Original Article
Cent Asian J Med Sci. 2019 June; 5(2): 88-97. https://doi.org/10.24079/CAJMS.2019.06.003

Central Asian Journal of Medical Sciences
CAJMS

The Assessment of Vitamin B1 and B12 Deficiencies in Patients with Alcohol Withdrawal Syndrome

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Submitted: January 14, 2019
Revised: February 27, 2019
Accepted: May 24, 2019

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Objectives: We examined the oral intake and serum concentration of vitamins B1 and B12 in patients with alcohol withdrawal syndrome to determine some risk factors leading to deficiencies of those vitamins and their impact on memory function. **Methods:** Data were collected from patients with alcohol withdrawal syndrome admitted to 3 addiction care clinics in Ulaanbaatar. A total of 162 subjects were selected with a ratio 1:1 in this case-control study. Nutrient intake of B1 and B12 were estimated by questioning the patients regarding their food intake during the 24 hours prior to admission. **Results:** Drinking while hungry resulted in inadequate oral vitamin B12 consumption (OR 9.43; 95% CI 2.42;36.64, p=.001) as did refusing food while drinking (OR 4.76; 95% CI 1.23;18.30, p=.02). Inadequate B1 intake was higher in women (OR 0.16; 95% CI 0.05;0.49, p=.001) and if the patient refused food while drinking (OR 3.38; 95% CI 1.24; 9.21, p=.01). Serum B1 and B12 deficiency were associated refusing food while drinking (OR 4.53; 95% CI 1.81; 11.31, p=.001 and OR 3.43; 95% CI 1.22; 9.61, p=.01). Short term memory impairment was associated with dietary B1 and B12 deficiency (OR 8.59; 95% CI 3.10;23.82, p=.0001 and OR 3.14; 95% CI 1.02;9.61, p=.04) and low serum B12 levels (OR 2.45; 95% CI 0.98;6.11, p=.05). Long-term memory impairment was associated with deficient B12 and B1 intake (OR 19.49; 95% CI 3.19;118.86, p=.0001 and OR 4.73; 95% CI 1.91; 11.68, p=.0001) and low serum B12 and B1 levels (OR 3.32; 95% CI 1.31;8.42, p=.01 and OR 2.64; 95% CI 1.06;6.59, p=.03.)

Keywords: Alcohol Consumption, Alcohol Withdrawl, Nutrient, Thiamine, Cobolamine

Introduction

Alcohol withdrawal syndrome is an important cause of disease and death associated with alcohol consumption. One of the main criteria for diagnosing alcohol dependency is alcohol withdrawal syndrome. Alcohol withdrawal syndrome is characterized by restlessness, anxiety, irritability, tremor, nausea, vomiting, increased heart rate, and arterial hypertension[1,2]. Severe symptoms such as grand mal seizures, delusions and hallucinations may appear as secondary complications in severe cases. Other complications such as Wernicke-Korsakoff syndrome, Marchiafava-Bignami syndrome can arise due to

deficiency of vitamins B1 and B2 in patients with severe alcohol dependency[3,4].

Ethanol can replace almost 60 percent of the daily calorie intake of chronic adult drinkers in some cases leading to malnutrition. Excessive alcohol consumption may result in nutrient deficiencies of variable clinical significance. Alcohol consumption changes the metabolism of many nutrients and the consequences of these distortions may play a significant role in the pathogenesis of liver disease[5].

The relationship between alcohol use and cognitive impairment has been too difficult to disentangle. In the past 65 years, as a consequence of rising alcohol consumption in many countries, alcohol-related brain diseases has again become a major concern[6,7]. Nutritional deficiency can cause Wernicke encephalopathy in chronic alcoholics and its chronic neurological sequelae related to thiamine deficiency, Wernicke-Korsakoff syndrome[8].

Several studies have found that deficiency of thiamine, also known as vitamin B1, is prevalent in both critically ill patients and alcoholic patients. In patients with alcohol abuse disorder, the decrease in dietary intake and malabsorption of thiamine often causes an inadequate supply of thiamine to the brain. Thiamine plays a vital role as coenzyme, in glycolysis and the citric acid cycle. It is well established that excessive and long-term alcohol use lead to permanent damage to the structure and function of the brain[9-12].

