

LIVER FUNCTION TESTS IN TUBERCULOID LEPROSY

R. V. KORANNE,* RATAN SINGH† B. IYENGAR‡

Summary

A total of 24 patients with untreated tuberculoid leprosy were taken up for study. They were the same group of patients in whom the authors had earlier reported involvement of liver in 85% cases. Five healthy controls studied also belonged to the same series.

Liver function tests included prothrombin time, serum bilirubin, zinc sulphate turbidity, serum proteins and serum transaminases.

No significant alterations in the liver function were observed. This is because the changes in the liver were so minimal and focal that they were not reflected in the various liver function tests.

Literature is replete with the studies of hepatic function in lepromatous leprosy but little information is available with reference to tuberculoid leprosy. There are contradictory reports about the prevalence and extent of abnormal functioning of the liver in tuberculoid leprosy. Verghese and Job¹ studied three cases of tuberculoid leprosy. They found impaired liver function in one of them but the disturbance had no significant correlation with either the duration of illness or the extent of leprotic granuloma in the liver. Minimal changes in some of the tests in tuberculoid leprosy were also reported by Dhople and Balkrishna² and Mukerjee and Ghosh³. Most of the reports are based on two or three tests. It is difficult to assess the function of the liver on such isolated investigations. Koranne et al⁴ reported

the presence of leprosy pathology and acid fast bacilli in liver in 85% of cases of tuberculoid leprosy. It was decided to study in detail, in the same group of patients the liver function tests and to evaluate the extent of liver dysfunction, if any.

Material and Methods

A total of 24 untreated proved tuberculoid leprosy patients which included 20 earlier cases of Koranne et al⁴, who attended the Hansen's Clinic of the department of Dermatology and Venereology, Maulana Azad Medical College and Associated Irwin & G.B. Pant Hospitals, New Delhi constituted the subject material for this study. Cases in reaction or with concomitant tuberculosis or those with recent history of malaria, kala azar, infectious hepatitis, amoebic hepatitis or syphilis were not included in the study. Five normal healthy persons formed the control group.

10 c.c. of venous blood was drawn from the cubital vein in an oxalated bottle. Following investigations were carried out:-

* Ex-resident

† Professor & Head of the Department

‡ Assistant Professor of Pathology

Department of Dermatology and Venereology
Maulana Azad Medical College,
New Delhi-2 (India)

Received for publication on 27-1-78.

1. Prothrombin time Was determined by Quick's method⁵.
2. Serum bilirubin By the method of Malloy and Evelyn as described by Wootton^{6-a}.
3. Zinc Sulphate turbidity By the method of Wootton^{6-b}.
4. Serum proteins Total albumin and globulin were determined by the method of Biuret as described by Wootton^{6-c}.
5. Serum transaminases Were determined by the method of Reitmen and Frankel, as adopted by Wootton^{6-d}.

Zinc sulphate turbidity

The mean value in the patients was 3 units and the range was 2-5 units. One patient (case number 5) had raised level of zinc sulphate turbidity. The mean value and the range in the healthy controls were 4 units and 3-5 units respectively. One control (case number 2) had raised level of zinc sulphate turbidity.

Serum proteins

The mean value of total serum proteins in the patients was 6.1 gms.% and the range was 5.1 - 7.2 gms.%. The values were within the normal range in all the 24 patients.

The mean value and the range of total serum proteins in the healthy controls were 6.4 gms.% and 5.7 - 7.6 gms.% respectively. The values were within the normal range in the controls. The mean value and the range of serum albumin in the patients were 3.6 gms.% and 3.0 - 4.2 gms.% respectively. The values were within the normal range in the patients. In the healthy controls, the mean value and the range of serum albumin were 3.6 gms.% and 3.2 - 4.6 gms.% respectively. All the healthy controls showed values within the normal range. The mean value and the range of serum globulin in the patients were 2.5 gms.% and 2.0 - 3.5 gms.% respectively. The values were within the normal range in the patients. In the healthy controls, the mean value and the range of serum globulin were 2.8 gms.% and 2.4 - 3.0 gms.% respectively. The values were within the normal range in the controls.

Results

As shown in the Table, the evaluation of various liver function tests were as follows:

Prothrombin Time

The mean value in the patients was 18 seconds and the range was 16-19 seconds. The prothrombin time value was within the normal range in the patients.

The mean value in the healthy controls was 18 seconds and range was 17-19 seconds. The prothrombin time value was within the normal range in the healthy controls.

Serum bilirubin

In the study group, the mean value was 0.3 mg.% and the range was 0.2-0.7 mg.%. All the patients had serum bilirubin values within the normal range. The mean value in the healthy controls was 0.3 mg.% and the range was 0.2-0.9 mg.%. Healthy controls also had serum bilirubin value within the normal range.

The mean value and the range of A : G ratio in the patients were 1.4 and 0.9 - 1.7 respectively. One patient (case number 5) had reversal of A : G ratio. The mean value and the range of A : G ratio in the healthy controls were 1.3 and 1.2 - 1.5 respectively. The A : G ratio was within the normal range in the controls.

