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Lipids & lipoproteins in amoebic liver abscess

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Fasting blood samples from 70 patients of amoebic liver abscess during acute state of illness and 62 normal healthy controls were tested for lipids and lipoproteins *i.e.*, VLDL, LDL and HDL, and caloric intake from various nutrients was calculated. Mean caloric intake was significantly low in patients with amoebic liver abscess (ALA) as compared to control subjects. The levels of cholesterol and its lipoproteins were also significantly low with non-significant changes in triglyceride levels in patients with ALA. In 17 patients with ALA, fasting blood sample was also obtained 15-20 days after discontinuation of treatment, where clinical recovery had taken place. In these 17 patients, there was no change in lipids and lipoproteins level, before and after treatment. During this period there was no significant change in their caloric intake. The result of the present study showed failure of an early biochemical recovery in patients of amoebic liver abscess who had clinically recovered.

Protein-calorie malnutrition is the most common form of undernutrition in a large number of the countries of the third world[1] where amoebic infection is also very common[2]. Clinical manifestations and higher incidence of amoebic infection have been shown to be related to the malnutrition[3]. In acute amoebic liver abscess (ALA), where lesions of hepatocytes are marked, the synthesis of lipids and lipoproteins are significantly reduced. Low levels of plasma cholesterol and lipoprotein fractions have been demonstrated in patients with amoebic liver abscess[4] during the acute stage of infection. However, reports are not available on lipids and lipoproteins after clinical recovery from the disease. In this report we present data on lipids and lipoproteins in patients with amoebic liver abscess, during and after clinical recovery.

Material & Methods

Fasting blood samples were drawn from 70 patients of amoebic liver abscess (mean age 37±8 yr) admitted to the wards of Department of Gastroenterology, All India Institute of Medical Sciences (AIIMS), New Delhi. The diagnosis of amoebic liver abscess was according to the recommendations of the WHO Expert Committee[5]. In addition to the clinical findings, all patients had (*i*) liver scan or ultrasound showing the cold region; (*ii*) aspiration of 'anchovy sauce' pus from the swelling of the liver which was sterile for

bacterial culture; and (*iii*) an excellent therapeutic response to metronidazole treatment.

The pus was aspirated and the patients were treated with a dose of 2·4 g/day of metronidazole for 5 days. In 17 of these treated patients, fasting blood samples were also drawn 15 to 20 days after discontinuation of the drug. In addition, fasting blood samples were drawn from 62 normal healthy volunteers (mean age 35±10 yr) of both sexes, matched for age and socio-economic status. There was no recent or past history of amoebic liver abscess and intestinal amoebiasis in these subjects and no cysts and trophozoites of *Entamoeba histolytica* were seen in their stool samples. All of them showed negative serology by indirect haemagglutination assay (IHA).

The dietary history was obtained from 62 control subjects and 59 patients by recall method and various nutrients *i.e.*, carbohydrates, fats, proteins and total energy intake, were calculated. The height and weight was recorded.

After overnight fasting (12-14 h) blood samples were drawn in 1 mg/ml EDTA-disodium salt for lipids and lipoprotein estimation. The samples were centrifuged and stored at 4°C and processed with in 48 h of collection.

Quantification of lipoprotein fractions was carried out by the dual precipitation technique used by Wilson and Spiger[6]. Cholesterol and triglycerides were determined in plasma, HDL and HDL+LDL fractions by the method of Chaimori and Henry[7] and Van Handel and Zilversmit[8] respectively. The values for VLDL were obtained by substraction. Liver function tests, *viz.*, serum transaminases, alkaline phosphatase, bilirubin and proteins were carried out by standard techniques.

Results

With age and height being the same, the average weight of patients with amoebic liver abscess (55±9 kg) was significantly ($P<0·001$) lower than that of controls (65±7 kg). Total serum protein and serum albumin levels were also significantly ($P<0·001$) low (Table I).

In patients with amoebic liver abscess during the acute stage, the mean energy intake (1768 kcal) was significantly lower as compared to controls (2299 kcal; Table II).The intake of proteins, carbohydrates and fats by patients was also significantly low ($P<0·001$).

The levels of total cholesterol, and its lipoproteins *i.e.*, cholesterol in VLDL, LDL and HDL fractions were significantly lower in patients of amoebic liver abscess (Table III). However, the difference in values for triglycerides in these fractions

Table I. Height, weight, serum protein and albumin in patients with amoebic liver abscess (ALA)

Parameter	Control (n=62)	ALA (n=59)
Weight (kg)	65±7	55±9*
Height (cm)	171±7	168±10**
Serum protein (g/dl)	7·6±0·5	6·3±1·2*
Serum albumin (g/dl)	4·2±0·7	3·2±0·7*

P ... *0·001; **not significant

was non-significant except for HDL triglycerides where the difference was significant ($P<0{\cdot}001$).

