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Thiamine Deficiency in Alcoholics with Normal Body Mass Index

Research Article

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Abstract

Ethanol consumption leads to nutritional deficiency due to various causes. Well known theoretical as well as literature supports the hypothesis on dietary factors, metabolic derangement and alcohol per se produces empty calories. In this study completed in 2000 go hand in hand with the earlier observations and the available literature after 2000. Nutritional deficiency in turn leads to Alcoholic brain damage which is reversible if identified early. Among the nutritional deficiency Thiamine deficiency (Vitamin B6) appears as the most common findings across the studies. In this study also, thiamine deficiency was considered based upon the increased serum Pyrophosphate level an indirect indicator of thiamine deficiency. In this study all the 40 samples were moderate drinkers, diagnosed as Alcohol dependence syndrome according to ICD-10 DCR. No indicators of overt nutritional deficiency on clinical assessment by ICMR Scale, Biochemical assessment and anthropometric assessment. Though very preliminary and cross sectional analysis, thiamine supplementation of all the drinkers irrespective of the clinical and biochemical nutritional deficiency will prevent at large both acute and chronic alcoholic brain damage.

Background

Ethanol is a rich source of nutritional calories. So, that heavy drinking is often complicated by malnutrition and vitamin deficiency [1]. Malnutrition has been a well recognized accompaniment of alcoholism [2]. The low concentration of nutrients in alcoholic beverages cannot compensate for the reduced nutritional intake of patients, whereas the alcohol may provide two third of the daily caloric requirement, thereby producing nutritional imbalance. Nutritional and vitamin deficiency is suggested by analogy with Wernicke's Korsakoff's encephalopathy. Dietary inadequacy are often exaggerated by malabsorption of Thiamine secondary to the effect of circulating ethanol [3,4]. The chemical, biochemical and pathological observations which have been made in Wernicke's Korsakoff's syndrome strongly favor the contention that it represents the human counterpart of experimentally induced thiamine deficiency in animals [5]. Genetically determined abnormalities in the thiamine dependent enzyme transketolase may explain why only a subgroup of malnourished alcoholics have the Wernicke's Korsakoff's syndrome [6]. Current research suggests that ethanol related neurological disorders may be caused by a combination of neurotoxic effects of ethanol or its metabolites, nutritional factors and genetic predisposition. Therefore it would be important to study the relationship between alcohol consumption and nutritional status.

Ethanol intake appears to affect the thiamine status in three main ways [7]. First, the diet of alcoholics are frequently low in thiamine because the daily energy intake largely displaced by nutrient deficient alcoholic beverages. Secondly, the metabolic demand for thiamine is increased by the consumption of a diet rich in carbohydrate as primary source of energy. Thirdly, alcohol can inhibit the intestinal ATPase involved in the entire absorption of Thiamine. In young healthy non alcoholic individuals, subjective symptoms appear after 2-3 weeks of deficient diet [8].

This study has been done in 2000 at De-addiction centre, NIMHANS, Bangalore. There are very few studies actually focusing on vitamins and alcoholic brain damage in developing as well as developed countries. Hence we have retrospectively analyzed the relevance of this study with similar studies in the past and present. This study examined the relationship between nutritional parameters in a group of alcohol dependent individuals without any clinically manifest nutritional deficiency. The mechanism behind the nutritional deficiencies in alcoholics include inadequate dietary intake, vomiting,

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diarrhoea, steatorrhoea, reduced absorption of vitamins and minerals, increased metabolic demands and impaired utilization. Ethanol consumption in a bomb calorimeter indicates a caloric value of 7.1kcal/g. But, there are empty calories, since the alcoholic beverages are almost devoid of other nutrients like vitamins and trace elements. Alcohol induced gastritis, Pancreatitis and liver disease leads to malabsorption of major nutrients. Most of the alcoholic admitted for the management of medical problems are found to have evidence of malnutrition. Anthropometric measurements like Body Mass Index, Triceps Skin-fold thickness Creatinine/height index are found to be low in alcoholics. Individuals consuming more than 30 percentage of total calories as alcohol, significant decreases in protein and fat intake occurs, and their intake of Vitamin A,C and thiamine is below the recommended daily allowances. Periods of altered sensorium, Poor appetite and hangover during intoxication also leads to a reduction in the amount of food consumed. Various studies have demonstrated the high incidence of Vitamin deficiency [9].