TABLE :—Liver function tests in 24 Tuberculoïd leprosy patients and 5 healthy controls

Project Group Case No.	Age in Yrs.	Sex	Duration of disease in months	Clinical type	Prothrombin time (Sec.)	Serum-bilirubin (mg.)	Zinc Su'phate turbidity (Units) %	Total Serum proteins (gms.) %	Serum albumin (gms.) %	Serum globulin (gms.) %	A : G Ratio	Units	
												SGOT	SGPT
1.	35	M	9	T. Min.	19	0.4	3	5.5	3.3	2.2	1.5	32	28
2.	30	F	8	T. Maj.	18	0.3	3	6.5	3.5	3.0	1.1	28	26
3.	70	M	4	T. Maj.	18	0.2	2	5.7	3.6	2.1	1.7	26	22
4.	26	M	14	T. Maj.	19	0.4	4	6.4	4.0	2.4	1.6	24	28
5.	12	M	8	M.A.	18	0.2	5	6.8	3.3	3.5	0.9	24	28
6.	20	F	24	T. Maj.	18	0.3	3	6.0	3.8	2.2	1.7	24	20
7.	20	M	18	M.A.	17	0.2	3	5.7	3.0	2.7	1.1	13	24
8.	19	M	12	M.A.	18	0.2	3	7.2	4.2	3.0	1.4	36	30
9.	18	M	36	M.A.	17	0.2	4	5.5	3.5	2.0	1.7	26	28
10.	15	M	24	T. Maj.	18	0.7	3	5.1	3.1	2.0	1.5	28	20
11.	35	M	30	T. Min.	16	0.2	4	7.2	4.2	3.0	1.4	28	22
12.	21	M	24	T. Min.	18	0.3	4	5.8	3.3	2.5	1.3	40	24
13.	15	M	11	M. N.	17	0.2	4	6.0	3.8	2.2	1.7	32	28
14.	30	M	24	M. N.	19	0.4	4	6.8	4.0	2.8	1.4	60	48
15.	25	M	7	T. Min.	18	0.7	4	6.5	4.0	2.5	1.6	44	36
16.	25	F	16	M.A.	17	0.2	2	5.8	3.0	2.8	1.0	32	26
17.	22	M	3	T. Maj.	18	0.3	2	6.0	3.8	2.2	1.7	32	24
18.	27	M	5½	M.A.	16	0.3	2	6.2	3.6	2.6	1.4	26	22
19.	35	M	5½	M.A.	19	0.2	2	5.5	3.3	2.2	1.5	32	26
20.	60	F	12	T. Maj.	N.D.	0.2	2	7.0	4.0	3.0	1.3	36	30
21.	19	M	2	M.A.	18	0.2	4	6.7	4.0	2.7	1.4	30	28
22.	50	F	8	T. Min.	19	0.2	2	5.5	3.3	2.2	1.5	30	24
23.	35	M	2	T. Maj.	N.D.	0.4	2	5.8	3.4	2.4	1.4	32	22
24.	21	M	4	M.A.	18	0.3	3	6.4	4.0	2.4	1.6	28	24
			Mean		14	0.3	3	6.1	3.6	2.5	1.4	31	26
			Range		16	0.2	2	5.1	3.0	2.0	0.9	24	20
					19	0.7	5	7.2	4.2	3.5	1.7	60	48

Healthy Group

Case No.	Sex	T. Min	T. Maj	M. N.	Tuberculoid Major	Tuberculoid Minor	Maculo-Anaesthetic	M. A.	S.G.O.T.					Not Done
									(a)	(b)	(c)	(d)	(e)	
1.	M	17	0.3	4	5.7	3.2	2.5	1.2	26	22				
2.	M	20	0.2	5	7.0	4.0	3.0	1.3	20	16				
3.	M	19	0.3	4	6.0	3.3	2.7	1.2	32	36				
4.	M	25	0.9	4	7.6	4.6	3.0	1.5	28	24				
5.	M	23	0.2	3	6.0	3.6	2.4	1.5	20	24				
			Mean											
			Minimum											
			Maximum											
			18	0.3	4	6.4	2.8	1.3	25	24				
			17	0.2	3	5.7	2.4	1.2	20	16				
			19	0.9	5	7.6	3.0	1.5	32	36				

KEY:— (a) T. Min (b) T. Maj (c) M. N. (d) Tuberculoid Major (e) N. D. — Not Done

(c) M. A. (d) Maculo-Anaesthetic

Serum glutamic oxalacetic transaminase (S.G.O.T.)

The mean value and the range in the patients were 31 units and 24-60 units respectively. Two patients (case numbers 14 & 15) had raised level of S.G.O.T. In the healthy controls, the mean value and the range were 25 units and 20 - 32 units respectively. The values were within the normal range in the controls.

Serum glutamic pyruvic transaminase (S.G.P.T.)

The mean value and the range in the patients were 26 units and 20-48 units respectively. Two patients (case numbers 14 & 15) had raised level of S.G.P.T. The mean value and the range in the controls were 24 units and 16-36 units respectively. One control (case number 3) had raised S.G.P.T. level.

Discussion

The present study has shown normal levels of prothrombin time and serum bilirubin.