Thiamine is an essential vitamin that plays an essential role in the cellular production of energy from ingested food and enhances normal neuronal activities. Deficiency of this vitamin leads to a severe clinical condition known as delirium[2,12].

Mild to moderate thiamine deficiency plays a role in the neurodegeneration observed in chronic alcoholics, however it is often difficult to determine the dietary status of many alcoholic patients. The development of Wernicke's encephalopathy involves many factors. It has been hypothesized that a genetic predisposition is critical for the development of Wernicke's encephalopathy. The key elements are thought to be alterations in thiamine metabolism and susceptibility to alcohol neurotoxicity. Long term chronic ethanol abuse results in hippocampal and cortical cell loss. Thiamine deficiency also alters principally hippocampal and frontal cortical-dependent neurochemistry. The amnestic syndrome typical for Wernicke-Korsakoff syndrome is mainly due to the damage to the diencephalic-hippocampal circuitry, including thalamic nuclei and mammillary bodies[8,13].

The clinical aspects of alcohol use disorders in Mongolia have been studied previously. Erdenebayar in 1997 defined the prevalence and clinical characteristics of alcoholism in Mongolia and determined the scientific basics of prevention from alcoholism[14]. Gantumur in 2012 detected the pathopsychological manifestations of alcoholic patients[15]. Furthermore, Narantuya in 2012 studied alcohol consumption and its characteristics among the Mongolian population, and Odongerel 2014 defined the clinical features of the alcohol-related psychosis[16,17]. However, these previous studies did not focus on the deficiencies of dietary nutrients such as minerals, vitamins, essential amino acids, and the behavioral factors that cause them. In this paper, we sought to determine the relationship between some behaviors that cause deficiency of vitamins B1 & B12 and some memory functions in patients with alcohol withdrawal syndrome.

The objectives of this paper were to (1) assess the level of vitamins B1 and B12 in nutrition and serum of patients with alcohol withdrawal syndrome, (2) define some behaviors leading to deficiencies of those vitamins, and (3) to determine the impact of vitamin B deficiencies on memory function.

Material and Methods

Study design

A total of 162 people were assigned with ratio 1:1 to this non-randomized case-control study. The subjects were divided into alcohol withdrawal patients (case) and were relatively healthy adults who were being treated for mental health diagnoses not related to alcohol (control) groups. We randomly selected control group subjects from patients who were being seen for a preventive check-up at the National Center for Mental Health.

Inclusion criteria: Case group subjects were inpatients receiving treatment at the National Mental Health Center's Addiction Unit, Narcology Center and at "Borjigonii otoch" clinic, diagnosed with stage 2-3 alcohol withdrawal syndrome by addiction psychiatrists. These subjects reported they not had consumed alcohol for past 2-3 days before a structured examination by specialists, and they were diagnosed with alcohol withdrawal syndrome.

Control group subjects did not have any alcohol addiction symptoms and were rated as being non-consumers or non-hazardous users according to the Alcohol Use Disorders

Identification Test (AUDIT)[18].

Exclusion criteria: Individuals were excluded if they consumed alcohol the 2-3 days prior to the structured interview, were having disulfiram induced psychosis or were having psychosis not related to alcohol withdrawal syndrome, were diagnosed with severe somatic, neurological and psychiatric diseases, received alcohol dependency or pharmacological treatment previously, or refused to participate in the study.

Method of evaluating alcohol withdrawal syndrome

All participants of the case group patients were examined by an addiction psychiatrist. After assessing their mental health status, their stage of alcohol dependence was assessed using the Short Alcohol Withdrawal Scale (SAWS)[19]. Their degree of alcohol dependency degree and their stage of alcohol abuse disorder were defined by the Michigan Alcohol Screening Test (MAST)[20].