Thiamine is the vitamin commonly deficient in alcoholics. This is due to reduced intake as well as reduced absorption of thiamine. Thiamine deficiency leads to complications like Beriberi and Wernicke's Korsakoff's syndrome. Literature reports that most consistent reduction was in thiamine followed by folate. Early symptoms of thiamine deficiency include anorexia, weakness, itching, burning sensations in hand and feet, indigestion, irritability and depression [10]. A deficiency in the essential nutrient thiamine resulting from chronic alcohol consumption is one factor underlying alcohol induced brain damage. Studies found consistently low total vitamin B6 in chronic alcoholics. Riboflavin and nicotinic acid were the next most frequently reduced blood vitamins. Pantothenic acid and folate were least frequently affected. There were no significant reduction in folate level of the CSF [11]. Chronic alcohol intake increases the metabolism of vitamin A and leads to deficiency features like night blindness. Poor food intake, fat Malabsorption due to hepatic and pancreatic disease as well as lack of exposure to sunlight leads to Vitamin D deficiency in alcoholics.

Alcohol induced hepatic dysfunction leads to a reduction in the production of Vitamin K dependent clotting factors II, VII, IX and X leading to raised Prothrombine time and bleeding manifestations. Vitamin E is also found to be low in alcoholics with fat metabolism. Zinc deficiency found in alcoholics said to be the cause for night blindness and hypogonadism in alcoholics. Reduction in the concentration of zinc have been found in liver, Red blood cells and plasma following chronic ingestion of alcohol [12]. Patients with cirrhosis show that a greater proportion of zinc was bound to the alpha 2 Macroglobulin which is metabolically more inert and exaggerate zinc deficiency [13]. Iron deficiency anaemia often accompany the anaemia of liver disease [14]. It may be due to poor dietary intake or chronic Gastro intestinal blood loss either from peptic ulcer or from esophageal or gastric varices. Alcoholics also prone for Iron deficiency anaemia due to blood loss resulting from oesophagitis, gastritis, duodenitis and bleeding esophageal varices [15]. Many studies have documented low circulating magnesium concentration and a few studies have been demonstrating reduced tissue levels [16]. Magnesium deficiency may have pathogenic role in the symptomatology of delirium tremens, possibly resulting from cell cation bump failure in which magnesium deficiency plays a integral role [17].

Poorly nourished spirit drinkers are more liable to suffer to folate dependent megaloblastomas than beer drinkers, since the latter beverage contains fairly large quantities of folate [18]. The reduction in alcohol consumption and the restoration of normal diet has been shown to produce a reticulocytosis and the reversion of the marrow to normoplastic state.

Stomatocytosis may occur in isolated cases of alcoholics [19]. The stages of anaemia development in alcoholics includes, dietetic phase, megaloplastic and sideroblastic change [20]. Neutropenia in alcoholics usually transient. Cell count becomes normal within 24 hours of admission to hospital. A subsequent rebound leukocytosis has also been noted [21]. The total Granulocyte may be decreased and this was attributed to folate deficiency. A transient neutropenia, often with thrombocytopenia reported in non infected alcoholic patients with only mild hepatic disease [22]. Both lymphocyte function and macrophage activity are reduced in alcoholics [23]. Acute thrombocytopenia may follow excessive alcohol ingestion [24]. Thrombocytopenia appears to be due to a direct toxic effect of ethanol on the magakaryocytes. Thrombocytopenia appears to alleviate within a week of alcohol withdrawal [25]. In this study we retrospectively reviewed the studies related to nutritional deficiencies particularly thiamine deficiency in alcohol dependence syndrome.

Methods and Materials

Forty males aged 20 to 40 years attending the De-addiction services at National Institute of Mental health and Neuro-sciences, Bangalore were taken up for this study in the year 2000. In an Average 2-3 patients were recruited every week from the out-patient Screening Block. All the sample were males who were diagnosed to have Alcohol Dependence syndrome according to International Classification of Diseases tenth version Diagnostic Criteria for Research [26].

Subjects having independent medical disorders, independent psychiatric disorders, other substance use disorders except nicotine use, malnutrition and subjects having alcohol related psychiatric disorders were excluded from the study.

