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·Original Article·

An investigation of vitamin B12 deficiency in elderly inpatients in neurology department

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Abstract: Objective To investigate the status of vitamin B12 deficiency in elderly inpatients in the department of neurology. **Methods** A total number of 827 patients in the department of neurology of Shanghai Punan hospital, from March 2007 to July 2008, were employed in the present study. They were 60 years or older, and the average age was 77.1±7.5 years old. All the patients were diagnosed with no severe hepatic or renal dysfunction, without any usage of vitamin B12 during the previous 3 months before the detection. The levels of serum vitamin B12, folate and homocysteine (Hcy) were evaluated. The patients with vitamin B12 deficiency were screened. The resulting symptoms, positive signs of neurological examination, and the neuroelectricphysiological results were compared between patients with or without vitamin B12 deficiency. **Results** Vitamin B12 deficiency was found in 163 patients (19.71% of the total patients), and was more prevalent in female than in male patients, also with increased incidences with aging. Patients with low levels of serum vitamin B12 exhibited higher rate of gastrointestinal diseases, while only 9.82% of the vitamin B12 deficient patients had megaloblastic anemia. Symptoms of vitamin B12 deficiency included unsteadily walking in the darkness and hypopallesthesia, and some chronic diseases such as cerebral ischemia, hypertension, Parkinson's disease (Parkinsonism), diabetes mellitus and coronary heart disease. Most of the vitamin B12 deficient patients had neuroelectricphysiological abnormalities. **Conclusion** Vitamin B12 deficiency is remarkably common in elderly patients in neurology department, with various and atypical clinical manifestations, and the neurological symptoms are more common than megaloblastic anemia symptoms.

Keywords: aging; elderly inpatients; neurology; vitamin B12 deficiency

1 Introduction

Vitamin B12 deficiency, also named as cobalamin deficiency, was previously considered as a rarely occurring disease and was easily diagnosed because of the dramatic manifestations of megaloblastic anemia. Recently, accumulating evidences have shown that vitamin B12 deficiency is a relatively common disease, whose prevalence increases with aging[1,2]. Vitamin B12 deficiency is a worldwide problem, and some researchers regard vitamin B12 as a critical vitamin in old population[3]. Vitamin B12 deficiency occurs frequently 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. What's more, vitamin or nutrition deficiency usually leads to severe disorders, particularly the neuropsychiatric and hematological complications. However, there is little information of vitamin B12 deficiency in Chinese people, especially the prospective study. The present study aims to investigate the status of vitamin B12

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deficiency in elderly inpatients and to understand the neurological symptoms.

2 Subjects and methods

2.1 Subjects A total number of 827 patients in the department of neurology of Shanghai Punan hospital, from March 2007 to July 2008, were employed in the present study. They were 60 years or older, and the average age was 77.1±7.5 years old. All the patients in the present study were diagnosed with no severe hepatic or renal dysfunction, and without any B-vitamin supplements for 3 months before the detection. Informed consent of the participation was given by all the patients, or their descendents if the participants were demented.

2.2 Serum biomarker detection Elbow venous blood samples were collected from the right arm after overnight fasting. Blood was collected in vacuum tubes with EDTA for full blood count and in plain tubes for detecting vitamin B12, folate and total homocysteine (tHcy) levels, and assessing liver and renal functions. Full blood count was carried out using standard method. Serum vitamin B12 and folate concentrations were measured using the chemiluminescent microparticle immunoassay (CMIA) technology (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 vitamin B12 deficient. Patients with serum folate concentrations lower than 2.7 ng/mL were identified as folate deficiency.

2.3 Neurological examination A detailed neurological examination included bilateral assessments of knee jerks, ankle tendon jerks, joint position sense of the great toe and plantar responses. Loss of ankle tendon jerks was also analyzed separately as a more sensitive marker of peripheral neuropathy.

