A Case of Significant Malnutrition Presenting as Acute Sensorimotor Axonal Neuropathy

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Abstract

Nutritional status is an important, but often overlooked, component of care for patients being seen in any number of subspecialty clinics. The neurologic ramifications of nutritional deficiencies tend to present in the later stages of prolonged malnutrition. A 34 year-old woman with a history of alcoholism presented to the emergency department with two weeks of worsening gait imbalance, diffuse paresthesia, allodynia, and weakness of all extremities. The physical examination yielded diffuse loss of sensation to all sensory modalities in a length-dependent manner and complete lack of deep tendon reflexes in all extremities. There was also mild 4/5 weakness of proximal musculature of upper and lower extremities. Nerve conduction studies showed a diffuse sensorimotor axonal neuropathy that was worse in the lower extremities compared to the upper extremities. Laboratory evaluation yielded significant deficiencies that included calcium, pyridoxine, vitamin E, folate, borderline low thiamine, and elevated homocysteine levels. This was a unique case of severe malnutrition manifesting as an acute diffuse axonal neuropathy. Lower socioeconomic status with concurrent alcohol use can lead to a bevy of neurologic conditions as seen in our patient.

Introduction

Numbness is a common presenting symptom in the emergency department and in neurology clinics throughout the country. The underlying pathology of the numbness is often determined to be a neuropathy. The cause of the neuropathies can be due to various causes including toxicity, malnutrition, iatrogenic, and autoimmune. There are numerous vitamins and minerals whose deficiencies have been found to cause profound neuropathies including thiamine, cobalamin, pyridoxine, vitamin E, and copper to name a few. A singular nutritional cause is often difficult to determine as some patients may have coexisting alcoholism.

Case Presentation

A 34 year-old woman with a history of alcoholism presented to the emergency department with two weeks of worsening diffuse numbness, gait instability, allodynia, and coordination problems with her hands. Symptoms began on her bilateral face and proceeded to affect her arms and legs. She described the sensation of feeling “frost bit” on her extremities. She was unable to ambulate for several days prior to presentation due to the symptoms. She also noted having a flu-like illness four weeks prior to presenting to the emergency department, but her flu-like symptoms had resolved prior to presenting to our facility. Further history revealed that she was only eating one meal daily and drinking several alcoholic beverages per day. She was evaluated promptly for a rapidly progressive neuropathy with electromyography and nerve conduction studies (EMG/NCS). NCS showed a diffuse sensorimotor axonal neuropathy that was worse in the lower extremities compared to the upper extremities. The sensory nerve action potentials were absent in the right sural and peroneal nerves. No demyelinating changes were seen. There were no significant changes on EMG testing.

After admission, laboratory evaluation showed multiple nutrition deficiencies including protein calorie malnutrition as well. Nutritional abnormalities as follows: Pyridoxine was low at 4 mcg/L (ref 5-50), Vitamin E 4.0 mg/L (ref 5.0-18.0), folate 3.5 ng/mL (ref >7.0), homocysteine greater than 50.00 umol/L (ref 3.00-11.00), and thiamine 74 nmol/L (ref 70-180). Of note, she also presented with mild rhabdomyolysis with an elevated creatine kinase of 593 U/L (ref 25-190) which trended down with IV hydration. Supplementation was started while she was inpatient for the above deficiencies. Vasculitis panel, autoimmune evaluation, and HIV labs were all unremarkable. During her stay, she did have alcohol withdrawal symptoms of tachycardia and restlessness that resolved with a lorazepam taper. At clinic follow up in August 2017, patient is now able to ambulate with a walker and her proximal strength has improved significantly. She continues to attend physical therapy. She now has 5/5 strength throughout except mild 4+/5 weakness in bilateral deltoid and iliopsoas muscles. There has also been improvement in her neuropathic symptoms including pain and numbness which are being treated with gabapentin 900 mg three times per day and is on duloxetine 60 mg daily as well.
Her most recent limited vitamin labs were vitamin B12 of 716 pg/mL (ref 200-1000), thiamine of 274 nmol/L (ref 70-190), vitamin E 16.6 mg/L (ref 5.0-18.0). A repeat EMG/NCS will be performed in the future, but has not yet been completed.

Discussion

Neuropathy is a relatively common disorder with four to seven percent of adults over the age of 55 having symptoms. The most common causes for those symptoms being diabetes, alcoholism, liver disease, and neoplasms. As mentioned previously, nutritional deficiencies can cause significant neuropathies as well. Often times, these neuropathies tend to be axonal, but can have demyelinating features as well. These malnutrition-related neuropathies can also be complicated by comorbid alcoholism which makes diagnosing a singular cause of the neuropathy difficult. Patients with alcoholism are at a much higher risk of malnutrition which places those at risk for neuropathy from neurotoxic effects of alcohol and from malnutrition.

Current recommendations from the Journal of the American Medical Association prior to lab evaluation include determining if a known cause of neuropathy is present in the history without atypical features (acute presentation, asymmetry, non-length dependence, motor predominance, autonomic involvement). If a typical cause is found (diabetes or alcoholism) without atypical features, the recommendation is for no further lab testing or electrodiagnostic evaluation. The basic labs recommended are complete metabolic panel, complete blood cell count, serum protein electrophoresis, vitamin B12 with methylmalonic acid, and glucose tolerance testing. This is a reasonable initial evaluation for most patients who present with neuropathy. However, in patients with comorbid alcoholism, further testing would be reasonable to evaluate nutritional status and prevent significant neurologic sequelae including neuropathy.

Conclusion

The ultimate etiology of the patient’s presenting symptoms in our case was felt to be a culmination of years of alcohol use and malnutrition that reached a critical point several weeks prior to presenting to our facility. This case demonstrates that malnutrition with comorbid alcoholism can present with a profound acute sensorimotor axonal neuropathy that might appear like acute inflammatory demyelinating polyneuropathy or other acute neuropathy. This case highlights the importance of monitoring nutritional status not only in patients with alcoholism but in all patients. Further studies might be warranted to assess the feasibility of nutritional monitoring in patients with alcoholism to guide supplementation to prevent alcoholic and malnutrition-related neuropathy.
Acknowledgements

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Statement of value: This case report demonstrates a unique case of severe nutritional defects presenting with acute sensorimotor axonal neuropathy that appeared similar to other acute neuromuscular emergencies. However, this case was not autoimmune in nature and shows how prolonged malnutrition in the setting of alcoholism can have profound effects on the peripheral nervous system. This case is particularly pertinent to the state of West Virginia due to the high rate of poverty and lack of access to foods with nutritional value in many counties throughout the state.

Conflicts of interest: None

Patient permission: Permission was obtained from the patient via telephone to use electromyography, nerve conduction data, lab results, and clinical history for publication purposes only. Consent obtained by Vincent Amone, MD and is documented in electronic health record at West Virginia University.

References