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Sensory Motor Neuropathy due to Vitamin E Deficiency – A Case Study.

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Abstract

Diabetic patient presenting with features of neuropathy is at majority taken as features of diabetic neuropathy but in scenarios like described in this case we describe a case who presented us with weakness in lower limbs and features of neuropathy and history chronic of diarrhea.Retrospective evalaution was done and history of episode of pancreatitis followed by diabetes after 1 year and then weight loss and diarrhea followed in the chronology. Fat soluble vitamins like vitamin E levels were significantly reduced and neuropathy in this case was reversible with adequate management.

Keywords: Vitamin E, Lower Limb, Chronic, Neuropathy.

Introduction

Vitamin E deficiency can be observed in patients with malabsorption syndromes. It is unusual for it to be a result of dietary insufficiency due to its presence in a wide variety of foods. Patients with significant vitamin E deficiency can present with neuromuscular disorders such as ataxia, hyporeflexia as well as loss of vibration and proprioceptive sensation.

The Toxic-Metabolic, Idiopathic, Genetic, Autoimmune, Recurrent, and Severe Acute Pancreatitis, Obstructive classification system (TIGAR-O) categorizes the causes of chronic pancreatitis and pancreatic exocrine insufficiency. Amongst the toxic causes alcohol is a well-recognised cause and have a triple risk of developing chronic pancreatitis.

Diabetes developing secondary to pancreatitis is referred to as pancreatogenesis diabetes. In nearly 20% of cases, chronic pancreatitis seems to be the underlying disease, the prevalence and clinical importance of diabetes secondary to chronic pancreatitis has been underestimated and underappreciated and it being a causative factor is seldom considered.

Development of diabetes mellitus in chronic pancreatitis occurs due to the destruction of islet cells by pancreatic inflammation. Also, nutrient maldigestion leads to an impaired incretin secretion and therefore to a diminished insulin release of the remaining beta-cells.

Many patients with chronic pancreatitis manifest some degree of fat malabsorption, regardless of the presence of symptoms. In patients with pancreatogenesis diabetes mellitus exocrine pancreatic insufficiency is nearly ubiquitous present.[10] Since clinically overt steatorrhea is usually not observed until over 90% of exocrine pancreatic function have vanished, exocrine pancreatic insufficiency and maldigestion might remain undetected[9]. However, the relevant maldigestion, which is present in the majority of patients with chronic pancreatitis, may cause qualitative malnutrition. This is especially important concerning the absorption of fat-soluble vitamins (A, D, E and K).

Herein, we describe a case in which a previously healthy adult with no family history of genetic defects and presented with a characteristic Sensory-Motor Axonopathy, which was evaluated to be linked to Vitamin E deficiency and fat malabsorption.

Case History

A 34-year-old male presented to us with complaint of abnormal sensation with numbness and tingling in the bilateral lower extremities. Symptoms began 2 months back and involved distal lower and upper extremities. He noted slight lower extremity weakness and complained of difficulty in getting up from lying position.

Patient had been complaining of diarrhoea since the last 1 year and used to pass 5-6 stools daily. The episodes used to get relieved with fasting. It was not associated with any abdominal pain or fever.

Patient gave history of chronic moderate alcohol consumption up to 30-60 ml/day for 8 years and quit it following developing acute pancreatitis 5 years back. 1 year after the same, patient developed diabetes mellitus and has been on oral hypoglycaemics for same but complained of uncontrolled glycaemic levels since the past 6 months and switching to insulin.

He denied low back pain, incontinence of bowel or bladder, or muscle fasciculation, disruption of balance, gait, perceived upper extremity weakness, visual changes. No trauma at the back or leg has been reported and it was the first-time patient experience these symptoms. There was no headache, hearing nor visual difficulties, no abnormal sensation over the upper extremities. There were no constitutional symptoms such as fever, chills, night sweats, anorexia. Patient has no history of recent travel, tick bite, rash, flu-like symptoms. He has no known drug allergies. He denies tobacco, recreational drug use and over-the-counter prescriptions or medications.