In 17 patients of amoebic liver abscess, a second blood sample was drawn, 15-20 days after the cessation of drug therapy and detailed diet history was taken. Neither the difference in intake of total calories from nutrients (Table IV) nor the levels of lipids and lipoproteins (Table V) were found to be significant during the acute and recovery phase in patients suffering from amoebic liver abscess.

Table II. Nutrients intake in control subjects and patients with amoebic liver abscess
(Data are mean±SD)

Group	Amoebic liver abscess (n=59)	Control (n=62)
Calories (kcal)	1768±532*	2299±443
Proteins (g)	54±16*	72±15
Carbohydrates (g)	271±81*	334±78
Fats (g)	52±30*	75±26

*$P<0{\cdot}001$

Discussion

The results of the present study showed low values for cholesterol and its lipoprotein fractions in patients of amoebic liver abscess during the acute stage. The intake in terms of calories from proteins, carbohydrates and fats was significantly low. The significantly low body weight and low levels of serum albumin and total protein in patients of our series also support the underlying nutritional status of these patients. An association between malnutrition in amoebic liver abscess and abnormal lipids and lipoprotein fractions has been reported in clinical[9] and experimental[10] studies. Malnutrition in the host and a higher incidence of amoebic infection with greater severity of the disease has also been reported[3]. Clinical manifestations of amoebiasis have been shown to be more severe in poorly nourished individuals, as compared to well nourished ones[11]. Reduced dietary intake

Table III. Lipoprotein profile in control subjects and patients with amoebic liver abscess
(Data are mean±SD)

Group	Cholesterol, mg/dl				Triglycerides, mg/dl			
	Total	VLDL	LDL	HDL	Total	VLDL	LDL	HDL
Amoebic liver abscess (n=70)	90±38*	26±23*	43±22*	21±16*	144±53	43±42	33±27	63±33*
Controls (n=62)	156±30	39±17	83±24	35±15	129±37	47±34	35±19	47±20

*$P<0{\cdot}001$

and the subsequent tissue disturbances could make the liver more susceptible to invasion by amoebae[12].

Low levels of serum cholesterol due to dietary deficiencies in ALA patients are said to aggrevate the illness and contribute in reducing lipid levels in blood during acute infection[13,14]. Reduced synthesis of endogenous cholesterol has also been reported in rats[15] and chicks[16] infected with trophozoites. The changes in cholesterol and its lipoprotein fractions could also be attributed to reduced endogenous synthesis by the paranchymal cells whereas triglycerides, which are more related to exogenous lipids remain unaltered. Low levels of lipids and lipoproteins have also been demonstrated[4] in patients with ALA having extensive liver lesions.

Lower levels of lipids and its lipoprotein fractions seen after treatment in the present study could not be attributed to the effect of metronidazole as it has already been reported[17] that lowering effect of metronidazole on cholesterol and triglycerides persist only for a maximum period of one week after the cessation of drug therapy. A number of reports reviewed by Diamond[3], report alleviation of symptoms and eradication of the parasite by improvement in the diet. In hamsters infected with trophozoites, which produced liquification of large parenchymal area, after treatment with metronidazole, the shift in values of lipids towards control levels has also been demonstrated by Gujral *et al*[18].

Table IV. Nutrient intake in patients with amoebic liver abscess during acute and recovery stage (n=17)*

(Data are mean±SD)

Group	ALA (acute)	ALA (recovered)
Calories (kcal)	2156±745	2629±1026
Proteins (g)	64±18	72±28
Carbohydrates (g)	304±86	358±132
Fats (g)	76±44	101±47

*Values are presented only for the same 17 patients where the data were available for acute stage and recovered stage. There was no significant difference in the values before and after treatment

Table V. Lipoprotein profile in patients with amoebic liver abscess during acute and recovery stage (n=17)

(Data are mean±SD)

Group	Cholesterol, mg/dl				Triglycerides, mg/dl			
	Total	VLDL	LDL	HDL	Total	VLDL	LDL	HDL
ALA (acute)	90±43	23±22	42±20	25±16	147±37	28±37	22±20	94±21
ALA (recovered)	99±52	23±18	55±35	28±15	126±22	16±14	16±14	95±12

There was no significant difference in the values before and after treatment

During the recovery stage, although the active regeneration of the liver tissue starts at an early date[19], the process of synthesis and secretion of lipids and lipoproteins is yet to record. This inability for synthesis and secretion of lipids and lipoproteins in patients of amoebic liver abscess seems to be related to incomplete maturation of paranchymal cells due to malnutrition or the disease itself. This phenomenon may be responsible for failure of an early biochemical recovery.

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