2.4 Cognitive impairment screen A structured interview was employed to record the medical history, usage of medication and the neurological symptoms of the participants. Cognitive function was assessed using Mini-Mental State Examination (MMSE), and the cognitive impairment was assigned when the MMSE score was less than 24/30.

2.5 Neuroelectrophysiological examinations Neuroelectrophysiological examinations, including electroencephalogram, brainstem auditory evoked potential (BAEP), visual evoked potential (VEP), sensory evoked potential (SEP) and nerve conduction velocity, were performed in the vitamin B12 deficient patients to detect their subclinical neurological impairment.

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. Correlations between different markers were assessed by the Spearman test. Logistic regression was used to assess the associations of cognitive impairment or neuropathy with quartiles of vitamin status after adjustment for age and sex. Only two-tailed tests were used. $P < 0.05$ was considered as statistically significant.

3 Results

3.1 Prevalence of vitamin B12 deficiency A total number of 827 patients (402 male and 425 female), aged 60-96 years, with the mean age of 77.14±7.51 years old, fulfilled the enrolling criteria. As shown in Table 1 and Table 2, vitamin B12 deficiency was identified in 163 patients (19.71% of the total patients), 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 ($P > 0.05$). Furthermore, 50 patients (30.67% of the vitamin B12 deficient patients) also exhibited folate deficiency. The serum tHcy concentrations in the vitamin B12 deficient patients were all more than 15 μmol/L. There was no significant difference in the number of chronic diseases such as diabetes, hypertension, stroke, malignancy, vascular disease, arthritis / arthrosis, lung disease, and heart diseases between the patients with or without vitamin B12 deficiency ($P > 0.05$).

3.2 Clinical characteristics of the patients As shown in Table 3, the mean age of the patients with low vitamin B12

Table 1. Prevalence of vitamin B12 deficiency in the studied patients

Age	Number(*n*)			With folate	B12 deficiency		
(years)	Total	Male	Female	deficiency	Male	Female	Total
60-64	69	46	23	1 (1.45%)	6 (13.04%)	4 (17.39%)	10 (14.49%)
65-69	65	32	33	1 (1.54%)	5 (12.5%)	4 (15.15%)	9 (15.38%)
70-74	142	74	78	7 (4.93%)	10 (13.51%)	15 (19.23%)	25 (17.61%)
75-79	226	102	124	13 (5.75%)	16 (15.67%)	25 (20.16%)	41 (18.14%)
80-84	188	88	100	15 (7.98%)	19 (21.59%)	25 (25%)	44 (23.40%)
⩾ 85	137	60	77	13 (9.49%)	14 (23.33%)	20 (25.97%)	34 (24.81%)
Total	827	402	425	50 (6.05%)	70 (17.71%)	93 (21.88%)	163 (19.71%)

Table 2. Blood vitamin B12, folate, and tHcy levels and mean corpusular volume in vitamin B12 deficient patients (mean±SD)

Age	*n*	Folate (ng/mL)	VitB12 (pg/mL)	tHcy (μmol/L)	Red blood cell ($\times 10^{12}$/L)	Hemoglobin (g/L)	Mean corpusular volume (fL)	Number of chronic conditions*
60-64	10	4.96±2.73	148.4±31.59	37.25±18.38	4.39±0.51	132.65±11.07	91.51±4.86	1.7±0.82
65-69	9	5.52±4.36	145.22±20.57	37.08±19.69	4.81±0.45	134.96±23.48	85.67±7.51	2.44±0.88
70-74	25	4.98±2.97	139.68±25.24	37.02±12.09	4.28±0.61	129.89±17.65	92.33±3.96	1.92±0.76
75-79	41	4.51±3.01	138.19±29.95	33.27±11.51	4.09±0.47	124.46±12.79	93.08±5.10	2.02±0.86
80-84	44	4.16±2.70	136.72±32.83	42.02±20.73	4.07±0.46	123.00±15.66	92.04±5.43	2.07±0.80
⩾ 85	34	4.70±3.90	133.20±36.12	35.91±15.94	3.97±0.53	118.35±15.76	92.08±6.12	2.16±0.75
Total	163	4.6±3.15	138.10±30.84	37.25±16.43	4.15±0.53	124.84±16.04	91.98±5.54	2.04±0.81

