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Polyneuropathy due to Arsenic and Cadmium Toxicity: A Case Report

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Abstract

Polyneuropathy is a condition that can be caused by a variety of factors, including environmental, dietary, and lifestyle. This case study examines the impact of these factors on a patient's polyneuropathy symptoms. The objectives were to examine the symptoms and lab results of a patient with polyneuropathy symptoms and discuss the importance of physicians considering all aspects of a patient's lifestyle when determining the cause of such symptoms. We examined X-rays, lab results, and notes related to the patient's case, as well as conducted a web-based search, to describe the patient's history and condition. Based on the results of this research, factors such as smoking, alcohol use, arsenic and cadmium toxicity (i.e., through recently increased seafood intake or past occupational exposure), and pre-diabetes may have precipitated the patient's polyneuropathy symptoms. This case demonstrates that it is crucial to examine all layers of a patient's condition to recommend accurate treatment for the condition and effective alleviation of symptoms.

Keywords

Polyneuropathy, Cadmium, Arsenic, Seafood, Diabetes Mellitus, Occupational Exposure, Osteomyelitis

Introduction

Polyneuropathy is the most common disorder of the peripheral nervous system, especially among older adults [1]. This condition typically presents with sensory and motor symptoms, including numbness and tingling, burning, and weakness [1]. The most common causes of polyneuropathy include diabetes and alcohol use [2]. Other causes of polyneuropathy can include environmental toxins (i.e., heavy metals), chemotherapy, vitamin B12 deficiency, copper deficiency, HIV infection, and Hepatitis B or C infection [1,2].

Heavy metal exposure, specifically to cadmium and arsenic, has been associated with peripheral polyneuropathy. A case-control study of 41 metal industry workers and 36 healthy controls found that chronic exposure to multiple heavy metals, including cadmium and arsenic, was associated with peripheral nerve impairment when compared to healthy controls [3]. In general, cadmium and arsenic can both negatively impact the body [4]. Cadmium toxicity, often from occupational exposure in alloy, battery, and glass production, can lead to degenerative bone disease, kidney dysfunction, and disorders in the metabolism of

zinc and copper [4]. Cadmium can also be found in cigarette smoke [5]. Seafood, along with rice and grains, can also be contaminated with cadmium [5]. One population study found that individuals with cadmium poisoning had an increased likelihood of bone fractures and osteoporosis [6]. Arsenic is primarily absorbed through the small intestine and can lead to cardiovascular dysfunction, skin and hair changes, and central nervous system injury [4]. Furthermore, arsenic exposure can lead to the degeneration of axons in human peripheral nerves [7]. Both arsenic and cadmium can be found in seafood [8,9]. Non-toxic arsenic, like arsenobetaine and arsenosugars, can be found in most seafood and seaweed [9].

Chronic hyperglycemia, a hallmark of diabetes mellitus, plays a significant role in neuropathy development, affecting 60–70% of patients with diabetes [10]. Among diabetic patients, smoking is associated with diabetic peripheral neuropathy [11].

First-line treatment of polyneuropathy symptoms includes gabapentoids, tricyclic antidepressants, serotonin norepinephrine reuptake inhibitors, and non-pharmacologic treatments that typically include exercise, spinal cord stimulation, and acupuncture [12].

Osteomyelitis is a potential complication of polyneuropathy, usually in the setting of diabetic foot ulcers in patients with diabetic peripheral polyneuropathy [13]. These ulcers, if infected, can lead to osteomyelitis [13]. Antibiotics are used to treat osteomyelitis, but if the patient's condition worsens, amputation is required [13]. It is important to note that X-rays are often negative for osteomyelitis in the first few weeks of infection [14]. MRI and CT scans are more sensitive than X-rays and are often used to detect osteomyelitis [15]. A bone biopsy is considered essential to confirm an osteomyelitis diagnosis and identify the causative pathogen [15].

In this report, we present an unusual case in which a patient with long-standing neuropathy presented with high cadmium and arsenic levels following increased seafood consumption.

Case Summary

The patient is a 71-year-old male with past medical history of essential tremor, cervicalgia, post-traumatic stress disorder (PTSD), alcohol/cannabis/cocaine abuse, tobacco use, pre-diabetes, stage 2 chronic kidney disease, chronic headaches, depression, and alcohol-induced polyneuropathy presenting to Neurology for headaches, tremors, and lower extremity pain. A urine sample from two months prior was negative for heavy metals including lead, arsenic and mercury. Patient was advised to increase gabapentin to 1200 mg daily.