Method of determining serum vitamin B1 and vitamin B12 levels

After fasting for one day, 5 mL of venous blood was collected from all patients, anticoagulated, and centrifuged. The serum was frozen and stored in aliquots at -20°C for testing. Serum vitamin B1 and B12 were measured by enzyme-linked immunosorbent assay (ELISA) method (ELISA kit, human SL2644HU, SL1827Hu, Sunlong Bio-tech. Co LTD, China). Optical density (OD) of the results measured at a wavelength of 450 nm using a spectrophotometer device. Serum concentrations of vitamin B1 and B12 were determined using the OD value. The OD value was proportional to the concentration of Vitamin B1 and Vitamin B12. All related operations were performed by highly experienced personnel, in strict accordance with instrument instructions. The Vitamin B1 and B12 normal reference values in our hospital were 0.8 - 50 ng/ml and 0.8 - 30 ng/ml respectively, and a person was considered deficient when their values were below 0.8 ng/ml for either vitamin[21].

Method of determining estimated dietary intake of vitamin B1 and vitamin B12

Each participant was interviewed to complete a research questionnaire recalling their nutrient intake for the 24 hours before admission. Information regarding meal size, ingredients, variety, frequency of meals, and eating habits were collected. The nutrients in meals, and meal vitamin B1 and B12 concentration were estimated using ''Food ingredient chart" in Ministry of Health and the NUTRISURVEY program of the World Health Organization (WHO) in 2004[22,23]. A person's vitamin B1 and B12 intake were considered deficient when the estimated oral vitamin B1 and B12 intake were less than1.2 mg and 2.4 mg per day, respectively.

Method of determining vitamin B deficiency risk behaviors and memory impairment

Questions such as "Do you drink while you are hungry?", "Do you end up not eating while drinking?" were asked of alcohol-dependent participants to determine some risk factors leading to vitamin B deficiencies. Questions regarding their diet were asked when they affirmatively answered either of these questions.

Memory function was tested using Luria Memory 10 Words Test[24]. A person was considered to have a short term memory impairment when did not fully recall the memorized words in the next 1-5 minutes and long-term memory impairment when did not fully remember the memorized words after one hour.

Statistical analysis

Our data were initially subjected to the Kolmogorov test to determine the normality of the variables. Normally distributed variables were presented as mean, and standard deviation and were then analyzed using independent t-tests. A log transformation was used for non-parametric variables, and correlation was considered significant at the $p<.01$ and $p<.05$ levels (2-tailed). All reference categories were "No" except for the employment variable which used "Yes" as the reference. Multiple logistic regression analysis was used to examine some risk factors associated with low oral consumption of vitamins B1 and B12 in alcohol withdrawal patients; to define the relationship between vitamin B deficiencies and some memory functions. For the logistical regression, the reference category for all dichotomous variables was "No." The statistical analyses were done using SPSS 21.0.

Ethical statements

All subjects gave written informed consent. The research proposal was reviewed and approved by the Academic Committee of the Mongolian National University of Medical Sciences on January 5, 2018. Ethical approval was obtained from the Biomedical Ethics Committee of the Mongolian National University of Medical

Sciences on January 26, 2018 (№ 2108/3-01). Data were collected only after the administrative approvals were obtained.

Results

One hundred sixty-two people between 18 and 65 years of age participated in the study, half of whom were diagnosed with alcohol withdrawal syndrome (case subjects), and the other half were relatively healthy adults (control) in this non-randomized case-control study. The case group consisted of individuals with alcohol withdrawal syndrome, aged between 27 and 65 with amean age 42.43±8.28 years. Eighty-one relatively healthy adults with non-alcohol related mental diagnoses were included as control subjects. Men comprised 77.8% (n=63) of all subjects in each group, and gender ratio was 3:1. Regarding alcohol consumption, 1.2% (n=1) of all subjects had harmful levels of alcohol consumption for 1 year or less, 3.3% (n=27) of participants for 2-5 years, 28.4% (n=23) for 6-10 years, 19.8% (n=16) for 11-15 years, 2.3% (n=10) for 16-20 years and 4.9% (n=4) for more than 21 years (p<.001). Selected socio-demographic, job characteristics, and stage of alcohol abuse disorder of all participants are summarized in Table 1.