Only one patient showed raised level of Zinc Sulphate turbidity. Dhople and Balkrishna² also found negligible deviation from the normal in the turbidity tests.

The values of total serum proteins, serum albumin and serum globulin were within the normal range and compare favourably with the values in the controls. A : G ratio was reversed in one patient and nutritional factor may be responsible for it. These findings compare well with those of Shivde and Junnarkar⁷ and Dhople and Magar⁸ who did not find any abnormality in the serum protein values in tuberculoid leprosy.

Sobhanadri and Lalitendra Nath⁹ have given two explanations for raised level of S.G.O.T. in leprosy. One is sub-normal hepatic damage and the other is damage to skeletal muscle. Shivde and Junnarkar⁷ reporting raised level of S.G.O.T. in one tuberculoid leprosy

patient in their series have also indicated hepatic and skeletal muscle damage as a cause for raised S.G.O.T. levels. In the present study two cases showed raised S.G.O.T. levels. Both had atrophy of the hypothenar muscles. At the same time, mono-nuclear cell infiltration was observed in the liver in one of the two cases. The values of S.G.O.T. in rest of the 22 cases compare favourably with those in the control group.

Shivde and Junnarkar⁷ have reported raised S.G.P.T. level in lepromatous leprosy patients and attributed it to liver changes. One tuberculoid leprosy patient in their series, with definite cirrhotic changes in the liver showed an increase in the S.G.P.T. values. In the present study, two cases showed raised level of S.G.P.T., the values of S.G.O.T. also being raised in them. One of them had mono-nuclear cell infiltration in the liver. Both the cases had atrophy of hypothenar muscles. S.G.P.T. levels in the rest of 22 cases were comparable to those in the control group.

Koranne et al⁴ reported involvement of liver in 85% of cases of tuberculoid leprosy which belonged to the same series of patients in which L.F.T. have been studied. Fourteen cases (70%) showed mono-nuclear cell infiltration in the liver. The infiltration was focal located in the portal area in each case. Eleven cases (55%) showed the presence of bacilli which were located in the Kupffer's cells. Eight cases (40%) showed both cellular infiltration and the bacilli. Six cases (30%) showed infiltration only and three cases (15%) had bacilli without cell infiltration.

Thus, L.F.T. in tuberculoid leprosy is not significantly altered even with involvement of liver by leprosy pathology. This is understandable. Liver is known to possess tremendous reserve and regenerative power and unless this is gro-

ssly disturbed the liver function tests cannot be expected to give abnormal results.

Moreover, in all the cases studied by Koranne et al⁴ the nature of involvement of liver was focal in portal area with scanty mono-nuclear cell infiltrate. In three cases, AFB were seen without any cell infiltration. The liver cells in all the cases were normal. There was no atrophy, no fibrous changes and no fatty degeneration. Thus the changes in the liver in tuberculoid leprosy are minimal and focal and as such are not reflected in the various liver function tests.

Acknowledgement

This article has been abstracted from the thesis submitted by Dr. R. V. Koranne to the University of Delhi, Delhi in August, 1975, in part fulfilment for M.D. (Dermatology including leprosy & V.D.) degree.

References :

1. Verghese A and Job CK : Correlation of liver function with the pathology of liver in leprosy, *Internat J Lepr*, 33 : 342, 1965.
2. Dhople AM and Balkrishna S : Liver function tests in leprosy, *Ind J Med Sci*, 56 : 1552, 1968.
3. Mukherjee A and Ghosh S : A concomitant study of liver function and coagulation factors in leprosy. *Lepr India*, 1 : 19, 1973.
4. Koranne RV, Singh Ratan and Inggar B : Liver involvement in tuberculoid leprosy (Under publication).
5. Quick S : The prothrombin in haemophilia and in obstructive jaundice, *J Biol Chem*, 109 : IXXIII, 1935.
6. Wootton IOP 'Micro-analysis in Medical bio-chemistry'.
London, Churchili, 1964 (a), 79.
— Idem 1964 (b), 147.
— Idem 1964 (c), 138.
— Idem 1964 (d), 112.

7. Shivde AV and Junnarkar RV: Serum transaminase activity in leprosy in relation to liver damage, *Internat J Leprosy*, 35 : 366, 1967.
8. Dhople AM and Magar NG: Serum proteins in leprosy *Ind J Med Sci* 51 : 476, 1963.
9. Sobhanadri C and Lalitendra Nath K: Serological changes in leprosy. *Indian J Dermatol Venereol*, 38 : 108, 1972.

IMPORTANT NOTICE

We wish to bring to your attention that the annual subscription rate for the journal has been enhanced as follows with effect from January 1980.

Subscription Rates

		Ordinary Mail	Air Mail
INDIAN	Rs.	30-00	—
FOREIGN COUNTRIES	\$	8-00	\$ 14-00
	£	4-00	£ 7-00

The change in rate has become necessary owing to increase in cost of paper and printing.

Claims for copies lost in the mail must be lodged within 30 days (90 days for countries abroad) of the issue. If complaints are received after this time copies will be replaced subject to availability of the same.

Managing Editor