*Chronic conditions: diabetes, hypertension, stroke, malignancy, vascular disease, arthritis/ arthrosis, lung disease, and heart diseases.

levels (n = 163) was significantly older than that of patients with normal B12 levels (n = 664) (P = 0.03), and the prevalence of vitamin B12 deficiency seemed higher in female than in male patients. However, there was no significant difference in factor of gender or the number of chronic conditions between the 2 groups. Among the B12 deficient patients, the average age of female patients (79.25 years, n = 88) was a little older than that of male patients (77.19 years, n = 75), but with no significant difference (P = 0.063). In comparison with the patients with normal vitamin B12 levels, patients with low levels of serum vitamin B12 were more likely to experience unsteadily walking in the darkness and hypopallesthesia. Although there was no significant difference in the number of chronic conditions between the 2 groups, there were higher incidences of cerebral ischemia (P = 0.048), hypertension, Parkinson's disease (Parkinsonism), and coronary heart disease in vitamin B12 deficient patients. Moreover, the mean MMSE score of B12 deficient patients was remarkably lower than that of patients with normal B12 levels, and there were more patients with MMSE score < 24 in the former group. The vitamin B12 deficient patients exhibited significantly lower concentrations of hemoglobin, but also a higher mean corpuscular volume (P < 0.001) than patients with normal level of serum vitamin B12 did, however, only 9.82% of them had megaloblastic anemia.

3.3 Correlation of low serum vitamin B12 level with the clinical features Correlative analysis of the 163 patients with vitamin B12 deficiency showed that the serum vitamin B12 level decreased with aging, but with no statistical difference (r = -0.135, P = 0.086). There were no significant correlations between serum vitamin B12 level and levels of serum hemoglobin, folate and tHcy, but there was a negative correlation between B12 level and the mean corpuscular volume (r= -0.203, P = 0.009). Besides, there was no significant association of neuropathy with any of the laboratory measurements. The presence of macrocytosis or macrocytic anaemia did not

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

	Total (*n*=827)	Normal VitB12 Levels (*n*=664)	VitB12 deficiency (*n*=163)
Medical history			
Age (years)	77.14±7.51	76.8±7.59	78.2±7.13*
Sex: male (%)	402 (48.61)	322 (50)	75 (46.01)
Number of chronic conditions	2.02±0.74	2.01±0.59	2.04±0.81
Cerebral infarction (%)	458 (55.38)	356 (53.61)	102 (62.58)*
Transient ischemic attrack (TIA, %)	31 (3.75)	23 (3.46)	8 (4.91)
Parkinson's disease (%)	63 (7.62)	46 (6.93)	17 (10.43)
Hypertension (%)	546 (66.02)	444(66.87)	112(68.71)
Diabetes mellitus (%)	239 (28.9)	190 (28.61)	49 (30.06)
Coronary artery disease (%)	319 (38.57)	254 (38.25)	65 (39.88)
Cognitive impairment			
MMSE mean(SD)	27.34 (3.58)	27.45 (3.36)	26.54 (3.92)#
MMSE<24	127 (15.36)	93 (14.01)	34 (20.86)*
Neurological symptoms			
Unsteadily walking in the darkness (%)	173 (20.92)	129 (19.43)	44 (26.99)*
Altered sensation in feet on walking (%)	129 (15.6)	102 (15.36)	27 (16.56)
Paresthesia (%)	134 (16.32)	106 (15.96)	29 (17.79)
Neurological signs			
Absent knee tendon jerk (%)	152 (18.38)	114 (17.17)	38 (23.31)
Absent ankle tendon jerk (%)	335 (40.51)	282 (39.46)	73 (44.78)
Hypopallesthesia (%)	204 (24.67)	148 (22.29)	56 (34.36)#
Hematological findings			
Hemoglobin(g/L)	128.56±15.17	129.43±15.97	124.84±16.04#
Mean corpusular volume(fl)	91.65±4.86	91.34±4.36	91.98±5.54
Hemoglobin<120 g/L (%)	130 (15.72)	86 (12.95)	44 (26.99)#
Mean corpusular volume>95 fl (%)	115 (13.91)	62 (9.34)	53 (32.5)#
Mean corpusular volume>95 fl + Hemoglobin <120 g/L (%)	21 (2.54)	5 (0.75)	16 (9.82)#

