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Vitamin B12 And Folate Deficiency at Mercy Hospital Toledo: Studying Characteristics and Replacement Compliance for Deficient Patients

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Abstract

Background: Vitamin B12 and folate deficiencies are easily treatable deficiencies if identified on time. We report a study done in admitted patients in Mercy Hospital in Toledo, Ohio where we studied the prevalence of vitamin B12 and folate deficiencies in the area and looked at the compliance of physicians ordering Vitamin b12 and folate replacements for deficient levels.

Objective: The study aims to identify the patients with vitamin b12 and folate deficiencies and the physician compliance to identify the deficient levels and ordering appropriate replacement. Studying the prevalence of anemia, macrocytosis, thrombocytopenia, iron saturation in vitamin deficient patients.

Methods and materials: Retrospective, observational cohort study was designed to identify patients with vitamin b12, and/or folate deficiency who were diagnosed during the hospitalization and who received adequate treatment on discharge.

Results: Total of 295 cases were identified with Vitamin B12 and/or folate deficiencies in inpatient setting in a 6-month period. 12.5% cases had Vit B12 deficiency. 21% cases had Folate deficiency. 31.9% cases have either of the two deficiencies and only 1.7% patients have both vitamin deficiencies. 46% of patients did not get appropriate B12 replacement and 47% patients did not get folate replacement at the time of discharge. In folate deficient patients, 29% were thrombocytopenic, 52% were anemic, 52% had concomitant low iron saturation, 33% had macrocytosis. In vitamin B12 deficient patients, 16% were thrombocytopenic, 35% were anemic, 71% had concomitant low iron saturation, 22% had macrocytosis.

Conclusion: There was a definite gap between the deficiency and replacement orders for the deficient patients. Many of patients admitted in the hospital have more critical issues ongoing and the deficient levels can get ignored with more pressing issues. Multiple physicians taking part in the care of patient can also affect the decisions made at the discharge.

Keywords: Vitamin B12; folate deficiency; mercy hospital; Toledo; replacement; compliance; deficient patients

Background And Significance

Vitamin B12 and folic acid are water soluble vitamins. These deficiencies are often considered together and are less common in developed countries. These vitamins play a key role in hematopoiesis. Methylcobalamin and 5-deoxyadenosylcobalamin are the metabolically active forms of vitamin B12. However, two others form, hydroxycobalamin and cyanocobalamin, become biologically active after they are converted to methylcobalamin or 5-deoxyadenosylcobalamin [1]. Folate is also called Vitamin B9 and folic acid, technically, the vitamin is found in nature as folate, while folic acid is the synthetic form used therapeutically; it is an oxidized, water-soluble form that does not exist in nature [2]. In a report from the National Health and Nutrition Examination Survey (NHANES), the prevalence of vitamin B12 deficiency was 2.6 to 7.5 percent depending on the year [3]. A 2014 examination of folate testing performed on outpatients in a single center in Boston, Massachusetts (United States) found folate deficiency in only 47 of 84,187 (0.06 percent); another 166 (0.2 percent) had low-normal values (3.0 to 3.9 ng/mL) [4]. Classical findings include anemia, macrocytosis, variable neurological abnormalities including cognitive slowing, neuropathy, paresthesias, gait issues to name a few. Timely identification of subtle signs helps to prevent long term complications of these vitamin deficiencies. Main objectives of the study were as follows:

1. Identify the rate of vitamin deficiencies in admitted patients in Mercy Hospital in Toledo, Ohio.
2. Identifying the compliance of physicians ordering Vitamin b12 and folate replacements for deficient levels.

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3. Studying the prevalence of anemia, macrocytosis, thrombocytopenia, iron saturation in vitamin deficient patients.

Methods

Study Design

This retrospective cohort study was approved by the Institutional Review Board (IRB) for Mercy Health System, Toledo with a waiver of consent and a waiver of Protected Health Information.

Subjects And Inclusion Criteria

Adult patients admitted in the hospital with abnormal Vitamin B12 and folate levels were screened in a time period of 6 months. Charts were then screened for patients with low Vitamin B12 and folate levels. Discharge medications were reviewed as well.

Exclusion Criteria

Subjects less than 18 years of age and pregnant females were excluded from the study.

Primary And Secondary Outcomes

The primary outcome was prevalence of Vitamin B12 and Folate in admitted adult patients. The secondary outcomes studied was compliance of replacements ordered at the time of discharge and prevalence of anemia, macrocytosis, thrombocytopenia, iron saturation in vitamin deficient patients.

