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Acute axonal peripheral polyneuropathy following acute arsenic exposure in an industrial worker: A case report

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Abstract

Arsenic exposure can produce diverse neurological manifestations, with acute presentations being uncommon and often misdiagnosed. Acute arsenic-induced peripheral neuropathy may mimic immune-mediated conditions such as Guillain–Barré syndrome. We report a 44-year-old industrial worker who developed acute symmetrical distal paraesthesia and sensory ataxia following ingestion of contaminated water. Examination revealed a stocking–glove sensory pattern with preserved motor strength. Laboratory evaluation demonstrated markedly elevated blood and urinary arsenic levels, confirming toxic exposure. Initial nerve conduction studies were normal, while repeat testing showed sensory–motor axonal polyneuropathy. The patient was treated with oral succimer for 14 days, resulting in clinical improvement and reduction in urinary arsenic levels. This case highlights the importance of environmental exposure history and early chelation therapy in atypical acute neuropathies.

Keywords: Arsenic toxicity, peripheral neuropathy, sensory ataxia, Mees' lines, chelation.

INTRODUCTION

Arsenic contamination continues to pose significant health risks in both natural and industrial environments.[1,2] Arsenic exposure via water or occupation can affect multiple organs, including the peripheral nervous system. Acute or subacute sensory–motor neuropathy with ataxia is rare and may mimic Guillain–Barré syndrome or metabolic causes.[1] Early recognition is crucial, as timely chelation improves outcomes. We report an industrial worker with acute arsenic-induced axonopathy confirmed biochemically.

CASE REPORT

A 44-year-old male ordnance factory worker presented on 1 November 2025 with progressive tingling and numbness in all limbs since 24 October. He had been hospitalized from 17–23 October for acute gastroenteritis with jaundice and transaminitis. He reported consuming workplace drinking water for one month; coworkers had similar but self-limiting symptoms.

Examination showed normal motor strength and reflexes, with impaired lower-limb proprioception in a stocking–glove pattern and a wide-based ataxic gait. Cranial nerves and cerebellar signs were normal, and MRI brain and spine were unremarkable.

Differentials included Guillain–Barré syndrome and toxic neuropathy, but the pattern of symmetric sensory involvement favored arsenic toxicity, prompting heavy metal analysis. Blood arsenic levels were 31.56 µg/L and urinary arsenic levels were 300 µg/L, confirming significant exposure.

Extensive evaluation for other causes (HIV, hepatitis, HbA1c, thyroid, RPR/TPHA, ANCA, ACE, paraneoplastic markers, anti-GAD, anti-GQ1b) was unremarkable. Other heavy metals were within normal limits. CSF examination was unremarkable. Mees' lines and skin desquamation appeared 7–8 days after admission (Figure 1a and 1b).

Initial nerve conduction study on 3 November 2025 (10–12 days after onset) was normal, consistent with delayed electrophysiological changes that may appear after 2–3 weeks (Table 1a and 1b).[3]

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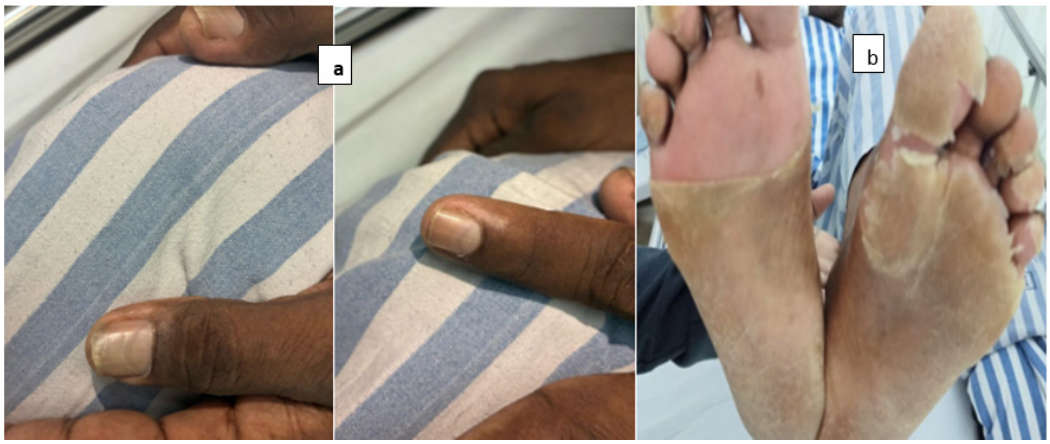

Figure 1. (a) Showing mee's lines. (b) Desquamation / peeling

Repeat nerve conduction study on 17 November 2025 showed acute sensory–motor axonal polyneuropathy with demyelinating features (Table 2a and 2b).

The patient was treated with oral succimer for 14 days, leading to gradual improvement in paraesthesia and balance. Urinary arsenic levels decreased to <50 µg/L with clinical recovery.