The age distribution of alcohol withdrawal patients was significantly different from the control subjects with alcohol withdrawal patients being older (p<.0001). The alcohol withdrawal patients were also less educated (p<.0001), and had a different distribution of employment being 3 times more likely to be unemployed (38.2 vs. 9.9%, p<.0001) and when they were employed they were 4 times more likely to have private sector employment compared to government job (49.3 vs. 12.4%, p<.0001). Their marital status distribution also differed with 43.2% of them being married compared to 69.1% of the control subjects (p<.0001).

Table 2 shows the results of the estimated oral intake and serum vitamin B1 and B12 concentration and determining some risk behaviors leading to that deficiency. The estimated levels of oral intake and the serum levels of vitamin B1 and B12 were all significantly lower in the patients with alcohol withdrawal syndrome compared to the control subjects.

Table 1. Some socio-demographic characteristics of case and control groups (N=162).

Demographic characteristics	Alcoholic withdrawal syndrome patients N (%)	Control patients N (%)	Total N (%)	p-value
Age				
≤29	4 (4.9)	26 (16.0)	30 (18.5)	
30-39	27 (33.3)	22 (27.2)	49 (30.2)	<.0001
40-49	31 (38.3)	24 (29.6)	55 (34)	
≥50	19 (23.4)	9 (11.1)	28 (34.5)	
Gender				
Male	63 (77.8)	63 (77.8)	126 (77.8)	.932
Female	18 (22.2)	18 (22.2)	36 (22.2)	
Educational status				
Secondary	49 (60.4)	28 (34.5)	76 (46.9)	
Specialized secondary	7 (8.6)	11 (13.6)	18 (11.1)	<.0001
Higher	26 (32.1)	42 (51.9)	68 (41.9)	
Employment status				
Governmental, NGO	10 (12.4)	63 (77.7)	73 (45.0)	
Private	40 (49.3)	10 (12.3)	50 (30.9)	<.0001
Unemployed	31 (38.2)	8 (9.9)	39 (24.1)	
Marital status				
Married	35 (43.2)	56 (69.1)	91 (56.2)	
Single	46 (56.8)	25 (30.9)	71(43.8)	<.0001
Total	81 (100)	81 (100)	162 (100)	

p-value was calculated with Chi-Square test; NGO-Non Governmental Organization

Table 2. Comparison of estimated oral intake and measured serum vitamin B levels in case and control subjects (N=162)

Nutrients	Average rate daily values for Mongolians[22,23]	Case (N=81)		Control (N=81)		t	p-value*	95% CI
		Mean	SD	Mean	SD			
Estimated oral intake								
Vitamin 1 (mg)	1.2	0.3	0.2	1.0	1.5	3.7	<.0001	[0.3;1.0]
Vitamin 12 (mkg)	2.4	2.7	2.4	9.7	8.6	6.9	<.0001	[5.0;8.9]
Measured serum level								
Vitamin B1ng/ml	0.8-50	0.8	0.9	11.1	14.2	6.5	<.0001	[7.2;13.5]
Vitamin B12 ng/ml	0.8-30	0.4	0.4	9.8	8.9	9.3	<.0001	[7.3;11.3]

*p-value from independent t-test comparing case and control groups after log transformation to compensate for non-normal data distribution. CI confidence interval.

Driven by our vitamin B deficiency findings, we determined the effect of some demographic variables (age, employment, marital status) and some risk behaviors related to drinking (drinking while hungry and refusing food while drinking) on the oral vitamin B1 and B12 intake and serum vitamin B levels using multiple logistic regression (Table 3).

As shown in Table 3, the risk of deficient vitamin B1 intake in alcohol withdrawal patients was 6 times higher in women (OR 0.16; 95% CI 0.05;0.49, p=.001) and 3 times higher if the patient refused food while drinking (OR 3.38; 95% CI 1.24;9.21, p=.01).

