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Status of Vitamin B12 Deficiency in the Elderly Chinese Community People

Yuhui Wang, Yunyun Zheng, Fang Yan, Wenbo Zhang

Department of Neurology, Shanghai Punan Hospital, Shanghai, China
Email: yhwangch@sina.com

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Abstract

This study was aimed to investigate the vitamin B12 deficiency prevalence and symptoms in elderly people lived in a community of Shanghai, China. A total number of 962 elderly people resided in Shanghai community were recruited in the present study. They were 60 years and older, and the average age was 76.38 ± 13.68 years old. Information on previous and present diseases, currently prescribed and over-the-counter medication, and the presence or absence of symptoms relating to vitamin B12 deficiency were obtained by questionnaire. The levels of serum vitamin B12, folate and homocysteine (Hcy) were estimated. The patients with vitamin B12 deficiency were screened. The results of symptoms and positive signs of neurological examination were compared between subjects with or without vitamin B12 deficiency. The results showed that vitamin B12 deficiency was found in 130 persons (13.53% of the total subjects), with an increase in incidence with aging, only 10% of the vitamin B12 deficient subjects had megaloblastic anemia. The reported symptoms of vitamin B12 deficient subjects included fatigue, memory decline, dizziness, unsteadily walking in the darkness and hypopallesthesia. In conclusion, vitamin B12 deficiency was remarkably common in Chinese elderly people, with various and atypical clinical manifestations, and the neurological symptoms were more common than those of megaloblastic anemia.

Keywords

Aging, Community, Vitamin B12 Deficiency

1. Introduction

Vitamin B12 (cobalamin) deficiencies are no longer a rare disease but a relatively common disease, whose prevalence increases with aging [1] [2]. Vitamin B12 deficiency is a common problem worldwide, and some researchers regard vitamin B12 as a critical vitamin in old population [3]. Vitamin B12 deficiency occurs fre-

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quently among elderly patients (about 5% - 40% of the aged population), depending on the diagnostic criteria used [4]-[6], but it is often unrecognized or not investigated due to the subtle clinical manifestations. However, vitamin or nutritional deficiency usually leads to severe disorders, particularly the neuropsychiatric and hematological complications. In the previous study, we find a prevalence of 17.91% of vitamin B12 deficiency in elderly inpatients in neurology department in Shanghai, China [4]; there is little information about the status of vitamin B12 deficiency in Chinese community people, especially the prospective study. The aim of this study is to investigate the status of vitamin B12 deficiency in elderly people, and to understand the neurological symptoms.

2. Methods

2.1. Study Populations

The study population was randomly selected from the permanent residents aged 60 and over who lived in Nanmatou community located in Pudong New Area of Shanghai, China, by the end of December 31, 2012. The age and sex distributions in this community are similar to those of the Shanghai population. All the participants were diagnosed with no severe hepatic or renal dysfunction, and without any B-vitamin supplements for 3 months. Altogether 1108 persons were included in the study, 62 were hospitalized, 84 refused to participate the study, 962 persons finally finished the study. Informed consent of the participation was given by all the patients or their descendents if the participants were demented.

2.2. Questionnaire Investigation

Information on previous and present diseases, ongoing prescribed and over-the-counter medication, and the presence or absence of symptoms relating to vitamin B12 deficiency was obtained by questionnaire. An interview was performed by a general practitioner, during which the questionnaire responses were verified. For persons with dementia or memory disturbances, permission for inclusion was obtained from their children, who also supplemented some of the information that the respondent could not provide him or herself. In addition, information from medical records was used in these instances.

The symptoms were grouped into gastrointestinal, neurological, psychiatric and miscellaneous symptoms. Gastrointestinal symptoms included reduced appetite, mouth angle stomatitis or tongue mucosa atrophy, nausea, vomiting, diarrhea and heartburn. Neurological symptoms included numbness, paraesthesias in the feet or fingers, walking difficulties, dizziness, impaired touch perception, and the psychiatric symptoms included irritability, memory impairment, depression and being easily moved to tears. Miscellaneous symptoms included fatigue, shortness of breath, palpitations and ankle oedema.

2.3. Physical Examination

A physical examination was performed, including oral cavity examination, where special attention was paid to atrophy of the tongue mucosa and mouth angle stomatitis. The biceps, brachioradial, triceps, patellar and achilles reflexes were tested bilaterally at standardized sites. The responses were recorded as 0 - 3, where 0 meant no response and 3 meant a very strong one. For this report, the mean value of the bilateral responses was computed, rounded off to the nearest integer.

