Section: Pathology

Original Research Article

STUDY OF PERNICIOUS ANEMIA IN FEMALES OF WESTERN MAHARASHTRA

Jafar Isakhan Pathan[1], Rubina Rahman[2]

[1]Assistant Professor, Department of Pathology, JIIU's Indian Institute of Medical Sciences and Research (IIMSR) Warudi Badnapur, (dist) Jalna, Maharashtra, India.
[2]Consultant Pathologist, Healthworld Hospitals, Durgapur, West Bengal, India

Received : 17/04/2024
Received in revised form : 10/06/2024
Accepted : 26/06/2024

Keywords:
CLIA method, bone marrow aspiration, neuro-imaging, electrophysiological, megaloblastic anemia

Corresponding Author:
Dr. Jafar Isakhan Pathan,
Email: pathanjafar@gmail.com

DOI: 10.47009/jamp.2024.6.3.135

Source of Support: Nil,
Conflict of Interest: None declared

Int J Acad Med Pharm
2024; 6 (3); 654-656

Abstract

Background: The clinical onset of pernicious anemia is usually insidious and vague. If not treated in due course, it may be fatal. **Materials and Methods:** 85 (eighty-five) adult females suffering from pernicious anemia were studied on Vit-B12 and routine blood examinations were carried out. Other investigations, including neuroimaging and electrophysiological studies, were also carried out. Some patients with a normal B12 level but clinically suspected pernicious anemia underwent bone marrow study. The diagnosis was established by a low serum Vit-B12 level (< 145 pg/ml) assessed by (1) the CLIA method and (2) bone marrow aspiration showing megaloblastic change. **Result:** In the study of neurological signs, The highest was 27 (31.7%) followed by vibration and 15 (17.6%) touché. In clinical symptoms, the highest was giddiness (32.9%), and the least was ataxic gait (5.8%). The highest clinical problem pallor, (41%) and least was 13 (15.2%) hyper pigmentation. **Conclusion:** The present pragmatic study has revealed the clinical features of pernicious anemia. It will help the clinician treat such patients efficiently to avoid morbidity and mortality.

INTRODUCTION

The term "pernicious anemia" is an anachronism. It is observed from the era when treatment had not yet been discovered and disease was fatal, but it was believed to be an autoimmune disorder that affects. The production of intrinsic factor by the gastric mucosa is impaired because vitamin B12 facilitates its transport to the terminal ileum for absorption, and impaired production leads to vitamin B12 deficiency and megaloblastic anemia. Intrinsic factors that cause megaloblastic anemia include folic acid deficiency, altered PH in the small intestine, and a lack of absorption of B12 complexes in the terminal ileum. Thus, pernicious anemia must be differentiated from the other disorders that interfere with the absorption and megaloblasticity of vitamin B12 deficiency.[1-3] The clinical onset of pernicious anemia is usually insidious and vague. The classic triad of weakness, soreness of tongue, and parasthesia may be elicited but is usually not the chief symptom complex. Hence, an attempt is made to evaluate the neurological and clinical symptoms and problems to rule out the manifestation of pernicious anemia in females because females are often victims of all vitamin deficiencies.[4,5]

MATERIALS AND METHODS

85 (eighty-five) adult female patients aged between 20-55 years who were regularly visited to JIIU's Indian Institute of Medical Sciences and Research (IIMSR) in Warudi Badnapur (dist), Jalna Maharashtra-431202 were studied.

Inclusive Criteria

The female patients diagnosed with deficiency of B12 or pernicious anemia and given written consent for study were selected for study.

Exclusion Criteria

The patients are already on treatment. Patients not willing to consent and those with foliate deficiency were excluded from the study.

Method: Every patient was examined thoroughly for detailed history, demographics, clinical features, and associated co-morbidities. vitamin B12 and routine blood examinations were carried out in both OPD and indoor patients. Other relevant investigations, such as neuro-imaging and electro-physiological studies, were performed if necessary. In patients with a normal vitamin B12 level but clinically suspected to have undergone bone narrow examination, megaloblastic changes in bone marrow were ruled out. The diagnosis was established by one or both of the of the following: (i) low serum vitamin B12 level (< 145 pg/ml) assessed by the chemilumine scene (CLIA) method; (ii) bone narrow aspiration showing megaloblastic changes. Mini-mental status (MMSE) was to study dementia.

Intra muscular B12 injections (cynacobalmin 1000 micrograms) were given daily for 2 weeks, followed by once a week for 4 weeks, and then monthly for a long time.

ISSN (O): 2687-5365; ISSN (P): 2753-6556

The duration of study was from December 2022 to May 2024.

Statistical analysis: Clinical parameters, i.e., neurological signs, clinical symptoms, and clinical problems, were studied and classified by percentage. The statistical analysis was done in SPSS software.