All the study subjects were administered a semi-structured interview proforma including demographic data. Diet history was assessed by 24 hour recall method. Average daily intake of calories, protein, fat, carbohydrates, minerals and vitamins were calculated from the collected diet history. A detailed physical examination was done by administering Indian Council of Medical Research Score card. All the subjects were subjected to anthropometric measurements including weight, height, Mid-Arm Circumference (MAC), Triceps Skin-fold thickness (TKFT) and Body Mass index (BMI). All the subjects were subjected to the following Biochemical investigations before vitamin supplementation;

(a) Hematological parameters

(b) Bio-chemical parameters including fasting blood Sugar, Serum Bilirubin, Total Protein, Serum Albumin, Serum Glutamte Oxaloacetae (SGOT), Serum Glutamate Pyruvate Transaminase (SGPT), Serum Alkaline Phosphatase, Serum Gamma Glutamate Transferase (GGT), blood Urea, Serum Creatinine.

(c) Fasting Lipidogram

(d) Serum Calcium, Phosphorous, Magnesium, Zinc and Iron

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(e) Serum Sodium and Serum Pottasium

All the data were analyzed by using descriptive statistics, Pearson Correlation Co-efficient, Student's-t test and multiple linear regression.

Results

Demographic variables

All the study sample were men with mean age of 33 years+4.59. The mean number of education was 7 years and their monthly income ranged from 600 INR to 6000 INR. Eighty five percentage of the sample were married. Among the study sample skilled workers were 45 percentage, unskilled workers were 42.5 percentage. Ninety percentage of the subjects belong to Hindu religion. Sixty percentage from the Urban Background. All the sample were 100 percent Non-Vegetarien (Table 1 and 2).

Drinking variables

The mean duration of drinking was 12+4.70 years. The mean daily consumption of alcohol was 128 grams of alcohol. Mean lifetime consumption of alcohol was calculated as 1317 Kilogram of ethanol. All the study subjects fulfilled more than five out of six criteria for Alcohol dependence syndrome as per ICD-10 DCR. Seventy five percentage of the subjects have consumed Indian Made Foreign Liquor (IMFL) like Whisky, Brandy and Rum. The remaining twenty five percentage of the subjects consumed arrack. The drinking pattern reveal that 5 percentage of them had binge drinking. Sixty percentage were smokers.7.5 percentage used Panparag. Thirty percent had a positive family history of Alcohol Dependence in parents, 15 percent in sibling 2.5 percent in Off-spring (Table 3 and 4).

The mean caloric intake was 2089+537.26 which is slightly below the norms for the average Indian male (2400 Calories). Sixty percent of the total calories (mean 1258.23+349.960) derived from ethanol. Only 40 percent from the food intake. Dietary intake also appears deficient in Protein, Calcium, Iron and Thiamine.

Nutritional variables

Clinical assessment reveals no evidence of malnutrition in all the study population. However anorexia was the commonly reported symptom in 47.5 percent of the study population. The next common presentation was deficient adipose tissue quantity. The mean Body Mass Index (BMI) was 19.4 which falls within the normal range of Indian males (18.5 to 25). The mean anthropometric measures of Triceps skin fold thickness and mid-arm circumference were within normal range (Tables 5-7).

Biochemical variables

All the study subjects had normal heamogram. Liver function tests were abnormal as reflected by the elevated liver enzymes. SGOT/SGPT ratio was 1.2.Serum protein and Albumin Globulin ratio was in the normal range. Lipid profile showed an increase in HDL cholesterol. Serum Pyruvate was raised to 318.47 mmol/litre, (Normal range 0.08 to 0.16 mmol/l) indicating Thiamine deficiency. All other trace elements were within normal range (Table 8).

Table 1: Demographic variables.

	N	Mean	SD	Range
Age	40	33.20	4.59	26-40
Number of years of education	40	7.80	4.61	0-15
Income	40	1892.50	1463.46	600-6000

Table 2: Demographic variables.

Marital status	N	%
Married	34	85
Unmarried	4	10
Separated	1	2.5
Widowed	1	2.5
occupation		
Unskilled	17	42.5
Skilled	18	45
Clerical	4	10
Self-employed	1	2.5
Religion		
Hindu	36	90
Christian	4	10
Residence		
Rural	6	15
Semi-urban	10	25
Diet habits		
Mixed	40	100

Table 3: Drinking Variables.