Patient had a follow-up visit with Neurology less than two months later. He discontinued his use of gabapentin as he felt that this was not helping his paresthesia symptoms. Laboratory studies reviewed at that time revealed high serum cadmium and arsenic levels, measured at 4.2 ug/L (normal range: 0.0-1.2 ug/L) and 32 ug/L (normal range: 0-9 ug/L), respectively. Other screenings appeared relatively normal: hemoglobin A1c was measured at 5.5%, vitamin B12 was measured at 683 pg/mL, folic acid was measured at 3.3 ng/mL and TSH was measured at 1.32 mIU/L. Mercury and lead levels were determined to be normal by the physician. Patient reported eating large amounts of seafood, particularly crabs, and had a history of occupational exposure to heavy metals. The patient was recommended to undergo electromyography (EMG) of the bilateral lower extremities, consult Integrated Medicine for acupuncture, start folic acid supplementation (1 mg daily), and avoid seafood for at least two weeks before repeating laboratory studies to monitor cadmium and arsenic levels.

Laboratory studies were repeated about two weeks later. Serum cadmium and arsenic levels were measured at 2.7 ug/L (normal range: 0.0-1.2 ug/L) and 11 ug/L (normal range: 0-9 ug/L), respectively. Another blood test collected on the same day measured cadmium at 3.0 ug/L and arsenic at 12 ug/L. Mercury and lead levels were normal, measured at 5.2 ug/L (normal range: 0.0-14.9 ug/L) and 2.0 mcg/dL (normal range: 0-19 mcg/dL), respectively. Serum copper levels were high, at 139 ug/dL (normal range: 69-132 ug/dL). Serum vitamin B1 and B2 levels were measured at 81.3 nmol/L (normal range: 66.5-200.0 nmol/L) and 231 ug/L (normal range: 137 – 370 ug/L). ESR was measured at 8 mm/hour (normal range: 0-15 mm/hour). HCV, HIV, and Hepatitis B and C

screenings were negative.

About two months later, the patient's serum glucose level was measured at 118 mg/dL (normal: 65-99 mg/dL), serum creatinine was measured at 1.4 mg/dL (normal range: 0.7-1.3 mg/dL), and eGFR was measured at 54 (normal range ≥90). Hemoglobin A1c was measured at 6.0% (normal: 4.8-5.6%). Lymphocyte count was normal, measured at 28.8% (normal: 19.1-53.0%). Regarding cholesterol levels, LDL was high at 119 mg/dL while HDL was within normal limits at 64 mg/dL. Electrolytes, including sodium at 140 mEq/L (normal: 136-145 mEq/L), potassium at 4.1 mEq/L (normal: 3.5-5.1 mEq/L), chloride at 100 mEq/L (normal: 98-107 mEq/L), and calcium at 9.6 mg/dL (normal: 8.6-10.3 mg/dL) were all normal. Liver function tests, including albumin at 4.2 g/dL (normal 3.5-5.7 g/dL), AST at 18 U/L (normal: 13-39 U/L), ALT at 10 U/L (normal: 7-52 U/L), and alkaline phosphatase at 92 U/L (normal: 34-104 U/L), were all normal.

The patient then presented to the emergency department two weeks after this updated blood work with a flare up of his bilateral lower extremity pain. His vital signs were as follows: temperature 97°F, heart rate 99 beats/minute, respiratory rate 20 breaths/min, O_2 saturation 98%, blood pressure 171/97 mm Hg.

During the emergency department visit, laboratory studies were negative for Hepatitis B. Additional studies revealed an elevated serum glucose of 107 mg/dL (normal range: 65-99 mg/dL), low eGFR of 80 (normal range: ≥90), and ESR of 16 mm/hr (normal range: 0-15 mm/hr). White blood cell and red blood cell counts were normal, measured at 7.28 x 10^3/uL (normal: 4.3-10 x 10^3/uL) and 4.62 x 10^6/uL (normal: 4.5-6 x 10^6/uL), respectively. Lymphocyte count increased significantly, measured at 54.7% (normal range: 19.1-53.0%), with the majority accounting for absolute lymphocytes at 3980/uL (normal range: 1320-3570/uL). Plasma lactic acid was measured at 1.1 mmol/L (normal: 0.5-2.2 nmol/L). While prothrombin time was normal, measured at 11.5 seconds (normal: 10-13.8 seconds), prothrombin INR was low, measured at 1.05 (normal: 2.0-3.0), and partial thromboplastin time was high measured at 36.7 seconds (normal: 23.3-35.6 seconds). Electrolytes, including sodium measured at 138 mEq/L (normal: 136-145 mEq/L), potassium at 3.8 mEq/L (normal: 3.5-5.1 mEq/L), chloride at 100 mEq/L (normal: 98-107 mEq/L), and calcium at 9.5 mg/dL (normal: 8.6-10.3 mg/dL) remained normal. Liver function tests, such as albumin at 4.2 g/dL (normal 3.5-5.7 g/dL), AST at 18 U/L (normal: 13-39 U/L), ALT at 16 U/L (normal: 7-52 U/L), and alkaline phosphatase at 91 U/L (normal: 34-104 U/L), were all normal. Physical examination showed both lower extremities had distal pulses and sensation intact without crepitus or tenderness. The radiologist's interpretation of the right foot X-ray noted a deformity and subtle cortical irregularity of the first distal phalanx, raising concern for osteomyelitis (**Fig-1**). The patient was referred to primary care for follow up heavy metal testing and Podiatry to review the X-ray imaging of his feet. He was treated with Tylenol and Flexeril. The patient was also counseled to drink fluids, rest both feet, and wear support shoes. Patient was recommended to continue following with Neurology to monitor his symptoms.