Intake deficiency of vitamin B12 was only significantly

Table 4. Multiple logistic regression of some factors affecting serum vitamin B deficiency in alcohol withdrawal patients (N=81)

Variables	Risk of B1 deficiency (ng/ml)			Risk of B12 deficiency (ng/ml)		
	cOR	CI, 95%	p-value	cOR	95% CI	p-value
Age 30-39						
No	1		.34	1		.14
Yes	1.57	[0.61;4.04]		0.50	[0.20;1.24]	
Age 40-49						
No	1		.12	1		.90
Yes	2.00	[0.81;4.92]		0.94	[0.39;2.25]	
Male						
No	1		.56	1		.006
Yes	1.77	[0.31;1.88]		0.21	[0.07;0.64]	
Unemployment						
No	1		.83	1		.16
Yes	0.91	[0.37;2.22]		2.01	[0.75;5.37]	
Single						
No	1		.41	1		.59
Yes	1.37	[0.64;2.92]		1.24	[0.56;2.73]	
Drinking while hungry*						
No	1		.65	1		.31
Yes	0.78	[0.26;2.31]		2.14	[0.48;9.50]	
Refusing food while drinking*						
No	1		.001	1		.01
Yes	4.53	[1.81;11.31]		3.43	[1.22;9.61]	

The reference category is "No." cOR – Crude Odd ratio. *Risk behavior for vitamin B deficiency related to drinking.

associated with the two risk behaviors related to drinking. Answering affirmatively the question "drinking while hungry" resulted in nearly a 10-fold increase risk of below normal oral consumption (OR 9.43; 95% CI 2.42;36.64, p=.001) while refusing food while drinking led to almost 5-fold risk (OR 4.76; 95% CI 1.23;18.30, p=.02) in alcohol withdrawal patients. Inadequate consumption of B12 was not associated with the demographic variables related to age, employment, or marital status.

Table 4 shows the results of multiple logistic regression analysis on some risk factors associated serum deficiency of vitamins B1 and B12 in alcohol withdrawal patients.

Decreased serum concentration of vitamin B1 was associated affirmatively answering the question "refusing food while drinking" resulted in a nearly 4-fold increased risk of deficiency in alcoholic withdrawal patients (OR 4.53; 95% CI 1.81;11.31, p=.001). None of the other factors examined were associated with a risk of vitamin B1 deficiency.

Serum vitamin B12 deficiency was significantly reduced by almost 80% with being male (OR 0.21; 95% CI 0.07;0.64, p=.006) and again with "refusing food while drinking" (OR 3.43; 95% CI 1.22; 9.61, p=.01) in alcohol withdrawal patients. Serum B12 deficiency was not associated with any of the other factors examined.

In Table 5, short term memory decline in alcohol withdrawal patients was most significantly associated with nutritional vitamin B1 (OR 8.59; 95% CI 3.10;23.82, p=.0001) followed by B12 (OR 3.14; 95% CI 1.02;9.61, p=.04) and serum vitamin B12 deficiencies (OR 2.45; 95% CI 0.98;6.11, p=.05).

However, long-term memory impairment in alcohol withdrawal patients was associated with both nutritional and serum deficiency of both B vitamins. Most striking was the almost 20-fold increased risk in patients with B12 intake deficiency (OR 19.49; 95% CI 3.19;118.86, p=.0001) followed by a nearly 5-fold increase in risk with B1 intake deficiency (OR 4.73; 95% CI 1.91;11.68, p=.0001). Serum vitamin B12 deficiency was likewise associated with significant risk of long term memory impairment (OR 3.32; 95% CI 1.31;8.42, p=.01) followed by serum B1 deficiency which was associated with a nearly 3-fold risk (OR 2.64; 95% CI 1.06;6.59, p=.03). Unlike short term memory impairment, long term memory decline was reduced by almost two-thirds with being male (OR 0.33; 95% CI 0.11; 1.00, p=.05).

