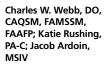
## **CASE REPORT**

## > THE PATIENT

34-year-old man

# > SIGNS & SYMPTOMS

- Chronic lower back pain
- Peripheral neuropathy
- Leg spasms with increasing weakness



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### THE CASE

A 34-year-old man was referred to the sports medicine clinic for evaluation of lumbar radiculopathy. He had a 2-year history of chronic lower back pain that started while he was working on power line towers in Puerto Rico. The back pain was achy, burning, shooting, and stabbing in nature. He had been treated with anti-inflammatories by a company health care provider while in Puerto Rico, but he did not have any imaging done.

At that time, he had tingling and burning that radiated down his left leg to his ankle. The patient also had leg spasms—in his left leg more than his right—and needed a cane when walking. His symptoms did not worsen at any particular time of day or with activity. He had no history of eating exotic foods or sustaining any venomous bites/stings. Ultimately, the back pain and leg spasms forced him to leave his job and return home to Louisiana.

Upon presentation to the sports medicine clinic, he explained that things had worsened since his return home. The pain and burning in his left leg had increased and were now present in his right leg, as well (bilateral paresthesias). In addition, he said he was feeling anxious (and described symptoms of forgetfulness, confusion, and agitation), was sleeping less, and was experiencing worsening fatigue.

Work-ups over the course of the previous 2 years had shed little light on the cause of his symptoms. X-rays of his lumbar spine revealed moderate degenerative changes at L5-S1. A lab work-up was negative and included a complete blood count, testing for HIV and herpes, a hepatitis panel, an antinuclear antibody screen, a C-reactive protein test, and a comprehensive metabolic panel. Thyroid-stimulating hormone, creatine kinase, rapid plasma reagin, and human leukocyte antigen B27 tests were also normal.

Magnetic resonance imaging (MRI) revealed a cystic lesion in the right ilium near the sacroiliac joint. A more recent follow-up MRI and computed tomography scan of the pelvis found the cyst to be stable and well marginalized, with no cortical erosion. Attempts at physical therapy had been unsuccessful because of the pain and decreasing muscle strength in his lower extremities. The patient's primary care provider was treating him with meloxicam 15 mg/d and duloxetine 60 mg/d, but that had not provided any relief.

Our physical examination revealed a patient who was in mild distress and had limited lumbar spine range of motion (secondary to pain in all planes) and significant paraspinal spasms on the right side in both the lumbar and thoracic regions. The patient had reduced vibratory sensation on his left side vs the right, with a 256-Hz tuning fork at the great toe, as well as reduced sensation to fine touch with a cotton swab and a positive Babinski sign bilaterally. Lower extremity reflexes were hyperreflexic on the left compared with the right. He had no pronator drift; Trendelenburg, straight leg raise, Hoover sign, and slump tests were all negative. His gait was antalgic with a cane, as he described bilateral paresthesias.

#### THE DIAGNOSIS

The differential diagnosis for low back pain is quite extensive and includes simple mechanical low back pain, lumbar radiculopathy, facet arthritis, spinal stenosis, spondylolysis/spondylolisthesis, and referred pain from the hip, knee, or upper back. It can also be caused by referred pain from visceral organs such as the liver, colon, or kidneys. Low back pain can also signal primary or metastatic disease. However, most of these potential diagnoses had been ruled out with imaging and lab tests.

• Two things caught our attention. First: Mechanical low back pain and the associated discogenic radiculopathy would be unilateral, manifesting with asymmetric paresthesias and pain. Our patient had weakness in gait and pain and burning in both of his legs. Second: Our patient described decreased sleep and feeling anxious, with symptoms of forgetfulness, confusion, and agitation. These factors prompted us to look beyond the normal differential and consider a potential toxicity. A heavy metal screen was ordered, and the results were positive for arsenic toxicity.

#### **DISCUSSION**

Arsenic toxicity is a global health problem that affects millions of people. 1.2 Arsenic has been used for centuries in depilatories, cosmetics, poisons, and therapeutic agents. Today it is used as a treatment for leukemia and in several ayurvedic and homeopathic remedies. 3.7 It is a common earth element found in ground water and a waste product from mining and the manufacturing of glass, computer chips, wood preservatives, and various pesticides. 2.3.7.8

■ A great masquerader. Once in the body, arsenic can cause many serious ailments ranging from urinary tract, liver, and skin cancers to various peripheral and central nervous system disorders.<sup>2</sup> Arsenic can cause symmetrical peripheral neuropathy characterized by sensory nerves being more sensitive than motor nerves.<sup>2,3,5,6</sup> Clinically, it causes numbness and paresthesias of the distal extremities, with the lower extremities more severely affected.<sup>3,6</sup> Symptoms can de-

velop within 2 hours to 2 years of exposure, with vomiting, diarrhea, or both preceding the onset of the neuropathy.<sup>2,3,5,6</sup> Arsenic is linked to forgetfulness, confusion, visual distortion, sleep disturbances, decreased concentration, disorientation, severe agitation, paranoid ideation, emotional lability, and decreases in locomotor activity.<sup>3,5,6</sup>

■Testing and treatment. Arsenic levels in the body are measured by blood and urine testing. Blood arsenic levels are typically detectable immediately after exposure and with continued exposure, but quickly normalize as the metal integrates into the nonvascular tissues. Urine arsenic levels can be detected for weeks. Normal levels for arsenic in both urine and blood are ≤ 12 μg/L.³ Anything greater than 12 μg/L is considered high; critically high values are those above 50 μg/L.³.5 Our patient's blood arsenic level was 13 μg/L.

Treatment involves removing the source of the arsenic. Chelation therapy should be pursued when urine arsenic levels are greater than 50  $\mu g/L$  or when removing the source of the arsenic fails to reduce arsenic levels. Chelation therapy should be continued until urine arsenic levels are below 20  $\mu g/L.^{5.6}$ 

■ After discussing potential sources of exposure, our patient decided to move out of the house he shared with his ex-wife. He started to recover soon after moving out. Two weeks after his clinic visit, he no longer needed a cane to walk, and his blood arsenic level had dropped to 6  $\mu$ g/L. Two months after his clinic visit, the patient's blood arsenic level was undetectable. The patient's peripheral neuropathy symptoms continued to improve.

The source of this patient's arsenic exposure was never confirmed. The exposure could have occurred in Puerto Rico or in Louisiana. Even though no one else in the Louisiana home became ill, the patient was instructed to contact the local health department and water department to have the water tested. However, when he returned to the clinic for follow-up, he had not followed through.

#### THE TAKEAWAY

When evaluating causes of peripheral neuropathy, consider the possibility of heavy metal toxicity, which can be easily over-

Several of the patient's symptoms prompted us to look beyond the normal differential and consider a potential toxicity. looked by the busy clinician. In this case, the patient initially experienced asymmetric paresthesia that gradually increased to burning pain and weakness, with reduced motor control bilaterally. This was significant because mechanical low back pain and the associated discogenic radiculopathy would be unilateral, manifesting with asymmetric paresthesias and pain.

Our patient's leg symptoms, the constellation of forgetfulness, confusion, and agitation, and his sleep issues prompted us to look outside our normal differential. Fortunately, once arsenic exposure ceases, patients will gradually improve because arsenic is rapidly cleared from the bloodstream.<sup>3,6</sup>

#### CORRESPONDENCE

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