2.4. Cognitive Function Assessment

A Mini Mental State Examination (MMSE), 30 questions for total of 30 point, was performed to assess the cognitive function. Possible scores were 0 - 30, with low scores indicating cognitive impairment. In the analyses MMSE scores were used as a continuous variable, i.e. no reference value indicating dementia was used.

2.5. Hematologic Biomarkers Detection

Elbow venous blood samples were collected from the right arm after overnight fasting. Blood was collected in vacuum tubes containing EDTA for blood routine test and in plain tubes for detecting vitamin B12, folate and total homocysteine (tHcy) levels. Blood routine test, including full blood cell count, mean corpuscular volume (MCV) and hemoglobin, was carried out using standard methods by blood cytoanalyzer. Serum vitamin B12 and folate concentrations were measured using the chemilumimescent microparticle immunoassy (CMIA) technolo-

gy (Architect B12 assay, Architect Folate assay), with reference ranges of 189 - 883 pg/mL for vitamin B12 and 2.7 - 34 ng/mL for folate. Serum tHcy level was measured using enzymatic cycling assay (Beijing Strong Biotechnologies, Inc) with a reference range of 10 - 15 μmol/L. Patients with vitamin B12 concentrations lower than 189 pg/mL and tHcy concentrations higher than 15 μmol/L were identified as with vitamin B12 deficiency. Serum folate concentrations lower than 2.7 ng/mL were identified as folate deficiency.

2.6. Statistical Analysis

Continuous variables were summarized as means and standard deviations. One-way ANOVA was applied for multiple comparisons. The differences between the groups were tested with the Tamhane-T2 test for quantitative data and the chi-square test for categorical data. Only two-tailed tests were used. $P < 0.05$ was considered as statistically significant.

3. Results

3.1. Prevalence of Vitamin B12 Deficiency

Among the 1108 subjects, 62 were hospitalized, 84 refused to participate the study, leaving 962 (454 male and 508 female), aged 60 - 97 years, with the mean age of 76.38 ± 13.68 years, finished the study. As shown in **Table 1** and **Table 2**, vitamin B12 deficiency was identified in 130 persons (13.53% of the total subjects), with an increasing tendency with aging. Besides, the prevalence of vitamin B12 deficiency seemed higher in female than in male patients, however, there was no statistically significant difference ($P > 0.05$).

3.2. Clinical Characteristics of the Subjects

As shown in **Table 3**, the mean age of the participants with low vitamin B12 levels (n = 130) was significantly

Table 1. Prevalence of vitamin B12 deficiency in studied participants.

Age (years)	Number (n)			VitaminB12 deficiency [n (%)]		
	Total	male	femal	male	female	Total
60 - 64	124	65	59	6 (9.2%)	6 (10.2%)	12 (9.7%)
65 - 69	102	49	53	5 (10.2%)	6 (11.3%)	11 (10.8%)
70 - 74	169	83	86	9 (10.8%)	11 (12.8%)	20 (11.8%)
75 - 79	204	93	111	11 (11.8%)	17 (15.3%)	28 (13.7%)
80 - 84	196	92	104	13 (14.1%)	17 (16.3%)	30 (15.3%)
≥85	167	72	95	12 (16.7%)	18 (18.9%)	30 (17.96%)
Total	962	454	508	55 (12.1%)	75 (14.8%)	130 (13.53%)

Table 2. Blood vitamin B12, folate, Hcy and MCV in vitamin B12 deficient subjects (mean ± SD).

Age (years)	n	Folate (ng/mL)	VitB12 (pg/mL)	Hcy (μmol/L)	Red Blood Cells ($\times 10^{12}$/L)	HGB (g/L)	MCV (fL)
60 - 64	12	4.92 ± 2.53	151.12 ± 32.42	36.24 ± 18.46	4.36 ± 0.55	133.69 ± 11.36	91.56 ± 4.87
65 - 69	11	5.34 ± 4.37	148.23 ± 21.79	36.14 ± 19.76	4.87 ± 0.48	135.97 ± 23.89	86.67 ± 7.59
70 - 74	20	4.92 ± 3.09	142.67 ± 26.35	36.09 ± 12.35	4.31 ± 0.67	130.82 ± 17.33	92.39 ± 3.98
75 - 79	28	4.52 ± 3.14	139.78 ± 29.98	37.28 ± 12.49	4.13 ± 0.48	125.48 ± 12.91	93.21 ± 5.37
80 - 84	30	4.12 ± 2.89	137.94 ± 32.47	41.07 ± 21.72	4.05 ± 0.47	124.03 ± 15.88	92.88 ± 5.75
≥85	30	4.69 ± 3.84	135.26 ± 37.24	36.94 ± 16.99	3.98 ± 0.55	119.39 ± 15.84	92.17 ± 6.44
Total	130	4.58 ± 3.26	141.16 ± 31.89	37.12 ± 16.85	4.18 ± 0.59	125.89 ± 16.72	92.46 ± 5.78

Hcy: homocysteine; Vit B12: vitamin B12; HGB: hemoglobin; MCV: mean corpuscular volume; SD: standard deviation.