Main Results

Total of 295 cases were identified with Vitamin B12 and/or folate deficiencies in inpatient setting in 6-month period of a tertiary hospital in Toledo, Ohio. Per hospital lab data vitamin B12 levels were flagged when B12 levels were lower than 232, folate levels were flagged when lower than 4.8. Out of total 295 cases studied 12.5% cases had Vit B12 deficiency, 21% cases had Folate deficiency. 31.9% cases have either of the two deficiencies and only 1.7% patients have both vitamins deficient. These deficient patients were further evaluated, and associations were studied between deficiency patients and thrombocytopenia, anemia, low iron saturation, macrocytosis. Mean age identified for B12 deficient patient was 66 years. Out of the 37 deficient patients, 16% patients had thrombocytopenia with platelet count of <150, 35% patients were anemic with hemoglobin level of 10gm/dl, 71% patients had iron saturation less than 20%, 22% had macrocytosis with MCV>100 fl/cell. Only 54% patients with B12 deficiency received replacement on discharge. Mean age identified for folate deficient patient was 63 years. Out of the 62 deficient patients, 29% patients had thrombocytopenia with platelet count of <150, 52% patients were anemic with hemoglobin level of 10gm/dl, 52% patients had iron saturation less than 20%, 33% had macrocytosis with MCV>100 fl/cell.

Statistical Methods

Results are shown as number of cases and percentages. Data were analyzed with SAS v9.4.

Table 1: B12 and Folate Deficiency among 295 Cases

	Number of Cases	Percentage of Cases
B12 Deficient (<232)	37	12.5%
Folate Deficient (<4.8)	62	21.0%
Both B12 and Folate Deficient	5	1.7%
B12 and/or Folate Deficient	94	31.9%

Table 2: Characteristics of B12 Deficient or Folate Deficient Cases

	B12 Deficient	Folate Deficient
No. Cases	37	62
Age, mean years (SD)	66 (14)	63 (15)
Was B12 Replacement Given?		
Yes	20 (54%)	4 (7%)
No	17 (46%)	57 (93%)
Type of B12 Replacement Given		
Oral	17 (46%)	4 (7%)
IM	3 (8%)	0 (0%)
None	17 (46%)	57 (93%)
Was Folate Replacement Given?		
Yes	7 (19%)	33 (53%)
No	30 (81%)	29 (47%)

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Low Platelet (<150)	6 (16%)	18 (29%)
Low Hemoglobin (<10 g/dl)	13 (35%)	32 (52%)
Low Iron Saturation (<20%)	17 (71%)	24 (52%)
High MCV (>100 fl/cell)	8 (22%)	20 (33%)