*$P < 0.05$, #$P < 0.01$ *vs* patients with normal vitamin B12 levels.

increase the probability of vitamin B12 deficiency.

3.4 Neuroelectrophysiological examinations The neuroelectrophysiological examinations revealed that 67% of the vitamin B12 deficient patients exhibited increased slow waves in electroencephalogram, 30% with abnormal visual evoked potential, 44.83% with abnormal sensory evoked potential, and 43.64% with abnormal brainstem auditory evoked potential. Besides, 10% of the patients were detected with peripheral nerve lesions in both lower extremities by electromyologram record.

4 Discussion

Accumulating evidence has indicated that instead of being a rare and easily diagnosed disease, vitamin B12 deficiency occurs frequently among elderly patients, 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[4]. It is reported that the prevalence of vitamin B12 deficiency in the elderly population in developed country is 5%-15%[4-6], with

12% being the old community residents[2] and up to 30%-40% being the old hospitalized patients[7]. Similar results have also been reported in the developing countries[8-12]. In the present study, we found that the prevalence of vitamin B12 deficiency in the elderly hospitalized patients was 19.71%, which was increased with aging, and the same tendency was also found in complicating folate deficiency. The mean age of the patients with low serum vitamin B12 levels was older than that of the patients with normal B12 levels, implicating that B12 deficiency has a liability in the aged. It is 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, but it cannot be synthesized by human bodies and is completely dependent on dietary sources. Vitamin B12 is an essential cofactor and coenzyme mediating 2 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[1]. 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 that are believed to be of primary importance in explaining the pathophysiological aspects of vitamin B12 and folate deficiencies[13,14].

Vitamin B12 deficiency may be entirely asymptomatic or present the 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, after which the spinal cord, the brain, the optic nerves and the peripheral nerves are all affected. Also, these impairments may be accompanied by 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)[12-15]. The spinal cord is usually the first and the exclusive site of affection. Visual impairment resulting from optic neuropathy may occasionally be the earliest or the sole manifestation.

It may take decades for the development of vitamin B12 deficiency. 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 or neuropathy. Instead, they present more often the non-specific symptoms of fatigue and cognitive impairment that can be attributed to 'old age'. The uncertainty about the importance of vitamin B12 deficiency may be partly due 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[2,3,16], 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[7]. In this study, we found that almost all the vitamin B12 deficient patients lacked previous diagnosis, and only 9.82% of the patients had megaloblastic anemia. The patients with low vitamin B12 levels all exhibited abnormal homocysteine metabolism. Moreover, vitamin B12 deficiency could induce unsteadily walking in the darkness, hypopallesthesia and neuroelectrophysiological abnormalities, which indicated the vitamin B12 deficiency at cellular level. It is likely that both the pre-clinically cellular B12 deficiency in the central nervous system and the subclinical neuroelectricphysiological abnormalities are involved. Other studies[17-19] have shown similar results.

