ORIGINAL RESEARCH PAPER
Volume-8 | Issue-12 | December - 2019 | PRINT ISSN No. 2277 - 8179 | DOI : 10.36106/ijsr
INTERNATIONAL JOURNAL OF SCIENTIFIC RESEARCH

TO STUDY THE RELATIONSHIP BETWEEN VITAMIN B12 DEFICIENCY AND DEGENERATIVE CERVICAL MYELOPATHY IN INDIAN VEGETARIAN POPULATION AT A TERTIARY LEVEL HOSPITAL

Neurosurgery

Dr D.P.Tiwari

Dr. Ankit Jain* *Corresponding Author

ABSTRACT

STUDY DESIGN : Retrospective study
OBJECTIVE : to discuss the relationship between degenerative cervical myelopathy and vitamin B12 deficiency in indian vegetarian population.
METHODS : a retrospective case study was carried out at Government Doon Medical College, Dehradun , a tertiary level hospital.
RESULTS : in the study of 135 patients, 64 were found to having vit B 12 deficiency. 56 patients out of these were vegetarians.
CONCLUSION: Vitamin B12 deficiency is found in significant number of patients with degenerative cervical myelopathy. Significant association was found with vegetarian diet. Vitamin B12 level optimization can improve results of surgical outcome. Routine assessment of Vit B12 levels in vegetarian patient in cases of degenerative cervical myelopathy is considered.

KEYWORDS

INTRODUCTION

Vitamin b12 is an essential vitamin which is mostly derived from non-vegetarian source. Indian population is more prone to have deficiency of vit B12 because of a largely vegetarian diet. Lack of balanced diet and inadequate assess to vegetable and animal food which are a major source of micronutrients is a basic cause for deficiency.

The clinical spectrum of vitamin B12 deficiency include chiefly megaloblastic anemia and neurological deficits. Megaloblastic anemia is characterized by enlarged red blood cell precursors. It involves asynchronous maturation of the cytoplasm and nucleus. The clinical picture include weakness, palpitations, dyspnea on exertion, fatigue, light-headedness, jaundice, and shortness of breath. These symptoms typically do not arise until the anemia is quite severe, as cardiopulmonary adaptations can alleviate hypoxia.1

Neurological deficits due to vitamin B12 deficiency include peripheral neuropathy, mental status changes, myelopathy, optic neuropathy, or a combination of these.2,3

Patients with both DCM and B12 deficiency are most frequently diagnosed above the age of 50 years. The prevalence of B12 deficiency is about 20% in industrialized nations.4 Since B12 is essential for myelination , investigation of this relationship is important as deficiency of B12 may not only exacerbate myelopathic symptoms in DCM but may also hinder neurological recovery.5

Mechanism of action of vitamin B12

Vitamin B12 is synthesized exclusively by anaerobic bacteria, and it is obtained in foods of animal origin. Gastrointestinal uptake of vitamin B12 requires binding of a glycoprotein secreted by gastric parietal cells, called intrinsic factor. The B12-intrinsic factor complex binds to "cubam" receptors expressed on enterocytes in the distal ileum and is absorbed via receptor-mediated endocytosis. Given the important role of intrinsic factor in vitamin B12 uptake, deficiencies of this due to an autoimmune gastritis leads to a severe B12 deficiency, with haematological and neurological manifestations.6 This condition is known as "pernicious anemia"

Intracellular B12 is stored as 2 active coenzymes: deoxyadenosy lcmobalamin and methylcobalamine. Deoxyadenosy lcobalamin is a cofactor for methylmalonyl-CoA mutase, which catalyzes the conversion of methylmalonyl-CoA to succinylCoA in the mitochondria. Succinyl-CoA subsequently enter the Krebs cycle and is important for the synthesis of lipids and carbohydrates7. Methylcobalamin acts as a coenzyme for cytoplasmic methionine synthase, which catalyzes the methylation of homocysteine to methionine. This transmethylation reaction also involves folate (vitamin B9) and is therefore critical for nucleic acid synthesis.

Methylcobalamin is also important for the synthesis and maintenance of the myelin sheath. A number of studies have reported significant association of vitamin B12 deficiency with the development of white-matter lesions or retarded myelination.8-10 This may be due to a number of pathways, including increased synthesis of lecithin (the primary component of myelin sheath lipids)11-12; increased synthesis of myelinotrophic cytokines and growth factors such as IL-6 and EGF13; downregulation of Erk1/2 and upregulation of myelin basic protein14;upregulation of neurotrophic gene factors15; and regulation of normal prion protein concentration in the central nervous system.16

Pathophysiology of DCM

There are a number of pathophysiological factors that result in DCM: (1) static compression of the spinal cord, (2) dynamic injury resulting from mobile degenerative cervical spine elements compressing the cord, and (3) tethering of the cord or altered cord tension due to changes in the cervical spine alignment or cord compression.17 All these mechanisms causes reversible and irreversible injury leading to spinal cord dysfunction. Reversible tissue injury includes demyelination, Wallerian degeneration, edema, and inflammatory changes. Whereas irreversible injury manifests after frank loss of neuronal tissue has occurred.18

The clinical picture of Degenerative cervical myelopathy includes a number of upper and lower limb neurological complaints. These include corticospinal motor deficits,clumsiness, gait impairment, numbness ,atrophy of hand muscles,hyperefflexia and spasticity , paresthesia and urinary incontinence in severe cases Objective myelopathic signs such as Hoffmann's sign, Babinski's reflex, and ankle clonus ,L'hermitte's phenomenon, Romberg s sign can also be elicited.

AIM AND OBJECTIVE

To discuss the relationship between degenerative cervical myelopathy and vitamin B12 deficiency in Indian vegetarian population.