Drinking variable	n	mean	SD	Range
Years of drinking	40	12.35	4.70	6-25
Average daily intake of alcohol (grams)	40	128.41	55.43	22.35-251.50
Lifetime intake of alcohol (kilograms)	40	1317.17	1000.53	156.80-5364.00
Number of ICD-10 ADS criteria met	40	5.12	0.33	5-6

Table 4: Drinking variable.

Drinking Variable	n	%
Type of alcohol		
IMF	30	75
arrack	10	25
Binge drinking	2	5
Frequency of drinking		
Every day	16	40
Nearly every day	24	60
Blackout	9	22.5

Table 5: Diet Variables.

Diet variable	n	mean	SD	Range	Normal*
Calories in food(K cal)	40	831.73	372.59	159-1586	-
Calories in ethanol(k cal)	40	1258.23	347.96	627-2115	-
Total calories(k cal)	40	2089.27	537.26	786-3067	2400
Protein(gms)	40	30.30	7.47	19-46	55
Fat(gms)	40	280.92	81.70	115-517	-
Calcium(mg)	40	320.72	224.28	90-959	400
Phosphorous9mg)	40	631.90	186.77	-	800
Iron(mg)	40	13.94	4.75	6-24	24
Thiamine(mg)	40	0.85	0.44	O.5-2.0	1.2

*Recommended daily intake of nutrients, ICMR, 1981.

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Table 6: Clinical Variable.

General	n	%
Appetite poor	3	7.5
Skin(loss of lustre)	10	25
Skin elasticity (diminished)	10	25
Face (nasolabial seborrhoea)	1	2.5
Face(Symmetrical sub-orbital pigmentation	1	2.5
Adipose tissue quantity(deficient)	9	22.5
Appetite(anorexia)	19	47.5
Stools(diarrhoea)	3	7.5
Paresthesia (present)	2	5
Extremities(symmetrical dermatitis)	1	2.5
Hair(loss of lustre)	3	7.5
Eyes		
Conjunctival xerosis	3	7.5
Night blindness	1	2.5
Mouth		
Tongue colour(red)	3	7.5
Tongue surface(fissured)	1	2.5
Buccal mucosal(stomatitis)	1	2.5
Caries(mild)	2	5
Caries(moderate)	1	2.5

@Rao K.S et al. Indian food medical Research,1954.

Table 7: Anthropometric variables.

Anthropometric variable	n	mean	SD	Range	Normal
Height(cm)	40	163.90	7.27	146-177	162-163
Weight(kg)	40	53.02	11.00	38-95	53-56
BMI	40	19.42	3.40	15.06-30.03	18.5-25
Triceps skin-fold thickness(mm)	40	8.08	9.92	8-12	8.7+03
Mid arm circumference	40	23.41	3.29	18-26	25+02

Relationship between drinking variables and biochemical variables

Duration of drinking is positively correlated with A/G ratio (P<0.05) and Mean Corpuscular Volume (P<0.05).Life time alcohol intake is positively correlated with serum bilirubin (P<0.05) (Table 9).

Discussion

Chronic alcohol use is known to affect various system in human body producing Liver function abnormalities, Hematological abnormalities, Mineral deficiencies and Electrolyte abnormalities. In this study we aimed to examine the relationship between drinking variables and nutritional variables in otherwise nutritionally healthy subjects. Dietary contribution of nutritional deficiency in alcoholism has been studied. Ethanol is a rich source of Non-nutritive calories. Heavy drinking is often complicated by malnutrition. Drinking variables correlated significantly with Liver function tests. Compromised Liver metabolism could interfere with nutrition, which in turn may contribute to nutritional deficiency. The mean total caloric content in this group was below the recommended daily average intake for an Indian Male. Majority, that is more than sixty (60%) percent derived from the ethanol, which is essentially known to produce empty calories. This Observation is comparable with other studies that ethanol may supply more than 50 percent of the dietary energy [27,28]. The 24 hour dietary intake indicated decreased intake of other nutrients such as Protein, Phosphorous, Calcium, Iron and thiamine. Acute alcohol exposure interferes with the absorption of thiamine from the Gastro intestinal tract at low thiamine concentration [29]. Biochemical investigations reveal that most of the nutrients were in normal range except Serum Pyruvate, an indirect indicator of thiamine deficiency. The Serum Pyruvate this study is 318.47 mmol/L (Normal Value is 0.08 to 0.16mmo/L).Thiamine is required for the Pyruvate Metabolism. The estimation of Serum

Table 8: Biochemical Variables.