RESULTS

[Table 1] Study of neurological signs in vitamin B12 deficiency in female patients Touch 15 (17.6%) upper limb, 10 (11.7%) in lower limb (LL), 27 (31.7%) reflex, 10 (11.7%) in UL, 17 (20%) in LL, 23 (27%) position, 9 (10%) in UL, 14 (16.4%) in LL, 20 (23.5%) vibration, 20 (23.5%), 8 (9.4%) in UL, and 12 (14.1%) in LL.

[Table 2] Clinical symptoms in female patients with vitamin B12 deficiency: 17 (20%) chronic headache, 9 (10.5%) palpation, 28 (32.9%) giddiness, 11 (12.9%) altered bowel habit, 15 (17.6%) tingling, and 5 (5.88%) ataxic gait.

[Table 3] Clinical problems observed in deficiency of vitamin B12 in female patients: 35 (41.1%) pallor, 22 (25.8%) stomatatis, 15 (17.6%) glossitis, and 13 (15.2%) hyperpigmentation of the skin.

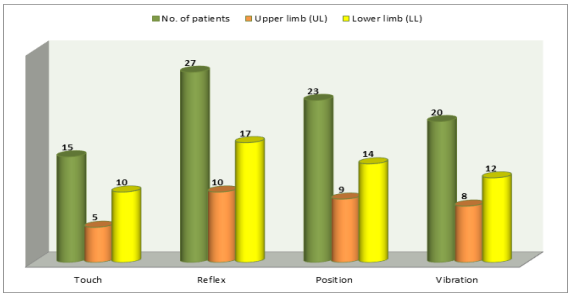

Figure 1: Study of Neurological signs in Vitamin B12 deficiency female patients

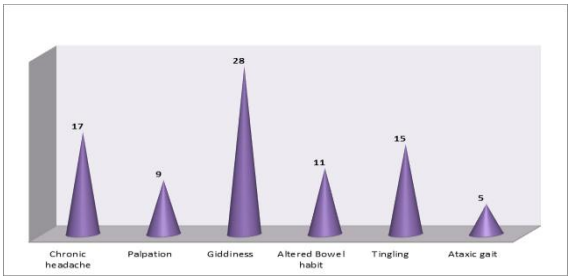

Figure 2: Clinical symptoms in female patients with vitamin B12 deficiency

Table 1: Study of Neurological signs in Vitamin B12 deficiency female patients. No. of patients: 85.

Sl. No	Neurological signs	No. of patients		Upper limb (UL)		Lower limb (LL)	
		No	%	No	%	No	%
1	Touch	15	17.6	5	5.8	10	11.7
2	Reflex	27	31.7	10	11.7	17	20
3	Position	23	27	9	10.5	14	16.4
4	Vibration	20	23.5	8	9.4	12	14.1

Table 2: Clinical symptoms in female patients with vitamin B12 deficiency. No. of patients: 85

Sl. No	Clinical symptoms	No. of patients	Percentage (%)
1	Chronic headache	17	20
2	Palpitation	9	10.5
3	Giddiness	28	32.9
4	Altered Bowel habit	11	12.9
5	Tingling	15	17.6
6	Ataxic gait	5	5.88

Table 3: Clinical problems observed in deficiency of vit-B12 in Female patients. No. of patients: 85

Sl. No	Clinical problems	No. of patients	Percentage (%)
1	Pallor	35	41.1
2	Stomatatis	22	25.8
3	Glossitis	15	17.6
4	Hyper pigmentation of skin	13	15.2

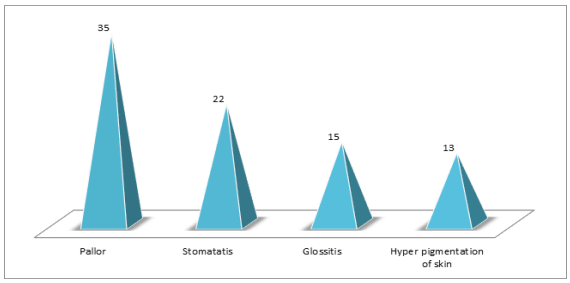

Figure 3: Clinical problems observed in deficiency of vit-B12 in Female patients

DISCUSSION

Present study of pernicious anemia in females of western Maharashtra. In the neurological signs studies, the highest sign was 27 (31.7%), followed by 23 (27%) position, 20 (23.5%) vibration, and the least was 15 (17.6%) touch [Table 1]. In the study of clinical symptoms, 28 (32.9%) giddiness was observed, and the least was 5 (5.8%) ataxic gait [Table 2]. Study of clinical problems in pernicious anemia patients. The highest problem was 35 (41.1%) pallor, followed by stomatatis, 15 (17.6%) glossitis, and 13 (15.2%) hyperpigmentation of the skin [Table