Table 3. Clinical characteristics of the subjects with or without vitamin B12 deficiency.

	Total (n = 962)	Normal VitB12 Levels (n = 832)	VitB12 deficiency (n = 130)
Medical history			
Age (years) (mean ± SD)	76.38 ± 13.68	76.71 ± 7.34	78.35 ± 8.13*
Sex: male [n (%)]	454 (47.19)	399 (47.96)	55 (42.31)
Number of chronic diseases[a] (mean ± SD)	1.64 ± 0.73	1.41 ± 0.52	1.79 ± 0.85
Symptoms			
Memory decline [n (%)]	258 (26.82)	209 (25.12)	49 (37.69)*
Fatigue [n (%)]	201 (20.89)	159 (19.11)	42 (32.31)*
Unsteadily walking in the darkness [n (%)]	194 (20.17)	158 (18.99)	36 (27.69)*
Altered sensation in feet on walking [n (%)]	147 (15.28)	125 (15.02)	22 (16.92)
Paresthesia [n (%)]	154 (16.01)	130 (15.62)	24 (18.46)
Dizziness [n (%)]	185 (19.23)	155 (18.63)	30 (23.08)
Neurological signs			
Ankle tendon jerk (mean ± SD)	1.58 ± 0.89	1.86 ± 0.77	1.36 ± 0.69
Hypopallesthesia [n (%)]	240 (24.95)	182 (21.88)	45 (34.62)#
Cognitive impairment			
MMSE (mean ± SD)	27.95 ± 6.59	28.41 ± 6.98	26.51 ± 5.95#
Hematological findings			
HGB (g/L) (mean ± SD)	129.57 ± 15.74	130.41 ± 16.24	125.37 ± 16.88#
MCV (fl) (mean ± SD)	91.61 ± 4.89	91.37 ± 4.39	91.97 ± 5.62
HGB < 120 (g/L) [n (%)]	143 (14.86)	108 (12.98)	35 (26.92)#
MCV>95 (fl) [n (%)]	121 (12.58)	78 (9.37)	43 (33.08)#
MCV>95 (fl) + HGB < 120 (g/L) [n (%)]	19 (1.98)	6 (0.72)	13 (10)#

[a]Chronic diseases: diabetes, hypertension, stroke, malignancy, vascular disease, arthritis/arthrosis, lung disease, and heart diseases.*$P < 0.05$, #$P < 0.01$ *vs*. persons with normal vitamin B12 levels.

higher than that of participants with normal B12 levels (n = 832) ($P = 0.04$), and the prevalence of vitamin B12 deficiency seemed higher in female than in male subjects, however, there was no significant difference in factors of gender and number of chronic diseases between the 2 groups. In comparison with the subjects with normal B12 levels, those with low level of serum vitamin B12 were more likely to experience memory decline, fatigue, dizziness, unsteadily walking in the darkness and hypopallesthesia. Moreover, the mean MMSE score of B12 deficient subjects was remarkably lower than that of subjects with normal B12 level. The vitamin B12 deficient persons exhibited significantly lower concentrations of hemoglobin, but also a slightly higher mean corpuscular volume ($P < 0.001$) than those with normal level of serum vitamin B12 did, however, only 10% of them had megaloblastic anemia.

4. Discussion

Vitamin B12 deficiency was previously considered as a rarely occurring but easily diagnosed disease, because of the dramatic findings of megaloblastic anemia. Recently, accumulating evidence has indicated that vitamin B12 deficiency occurs frequently among elderly people, who were usually unrecognized or not investigated because of the subtle clinical manifestations, and it is estimated to affect 5% - 40% of the aged people, depending

on the diagnostic criteria [5]. It is reported that the prevalence of vitamin B12 deficiency in the elderly population in developed country is 5% - 15% [5]-[7], with 12% being the old community residents [2] and up to 30% - 40% being the old hospitalized patients [8]. Similar results were reported in the developing countries [9]-[12]. In this study, we found that the prevalence of vitamin B12 deficiency in the elderly people resided in Shanghai community was 13.53%, which was lower than that of inpatient in neurological department [4]. We also found that the prevalence of vitamin B12 was increased with aging, and the same tendency was found in complicating folate deficiency with aging. The mean age of the persons with low serum vitamin B12 levels were a little older than that of the persons with normal B12 levels, implicating that vitamin B12 deficiency has a liability in the aged. It was considered that older average age of the female was a risk factor of higher prevalence of vitamin B12 deficiency.

