Lipid Pattern in Liver Disease

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Abstract

Lipid pattern was determined in ninety six control subjects and one hundred and thirty patients with liver diseases i.e., hepatitis, cirrhosis and cancer.
Serum cholesterol, triglycerides and low density lipoprotein were significantly elevated in hepatitis and cancer. Hypertriglyceridaemia was found in 60% cases of both diseases. A significant decline in total lipids, cholesterol, triglycerides, phospholipids and low density lipoprotein was observed in cirrhosis. Hypocholcsterolaemia was found in 48% and hypolipoproteinaemia in 80% cirrhotics.
A positive correlation coefficient was found between bilirubin and triglyceride in hepatitis and cancer and the result was statistically significant (JPMA 32:275, 1982).

Introduction

Liver plays a paramount role in metabolic functions of the body and is actively involved in many phases of lipid metabolism. The metabolism of lipid embraces the metabolism of free fatty acids, fats, phosphatides, sterols and other complex lipids.
Alterations have been observed in various fractions of serum lipids in patients with liver disease (Man et al., 1945; Kunkel and Slater, 1952;Ederetal., 1955; Phillips, 1960; Levyetal., 1966; Smith et al., 1967; Wallach, 1974; Eastham, 1975; Feher, 1976; Vialet et al., 1962; Santer et al., 1967; Alport et al., 1969; Mclntyrc, 1978; Teloh, 1978).
This study was undertaken to determine the levels of cholesterol, triglycerides, phospholipids and lipoproteins in patients with hepatitis, cirrhosis and liver cancer and their correlation with other liver function tests.

Material and Methods

Serum total lipids, cholesterol, triglycerides, phospholipids and low density lipoproteins were estimated in 96 healthy subjects, 50 patients with acute viral hepatitis, 50 cirrhotics and 30 with liver cancer. The subjects were of both sexes and represented various age and socio economic groups.
Controls were selected on the basis of normal liver function tests whereas liver diseases were classified on clinical examination, biochemical investigations and liver biopsy. The biochemical tests were determined by the following methods.
Total lipids by the modified method of Kunkel et al. (1948), Cholesterol by Ferro and Ham (1960), Phospholipids were estimated by the method of Youngberg and Youngberg (1930) and lipoproteins were determined using the method of Walton and Scott (1964). Triglycerides were estimated using Biomerieux kit.

Results

The age and sex distribution in control subjects and patients with liver disease is presen ted in tabje I.

Table I

Age and Sex Distribution in Controls and Cases of Hepatitis, Cirrhosis and Cancer

Age Groups	*Controls* (96)		*Hepatitis* (50)		*Cirrhosis* (50)		*Cancer* (30)	
(Years)	*Male*	*Female*	*Male*	*Female*	*Male*	*Female*	*Male*	*Female*
20—29	17	25	22	4	1	4	—	—
30—39	9	13	9	2	7	6	—	—
40—49	5	11	6	2	5	10	3	5
50—55+	4	12	4	1	13	4	9	13

Figures in parenthesis indicate total number of cases.

Table II **Lipid Pattern in Controls and Cases of Hepatitis, Cirrhosis and Cancer**

Groups	*Number of Cases*	*Total Lipids (mg)*	*Cholesterol (mg)*	*Triglycerides (mg)*	*Phospho Lipids (mg)*	*Low Density Lipoprotein (mg)*
		Mean±S.E.	*Mean±S.E.*	*Mean±S.E.*	*Mean±S.E.*	*Mean±S.E.*
Controls	96	673.7 ±15.04 (377—887)	194.9 ± 4.2 (122—272)	123.9 ± 3.7 (44—195)	147.4 ± 5.11 (62—308)	439.8 ±12.6 (220—694)
Hepatitis	50	* 789.08 ±42.88 (387—1961)	** 241.5 ±12.95 (136—695)	*** 218.3 ±15.8 (44—470)	162.2 ±15.31 (60—750)	** 590.984 ±1.4 (242—1400)
Cirrhosis	50	*** 553.84 ±18.44 (327—906)	*** 152.62 ± 6.47 (75—285)	** 97.1 ± 6.5 (34—306)	* 125.72 ± 7.4 (52—289)	*** 285.08 ±14.8 (180—696)
Cancer	30	766.8 ± 59.65 (358—1781)	* 235.53± 19.5 (110—562)	*** 193.9± 14.6 (87—382)	205.36± 34.6 (39—980)	* 602.66± 68.33 (180—1890)

Range is shown in parenthesis

* P <0.05 as compared to controls.

** P <0.01 as compared to controls.

*** P <0.001 as compared to controls.

Table II represents lipid profile in healthy subjects and the Patients.