On Examination, patient was calm, conscious and fully oriented to time ,place and person .He had a regular normal volume pulse =78/min .BP was 130/90 in supine position and no postural drop was evident on standing at 2 and 3 min .Patient had pallor present .No evidence of icterus, cyanosis or clubbing seen. Patient had B /l Pedal, painless, pitting edema.

On Neurological examination, patient had generalised muscle wasting with normal tone. Patient had weakness in hip flexion and adduction with power 4/5. At the ankle patient had weakness in plantar flexion with power 4/5. Rest all movements in lower limb had normal power. He had a limping gait. Deep tendon reflexes in lower limb were 2+ except ankle reflex which was absent. There was normal extensor response. Sensory examination showed impaired vibration up to mid of tibia. Proprioception was impaired at Metatarso-tarsal

joint. Fine touch and pin prick, pain and temperature were intact B/L.

Further investigation showed Hb=6.5 TLC=9700 Platelet count=2.54 lakhs/cumm. PTI was 50%. Liver function test showed normal bilirubin, AST and ALT levels. However Alkaline phosphate levels were raised =976(46-116 U/L). Patient was non-reactive for HBsAg, HIV, HCV and negative for ANA.RFT were normal. UACR was 55.4 s/o microalbuminuria.

Patient's serum lipase levels were almost no detectable and by colorimetric method value of 1 U/L was estimated (Normal=73-393 U/L). Fecal matter was positive for Sudan staining s/o Steatorrhea. IV replacement of vitamin K corrected PTI in 48h. Hence deficiency of vitamin K was evident.

CT Abdomen imaging confirmed pancreatic insufficiency. Pancreas appeared atrophied with features s/o chronic calcified pancreatitis.

Nerve conduction study was s/o predominantly sensory motor axonal neuropathy with nerves showing decreased CMAP, decreased conduction velocity, however no conduction defect was seen.

Features were suspected of arising from Vitamin E deficiency and it was confirmed when Tocopherol levels were found to be 1.72(5.0-18.0 mg/L) by HPLC s/o Significant deficiency .

Discussion

Vitamin E deficiency is rare in humans due to the wide availability of tocopherols in the food. Even vegetarian and vegan diets are replete with sources of vitamin E. The main cause of vitamin E deficiency is from fat malabsorption. A deficiency in vitamin E can result in neuromuscular disorders, haemolysis and chronic kidney disease which may lead to dialysis Low serum levels are defined as below 0.5 mg/dl and often cause no appreciable symptoms.[9] When symptoms do appear, they usually present as a neuropathy or myopathy. The neuropathy consists of a spinocerebellar syndrome with variable peripheral nerve involvement. Clinical manifestations include ataxia, hyporeflexia, and decreased proprioceptive or vibratory sensation.

This case described a case of neurological manifestations arising from significant vitamin E deficiency. The history of loose and frequent bowel motions that are present for the last year with chronology of diabetes following the development of pancreatitis made us suspect pancreatic insufficiency causing diabetes and fat malabsorption .Serum lipase levels were non detectable which made us investigate in this direction .Hence diagnosis pancreatogenesis diabetes was made as per the major criteria mentioned above Replacement with pancreatic enzymes improved the appetite of patient and stool frequency as well .Vitamin E supplementation improved the positive and negative sensory symptoms and also the ambulation improved

Conclusion

It is capable of mimicking a number of other conditions, and oftentimes there is no clear pathogenesis behind it. In this case, the patient's primary symptom was sensory neuropathy; however, the most common symptom of spinocerebellar ataxia was absent. The patient also lies outside of the age range that genetic disorders of vitamin E metabolism usually present, and he did not display evidence of malabsorption. This indicates that there may still be unexplained mechanisms behind vitamin E deficiency. A more in-depth understanding of vitamin E metabolism and the pathogenesis behind vitamin E deficiency may lead to better diagnostic tools and earlier detection in at-risk patients, thus improving morbidity in the condition.

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