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A STUDY OF CORRELATION BETWEEN SERUM VITAMIN B12 AND FOLIC ACID IN ALCOHOLIC LIVER DISEASE

Medicine

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ABSTRACT

Alcohol remains a major cause of liver disease worldwide. It is common for patients with ALD to share the risk factors for simultaneous injury from other liver insults (e.g., coexisting non-alcoholic fatty liver disease or chronic viral hepatitis). Many of the natural history studies of ALD and even treatment trials were performed before these other liver diseases were recognized, or specific testing was possible. The study was done on 25 patients admitted to Tertiary care hospital, Bengaluru during the period of January 2019 to December 2019. In the present study 11 cases were belongs to 21 to 30 year age group followed by 9 cases belongs to 31-40 year age group. Among patients studied, 88% were males and 12% were females, 11 patients had high Vitamin B12, 14 patients had normal Vitamin B12 & none had low Vitamin B12, 8 had low folic acid, 17 patients had normal folic acid & none had High folic acid and 6 patients were alcoholic fatty liver, 6 patients were alcoholic steatohepatitis, 7 were cirrhosis and 6 were cirrhosis in hepatic encephalopathy. Plasma levels of vitamin B12 in patients with decompensated chronic liver disease are high, whereas plasma folate levels are low. The ratio between vitamin B12 and folic acid may be useful in the differential diagnosis of the etiology of chronic liver disease.

KEYWORDS

Serum Vitamin B12, Folic Acid, Alcoholic Liver Disease

INTRODUCTION:

Alcohol remains a major cause of liver disease worldwide. It is common for patients with ALD to share the risk factors for simultaneous injury from other liver insults (e.g., coexisting non-alcoholic fatty liver disease or chronic viral hepatitis). Many of the natural history studies of ALD and even treatment trials were performed before these other liver diseases were recognized, or specific testing was possible. Thus, the individual effect of alcohol in some of these studies may have been confounded by the presence of these additional injuries. Despite this limitation, the data regarding ALD are robust enough to draw conclusions about the pathophysiology of this disease. The possible factors that can affect the development of liver injury include the dose, duration, and type of alcohol consumption, drinking patterns, gender, ethnicity, and associated risk factors, including obesity, iron overload, concomitant infection with viral hepatitis, and genetic factors. Geographic variability exists in the patterns of alcohol intake throughout the world. The majority drinks small or moderate amounts and do so without evidence of clinical disease.[1,2,3]

The population-level mortality from ALD is related to per capita alcohol consumption obtained from national alcoholic beverage sales data. There are conflicting data regarding a possible lower risk of liver injury in wine drinkers. One epidemiological study has estimated that for every increase in per capita alcohol consumption (independent of the type of beverage), there was a 14 % increase in cirrhosis in men and 8 % increase in women. These data must be considered in the context of the limitations of measuring alcohol use and defining ALD. The scientific literature has also used a variety of definitions of what constitutes a standard drink. Most studies depend on Interviews with patients or their families to quantify drinking patterns, a method that is subject to a number of biases, which may lead to invalid estimate of alcohol consumption.[4]

Although there are limitations of the available data, the World Health Organization s Global Alcohol database, which has been in existence since 1996, has been used to estimate the worldwide patterns of alcohol consumption and allow comparisons of alcohol-related morbidity and mortality10. The burden of alcohol-related disease is the highest in the developed world, where it may account for as much as 9.2 % of all disability adjusted life years. However, even in the developing regions of the world, alcohol accounts for a major portion of the global disease burden, and is projected to take on increasing importance in those regions over time.The spectrum of alcohol-related liver injury varies from simple steatosis to cirrhosis. Fatty liver develops in about 90 % of individuals who drink more than 60 g /day of alcohol, but may also occur in individuals who drink less. Simple, uncomplicated fatty liver is usually asymptomatic and selflimited, aand may be completely reversible with abstinence after about 4 – 6 weeks.[5]

However, several studies have suggested that progression to fibrosis and cirrhosis occurs in 5 – 15 % of the patients despite abstinence. In one study, continued alcohol use (>40 g /day) increased the risk of progression to cirrhosis to 30 %, and fibrosis or cirrhosis to 37%.

OBJECTIVES:

To measure serum vitamin B12 in various stages alcoholic liver disease.