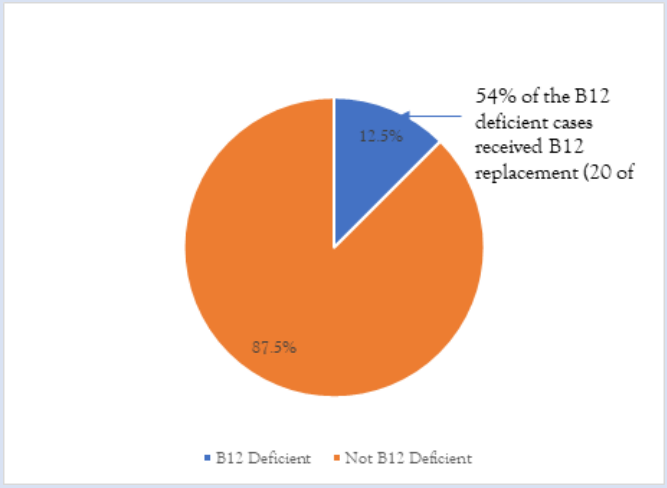

Figure 1: B12

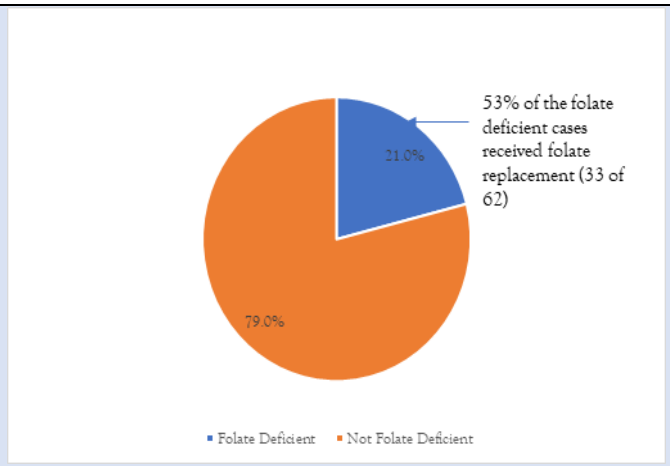

Figure 2: Folate

Discussion

Vitamin B12 is a water-soluble vitamin which is absorbed in terminal ileum. It is obtained from dietary sources like dairy products, eggs and meat [5]. Adult deficiency commonly present with anemia, neurological dysfunction and rarely glossitis. Cobalamin deficiency rarely requires instant therapy and the damage is mostly reversible but treatment should be started expeditiously for severe neurologic symptoms due to the risk of irreversibility (eg, extensive sensory defects, gait disturbances, mental changes) [6]. Diagnostic criteria include a serum cobalamin <148 pmol/l (200 ng/l) in the presence of signs and symptoms and/or hematological indices of vitamin B_{12} deficiency, or a serum cobalamin <148 pmol/l in conjunction with elevated serum homocysteine or methylmalonic acid (MMA) [7]. Food-bound cobalamin malabsorption is the commonest cause of low serum cobalamin levels, while pernicious anemia accounts for most cases of severe vitamin B_{12} deficiency [8]. Some common causes of B12 deficiency include low dietary intake eg in patients with vegetarian diet, chronic alcoholics and elderly age; autoimmune diseases like pernicious anemia and Sjogren syndrome; food bound cobalamin mal-absorption like in atrophic or chronic gastritis; Cobalamin malabsorption like celiac disease, achlorhydria and crohn disease; drugs like metformin and PPI; pregnant females and use of oral contraceptives [9].

Folic acid (FA), also termed folate, is an essential vitamin used in the biosynthesis of nucleotides, amino acids, neurotransmitters, and certain vitamins [10]. Folate is essentially important in development of nervous system. Humans cannot synthesize folate in body and depend on external supplementation to meet the demands. Folate deficiency has been linked

with an increased risk of neural tube defects, cardiovascular disease, cancer and cognitive dysfunction [10]. Deficiencies of folate can occur for many reasons, including reduced intake, increased metabolism, and/or increased requirements as well as through genetic defects. The effects of folate deficiency include hyper homocysteinemia, megaloblastic anemia, and mood disorders [12]. The major food sources of folate include cooked dried beans, leafy green vegetables, and fortified cereals [13]. Vitamin B12 and folate deficiencies are easily treatable when diagnosed on time and managed appropriately. A blood film showing oval macrocytes and hyper segmented neutrophils in the presence of an elevated MCV may alert the clinician to the presence of underlying cobalamin or folate deficiency [14]

Conclusion

In our study we saw the prevalence of Vitamin B12 and Folate deficiencies. Study showed the average age was 63-66 years for the deficiencies. There was a significant gap between the physician compliance regarding replacement orders and was a useful information to work on as a quality improvement project.

References

1. Institute of Medicine (US) Standing Committee on the Scientific Evaluation of Dietary Reference Intakes and its Panel on Folate, Other B Vitamins, and Choline. (1998). Dietary Reference Intakes for Thiamin, Riboflavin, Niacin, Vitamin B6, Folate, Vitamin B12, Pantothenic Acid, Biotin, and Choline. Washington, DC: *National Academies Press*.
2. Scaglione, F., & Panzavolta, G. (2014). Folate, folic acid and 5-methyltetrahydrofolate are not the same thing. *Xenobiotica*, 44(5):480.
3. Bird, J. K., Murphy, R. A., Ciappio, E. D., & McBurney, M. I. (2017). Risk of deficiency in multiple concurrent micronutrients in children and adults in the United States. *Nutrients*, 9(7):655.
4. Theisen-Toupal, J., Horowitz, G., & Breu, A. (2014). Low yield of outpatient serum folate testing: Eleven years of experience. JAMA Internal Medicine, 174(10):1696-1697.
5. Antony, A. C. (2003). Vegetarianism and vitamin B-12 (cobalamin) deficiency. The American *Journal of Clinical Nutrition*, 78(1):3-6.
6. Carmel, R. (2008). How I treat cobalamin (vitamin B12) deficiency, 112(6):2214-2221.
7. Devalia, V., Hamilton, M. S., & Molloy, A. M.; The British Committee for Standards in Haematology. (2014). Guidelines for the diagnosis and treatment of cobalamin and folate disorders. *Briti Journ of Haematology*, 166:496-513.
8. Shipton, M. J., & Thachil, J. (2015). Vitamin B12 deficiency - A 21st century perspective. Clinical Medicine (London, England), 15(2):145-150.
9. Jajoo, S. S., Zamwar, U. M., & Nagrale, P. (2024). Etiology, clinical manifestations, diagnosis, and treatment of cobalamin (vitamin B12) deficiency. *Cureus*, 16(1):e52153.
10. Sijilmassi, O. (2019). Folic acid deficiency and vision: A review. Graefe's Archive for Clinical and *Experimental Ophthalmology*, 257(8), 1573-1580.
11. Scaglione F, Panzavolta G. (2014). Folate folic acid and 5-methyltetrahydrofolate are not the same thing. Xenobiotica; The Fate of Foreign Compounds in Biological Systems, 44(5):480-488.
12. Donnelly, J. G. (2001). Folic Acid. Critical Reviews in Clinical Laboratory Sciences, 38(3):183-223.
13. Subar, A. F., Block, G., & James, L. D. (1989). Folate intake and food sources in the US population. *Amer Jour of Clin Nutr*, 50(3):508-516.
14. Devalia, V., Hamilton, M. S., & Molloy, A. M.; (2014). The British Committee for Standards in Haematology. Guidelines for the diagnosis and treatment of cobalamin and folate disorders. *British Journal of Haematology*, 166:496-513.

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