ISSN (O): 2687-5365; ISSN (P): 2753-6556

3]. These findings are more or less in agreement with previous studies.[5-7]

It is reported that the majority of pernicious anemia patients had hemoglobin levels less than 9/gm severe atrophic gastritis, d-xylose malabsorption, and jejunal mycoses that showed partial villus atrophy in the small intestine.[8] Histamine fast achlorhydria, absent or sub-normal intrinsic factor content of gastric juice, poor radioactive vit-B12 absorption improving with intrinsic factor, presence of partial cell antibody (PCA) and intrinsic factor antibody in serum and/or gastric was observed in the majority of patients.[9] In a pernicious anemia, failure of correction of vit-B12 mal-absorption with intrinsic factors may be due to either the presence of an intrinsic factor antibody (IFA) or saliva or to damage to the ileal musculature.

In developing countries like India, the definite diagnosis of pernicious anemia is difficult to establish because (a) macrocytic anemia, megaloblastic bone marrow, and low serum via-B12 levels are frequently not observed due to the common practice of injecting vit-B12 and B complex as tonics for any illness. (b) Improvement of radioactive vit-B12 absorption when administered with intrinsic factor may not be observed due to the prevalence of occult or manifest ileal lesions in tropical sprue. (c) Facilities for radioactive vitamin B12 absorption and detection of gastric antibodies are limited.[10] It is also reported that atrophy of the gastric mucosa was not observed in patients from western countries, but the incidence of partial cell antibodies is comparable in patients from western countries, but intransic factor antibodies (IFA) are significantly lower in western candy patients. Thyrotoxosis, diabetes mellitus, and iron deficiency were similar to Indian studies. In India, pernicious anemia is mainly due to nutritional deficiency, but in western countries, it is due to genetic factors.[11] It is also presumed that pernicious anemia is also due to environmental factors. It may damage the gastric mucosa or have a have a specific inherited effect, perpetuating damage to the gastric mucosa. The probable reason could be environmental factors responsible for the lesion of atrophic gastritis. It is also confirmed that the patient's atrophic gastritis and severely impaired vitamin B12 excretion should not be classified as pernicious anemia.[12]

CONCLUSION

The present study of pernicious anemia in females with different neurological symptoms, clinical problems, and symptoms is useful to clinicians to treat such patients, but this study demands further nutritional, environmental, genetic, and patho-physiological study because the exact mechanism and process of intrinsic factors that cause pernicious anemia are still unclear.

Limitation of study: Owing to the tertiary location of the research center, the small number of patients, and the lack of the latest techniques, we have limited findings and results.

REFERENCES

1. Kumar S: Vitamin B12 deficiency presents with an acute reversible extrapyramidal syndrome.
2. Klee GG: Calamine and foliate evaluation measurement of methylamine acid and hydroxycystine vs. vitamin B12 and foliate. Clin. Chem. 2000, 46 (13); 12–17.
3. Kiburtx KD, Giang d: Abnormal vitamin B12 metabolize in human immune deficiency virus infection Arch. Meurol. 1991, 48 (3); 12–14.
4. Kumar S: Recurrent seizes an unusual manifestation of vitamin B12 deficiency Neurol India 2004, 52; 507-9.
5. Robert Muir: Textbook of Pathology, 2nd edition, 1929 Edward Arnold and Company publication, London, 621-29.
6. Healton EB, Savage DG: Neurological aspects of cobalmine deficiency medicine (Baltimore), 1991, 70; 229–40.
7. Hissing LT, Whaling A: Further evidence on effects of vitamin B12 and foliate levels on episodic memory functioning population Biol. Psychiatry 1991, 45, 14; 72–80.
8. Mayes PA: Structure and function of water-soluble vitamins. Murray R.K. Grimnor, D.K. Harpers Biochemistry, 25th edition 2000, 635-7 Stamford, application, and large publication.
9. Carmel R: Re-assessment of the relative prevalence of antibodies to gastric parietal cells and intrinsic factors in patients with pernicious anemia Clin. Exp. Immune. 1992, 89; 74–7.
10. Jayaram N, Rao MG: Vitamin B12 levels and psychiatry symptomatology J. Neuropsychiatry Clin. Neuro Science 2013, 25; 150–2.
11. Senol MG, Somn Mez G: Reversible myelopathy with vitamin B12 deficiency, Singapore Med. J. 2008, 49; 330–2.
12. Vagar S, Shackelford KB: Pernicious anemia 2024J. an. (QX Medline) Full text.

ISSN (O): 2687-5365; ISSN (P): 2753-6556