Biochemical variable	n	mean	SD	Range	Normal
Hb	40	13.46	2.22	10-19	14-18gm%
Mcv	40	81.50	6.85	62-92	80-90cumic
Mch	40	28.71	4.05	19-37	27-33mic gm
Mchc	40	34.53	2.29	28-38	33-38%
Tc	40	8975.75	2480.39	4900-14,600	33-385
Serum bilirubin	40	1.06	0.54	0.2-2.06	0.2-1.0u/l
SGOT	40	107.27	88.06	38-556	6-40u/l
SGPT	40	88.40	59.88	25-283	35-130u/l
Alk.phos	40	112.60	38.74	64-234	35-130u/l
GGT	40	97.67	98.16	8-387	0-40u/l
Total protein	40	7.37	0.42	6.6-8.7	6-8.6mg/dl
Albumin	40	4.20	0.24	3.60-4.80	3.5-5.0mg/dl
A/G ratio	40	1.74	0.11	1.5-2.12	
Urea	40	23.20	8.79	11-45	20-45mg
Creatinine	40	0.96	0.16	0.6-1.3	0.5-1.5mg
RBS	40	92.45	35.95	47-210	80-120mg
Cholesterol	40	183.90	31.42	124-260	110-220mg
HDL	40	74.75	36.78	36-240	35-65mg
Triglyceride	40	136.70	87.52	52-437	50-150mg
Sr. Calcium	40	9.23	1.97	6-12.60	8-10.5mg/dl
Sr. Phosphorous	40	3.87	0.65	2.2-5.1	3-4.5mg%
Sr. Iron	40	124.63	33.23	48-213	60-150mg/dl
Sr. Magnesium	40	2.30	0.32	1.82-3.10	1.6-2.5mg/dl
Sr. Zinc	40	123.62	36.18	80-271	70-127mg/dl
Sr. Sodium	40	139.27	2.73	133-145	130-145mg/l
Sr. Pattasium	40	4.08	0.42	3-4.9	3.5-5.0ng/l
Sr. Pyruvate	40	318.47	128.94	32-782	60-100mmol/l

Hb: Hemoglobin; Alk.Phos: Alkaline Phosphatase; Tc: Total Count; GGT: Gamma Glutamic Transaminase; Mcv: Mean corpuscular volume; A/G: Albumin/ Globulin; Mch: Mean Corpuscular Haemoglobin; HDL: High Density Lipoprotein; Mchc: Mean Corpuscular Haemoglobin Concentration; Sr: Serum; Tc: Total Count; SGOT: Serum Glutamic Oxaloacetate; SGPT: Serum Glutamic Pyruvic Transaminase

Table 9: Relationship between drinking variable and biochemical variable.

	Duration of drinking	Lifetime alcohol consumption	Quantity of alcohol intake
A/G Ratio	0.371*	0.222	0.176
Albumin	-337*	-110	0.016
Bilirubin	-105	0.342*	-029
Mch	0.221	0.087	0.333*
Mcv	0.316*	-128	0.273
Sr. Pottasium	0.228	-323*	0.023

*Correlation is significant at 0.05 level (*p-value < 0.05*)

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Pyruvate level may be of help in suspected Thiamine deficiency. The symptom of mild thiamine deficiency are vague and can be attributed to other problems. So the diagnosis of sub clinical thiamine deficiency is difficult. [30] Mark in 1975 Reported that a useful sign of mild and moderate thiamine deficiency is Myotactic irritability. Anorexia is one of the early Symptoms of Sub-clinical thiamine deficiency is regarded to be a protective phenomenon, since high carbohydrate diet is a most dangerous in the presence of thiamine deficiency [31]. In this study, anorexia was the most common medical symptom (47.5%), which correlates with the elevated serum pyruvate level and indicator of sub-clinical thiamine deficiency.

