LIFESTYLE, NUTRITIONAL STATUS AND SEROCLINICAL PROFILE OF LIVER CIRRHOTIC PATIENTS

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Abstract

Liver cirrhotic patients were investigated for their lifestyle, energy intake, BMI, biochemical and serological indices and prevalent clinical signs. The study included 50 liver cirrhotic patients who were hospitalized in Bangabandhu Sheikh Mujib Medical University, Dhaka Medical College Hospital and BIRDEM Hospital during the period of 1999-2000. Of the fifty cirrhotic patients, hepatitis B virus infection was found responsible for 52% (n=26) of liver cirrhosis, hepatitis C was for only 6% (n=3) patients and etiology for the rest 42% (n=21) patients could not be identified. It was observed that literate, low-mid income and productive male were being suffering from the liver cirrhosis. The patients had good housing status. Low energy intake (1670.6±42.9) and low BMI (19.52±3.48) were present in almost all of the patients. Mean serum haemoglobin, total proteins, and albumin were 96.2±20.5g/L, 63.1±1.01g/L, and 31.30±0.93g/L respectively. Serum bilirubin was raised in 50% patients. Serum SGPT and SGOT levels were increased from 2 to 4 folds in 50% of the patients; alkaline phosphatase was increased only in 8% cirrhotic patients. Anaemia, PEM, ascities, oedema, muscle wasting were prevalently present in vast majority of the patients.

Key words: Liver cirrhosis, nutritional status, liver function determinants

Introduction

The liver is the second largest organ in human body. It is responsible for performing more functions than any other organ in the body, including metabolizing the food, filtering and detoxifying waste and toxic products, producing immune agents to control infection, and regenerating itself[1]. Its another important function is to produce prothrombin and fibrinogen (blood-clotting factors) and heparin (that helps prevent blood from clotting within the circulatory system). The liver also synthesizes proteins, cholesterol and enzymes; produces and secretes bile; and stores essential micronutrients.

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Liver cirrhosis is a chronic, diffuse, degenerative disease in liver, in which the parenchyma deteriorates, the lobules are infiltrated with fat and structurally altered, dense perilobular connective tissues, and often areas of regeneration develop. The surviving cells multiply in an attempt to regenerate and form "islands" of living cells that are separated by scar tissue. These islands of living cells have a reduced blood supply, resulting in impaired liver function. As the cirrhotic process continues, blood flow through the liver becomes blocked; portal hypertension may occur, glucose and vitamin absorption decrease, the manufacturing of hormones, enzymes and stomach and bowel function are affected, and noticeable facial veins may appear. Most patients die from cirrhosis in the fifth or sixth decade of life[2]. Approximately one-third of cirrhosis cases are "compensated," Cirrhosis is irreversible. Unless the underlying cause of cirrhosis is removed and the person takes measures to treat the condition, the liver will continue to incur damage, eventually leading to liver failure, ammonia toxicity, gastrointestinal hemorrhage, kidney failure, hepatic coma, and death. For some people, the only chance for a long-term cure is a liver transplant.

The most commonly markers of liver cirrhosis or hepatocyte injury are serum glutamic-oxaloacetic transaminase (SGOT]), serum glutamate-pyruvate transaminase (SGPT), alkaline phosphatase[3]. Anaemia, protein energy malnutrition, jaundice, ascities, fluid retention are the most frequent clinical symptoms associated with liver cirrhosis.

It is evident that most of the infections and nutritional deficiency diseases in developing countries are the "disease of poverty." Poverty predisposes to malnutrition, the major public health problem in developing countries like Bangladesh. In Bangladesh there have been several studies conducted on anaemia, vitamin A deficiency, iodine deficiency, but a very few attempts have taken to investigate into liver cirrhosis. Liver cirrhosis is one of the common health problems in Bangladesh. About 20 percent population has been suffering from different type of viral infections, one of the commonest cause is liver cirrhosis. This study has, therefore, investigated the lifestyle, nutritional status, liver function determinants and clinical signs of liver cirrhotic patients

Materials and Methods

Study population. It was a cross sectional study conducted among 50 liver cirrhotic patients who were hospitalized in Bangabandhu Sheikh Mujib Medical University, Dhaka Medical College Hospital and BIRDEM Hospital during the period of 1999-2000. Lifestyle, anthropometric data, energy intake data, clinical information were collected by interviewer administered

pre-tested questionnaire. The patients were explained about the purpose of the study, and on obtaining their consent, data were collected from the voluntarily participating patients. A 5ml venous blood specimen was collected from each of the patients for biochemical and serological analysis.

