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A study of the prevalence of serum vitamin B12 and folic acid deficiency in KIMS, Karad

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Abstract

Aim: To study the prevalence of vitamin B12 and folic acid deficiency in patients admitted in KIMS, Karad;

Material and Methods: 100 patients were selected on basis of below mentioned symptoms. Detailed history was taken regarding the presenting symptoms. A thorough clinical examination was done including central nervous system. They were investigated as per attached preforma [8]. Patients with a history of tingling and numbness in extremities, dizziness, unsteady gait, early tiredness, forgetfulness, proximal weakness, distal weakness, chronic headache, less interest in work, chronic loose stools, strict vegetarians, alcoholics, intake of medications like antitubercular treatment, surgery involving terminal ileum were included in this study.

Results: 100 cases of suspected vitamin B12 and folic acid deficiency were studied. There were 33 % patients who were vegetarian. Major cause of vitamin B12 deficiency was found to be folic acid deficiency. Deficiency of vitamin B12 and folic acid;

Conclusion: In a small study it was found that megaloblastic anaemia may have symptoms and signs referable to several systems including skin, gastrointestinal, neurology, neuropsychiatry

Keywords: serum vitamin B12, folic acid, KIMS, Karad

Introduction

Megaloblastic anaemias are a heterogenous group of disorders that share common morphologic characteristics. Erythrocytes are larger and have heavier nuclear-to-cytoplasmic ratios compared to normoblastic cells. Neutrophils can be hypersegmented and megakaryocytes are abnormal [1]. On the molecular level in the megaloblastic cells, the maturation of nuclei is delayed while cytoplasmic development is normal. Megaloblastosis is a generalised disorder because nonhematopoietic cells such as GI and uterine cervical mucosal cells can also have megaloblastic features [10-13]. The aetiology of megaloblastic anaemias is diverse but a common basis is impaired DNA synthesis [1-3].

Recent trends in medical care have emphasised early therapy. Folate supplementation is recommended to prevent the atherosclerosis and thromboembolic events by reducing homocysteine levels [6]. Folate is given during pregnancy to prevent developmental defects in foetus. Mild cobalamin deficiencies and incipient cobalamin-related neuropsychiatric abnormalities have recently been identified in some individuals and prompt early treatment with cobalamin is recommended to avoid progression of mental deterioration and neurological complications [14, 15]. There are a number of sporadic reports in the literature of compromised cellular immune status in patients with cobalamin and folate deficiencies affecting neutrophil functions [4, 5].

Material and Method

The present study is a cross sectional observation study carried out on patients seeking medical attention on outpatient and inpatient basis in the medicine department of a teaching institute in Karad. Large number of patients were studied and out of them, 100 patients were selected on basis of below mentioned symptoms. Detailed history was taken regarding the presenting symptoms. A thorough clinical examination was done including central nervous system. They were investigated as per attached proforma [8]. Patients with a history of tingling and numbness in extremities, dizziness, unsteady gait, early tiredness, forgetfulness,

proximal weakness, distal weakness, chronic headache, less interest in work, chronic loose stools, strict vegetarians, alcoholics, intake of medications like antitubercular treatment, surgery involving terminal ileum were included in this study.

It's a competitive assay in which vitamin B12 from patient's serum competes with vitamin B12 labelled with acridinum ester in the Lite Reagent, for a limited amount of purified [8, 9] intrinsic factor which is covalently coupled to paramagnetic particles in solid phase. The assay uses releasing agent (Sodium Hydroxide) to release vitamin B22 from the endogenous binding proteins in the samples and prevents rebinding after the solid phase is added to the sample Serum, heparinised plasma are recommended samples for this assay.

Observation and discussion

Table 1: Presence and severity of anaemia

Deficiency Group	Hb 6-11	Hb >11
Absolute B12	4	2
Folic acid	21	6
Borderline B12	9	0
Combined	7	3
Normal	38	10

100 cases of suspected vitamin B12 and folic acid deficiency were studied. There were 33 % patients who were vegetarian. Major cause of vitamin B12 deficiency was found to be folic acid deficiency. Deficiency of vitamin B12 and folic acid was studied in 4 groups:

- a. Absolute vitamin B12 deficiency (S. Vitamin B12<200pg/ml)
 - b. Absolute folic acid deficiency (S. Folic acid<5ng/ml)
 - c. Borderline vitamin B12 deficiency (S. Vitamin B12--200-250pg/ml)
 - d. Combined vitamin B12 and folic acid deficiency Of the tested 100 patients
- Absolute vitamin B12 deficiency was found in 6 % patients b. Borderline vitamin B12 deficiency was found in 9 % patients c. Absolute folic acid deficiency was found in 26 % patients d. Combined vitamin B12 and folic acid deficiency was found in 10 % patients Thus, folic acid deficiency forms a major chunk of deficiency group.