Vitamin B12 is essential for human, which cannot be synthesized by humans and completely dependent on dietary sources [13]. Vitamin B12 is an essential cofactor and coenzyme mediating2 enzymatic reactions. One involves the conversion of methylmalonyl-coenzyme A (CoA) to succinyl-CoA using adenosyl-Cbl (Ado-Cbl) as a cofactor, the other involves the synthesis of methionine from homocysteine using methyl-Cbl as a cofactor [2]. Impairment in the latter reaction leads to defects in DNA synthesis and a disruption in megaloblastic maturation pattern, as well as the defective productions of choline and choline containing phospholipids, which are believed to be of primary importances in explaining the pathophysiological aspects of vitamin B12 and folate deficiencies [14] [15].

Vitamin B12 deficiency may be entirely asymptomatic or present with hematological and neuropsychiatric manifestations. The neurologic manifestations begin pathologically with demyelization, followed by axonal degeneration and eventually the irreversible damage due to the axonal death. The neurologic manifestations can be the initial presenting complaint, and the spinal cord, the brain, the optic nerves and the peripheral nerves may all be affected. Also, these impairments may be accompanied with myelopathy (subacute combined degeneration of spinal cord), ataxia, spasticity and abnormal gait, dementia, depression, acute psychosis, reversible manic and schizophreniform states (megaloblastic madness), cerebrovascular disease (homocystenemia is an independent risk factor for stroke), and neuropathy such as motor-sensory polyneuropathy (parasthesias, numbness and weakness), mononeuropathy (optic or olfactory) and autonomic neuropathy (impotence, urinary or fecal incontinence) [13]-[16]. The spinal cord is usually the first affected site and often exclusively. Visual impairment resulting from optic neuropathy may occasionally be the earliest or sole manifestation.

Because of the liver reserve and intestinal liver circulation of cobalamin, it may take decades to develop vitamin B12 deficiency, and clinically, the suspected patients with typical features will be examined. However, old people with low vitamin B12 concentrations rarely exhibit the classical features of macrocytic anemia and neuropathy. Instead, they present more commonly with the non-specific symptoms off atigue and memory decline that can be attributed to “oldage”. The uncertainty about the importance of vitaminB12 deficiency may be partly related to the limitations of the standard vitamin B12 assays. Thus, the early diagnosis of vitamin B12 deficiency, which is important in preventing permanent neurological damage [2] [3] [17], is often delayed. Low plasma vitamin B12 levels in most of the elderly asymptomatic patients are at the subclinical deficient status rather than the manifestation of physiological aging [8]. In this study, we found that almost all the vitamin B12 deficient patients lacked previous diagnosis, and only 10% of the patients had megaloblastic anemia. All the subjects with low vitamin B12 levels exhibited abnormal homocysteine metabolism. Moreover, vitamin B12 deficiency could induce unsteady walking steps in the darkness and hypopallesthesia. Other studies [18]-[20] have shown similar results. Higher rates of Vitamin B12deficiency were reported among vegetarians [21].

This study has some limitations such as the study population is not large enough, and the serum vitamin B12, rather than its metabolites levels were detected, which would underestimate the incidence of vitamin B12 deficiencies. Further studies need to be undertaken to screen the subclinical insufficiency patients by detecting metabolites of cobalamin and seek for their risk factors, determine whether early screening and subsequent treatment can prevent them from developing neurological dysfunctions.

5. Conclusion

In summary, vitamin B12 insufficiency is relatively common finding in elderly people, with an increased incidence with age. The majority of cases results in mild symptoms and is due to food-bound cobalaminmal absorption, while pernicious anaemia is much rarer nowadays [22]. Vitamin B12 deficiency is often unrecognized or

not diagnosed promptly due to the subtleclinical manifestations. However, because of the potential seriousness of the complications (particularly neuropsychiatric and hematological complications), it is especially important to screen out and treat the vitamin B12 deficient patients, and decrease the risk of B12 deficiency-associated disabilities in elderly people. Therefore, we suggest that early diagnosis of vitamin B12 deficiency should be encouraged and vitamin B12 supplements should be given to the B12 deficient people to attenuate or prevent the neurological impairment, and to improve their life qualities. Conclusively, vitamin B12 deficiency is remarkably common in Chinese elderly people, with various and atypical clinical manifestations, and the neurological symptoms are more common than those of megaloblastic anemia.

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