METHODOLOGY:

The study was done on 25 patients admitted to Tertiary care hospital, Bengaluru during the period of January 2019 to December 2019.

INCLUSION CRITERIA:

Patients more than 18 years of age.
Patients who are satisfying AUDIT questionnaire and AASLD practical guidelines for alcoholic liver disease.

EXCLUSION CRITERIA:

Patients on methotrexate and metformin

RESULTS:

Table 1: Age Wise Distribution Of Cases:

Age in years	No. of patients	%
21-30	11	44
31-40	09	36
41-50	03	12
>50	02	08
Total	25	100

In the present study, 11 cases were belongs to 21 to 30 year age group followed by 9 cases belongs to 31-40 year age group. Among patients studied, 88% were males and 12% were females.

Table 2: Serum Vitamin B12 Levels Of Patients:

SERUM VITAMIN B12 LEVEL	GENDER		TOTAL
	MALE	FEMALE	
<187	0	0	0
187-883	12	2	14
>883	10	1	11
TOTAL	22	03	25

11 patients had high Vitamin B12, 14 patients had normal Vitamin B12 & none had low Vitamin B12.

Table 3: Serum Folic Acid Levels Of Patients:

SERUM FOLIC ACID LEVEL	GENDER		TOTAL
	MALE	FEMALE	
< 1.6	07	01	08
1.6 – 19.5	15	02	17
>19.5	00	00	00
TOTAL	22	03	25

8 had low folic acid, 17 patients had normal folic acid & none had High folic acid.

Table 4: Diagnosis:

DIAGNOSIS	GENDER		TOTAL
	MALE	FEMALE	
ALCOHOLIC FATTY LIVER	05	01	06
ALCOHOLIC STEOTOHEPATITIS	04	02	06
ALCOHOLIC CIRRHOSIS	07	00	07
ALCOHOLIC CIRRHOSIS IN HEPATIC ENCEPHALOPATHY	06	00	06
TOTAL	22	03	25

6 patients were alcoholic fatty liver, 6 patients were alcoholic steatohepatitis, 7 were cirrhosis and 6 were cirrhosis in hepatic encephalopathy.

DISCUSSION:

In the present study 11 cases were belongs to 21 to 30 year age group followed by 9 cases belongs to 31-40 year age group. Among patients studied, 88% were males and 12% were females, 11 patients had high Vitamin B12, 14 patients had normal Vitamin B12 & none had low Vitamin B12, 8 had low folic acid, 17 patients had normal folic acid & none had High folic acid and 6 patients were alcoholic fatty liver, 6 patients were alcoholic steatohepatitis, 7 were cirrhosis and 6 were cirrhosis in hepatic encephalopathy.

Sorensen et al[6] showed with increasing age severity of alcoholic liver disease increases and probably due to greater magnitude of alcohol consumption.

JiuFeng Dou et al[7] in his study showed that in chronic liver failure patients had significantly higher B12 levels at admission compared with healthy controls ($P < 0.001$).

Elevated B12 levels were associated with increased severity of liver disease and 3-month mortality rate. Multivariate analysis demonstrated that B12 levels and the model for endstage liver disease score were independent predictors for mortality (both $P < 0.001$)

Fragasso A et al[8] in their retrospective study of concluded that some alcohol-dependent patients with megaloblastic anemia may respond to vitamin B12 treatment despite normal or high cobalamin serum levels; therefore in alcoholics caution is urged in the interpretation of these vitamin assays, because of possible functional vitamin B12 deficiency. Medici V et al[9] in his study showed that Alcoholic liver disease (ALD) is typically associated with folate deficiency, which is the result of reduced dietary folate intake, intestinal malabsorption, reduced liver uptake and storage, and increased urinary folate excretion. Folate deficiency favors the progression of liver disease through mechanisms that include its effects on methionine metabolism with consequences for DNA synthesis and stability and the epigenetic regulation of gene expression involved in pathways of liver injury.

Wani NA[10] et al by immunohistochemical studies showed that alcoholic conditions deranged that localization of PCFT and RFC(folate transporters). These findings could explain the possible mechanistic insights that may result in folate malabsorption during alcoholism.

CONCLUSION:

Plasma levels of vitamin B12 in patients with decompensated chronic liver disease are high, whereas plasma folate levels are low. The ratio between vitamin B12 and folic acid may be useful in the differential diagnosis of the etiology of chronic liver disease.

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