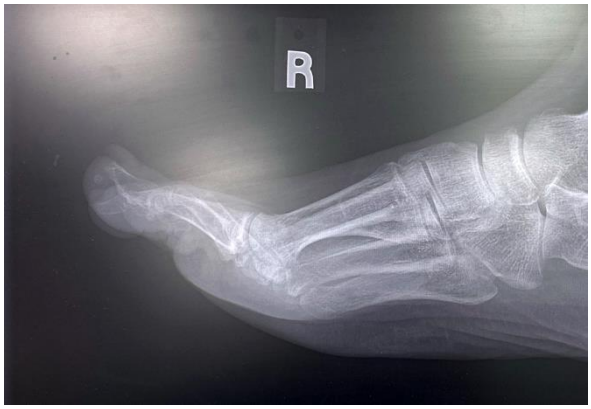

Fig-1:

Right foot radiograph showing cortical irregularity of the first distal phalanx.

Discussion

Polyneuropathy is a common disorder of the peripheral nervous system. While the relationship between heavy metal exposure and polyneuropathy has been established, heavy metal exposure is often secondary to occupational exposure, and exposure to high levels of heavy metals is relatively uncommon among the general public [3,16]. Seafood, particularly shellfish, can expose individuals to many different heavy metals, including cadmium and arsenic [8,9,16]. Cigarette smoke can also expose individuals to cadmium [5]. Cadmium has a half-life of 3-4 months in the blood, and inorganic arsenic has a half-life of 3-4 hours in the

blood [16]. Organic arsenic is considered relatively non-toxic to the body and is rapidly excreted in the urine 1-2 days after seafood consumption. While heavy metals may clear from the blood, some accumulate in other parts of the body for much longer [5]. For example, the half-life of cadmium in the body typically ranges from 5 to 30 years [5]. For this reason, urine, nail, or hair analyses are generally considered a better representation of heavy metal burden [16].

We present an unusual case of polyneuropathy likely exacerbated by cadmium and arsenic poisoning from seafood. His underlying history of occupational exposure to heavy metals could be a contributing factor, as heavy metals like cadmium can remain in the body for a long time. The diagnosis of his heavy metal poisoning was complicated by the patient having multiple risk factors for polyneuropathy. The patient had a history of alcohol-induced polyneuropathy, and his current drinking status was not discussed explicitly. The patient also had high blood glucose, and his hemoglobin A1c was indicative of pre-diabetes, which is also associated with polyneuropathy symptoms when not managed. While urine testing for multiple heavy metals came back negative five months prior, the patient subsequently had high arsenic and cadmium levels in the blood after increased seafood intake. This patient also discontinued his gabapentin regimen at the same time he began eating more seafood. This case demonstrates that treatment of longstanding polyneuropathy can be complex as potential causes can be disguised among others. Furthermore, early counseling on specific lifestyle modifications and possible exacerbating factors for patients with polyneuropathy could improve patient outcomes overall. Earlier questioning of a patient's previous and current heavy metal exposures can guide treatment and counseling. Physicians should educate patients with polyneuropathy to monitor potential heavy metal exposures from seafood or cigarette smoke, as well as encourage proper diet and exercise in patients with high blood glucose.

The patient's X-rays were suspicious for possible osteomyelitis, and lab testing showed elevated ESR and lymphocyte count. While the development of these concerning signs could have been linked to the patient's arsenic and cadmium exposure, we are unable to conclude this definitively.

Conclusion

Overall, a 71-year-old male presented to a Neurology follow up appointment for long-standing lower extremity pain showed high serum cadmium and arsenic levels in the urine following increased seafood intake. The physician recommended the patient to have an EMG of the bilateral lower extremities, consult Integrated Medicine, start folic acid supplementation, avoid seafood, and repeat blood studies. Bilateral foot radiographs raised concerns for osteomyelitis based on an irregularity in the distal phalanx of the left foot.

This case highlights the importance of considering all aspects of a patient's life, including their occupation, environment, and habits, to determine the origin of the patient's symptoms and, as a result, what treatments are most effective. For example, in this case, the patient's neuropathy symptoms could be due to multiple factors, including the patient's history of smoking, high seafood diet exposing the individual to excess arsenic and cadmium, previous occupational exposure, alcohol use, and pre-diabetes. Based on this case, providers should be more aware that a high seafood diet may predispose individuals to heavy metal exposure, particularly cadmium and arsenic. In general, healthcare providers should consider the importance of a more holistic and multifactorial approach to treatment, such as encouraging smoking cessation, limiting excessive seafood consumption and alcohol use, and outlining the importance of glucose management in patients with polyneuropathy symptoms.

Conflict of Interest

All the authors have read and approved the final version of the manuscript. All the authors declare no conflicts of interest.

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