Table 1a and 1b: Initial nerve conduction study (03 November 2025)

Table 1a. Motor nerve conduction study (Motor NCS)

Nerve	Distal Latency (ms)	F-wave/Diff Latency (ms)	Amplitude (mV)	Distance (mm)	Conduction Velocity (m/s)
Right Common Peroneal	3.18	8.49	9	360	42.4
Right Posterior Tibial	3	10.26	8.2	440	42.9
Left Common Peroneal	2.86	8.39	8.4	360	42.9
Left Posterior Tibial	3.18	9.95	8.4	410	41.9
Right Median	4.48	5.1	6	260	50.9
Right Ulnar	2.45	4.64	6.6	270	58.2
Left Median	3.54	4.95	9.4	240	48.5
Left Ulnar	2.6	4.8	8.8	250	56.0

Table 1b. Sensory nerve conduction study (Sensory NCS)

Nerve	Distal Latency (ms)	Amplitude (mV)	Distance (mm)	Conduction Velocity (m/s)
Right Sural	2.24	8.5	120	53.6
Left Sural	2.08	12.1	130	61.4
Right Median	Absent	—	—	—
Right Ulnar	1.93	13.1	105	54.5
Left Median	1.72	12.9	110	54
Left Ulnar	2.5	12.9	135	54

Table 2a and 2b: Repeat nerve conduction study (17 November 2025)

Table 2a. Sensory nerve conduction study (Sensory NCS)

Nerve	Distal Latency	Amplitude (mV)	Distance (mm)	Conduction Velocity (m/s)
Right Sural	Absent	Not Recordable	Not Recordable	Not Recordable
Left Sural	Absent	Not Recordable	Not Recordable	Not Recordable
Right Median	Absent	Not Recordable	Not Recordable	Not Recordable
Right Ulnar	Absent	Not Recordable	Not Recordable	Not Recordable
Left Median	Absent	Not Recordable	Not Recordable	Not Recordable
Left Ulnar	Absent	Not Recordable	Not Recordable	Not Recordable

Table 2b. Sympathetic skin response (SSR Study)

Nerve	Distal Latency (s)
Right Median	Absent
Left Median	Absent
Right Tibial	0.06
Left Tibial	0.17

DISCUSSION

Heavy metal–induced neuropathies are relatively common in India, with arsenic, lead, mercury, thallium, organophosphates, and some traditional medications as key causes.[4] Arsenic is widely present in soil, groundwater, and industrial effluents, and is rapidly absorbed after ingestion, reaching peak levels within about one hour.[4,5] Its trivalent form disrupts cellular energy metabolism by inhibiting thiol-dependent mitochondrial enzymes, impairing oxidative phosphorylation and causing neuronal injury.[6]

The peripheral nervous system is particularly susceptible to damage. Sensory manifestations typically precede motor involvement, initially affecting distal regions and progressively extending proximally in a length-dependent pattern.[3,4] Patients may present with sensory ataxia due to impaired proprioception, while reflexes can remain preserved early. Features may mimic Guillain–Barré syndrome or chronic inflammatory demyelinating polyneuropathy, leading to diagnostic delay without exposure history. Mees' lines—transverse white nail bands from arsenic deposition—support the diagnosis, typically appearing after 3–6 weeks and seen in both chronic and acute/subacute exposure.[5]

Diagnosis is confirmed by elevated blood or urinary arsenic levels, as imaging studies are typically unremarkable.[12] Electrophysiological changes may be delayed, explaining the initially normal nerve conduction study. Repeat testing showed sensory–motor axonal neuropathy with demyelinating features, consistent with toxic neuropathy.[3]

Chelation is the mainstay of treatment. Succimer is preferred due to oral use and better tolerability than dimercaprol in stable patients. Early therapy improves recovery, though some may have residual sensory deficits.[13,14]

Several Indian studies have reported arsenic-induced neuropathy associated with contaminated groundwater or traditional medicines, with outcomes varying according to the duration of exposure and the timeliness of treatment initiation.[2,8]

Our patient showed clinical and biochemical improvement after chelation, consistent with prior reports. This case highlights the need to consider toxic exposures in acute or subacute neuropathies, especially in industrial settings or contaminated water areas. Chronic arsenic-related neuropathy has also been reported in India (Table 1).

In conclusion, arsenic neuropathy should be suspected in patients with sensory ataxia and a stocking–glove sensory pattern, especially with occupational or environmental exposure. Early diagnosis with arsenic testing and prompt chelation can markedly improve outcomes.

DISCLOSURE

Ethics: Appropriate patient consent was obtained.

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Conflicts of interest: None

Table 3 : Previous reports of arsenic toxicity–related neuropathy in India

Author (Year, Location)	No. of Cases	Source of Exposure	Age	Principal Clinical Features	Therapy	Outcome
Mukherjee *et al.* (2003, West Bengal)[2]	187	Contaminated water	21–30 years	Distal hypesthesia, paresthesia	Not specified	Improvement (33%), deterioration (10%), static (57%)
Valappil and Mammen (2019, Kerala)[3]	1	Indigenous medicine	9 years	Progressive weakness and numbness of all limbs	Antioxidants and vitamins	Upper limb deficits resolved; full recovery at 18 months
Ghosh (2013, West Bengal)[7]	5	Contaminated water	31–53 years	Sensory neuropathy	Not specified	Not reported
Chakraborti *et al.* (2016, Patna)[8]	15	Contaminated water	16–75 years	Sensory neuropathy	Not specified	Not reported
Pinto *et al.* (2014, Chandigarh)[9]	2	Ayurvedic medicine	20–32 years	Distal sensory loss and weakness of hands and feet	D-penicillamine (6 months)	Partial improvement with residual deficits
Mazumder *et al.* (2010, West Bengal)[10]	19	Contaminated water	Not reported	Weakness	Unithiol	Clinical improvement in 14 cases
Shinde *et al.* (2022, Pune)[11]	1	Ayurvedic medicine	53 years	Stocking–glove sensory loss	Withdrawal of offending agent	Clinical improvement with residual deficits

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