Introduction: Vitamin B12 deficiency is an important treatable cause of neurologic illness in our country which usually presents as non-compressive myelopathy, peripheral neuropathy, and higher function alteration. We herewith analyse 10 cases of Vitamin B12 deficiency for their clinical features and laboratory parameters.

Methods: 10 patients who were diagnosed to have Vitamin B12 deficiency at the Institute of Neurology between 2011 and 2014 were analysed for their clinical features and laboratory parameters.

Results: Age of patients ranged from 14-49 years. Nine were males and one female. Identifiable risk factors were noted in five patients. Nine patients had spastic weakness of limbs with exaggerated reflexes and posterior column signs. Sensory symptoms and signs were noted in nine patients. One patient presented with higher function alteration only without any myelopathy or neuropathy. Bladder symptoms were noted in four and loss of libido and erectile dysfunction in two. Sensory ataxia was prominent in eight patients. Low serum B12 level was noted in all patients. Peripheral smear was normal in four patients, normocytic normochromic in three, and dimorphic in three. MRI spine showed hyperintensities in spinal cord in one patient. MRI brain in the patient with dementia showed T2 and Flair Hyperintensities in the periventricular region.

Conclusions: This study highlights the classical clinical features of Vitamin B12 deficiency and also documents the occurrence of unusual symptoms like loss of libido, erectile dysfunction and bladder symptoms.

Keywords: Vitamin B12, Cobalamin, Paraparesis, ataxia, dementia

Abstract: Vitamin B12 deficiency is an important treatable cause of neurologic illness in our country which usually presents as non-compressive myelopathy, peripheral neuropathy, and higher function alteration. We herewith analyse 10 cases of Vitamin B12 deficiency for their clinical features and laboratory parameters.
Methods:
10 patients who were diagnosed to have Vitamin B12 deficiency at the Institute of Neurology between 2011 and 2014 were analysed for their clinical features and laboratory parameters.

Results:
Age and Sex Distribution

<table>
<thead>
<tr>
<th>S.No.</th>
<th>Age Group</th>
<th>Sex</th>
<th>No. of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td></td>
<td>Male</td>
<td>Female</td>
</tr>
<tr>
<td>1.</td>
<td>11 – 20 years</td>
<td>3</td>
<td>1</td>
</tr>
<tr>
<td>2.</td>
<td>21 – 30 years</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>3.</td>
<td>31 – 40 years</td>
<td>4</td>
<td>-</td>
</tr>
<tr>
<td>4.</td>
<td>41 – 50 years</td>
<td>1</td>
<td>-</td>
</tr>
<tr>
<td>5.</td>
<td>&gt; 50 years</td>
<td>-</td>
<td>-</td>
</tr>
<tr>
<td>Total</td>
<td></td>
<td>9</td>
<td>1</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Risk Factors</th>
<th>No. of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>Vegetarian Diet</td>
<td>10%</td>
</tr>
<tr>
<td>Surgery</td>
<td>10%</td>
</tr>
<tr>
<td>Alcohol</td>
<td>30%</td>
</tr>
<tr>
<td>Risk Factor not identified</td>
<td>50%</td>
</tr>
</tbody>
</table>

Clinical Spectrum:
Spastic Paraparesis            90%     9
Post Column symptoms           90%     9
Sensory ataxia                 80%     8
Bladder Symptoms               40%     4
Peripheral Neuropathy          30%     3

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Discussion:
Sources of Vitamin B12 are meat, fish, egg, milk, fortified cereals and legumes. Recommended Daily Allowance is 2.4 g/day. B12 deficiency can occur in pernicious anaemia as a result of malabsorption of intrinsic factor. Cobalamin deficiency is common in elderly due to atrophic gastritis and achlorhydria induced food-cobalamin malabsorption. Cobalamin deficiency is frequently seen following gastric surgery and bariatric surgery. These procedures cause food-bound cobalamin malabsorption. Partial gastrectomy is associated with loss of intrinsic factor. Acid reduction therapy using H2 blockers and prolonged use of drugs like metformin can cause cobalamin deficiency. Other causes of cobalamin deficiency are due to malabsorption secondary to ileal disease or resection, bacterial overgrowth pancreatic disease, celiac disease, crohn’s disease and tropical sprue. Helicobacter pylori infection of the stomach and parasitic infestation by fish tape worm, Diphyllobothrium latum can cause cobalamin deficiency. Certain hereditary enzyme defects can manifest with disorder of Vit B12 metabolism. These is increased prevalence of Vit B12 deficiency in HIV infected patients with neurologic symptoms. Nitrous oxide (N2O, “laughing gas”) is a commonly used inhalational anaesthetic that is abused due to its euphoriant properties. N2O irreversibly oxidizes the cobalt core of cobalamin and renders methyl cobalamin inactive. Post-operative neurologic dysfunction can be seen with N2O exposure during routine anaesthesia if subclinical cobalamin deficiency is present. N2O toxicity due to inhalant abuse was reported among dentists, other medical personnel and university students. Other causes are strict vegetarians and chronic alcohol abuse. Some unknown factors also contribute to deficiency. Clinical manifestations of Vitamin B12 deficiency appear when the body stores get depleted over a period of 2 to 10 years. Neurologic manifestation are the earliest and often the only manifestation of cobalamin deficiency.
Vitamin B12 deficiency presents usually as symmetrical paresthesia, numbness starting in the feet than in the hands with accompanying sensory ataxia. Patients have weakness and stiffness in the legs leading later on to ataxic paraparesis with variable spasticity and contractures. Other associated symptoms are impaired manual dexterity, memory loss and diminished visual acuity. Personality change, psychosis and rarely delirium are the neuropsychiatric manifestations.  

Rarely, reported neurologic manifestations related to cobalamin deficiency include cerebellar ataxia, orthostatic tremor, ophthalmoplegia, vocal cord paralysis, a syringomyelia like distribution of motor and sensory deficits and autonomic dysfunction in the form of impotence, incontinence and orthostatic light headedness.  

Examination of these patients reveals posterior and lateral column signs, loss of vibration and position sense more pronounced in the legs than in the arms. Visual impairment due to optic neuropathy may occasionally be the earliest or sole manifestation. Motor signs include weakness in the lower limbs, spasticity, increased, normal or reduced tendon reflexes especially ankle jerks and extensor plantar responses. The commonest clinical features noted in our patients were spastic paraparesis (9 patients), Posterior column signs (9), Sensory ataxia (8), Peripheral neuropathy in 3 patients. Autonomic symptoms in the form of bladder dysfunction (urgency, urge incontinence) was noted in 4 patients and loss of libido and erectile dysfunction was present in 2 patients. Only one patient presented with memory impairment, irritability and mild frontal lobe disturbances with wandering tendencies. Alcoholism was the commonest risk factor identified in our patients. Low serum B12 was found in all patients and the value ranged between 38 and 200 pg/l. MRI Spine was abnormal in only one patient. The patient with dementia had periventricular white matter hyper-intensities. With treatment, Vitamin B12 deficiency symptoms may gradually improve in about 6 months. The degree of improvement depends on the severity and duration of deficiency. Permanent nerve damage may occur if the deficiency is not corrected promptly.

Conclusion:
This study stresses that Vitamin B12 deficiency can present not only with classical features, but also have unusual symptoms like loss of libido, erectile dysfunction and bladder symptoms.

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