The most serious complication of thiamine deficiency is damage to the Central nervous system causing Wernicke's Korsokoff's Syndrome (WKS). That is characterized by the perventricular lesions including the mamillory bodies, other hypothalamic structures, periventricular thalamic nuclei and the structures from the floor of the fourth ventricle [32].

The discrepancy between the 24 hour dietary intake and biochemical investigation probably suggests that the dietary deficiencies have not yet resulted in obvious changes in biochemical measures. This also goes on hand with the observation of normal clinical assessment of nutrition and anthropometric measurements in this study sample. Research indicates that the majority of even the heaviest drinkers have few detectable nutritional deficiencies but many alcoholics who were hospitalized for medical complications of alcoholism do experience severe malnutrition [33,34]. This sample is moderate drinkers with no clinically evident medical complications. Hence,there is no clinically detectable nutritional deficiencies except serum pyruvate deficiency. The mean Body Mass Index (BMI) of this sample is 19.4 which falls within normal range for average Indian men. Fifty percent of the subjects had anorexia on clinical assessment which may have contributed for poor dietary intake.

Objective biochemical tests of thiamine status, particularly measurement of erythrocyte trasketolase activity (ETKA) and the Thiamine Pyrophosphate Effect (TPPE), provide a sensitive test for thiamine deficiency where facilities are available [35].

Thiamine is required for Pyruvate metabolism. Increased blood Pyruvate and Lactate levels can be caused by thiamine deficiency. The estimation of blood pyruvate can be of help in the diagnosis of suspected thiamine deficiency. Chronic Wernicke's Encephalopathy and korsakoff's Psychosis may result from episodes of Sub-clinical thiamine deficiency [36]. The clinician treating for Wernicks Encephalopathy has a window of opportunity when an adequate supply of thiamine to the brain can reverse the biochemical lesion and limit the permanent brain damage [37].

Countries where people normally receive adequate thiamine from their diet, thiamine deficiency is most commonly caused by alcoholism. The role of thiamine in the development of Wernicks korsokoff's syndrome is supported by reversal of symptoms by giving thiamine nutrient. Abstinence from alcohol and improved nutrition have been shown to reverse some of the impairments associated with thiamine deficiency including improving brain functioning [38]. In some studies researchers administered thiamine to alcoholic patients and Laboratory animals and found that this treatment reversed some of the behavioral and metabolic consequences of thiamine deficiency [39,40]. Researchers also administered different thiamine doses for two days to a group of alcoholics with no diagnosis of Wernicke's korsakoff's psychosis and tested the participants working memory. Participants who received highest thiamine dose performed well on tests of working memory [41]. Some people may exhibit more subtle neurological symptoms such as abnormalities in a brain region called cerebellum, inflammation and degeneration of peripheral nerves as well as changes in behaviour and problems with learning, memory and decision making. Cerebellum is particularly sensitive to thiamine deficiency [42]. Accordingly in their countries Wernicke's Encephalopathy is primarily found in alcoholics.

The elevated Serum Pyruvate in this study suggests an underlying Thiamine deficiency. Direct estimation of thiamine would be more appropriate, since that is more sensitive indicator of Thiamine deficiency.

Conclusion

This study of Moderate drinkers of Indian male with no clinical parameters indicating malnutrion reported Reduced Daily intake of calories than an average that is due to poor diet intake in alcoholics due to empty calories from ethanol and elevated serum pyruvate which is an indicator of sub-clinical thiamine deficiency. Also the common presentation of anorexia in this study, an indicator of sub-clinical thiamine deficiency improves the validity of sub clinical thiamine deficiency in this study. Hence, otherwise normal healthy drinkers both clinically and on routine biochemical evaluation, may have sub clinical thiamine deficiency. Anorexia also emerged as a clinical indicator as well as Protector against Wernicke's Encephalopathy and Wernicke's Korsakoff 's psychosis in this study. Alcohol related brain damage is reversible if thiamine supplementation is given. Hence, all the alcohol dependent subjects may benefit from thiamine supplementation, though they are nutritionally intact to prevent the precipitation of Wernicke's Encephalopathy and Wernicke's Korsakoff's Psychosis. Since this observation is cross sectional, more longitudinal study with direct thiamine estimation will be more convincing in future. In developing countries all the alcoholics irrespective of apparent nutritional deficiency may benefit from thiamine supplementation to prevent alcohol related acute brain damage.

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