

Liv.52 Therapy in Infective Hepatitis

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INTRODUCTION

Infective hepatitis though not one of the commonest diseases affecting children, certainly does not belong to the group of uncommon diseases. There are a few points about this disease which merit special consideration. They are:

1. Being viral in origin its treatment is not laid down in specific terms but treatment is imperative when it affects every year an appreciable percentage of the population in a country like India.
2. The most frustrating complaint in this disease is marked anorexia and the patient wants early relief from this symptom.
3. Infective hepatitis if not properly treated is likely to end in chronic active hepatitis, post-hepatitis cirrhosis, subacute necrosis and hepatic failure.

The therapy of viral hepatitis assumes immense importance as death from this disease is much more common in India, due to poor standards of nutrition, than in the West, though natural clinical cure may occur with or without residual liver cell damage. Since the liver performs many functions of the body—some understood well, some not so well, and others not at all—a drug that would help in keeping the liver functioning would go a long way in solving the problem of treating infective hepatitis.

In quest of such a drug we decided to evaluate the efficacy of Liv.52, an indigenous drug which has much published data heralding its beneficial effect in liver disorders. The drug Liv.52 has been observed to have experimental and clinical evidence of powerful hepatic stimulant and choleric actions, which markedly increase the functional efficiency of the liver. This drug is supposed to protect the hepatic parenchyma against toxic agents besides improving digestion and relieving flatulence and discomfort. (Sule *et al.*, 1956; Murkibhavi and Sheth 1957; Sheth *et al.*, 1960; Joglekar *et al.*, 1963; Patel and Sadre, 1963; Karandikar *et al.*, 1963; Captain and Syed, 1966 and Joglekar and Leevy, 1970).

MATERIAL AND METHODS

The present trial on Liv.52 was carried out in 49 cases of infective hepatitis.

Males	Females
34	15
Total: 49 cases	
Ratio: 2 : 1 (approximately)	

In none of the 49 cases was there a history of ingestion of any hepatotoxic drug, pruritus, diarrhoea or clinical evidence of rash, bleeding episode, oedema, ascites and collateral veins. Detailed history pertaining to infections, blood transfusion within the past 50 to 160 days was asked and in none was there a positive history.

In all the cases liver was palpable by about 3 to 5 cm. There was no evidence of liver being displaced downwards. In 41 cases liver was soft in consistency with soft edge and smooth surface. In 8 cases liver was firm in consistency. In all the cases there was hepatic tenderness.

The 49 cases were divided into two groups:

Group A

30 cases—they were given Liv.52 drops, B complex tablets and Vitamin C tablets.

Group B

19 cases—they were given B complex and Vitamin C tablets. This group served as control.

DOSAGE

Dose of Liv.52 was 20 drops t.i.d. in younger age groups and one teaspoonful t.i.d. in the older age group. Diet in each group was essentially the same fat-free, rich carbohydrate diet.

In this trial emphasis was laid on the following points.

1. Disappearance of nausea
2. Control of vomiting
3. Improvement of appetite
4. Disappearance of fever
5. Disappearance of jaundice
6. Disappearance of hepatic tenderness
7. Clearance of bile salts and pigments from urine
8. Normalcy of Liver function tests.

Age	Number of cases
Above 3 years to less than 5 years	18
Above 5 years to less than 8 years	22
Above 8 years to less than 10 years	9
Total	49

1.	Fever, jaundice and anorexia	44
2.	Fever and anorexia — Jaundice appearing 1-3 days after the admission	5

Sl. No.	Clinical features	No. of cases
1.	Jaundice	49
2.	Passage of yellow urine	49
3.	Fever	49
4.	Anorexia, nausea, vomiting	49
5.	Clay coloured stools	40
6.	Palmar erythema	32
7.	Palpable spleen	30
8.	Irritability	30
9.	Peevishness	30

10.	Lack of playfulness	30
11.	Abdominal discomfort	26
12.	Headache	25
13.	Anaemia	25
14.	Halitosis	5
15.	Constipation	3

Table V: Showing results of liver function tests

Sr. No.	L.F.T.	Range	Average
1.	S. bilirubin	3.2 mg% - 6 mg%	4.37 mg%
2.	Thymol turbidity	4 - 9 units	5.95 units
3.	Cephalin cholesterol	1 + to 3+ (after 48 hours)	2+
4.	Alkaline phosphatase	7 - 10.5 B.U.	9.25 B.U.

Table VI: Showing liver biopsy reports

Group	Biopsy report	No. of cases
1st group	Mild to moderate degeneration of the liver cells with mild to moderate infiltration by chronic inflammatory cells in the portal areas	8
2nd group	Features of first group and in addition, cholestasis	4
3rd group	Features of second group with added necrosis of liver cells	3

Table VII: Showing the average period for the improvement of symptom/sign

Sr.No.	Symptom or sign	Average period for the improvement of symptom./sign	
		Group A Liv.52 group	Group B (Control) without Liv.52
1.	Disappearance of nausea	6.0 days	19.0 days
2.	Control of vomiting	1.4 days	2.2 days
3.	Improvement of appetite	9.4 days	23.7 days
4.	Disappearance of fever	3.6 days	5.2 days
5.	Disappearance of jaundice	10.0 days	22.6 days
6.	Disappearance of hepatic tenderness	12.6 days	24.3 days
7.	Clearance of urine	11.0 days	24.6 days

Liver biopsy was done in 15 cases. In the rest of the patients it was considered unsafe or was not permitted. All the biopsies showed the findings consistent with histopathological picture caused by acute viral hepatitis. The biopsy results were classified into three groups.