Blood analysis. A portion of blood was used for haemoglobin estimation. The remaining blood was spun at 3000rmp for 10min to extract serum, which was then aliquoted in eppendorf tubes and stored at -20^0C for analysis of biochemical and serological determinants.

Haemoglobin was estimated by cyanomethaemoglobin method using a commercial kit. Analyses of serum bilirubin, SGPT, SGOT, alkaline phosphatase, total protein and albumin were carried out by Hitachi 911 Automated Analyzer. Calibration, calculations were performed by a built-in-computer program. The analyzer was programmed for the tests. One ml serum was pipetted into reaction cells of the analyzer. The analyzer performed all programmed tests on each sample without operator intervention. After analysis, results were printed out in numerical values.

Statistical analysis. SPSS software package (version 10.0, SPSS Inc. Chicago, USA) was used to analyse the data. Descriptive statistics were calculated for all variables. Values were expressed as percentage and mean±SD.

Results

Of the fifty liver cirrhotic patients, hepatitis B virus infection was responsible for 52% (n=26) of liver cirrhosis, hepatitis C was for only 6% (n=3) patients and aetiology for the rest 42% (n=21) liver cirrhosis could not be identified. Socioeconomic status of the liver cirrhotic patients is presented in table 1. It was observed that the most of the patients investigated were literate (class viii to graduate and above), low to mid income (US$50-100) and were productive aged (19-49 years) male. The majority (64%) of the patients had their own house, most of which were pucca and tin built and had multiple room facilities (3 to above). Water supply and latrine facilities were also found to be adequate.

Table 2 shows the average energy intake, BMI and MUAC values of the liver cirrhotic patients. Mean energy intake was 1870.6±47.6 comprising carbohydrate 411.6±15.6g/day, protein 25.8±2.7g/day and fat 13.41±1.8g/day. The mean BMI of the patients was 19.52±3.48, of which 52% were being suffering from chronic energy deficiency. MUAC value (mean 19.81±2.71cm) showed 54% cirrhotic patients were malnourished.

Table-I: *Socioeconomic status of liver cirrhotic patients (n=50)*

Socio economic condition	Case subjects	
	Number	Percentage
Education		
Illiterate	14	28
Literate		
Upto class viii	12	24
Class IX-HSC	7	14
Graduate & above	8	16
Monthly income in taka		
Upto 3000	26	46
3001–6000	20	40
>6000	4	8
Age in year		
Upto 19	2	4
20-29	5	10
30-39	9	18
40-49	14	28
50 & above	20	40
Age in year		
Upto 19	2	4
20-39	19	38
40-59	24	48
60 & above	5	10
Sex		
Male	38	76
Female	12	24

Table-II: *Residential style of liver cirrhotic patients*

Residential pattern	Case subjects	
	Number	Percent
House occupied		
Own	32	64
Rented	18	36
House type		
Pucca	18	36
Pucca with tinroof	8	16
Tin	14	28
Thatched	10	20
Number of rooms		
1-2	21	42
3-6	28	56
>6	1	2
Source of water		
Tape 15	30	
Tube well	35	70
Latrine facilities		
Sanitary	33	66
Service	12	24
Others	3	6
Living at		
Union	23	46
Thana	9	18
District	18	36

Table-III: *Nutritional indices (energy intake, BMI, MUAC) of liver cirrhotic patients*

Energy intake	Carbohydrate Gm/day	Protein	Fat	Kcal/day
	411.6±15.6	25.8±2.7	13.41±1.8	1870.6±47.6
BMI (kg/m^2)	Number	Percent	Mean±SD	
<16	9	18		
16-18.4	17	34	19.52±3.48	
>18.4	24	48		
MUAC (cm)				
<20	10	20	19.81±2.71	
20-23	17	34		
>23	23	46		

Table-IV: *Biochemical and serological indices of liver cirrhotic patients*

Parameter	Subjects	
	n(%)	Mean± SD
Biochemical		
Haemoglobin (g/L)		
60-89	17 (34)	96.2±20.5
90-109	21(42)	
> 110	12(24	
Total Protein (g/L)		
< 60	22(44)	63.1±1.01
60-69	19(38)	
>70	9(18)	
Albumin (g/L)		
<30	30(60)	31.30±0.93
30-34	12(24)	
35 and above	8(16)	
Serological		
Serum bilirubin (mg/dl)		
Normal (<1.0)	25(50)	
Mild (1.1-6.0)	23(46)	2.05±2.98
Severe (>12.1)	2(4)	
SGPT (U/l)		
<40	21(42)	
40-80	24(48)	52.89±26.72
81-160	5(10)	
SGOT (U/l)		
< 35	25(50)	
36-80	21(42)	51.61±37.36
>81	4(8)	
Alkaline phosphatase (U/l)		
200-400	46(92)	183.18±134
>400	4(8)	

The cut off values for anaemia and PEM are haemoglobin <130g/L (FAO, 1995)[4], serum proteins <70g/L (Rand and Murray, 2000)[5] and albumin <35g/L (Ganong, 1997)[6] respectively.