There are many causes for vitamin B12 deficiency, among which the inadequate dietary intake and food-cobalamin malabsorption (FCM) are especially common. FCM is the main cause of vitamin B12 deficiency in the elderly and explains why vitamin B12 depletion occurs with aging. Under FCM condition, vitamin B12 deficiency is caused by atrophy of the gastric mucosa and the gradual loss of gastric acid, which releases the vitamin from food[20]. In elderly patients, FCM may be associated with significant neurologic, psy-

chologic and hematologic abnormalities[21]. The causes for FCM are uncertain. Carmel *et al.*[22] summarized 9 studies in the United States and found that FCM was present in about 40% of patients with unexplained low serum vitamin B12 concentrations. Not all of those patients were elderly, and some had other risk factors such as gastric resection. A subsequent comparison of 43 normal elderly and 159 elderly persons with low serum vitamin B12 levels[23] revealed that malabsorption primarily affected those aged 60 years and was not obviously related to markers of gastric function (e.g. serum gastrin). Helicobacter pylori infection was present in 78% of those with severe malabsorption, in 50% with mild malabsorption, and in 44% with normal absorption, but any effect of H. pylori was independent of its associations with atrophic gastritis or gastric acid production, although H. pylori infection is generally accepted as the main cause of chronic atrophic gastritis and affects about 50% of those aged 60 years in industrialized countries, with a far greater proportion in developing countries. The low gastric pH that occurs as a result of gastric atrophy can also increase bacterial overgrowth in the upper intestine, which results in less absorption of protein-bound (but not crystalline) vitamin B12[24]. In this study, gastroscope examination was performed in 20 vitamin B12 deficient patients. Results showed that atrophic gastritis was found in 14 cases, and gastric mucosa anabrosis was in 2 cases. The results support that FCM-associated gastric mucosa atrophy is the main cause of vitamin B12 deficiency in the elderly patients.

Moreover, Robertson has reported that vitamin B12 deficiency is common in patients with vascular diseases[25], and folate and vitamin B12 supplements can decrease the risk of ischemic cerebral disease[26]. Vitamin B12 deficiency is also involved in the development and progression of dementia[27]. In this study, we found that patients with low serum vitamin B12 levels also suffered from some chronic diseases, such as cerebral ischemia, hypertension, Parkinson's disease or Parkinsonism, and coronary heart disease. Cognitive impairment and cerebrovascular disease may be related aetiologically with low serum vitamin B12 concentration, possibly through elevated blood tHcy concentration resulting from vitamin B12 deficiency or by other mechanisms. However, it is also possible that low level of vitamin B12 or folate may be an effect rather than a cause of cognitive impairment.

In conclusion, vitamin B12 deficiency occurs frequently among elderly patients, but is often unrecognized or not diagnosed promptly due to the subtle clinical manifestations. However, because of the potential consequences of the complications (particularly neuropsychiatric and hematological complications), it is especially important to screen out and treat 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 the B12 deficient patients should take vitamin B12 supplements to attenuate or prevent the neurological impairment, and to improve their life qualities.

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神经科住院老年患者维生素 B12 缺乏的调查

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摘要：目的 调查神经科老年住院患者维生素(Vit)B12 缺乏的状况。**方法** 对本院神经内科 2007 年 3 月—2008 年 7 月间连续住院的、年龄≥ 60 岁的患者进行血清叶酸、VitB12、同型半胱氨酸(Hcy)等指标的检测，筛查 VitB12 缺乏者。共 827 例患者入组，平均年龄 77.1±7.5 岁。所有患者均无严重肝肾功能障碍，且近 3 个月内未补充 VitB12。对 VitB12 正常患者和缺乏患者的神经系统症状、体征及神经电生理检查结果进行比较和分析。**结果** VitB12 缺乏者有 163 例(占所有患者总数的 19.71%)，神经电生理检查多有异常，患病率女性高于男性，并随年龄增长有增加的趋势。VitB 12 缺乏者中伴有胃肠道疾病的比率较高，巨红细胞贫血仅为 9.82%。VitB12 缺乏患者更易出现行走不稳及振动觉减退等症状，更多还伴有脑梗死、高血压、冠心病等慢性疾病。**结论** 神经科住院老年患者常伴有 VitB12 缺乏症，症状表现多样，神经系统表现较之巨红细胞贫血更常见。

关键词： 衰老；住院老年患者；神经科；维生素 B_{12} 缺乏