Discussion

The study identified low oral consumption of vitamin B1 and B12 resulting in low serum levels of these vitamins. Logistic regression showed that drinking while hungry and refusing food while drinking were associated with inadequate intake and low

Table 5. Multiple logistic regression of some factors associated with memory impairment in alcohol withdrawal patients.

Variables	Short term memory decline*			Long term memory decline*		
	cOR	CI, 95%	p-value	cOR	95% CI	p-value
Age 30-39						
No	1		.36	1		.40
Yes	0.61	[0.21;1.76]		1.57	[0.54;4.56]	
Age 40-49						
No	1		.44	1		.16
Yes	0.67	[0.24;1.85]		2.15	[0.73;6.30]	
Male						
No	1		.95	1		.05
Yes	0.97	[0.38;2.49]		0.33	[0.11;1.00]	
B1 intake deficiency, mg						
No	1		.0001	1		.001
Yes	8.59	[3.10;23.82]		4.73	[1.91;11.68]	
B12intake deficiency, mg						
No	1		.04	1		.001
Yes	3.14	[1.02;9.61]		19.49	[3.19;118.86]	
B1 serum deficiency, ng/ml						
No	1		.17	1		.03
Yes	1.83	[0.77;4.35]		2.64	[1.06;6.59]	
B12 serum deficiency, ng/ml						
No	1		.05	1		.01
Yes	2.45	[0.98;6.11]		3.32	[1.31;8.42]	

The reference category is "Yes". cOR – crude odds ratio.

serum levels these vitamins and that their deficiencies adversely effected memory function in alcohol withdrawal patients.

Heavy alcohol consumption not only influences the dietary pattern of alcohol abusers but also reduces the absorption of proteins, fats, vitamins including B1, B6, and B12 and minerals such as iron, selenium, and zinc from food in the gastrointestinal tract[5-27]. This leads to nutritional deficiencies that are essential to mental health, contributing to depression, fatigue, attention deficiency, and sleep deprivation[28,29].

Vitamin B1 also known as thiamine, is one of the eight essential B vitamins that help convert food (carbohydrates, fat, and protein) into energy. Thiamine deficiency leads to decreased activity of thiamine-dependent enzymes that triggers a sequence of metabolic events leading to energy compromise. Neuronal death often occurs in specific neuronal populations that have high metabolic requirements and high thiamine turnover. Of all B vitamins, vitamin B12, niacin, and thiamine have the most clearly established relationship with mental deterioration. For example, vitamin B12 deficiency has a well-recognized neurologic syndrome that is characterized by cognitive and psychiatric disturbances, as well as by subacute combined degeneration of the spinal cord, and peripheral neuropathy[2,30-32].

We found that oral consumption and serum levels of vitamin B1 & B12 were significantly lower in alcohol withdrawal patients than in the control group. Dietary habits are a significant aspect of people's lifestyles that influence health, morbidity, and mortality for a range of conditions. Several studies have focused on patterns of food consumption and their relation to mental well being. Diet affects mood and other various cognitive functions. The anorectic influence of alcohol on the satiety centers of the hypothalamus leads alcoholic abusers to refuse food. Furthermore, alcoholic patients lack adequate nutrition in their diet and attempt to meet their caloric needs from ethanol consumption leading to alcoholics being hungry many days while drinking heavily[27,28,33].

Therefore, we determined the relationship of two behavioral risk factors on B vitamin deficiency in alcohol withdrawal patients. First, drinking while hungry and refusing food while drinkinghad a mild but statistically significant effects on oral consumption and serum levels of vitamin B1 & B12. Second, drinking while hungry significantly decreased the dietary intake ofvitamin B12, but not B1. Third, refusing food while drinking significantly reduced the dietary intake of B1 and B12 vitamins and was associated with low serum levels of these vitamins inalcohol withdrawal patients.

These outcomes are similar to the results international studies mentioned above, which described the reduced dietary nutrient and calorie consumption caused by alcohol dependence.