Biochemical analysis showed that mean haemoglobin value of liver cirrhotic patients was 96.2±20.5g/L (table 4) indicating they were anaemic. Mean protein and albumin values were 63.1±1.01g/L and 31.30±0.93g/L (table 4). Low protein value was present in 82% (n=41) patients and low serum albumin level was found in 84% (n=42) patients representing a highly prevalent multiple PEM in them. Liver function determinants are described in table 5. There had significant increase in serum SGPT (52.89±26.72U/L) and SGOT (51.61±37.36U/L). Two fold (41-80U/l) and four fold (81-160U/l) rises of SGPT were found in 40% and 10% cirrhotic patients. Serum SGOT value was also increased in the same pattern. Mild to severe rise of serum bilirubin were noted in 50% of the patients. Alkaline phosphatase was increased only in 8% patients. Mild to severe signs of anaemia, PEM, jaundice, ascities, oedema, muscle wasting were present in vast number of cirrhotic patients (table V).

Table-V: *Clinical signs in liver cirrhotic patients*

Clinical sign	Severity	Number	Percent
Anemia	Severe	17	34
	Moderate	21	42
	Mild	12	24
PEM	Severe	22	44
	Moderate	19	38
	Absent	9	18
Joundice	Severe	2	4
	Moderate	7	14
	Mild	16	32
	Absent	25	50
Oedema	Severe	0	0
	Moderate	6	12
	Mild	26	52
	Absent	18	36
Ascitis	Severe	4	8
	Moderate	17	34
	Mild	22	44
	Absent	7	14
Muscle wasting	Present	46	92
	Absent	4	8

Discussion

Liver cirrhosis is one of the common health problems in Bangladesh. Infections and nutritional deficiency diseases are the “disease of poverty.” Poverty predisposes to malnutrition, the major public health problem in the developing countries like Bangladesh. Likely multiple malnutritions are frequent and common in liver cirrhosis. Occurrence of malnutrition in cirrhotic patients is however strongly associated with the progressive deterioration of liver function[7]. It was noted that

the prevalence of malnutrition in chronic liver failure is highly influenced by the severity of the disease.

In the present study, most of the cirrhotic patients were literate, low-mid income class and were in productive age group, but they had apparently good living and sanitation facilities. However, information on their socioeconomic and living status or lifestyle is scarce in Bangladesh. Dooley (1993)[8] reported that this disease is prevalent in low socioeconomic people, which is nearly similar to the present findings.

The energy intake by the cirrhotic patients was found to be low as compared to the RDA. The main source of energy was carbohydrate. Protein and fat consumption was very poor. This result is somewhat consistent with the report of Muller (1995)[6]. In general, malnourished patients with cirrhosis have an increase protein requirement in order to achieve nitrogen balance[9-10]. The poor energy intake was due to poor appetite, in taking poor nutrient containing foods and also because of ascities, odema etc. This further aggravates the nutritional status.

Poor nutritional status, which was prevalent in the cirrhotic patients, might be associated nutritional and metabolic disorders. Biochemical determinants and clinical signs further identified a high prevalence of anaemia and PEM in the liver cirrhotic patients, association of which has also been documented by several investigators[7, 11-13].

Serological analysis of liver function determinants showed a significant increase of bilirubin, SGOT and SGPT. Alkaline phophatase was unchanged. These are common outcome in cirrhotic patients. In compensated cirrhosis, serum bilirubin, SGPT, SGOT, ALP remains unchanged[14]. Presence of ascities, oedema, muscle wasting are also associated with liver cirrhosis.

It was noted in the present study that hepatitis B viral infection was responsible for 52% of liver cirrhosis, hepatitis C was for only 6% cirrhosis and the aetiology for the rest 42% liver cirrhosis could not be identified. This is well matched with the report for aetiology of liver cirrhosis for Asia and Africa. The 42% liver cirrhosis may be of autoimmune hepatitis, alcoholic hepatitis, or because of drugs or toxins, liver muscle injury, tumour growth, fatty liver etc[3].

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