An attempt was made to estimate liver function tests at least once in a week. But this was not possible due to practical difficulties like—(1) Patients (patient's-attendants) were not prepared for giving repeated blood samples. (2) Parents of the diseased frequently felt that their children were "obviously normal" after few days of treatment and refused to give blood for L.F.T. studies.

DISCUSSION

Patro (1957) and Mathur (1957) reported that Liv.52 helps in improving the outlook in severe hepatic damage. Sheth (1963) observed that it has a salutary effect on anorexia of infective hepatitis. Qazi (1965) stated that in animal experiments Liv.52 has a good influence on biochemical and functional abnormalities of the liver. He reported that the drug has a protective action against hepatotoxicity of tetracyclines in rats. Arora (1969) reported that Liv.52 adds "materially to patient's comfort and accelerates recovery". According to the study of Prasad and Tripathi (1969) Liv.52 brought an immense increase in appetite and power to assimilate without bowel disturbances in cases of infective hepatitis and malnutrition. As for the work of Jaffari and Shyam Raj (1969)

Liv.52 clears jaundice earlier, improves appetite and gives a sense of well-being. Liv.52 is reported to offer quite considerable protection against carbon tetrachloride and many other toxic agents (Sheth *et al.*, 1960; Joglekar *et al.*, 1963; Joglekar and Leevy 1970). Microscopic examination of the liver of animals treated with Liv.52 and carbon tetrachloride showed that the peripheral cells of the liver parenchyma escape necrosis and definitely show less deglycogenation but the central cell necrosis cannot be prevented. Further, the drug prevents deglycogenation of the peripheral part of the liver lobule to synthesise serum albumin. Liv.52 markedly improves the functional activity of the liver by acting as a powerful hepatic stimulant (Mukerjee and Dasgupta, 1970). Dayal *et al.* (1971) reported improvement in general condition, regression in jaundice and improvement in liver function tests. Ramalingam *et al.* (1971) reported that in infective hepatitis with the therapy of Liv.52, symptoms improved earlier than in the Vitamin C plus B-complex group, better weight gain was recorded and earlier restoration of liver function to normal observed.

Table VII clearly shows that the improvement in symptomatology was remarkable as compared to the control group. After Liv.52 therapy patients had a subjective sense of well-being, appetite improved earlier, nausea disappeared more rapidly, jaundice and urine cleared earlier. Regarding the liver, size of the liver decreased much earlier than in controls and hepatic tenderness persisted for many days in the controls unlike in the Liv.52 given group.

These observations are in accordance with the earlier studies (Mukerjee and Dasgupta, 1970; Patel *et al.*, 1971; Deshpande *et al.*, 1971 and Ramalingam *et al.*, 1971).

Liv.52 was also tried in malnutrition in this part of the state by Khetarpal *et al.*, (1972), and they have found marked improvement of appetite and weight gain in cases on Liv.52 besides increase in total serum protein and haemoglobin percentage.

It is also felt from this trial that the shift of deranged L.F.T. to normal was earlier in group A although much emphasis cannot be laid on this aspect due to irregularities in L.F.T. study for reasons beyond our control.

Regarding control of vomiting and absence of fever, little difference between the two groups is noted.

Prasad and Prasad (1971) reported 85% cure rate while poor response in 15% of cases with Liv.52 in infective hepatitis. In this study, all the thirty cases showed better response with Liv.52.

Liv.52 was given for two months and no side-effect or toxicity was noted.

CONCLUSIONS

1. The symptoms improved earlier in the Liv.52 group than in the B-complex and Vitamin C group.
2. No side effect was noticed.

REFERENCES

1. Arora, J.K., *Armed Forces med. J.* (1969): 3, 362.
2. Captain, S.R. and Syed, A.H., *The Indian Veterinary Journal* (1966): 43, 11.
3. Dayal, R.S., *J. Ind. med. Prof.* 9(1970): 9, 7768.
4. Deshpande, R.S., Sheth, S.C. and Joykutty, M.D., *Curr. med. Pract.* (1971): 6, 810.

5. Jaffari, S.M.H. and Shyam Raj, *Antiseptic* (1969): 5, 353.
6. Joglekar, G.V. *et al*, *Acta pharmacol. et toxicol.* (1963): 20, 73.
7. Joglekar, G.V. and Leevy, C.M., *J. Ind. med. Prof.* (1970): 12, 7480.
8. Karandikar, S.M. *et al*, *Acta pharmacol. et toxicol.* (1963): 20, 274.
9. Khetarpal, S.K., Ramakumar, Leela and Lubhaya, Ram, *Curr. med. Pract.* (1972): 16, 481.
10. Mathur, P.S., *Curr. med. Pract.* (1957): 2, 107.
11. Mukerjee, A.B. and Dasgupta, M., *Indian Practitioner* (1970): 6, 357.
12. Mukhibhavi, G.R. and Sheth, U.K., *Indian Veterinary Journal* (1957): 4, 276.
13. Patel, J.R. and Sadre, N.L., *Probe* (1963): 1, 19.
14. Patrao, S., *J. Ind. med. Prof.* (1957): 8, 1878.
15. Prasad, L.S. and Prasad, K., *Probe*, (1969): 3, 114.
16. Prasad, L.S. and Tripathi, D., *Probe* (1969): 1, 1.
17. Qazi, I.H., *Probe* (1965): 5, 1.
18. Ramalingam, V. *et al*, *Ind. Paed.* (1971): 12, 839.
19. Sheth, S.C., Northover, B.J., Tibrewala, N.S., Warekar, U.R. and Karande, V.S., *Ind. J. Paed.* (1960): 149, 204.
20. Sheth, S.C., Tibrewala, N.S., Warekar, U.R. and Karande, V.S., *Probe* (1963): 4, 137.
21. Sule, C.R. *et al*, *Ind. Practit.* (1956): 4, 357.