Research describing the decline in cognitive function due to the harmful effects of alcohol date back to the 19th century. Gayet–Wernicke encephalopathy is a chronic neurological disease caused by vitamin B1 deficiency. Its symptoms were documented in ancient Chinese literature as result of eating mainly raw rice about 1000 hundred years ago[3,7]. But, Gayet–Wernicke encephalopathy was described by the German scientist Korsakoff who wrote about this condition in detail and published it in late 19th century[34,35].

Prolonged heavy alcohol consumption results in thiamine intake deficiency, reduced absorption, and increased excretion of thiamine and leads to Wernicke's encephalopathy. Wernicke's encephalopathy is characterized by delirium with prominent partial amnesia, and short-term memory loss associated with compensatory confabulation, with relative preservation of long-term memory and other cognitive skills. Chronic alcoholism causes a nutritional deficiency in vitamin B1 or thiamine, reducing its intestinal absorption and usage in cells. Moreover, it causes deformities in the brain cells and in their biochemical pathways, which is the foundation for cognitive, behavioral, and especially memory impairments[11,13,36-38].

Another disease related to chronic alcoholism is Marchiafava-Bignami disease, and it was first described by Italian pathologists Amico Bignami and Ettore Marchiafava in 1903. Marchiafava-Bignami disease is a rare complication of long-term, heavy alcohol abuse leading to a decreased level of consciousness, seizure, and death. This disease is characterized by chronic alcoholism, corpus callosum demyelination, and necrosis and subsequent atrophy. It may involve adjacent while matter and subcortical regions. The most accepted etiologic factor is multiple vitamin B deficiencies. Symptoms include psychosis, depression, apathy, hemiparesis, and apraxia and in severe cases coma and death. Researchers from Portugal, Iran, India noted that in patients diagnosed with Marchiafava-Bignami disease serum vitamin B12, and folic acid (vitamin B9) levels were decreased and patients started to feel better with the administration of treatments containing vitamin B[4,39,40].

Our study shows that patients with decreased levels of

both vitamin B1 and B12 developed short term and long term memory loss. Short term memory decline was most significantly associated with nutritional vitamin B1 and B12 and serum vitamin B12 deficiencies with over a 2-8-fold risk of these occurring in alcohol withdrawal patients.

Nutritional and serum vitamin B1 and B12 deficiencies were most significantly associated with long-term memory decline with an over 2-19-fold risk in patients with alcohol dependency. When the oral intake of vitamin B12 and serum vitamin B12 concentration-were low, the long-term memory decline 3-19 times compared to the control group.

Therefore, the results of our study confirm that patients with alcohol dependency develop memory function impairments have nutritional and serum vitamin B level deficiencies. Furthermore, we should study the nutritional problems not only in alcoholic patients but also of other psychiatric patients to develop nutritional therapy for these patients.

To our knowledge, our study is the first focused on oral consumption of specific B vitamins in and their serum levels in alcohol withdrawal syndrome in Mongolia. Our research shows that determining essential vitamin deficiencies in alcohol abuse disorder is an area for further study in the field of addiction medicine. Therefore, we believe that the nutritional therapies can very important in treatment for alcoholic patients, and it may be possible to prevent many alcohol-induced mental, behavioral and somatic problems by providing adequate specific B vitamins. Furthermore, we need a study of the nutritional needs of alcoholic patients to better treat them.

This study has a few limitations. First, our sample size was limited by the high cost the laboratory tests as this study conducted using the personal funds of the lead author. Second, alcohol withdrawal syndrome subjects had difficulties recalling their meal ingredients and variety because most of the subjects had memory problems related to chronic alcohol consumption. Third, many of the alcohol withdrawal syndrome subjects had a period of psychosis and confused consciousness related to heavy alcohol consumption, and it was challenging to collect data from them. Despite these challenges, we will further investigate changes in cognition and behavior caused by alcohol dependency disorder.

Conclusions

Oral consumption and serum vitamin B1 and B12 levels were markedly decreased in patients with alcohol withdrawal syndrome. Risk behaviors related to drinking affected the deficiency of those vitamins. The memory function of alcohol withdrawal patients was significantly impaired.

Conflict of Interest

The authors state no conflict of interest.

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