Total fipids

Peak level was found in hepatitis. Highly significant (P<0. oo1) decline in serum total lipids concentration was observed in cirrhosis and statistically less significant (P Cholesterol

Minimal scrum cholesterol level was found in cirrhosis and the decrease was statistically significant (P<0.001). Significant elevation in serum cholesterol level occurred in patients with hepatitis and cancel. The rise was more significant (P <0.01) in hepatitis and less significant (P<0.05) in cancer. Peak

level was observed in hepatitis and the concentration found in the descending order from hepatitis to cancer to cirrhosis.

Triglycerides

Marked changes in serum triglycerides concentrations were found in hepatitis, cirrhosis and cancer. Peak rise was found in hepatitis. Highly significant (P<0. 001) elevation was observed in hepatitis and cancer. In cirrhosis the decline from the control value was found significant (P Phospholipids

Peak elevation was found in cancer. Significant decline (P<0.05) in serum phospholipids level was observed in cirrhosis.

Low Density Lipoprotein

The concentration was found significantly (P<0.001) reduced in cirrhosis whereas significant (P<0.01) elevation was observed in hepatitis and less significant (P<0.05) rise in cancer.

The data presented shows that all serum lipid levels were significantly reduced in cirrhosis. Serum total lipids, cholesterol and triglyceride levels were significantly elevated in hepatitis whereas serum phospholipids and serum low density lipoprotein levels were raised in cancer.

Correlation between Bilirubin and Triglycerides

A positive correlation coefficient (r=0.42) between bilirubin and triglycerides in hepatitis was observed and the result was statistically significant (P<0.01). Similarly bilirubin and triglyceride in cancer also showed a positive correlation coefficient (r=0.6) and the result was significant (P<0.001).

Discussion

Hepatic damage or dysfunction causes biochemical alterations in the body which arc reflected in several biochemical tests. Assessment of these changes can be of value in the diagnosis of various hepatic diseases.

Disorders of lipid metabolism in acute hepatitis are common.

Serum lipid pattern observed in the present study indicates a significant (P<0.01) elevation in serum cholesterol level in hepatitis than in cirrhosis and cancer. Serum triglycerides (P<0.001) and serum low density lipoprotein (P<0.01) were also significantly raised. However serum phospholipids elevation was not statistically significant in these patients. Similar findings have previously been reported (Eder et al., 1955; Phillips, 1960; Thalassinos et al., 1975). In the present scries, sixty percent cases of hepatitis had hypertriglyceridaemia.

Hyperlipidaemia could result from an inability of the liver to excrete lipids into the bile as a result of parenchymal damage (Phillips, 1960).

Serum lipid profile in cirrhosis indicates a significant decrease in the scrum levels of total lipids, cholesterol, triglycerides, phospholipids and scrum low density lipoprotein. Highly significant decline in serum cholesterol (P<0.001) and scrum low density lipoprotein (P<0.001) was observed. Hypocholesterola-emia was found in forty eight percent and hypolipoproteinaemia in eighty percent cases. This decline might be due to decreased synthesis in liver cells as cirrhosis involves considerable destruction of hepatic cells. Similar findings were reported by Feher (1976) and Ellefson and Caraway (1976). It was suggested that decreased cholesterol in patients with cirrhosis was due to diminished hepatic cholesterol synthesis (Ellefson and Caraway, 1976). Decreased lipoprotein concentration was observed in cirrhosis and the level was found related to liver's synthetic capacity (Eder et al., 1955).

A significant elevation in serum levels of triglyceride (P<0.001), cholesterol (P<0.05) and serum low density' lipoprotein (P<0.05) was found in cancer patients. Hypertriglyceridaemia was observed in sixty percent and hypercholesterolaemia in twenty seven percent cases. Similar findings have previously been reported by Figelson et al. (1944), Vialet et al. (1962), Santer et al. (1967) and Alpert et al. (1969).

An increased mobilization of fatty acids in cancer patients and fatty infiltration of the liver causes

triglyceride to accumulate in the liver (Figelson et al., 1944).
Hypercholesterolaemia in hepatocellular carcinoma is related to the absence of a negative feed-back system (Siperstein and Guest, 1960; Siperstein and Fagan, 1964). Deleted control mechanism was found fin primary human hepatoma, mouse hepatoma and morris hepatoma (Siperstein and Fagan, 1964). Later findings are in contrast to previous observations and it was found that feed-back inhibition of cholesterol synthesis does not occur in hepatomas though impaired tissue uptake and storage of cholesterol mav be an alternate mechanism (Harry et al., 1971).
A positive correlation was found between bilirubin and triglycerides in hepatitis and cancer and the result was highly significant.
This clearly indicates the relationship between bilirubin and triglyceride levels in liver diseases.

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