Table 2: Neurological Signs (ABS/Loss) In Normal Levels of Vitamin B 12 and Folic Acid

Neurological signs	UL	LL
Reflex	0	28
Touch	0	0
Vibration	0	29
Position	0	29
Romberg's	30	0

Table 3: Peripheral Blood Smear Findings IN Deficiency Group

Deficiency Group	Macrocytes	Hypersegmented neutrophils
Absolute B12	2	1
Folic acid	10	2
Borderline B12	2	0
Combined	2	0
Normal	0	2

The clinical features in megaloblastic anaemia were found to be tingling numbness in 11 % patients, unsteady gait in 3 % patients, giddiness in 41% patients, pallor in 85% patients, diffuse hyperpigmentation on skin in 52 % patients, glossitis in 40% patients and stomatitis in 57 % patients.

Table 4: Platelet count deficiency group

Deficiency Group	<1.5 lakh	>1.5 lakh
Absolute B12	2	4
Folic acid	1	26
Borderline B12	1	8
Combined	4	7

MCV was raised in 23% patients, 37% patients had macrocytosis and 6% patients had hypersegmented neutrophils on peripheral blood smear and 8% patients had thrombocytopaenia. 28 % patients had low RBC count i.e. <3x106 cu/mm 6% patients had neuropsychiatric manifestations [7]. Depressive illness in 1% patients, dementia in 0% patients, forgetfulness in 1% patients, mania/hallucination in 0 % patients each and chronic headache in 1% patients. Neuropathy in form of loss of reflexes, decreased touch sensation was present in 9% patients. Posterior column involvement viz. Loss of joint position, vibration, positive Romberg's sign were present in 34% patients of vitamin B12 and folic acid deficiency.

Conclusion

In a small study it was found that megaloblastic anaemia may have symptoms and signs referable to several systems including skin, gastrointestinal, neurology, neuropsychiatry. The neurological syndrome is variable and includes headache to simple tingling and numbness. The neurological features may not be proportionate to the degree of haematological changes and may be independent of each their. Hence a high index of suspicion may avert the serious neurological damage with reversal to normal status if treated early.

Conflict of interest: No conflict of Interest

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References

1. Sharvon SD, Carney MW, Chanarin I, Reynolds EH. The neuropsychiatry of megaloblastic anaemia. Br Med J. 1980; 281(6247):1036-8.
2. Babu S, Srikantia SO. Availability of folates from some foods. Am J Clin Nutr 1976; 29:376-9
3. Smith EL. Vitamin 12. London: John Wiley and Sons, 1960, 20.

4. Allan Diamond, Revie Diamond, Florian Thomas. Vitamin B12 associated Neurologic Diseases. Movement and Neurodegenerative Diseases Oct 11, 2004.
5. Victor Hoffbrand, Megaloblastic Anaemia. Postgraduate Hematology pg47-67.6. Schilling R F: Is Nitrous Oxide a dangerous anesthetic for Vitamin B12 deficient subject? JAMA. 1986; 255(12):1605-6.
6. John Coleman: An Introduction to Cobalamin Metabolism-Cobalamin Form Function, Inhibitors, a vegan perspective, 2002.
7. Robert S. Hillman: Hematopoietic Factors in Goodman and Gillman: Textbook of Pharmacology. 54,1487-1519.
8. Gardner W, Osier W. A case of progressive pernicious anaemia (Idiopathic of Addison). Can Med Surg J. 1877; 5:385-404.
9. Russell JSR, Batten FE, Collier J. Subacute combined degeneration of the spinal cord. Brain. 1900; 23:39.
10. Woltmann HW. The nervous symptoms in pernicious anaemia: an analysis of one hundred and fifty cases. American Journal of Medical Science. 1919; 173:400-9.
11. Munot GR, Murphy WP. Treatment of Pernicious Anaemia by a special diet. JAMA. 926; 87:470-6.
12. Castle WB: Extrinsic factor in pernicious anaemia. American Journal of Medical Science. 1929; 178:148.
13. Greenfield JG, Carmichael EA: Peripheral nerves in cases of subacute combined degeneration of the cord. Brain.1935; 58:483-91.
14. Wills L, Clutterbuck PW, Evans PDF. A new factor in the production and cure of macrocytic anaemia and its relationship to the other haemopoetic principles curative in pernicious anaemia, Biochem. J. 1937; 31:2136-2147.