1-3]. Chronic arsenic exposure classically presents with distal symmetric sensorimotor neuropathy and may occur in the absence of overt dermatologic or gastrointestinal manifestations [2].

Interindividual variability in arsenic metabolism further complicates diagnosis, particularly in older adults. Genetic determinants of arsenic detoxification may render certain individuals vulnerable to toxicity at exposure levels tolerated by others [4]. This case illustrates the diagnostic, ethical, and institutional challenges that arise when such vulnerability is unmasked within a communal care setting.

Case Description

The patient was a 96-year-old female evaluated for continuation of care at a personal care facility, where she resided in a single room and consumed all meals prepared onsite. She ambulated independently with a walker and was cognitively intact. Her primary complaint was intermittent but progressively worsening neuropathy of the lower extremities.

At the patient's request, a second neurological opinion was obtained. As part of the neuropathy evaluation, blood testing revealed an arsenic level of 83 µg/dL (reference range < 20 µg/dL). The result was confirmed via mass spectrometry and gas chromatography and repeated with consistent findings.

An immediate and comprehensive investigation was initiated. The facility's municipal water supply demonstrated no detectable arsenic. Food vendors reported no recalls, including rice- or seafood-related products. The patient denied use of supplements, herbal preparations, or alternative therapies. Environmental review of housekeeping and maintenance materials revealed no arsenic-containing agents. No other

residents across multiple hallways exhibited symptoms suggestive of arsenic toxicity.

Repeat testing ten days later demonstrated a decreased but persistently elevated blood arsenic level of 63 µg/dL. Urinary arsenic speciation revealed predominantly inorganic arsenic. Screening of multiple residents residing along the same hallway revealed normal arsenic levels.

As a precautionary intervention, dietary rice and seafood were withheld for approximately two weeks. Concurrently, vitamin B12 and folate supplementation were initiated. Follow-up testing demonstrated normalization of arsenic levels, accompanied by stabilization of neuropathic symptoms.

Discussion

Chronic inorganic arsenic exposure is a well-recognized cause of peripheral neuropathy and may occur independently of other systemic manifestations [2,5]. In this case, the absence of a shared environmental source and the normalization of arsenic levels following dietary modification suggested impaired arsenic metabolism rather than excessive exposure.

The AS3MT gene encodes arsenic (+3 oxidation state) methyltransferase, the principal enzyme responsible for methylation of inorganic arsenic into excretable metabolites [4,6]. Polymorphisms affecting AS3MT activity occur in approximately 5-10% of the global population and are associated with reduced methylation efficiency and increased arsenic retention [6-8]. Although genetic testing was not performed, the patient's response to dietary restriction and methylation-supportive supplementation supports this working hypothesis.

Folate and vitamin B12 are essential cofactors in one-carbon metabolism and arsenic methylation. Supplementation has been shown to reduce blood arsenic levels and improve methylation efficiency, particularly in individuals with impaired detoxification capacity [9,10].

Implications for Long-Term Care and Personal Care Facilities

The identification of an elevated arsenic level in a single resident within a communal living environment raises immediate concerns regarding institutional safety and shared exposure. In the absence of geriatric-specific guidelines, facilities are often forced to extrapolate from occupational or public health frameworks that may not adequately address age-related metabolic vulnerability [3,11].

This case suggests that a structured, proportionate response is essential. Initial steps should include confirmation of abnormal results and urinary arsenic speciation to distinguish inorganic from dietary

organic forms [3,12]. Facilities should then conduct a focused environmental and dietary review, including water sources, food procurement, and cleaning or maintenance materials.

Importantly, this case supports targeted screening rather than universal testing. Screening should prioritize residents with overlapping dietary patterns, similar lengths of stay, or neurologic symptoms. Broad testing in the absence of corroborating findings may create unnecessary alarm without improving safety outcomes.

Pending clarification, interim dietary modifications—such as temporary reduction of rice-based products and certain seafood—may be reasonable, provided nutritional adequacy is maintained. Nutritional optimization, including adequate folate and vitamin B12 intake, may further mitigate risk in vulnerable individuals.

Reflective and Ethical Considerations

This case exposed a fundamental tension inherent in long-term care medicine: balancing the duty to protect the collective while honoring the individuality of each resident. The possibility that a single abnormal laboratory result reflects genetic susceptibility rather than institutional failure challenges conventional assumptions about environmental safety.

Equally significant is the psychological impact on staff, residents, and families when a potential toxic exposure is identified. Transparent communication and a methodical, evidence-informed response are essential to preserve trust while avoiding unnecessary disruption.

The paucity of data addressing arsenic exposure in geriatric populations represents a critical knowledge gap. Age-related changes in renal clearance, nutritional reserves, and metabolic capacity may amplify susceptibility to exposures traditionally considered benign. This case underscores the need for research and policy development specific to aging and institutionalized populations.

Conclusion

An elevated arsenic level in a resident of a personal care or long-term care facility should prompt a structured response emphasizing confirmation, speciation, targeted investigation, and individualized risk assessment. This case demonstrates that isolated arsenic toxicity in advanced age may reflect metabolic vulnerability rather than shared environmental exposure. Development of geriatric- and institution-specific clinical guidance is urgently needed to support patient safety while minimizing unnecessary institutional disruption.

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Statement of Equal Contribution

Both authors contributed to the conceptualization of the case, data collection, and analysis of the manuscript development. Both authors revised and approved the final version for submission.

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