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Original Research Paper

General Medicine

A CASE REPORT : SUBACUTE COMBINED DEGENERATION OF SPINAL CORD DUE TO VITAMIN B12 DEFICIENCY PRESENTED WITH BILATERAL LOWER LIMB WEAKNESS

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INTRODUCTION :

Subacute combined degeneration of the spinal cord is a neurological complication of vitamin B12 (cobalamin) deficiency. A deficiency of vitamin B12 can occur as a result of nutritional deficiency, reduced absorption due to altered gastrointestinal anatomy or function, or due to the intake of certain drugs. Subacute combined degeneration is characterized by degeneration of the dorsal columns and the lateral columns of the spinal cord due to demyelination. It commonly presents with sensory deficits, paresthesia, weakness, ataxia, and gait disturbance. In severe untreated cases, it can lead to spasticity and paraplegia. It is crucial to promptly identify and treat vitamin B12 deficiency to prevent the development of this serious neurological complication. Subacute combined degeneration is a preventable, reversible medical condition that affects the brain, spinal cord, and peripheral nerve with devastating effects if untreated.

The primary sources of vitamin B12 are dietary and include meat, eggs, and dairy products. Vitamin B12 obtained from these products is absorbed in the ileum along with the intrinsic factor produced from the parietal cells in the stomach. Alteration at the level of dietary intake, absorption, or intrinsic factor activity can cause vitamin B12 deficiency and, in turn, the neurological manifestations of subacute combined degeneration of the spinal cord.

Dietary deficiency is an unlikely cause of vitamin B12 deficiency as liver stores last up to 3 years. However, in patients that are strict vegans or the elderly, particularly those in assisted facilities, dietary deficiencies have been identified as causes for vitamin B12 deficiency.Vitamin B12 deficiency can be suspected as the cause of unexplained anemia in patients who have other autoimmune conditions such as vitiligo or thyroiditis. In such patients, pernicious anemia can be identified as the cause. It is an autoimmune condition where antibodies are formed against the parietal cells in the stomach, decreasing the production of the intrinsic factor and altering the absorption of vitamin B12.

Malabsorption as a cause of vitamin B12 deficiency can be due to loss of intrinsic factor seen in patients post gastrectomy or after gastric bypass surgery. Malabsorption is also found after the loss of absorption surface area in the ileum in patients with surgical resection following Crohn disease. It has also been associated with fish tapeworm infestations which compete with the host for vitamin B12 absorption. Certain medications such as metformin, proton pump inhibitors, and nitrous oxide have been associated with vitamin B12 deficiency as well.

Case Report :

A 25 year old male hindu patient residing at shobheshwar road morbi, gujarat belong to lower socioeconomic class came to general hospital morbi with complain of both lower limb tingling & numbness and weakness, unable to walk since 1 month. Weakness was insidious in onset and gradually increasing. Patient is chronic alcoholic since last 6 year. On admission his vitals were normal.

On examination, patient was fully conscious and oriented to time place and person. Mild pallor present. No icterus, clubbing, cyanosis, oedema, lymphadenopathy seen. On CNS examination, Motor- No visible atrophy/hypertrophy. Tone was increased in both lower limb and normal in upper limb, Power was 3/5 in both lower limb and 5/5 in upper limb.

Sensory- There were loss of vibration sense and loss of joint position in both lower limb. Pain, temperature, touch sensation were present on both side equally. Romberg sign was positive. Reflex: loss of ankle on both side, knee reflex were exacerbated on both side and Normal in both upper limb. Planter reflexes were extensor on both side. cerebellar sign-absent.

On investigation, HB- 9.6, WBC- 4200, Platelet count- 1.67lacs, PS examination- Megaloblastic RBCs and Hyper-segmented neutrophils seen. Creatinine-0.9, BILIRUBIN- 0.9, SGOT- 9, SGPT- 10, ALP- 8, LDH- 121. Serum Vitamin B12 level was 88pg/ml. MRI SPINE- T2WI demonstrated diffuse hyper-intensity in the posterior part of the spinal cord.The axial images revealed involvement of posterior columns bilaterally. Patient was treated with Inj cynocobalamine.

DISCUSSION :

This patient as described above, presented with features consistent with sub-acute combined degeneration of the spinal cord. The presentation was slowly progressive spastic paraparesis, proprio ceptive loss with mild cognitive impairment without any optic nerve involvement. MRI of dorsal spine showed T-2 hyper-intense lesions only in the dorsal column without any compressive or inflammatory lesions. There was no family history of hereditary spastic paraparesis.

The classic presentation of vitamin B12 deficiency is a combination of megaloblastic anemia and sub-acute combined degeneration of the spinal cord and peripheral neuropathy. About one third of patients having B12 deficiency, experienced only mild symptoms such as paraesthesia of the hands and feet. This can eventually progress to include sensory loss, gait ataxia, distal limb weakness and ataxic paraplegia. In the early stage of the disease, the spinal cord appears to be affected first. The extent of involvement usually includes the posterior and lateral columns of the lower cervical and upper thoracic spinal cord in a contiguous way. The predominant involvement of the posterior columns of the spinal cord results in impairment of position and vibration sense and paraesthesia.

Early diagnosis of sub acute combined degeneration of the spinal cord is important because the condition is curable with

prompt replacement therapy. The degree of resolution of clinical symptoms is inversely proportional to the duration and severity of vitamin B12 deficiency. In the event of delayed treatment, irreversible disabling neurological impairment may occur. In population those who are predominantly vegetarian, there is possibility of Vitamin B12 deficiency. Its diagnosis should be entertained if different parts of neural axis i.e. brain, spinal cord, optic nerve, peripheral nerves are involved; still the number cases which are reported are very few in India.

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