METHODS

Study type: Retrospective study.
Study Site: This study was conducted in the Department of Surgery GDMC, Dehradun.

Study period: The study was carried out in cases from 1st August 2018 to 30th July 2019.

Sample size: All the patients with degenerating cervical myelopathy(DCM) attending the OPD at GDMC, Dehradun from 1st August 2018 to 30 July 2019 were included in the study.

Inclusion Criteria: All patients with DCM were included in the study irrespective of age and sex.

Exclusion Criteria: Patients other than having DCM were excluded from study.

METHODOLOGY

Demographic data and information regarding the presenting ailment was collected.

History of presenting complaints and clinical symptoms along with MRI findings were assessed to confirm the diagnosis of DCM Vitamin B12 levels were recorded and patient categorized as deficient or normal. Values of less than 160 pg/mL (118 pmol/L) were considered deficient. The data collected was then analysed.

STATISTICALANALYSIS

Data is presented as frequency and percentages wherever applicable. Chi square test with or without Yates correction was used to compare descriptive variables. P value less than 0.05 was considered significant.

RESULT

The present study was aimed to study the relationship between degenerative cervical myelopathy and vitamin B12 deficiency in indian vegetarian population at GDMC, Dehradun. A total of 135 cases were enrolled in the study. The following observations were made :

There were 72 males and 63 females in the study.

Out of 135 cases vitamin B12 deficiency was found in 64 cases i.e. 47.4% of cases.(Figure 1)

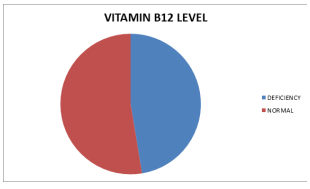

Figure 1: distribution based on vit B12 deficiency

98 patients (i.e. 72.6%) were vegetarians. Of these 56 patients have vitamin B 12 deficiency and 42 had normal levels i.e. 57% of vegetarians had B12 deficiency.

37 patients (27.4%) were non- vegetarians. Of these 08 patients have vitamin B12 deficiency and 29 had normal levels showing that only 21.6% of non- vegetarians have B12 deficiency.(Figure 2)

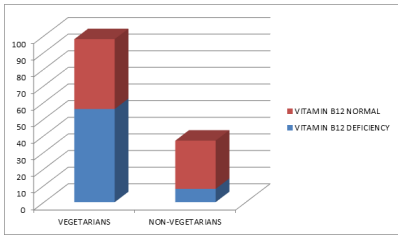

Figure2 : distribution of vitamin B12 deficiency in veg and non-vegetarians

DISCUSSION

In our study out of 135 patients with DCM, 72 were male and 63 females. There was no predilection towards any specific gender group. Of the 135 patients 47.4% presented with vitamin B12 deficiency. Of these 87.5 % 0f patients were having vegetarian diet. Of the remaining 52.6% patients who have normal blood vitamin B12 levels , only 59% were having a vegetarian dietary habit.

Vitamin B12 deficiency seems to be higher among vegetarians from Indian subcontinent compared with those living in other regions. In a retrospective study, Gupta et al.19 found 66.3% (deficiency defined as serum vitamin B12 ⩽132 pmol/l) of their sample having vitamin B12 deficiency. Similarly, Yajnik et al.20 reported deficiency rates of 81%, 68%, and 51%, among Indian men in different socioeconomic subgroups. Men from urban, middle-class had notably higher rates of deficiency compared with both men from rural and slum socioeconomic class. Yajnik et al.20 also suggested that the higher deficiency prevalence among male subjects from the middle-class may be reflective of ingestion of less microbial B12 from contaminated food and water sources. Although other studies confirmed that the contamination of water and unwashed products may contain B12, the high deficiency prevalence among individuals included in this study indicates that vegetarians cannot rely on them as means of vitamin B12 deficiency prevention.21

Treatment with B12 can optimize neurological recovery in patients with known B12 deficiency and superimposed DCM which usually show neurological impairment out of proportion of what would be expected based on imaging.22 In other case reports, patients with suspected diagnosis of DCM, but underlying SACD, experienced a resolution of symptoms after B12 administration.23 These findings imply that patients with DCM and concomitant B12 deficiency require treatment for both conditions to optimize neurological recovery. Care should be taken for patients with mild cord compression and possible B12 deficiency prior to surgical treatment, as cord compression may be a false positive finding and treatment with B12 may resolve symptoms.

A high index of suspicion for B12 deficiency among DCM patients should be placed among patient with history of gastrointestinal resection or comorbidities, such as atrophic gastritis and irritable bowel disease, which may be an underlying cause for unrecognized B12 deficiency.24 When suspected, laboratory findings of megaloblastic anemia, low B12 levels, and high levels of homocysteine may be helpful.

Conclusion

It is clear that B12 is necessary for maintaining spinal cord function, and deficiency can result in DCM. Given the high prevalence B12 deficiency in the population , the important role of B12 in myelination and B12 deficiency as a differential diagnosis of DCM, there is considerable rationale to conduct routine assessment of B12 levels in patients with DCM.

Since preoperative assessment includes routine blood work, this additional diagnostic measurement would not unnecessarily burden the patient or substantially increase costs. B12 is an essential vitamin, cheap, and readily accessible. We need to investigate the relationship between B12 and DCM to see whether :

- whether DCM patients with B12 deficiency present differently on clinical exam
- patients with deficiency who are supplemented with B12 achieve optimal outcomes
- patients with B12 deficiency and DCM have suboptimal surgical outcomes
- increasing B12 levels in patients with no deficiency improves surgical outcomes more than otherwise expected

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