[*Probe* (1979): (XVIII), (3), 157-166]

A Study of Therapeutic Actions of Liv.52 on Various Stages, Severity, and Activity of Portal Cirrhosis and Infective Hepatitis

Sinha, P.K., *M.B.,B.S.,*
Presently Demonstrator in Medicine, B.R.D. Medical College, Gorakhpur, U.P., India.
Ashok Kumar, *Ph.D.,*
Research Technician
and
Patney, N.L., *M.D., M.R.C.P., D.T.M. & H., D.T.C.D.,*
Post-graduate Department of Medicine, S.N. Medical College, Agra, India.

Infective hepatitis produces an acute inflammation of liver, which though mild and self-limiting in many cases, often runs an acute, fulminating and fatal course or results in severe, chronic and irreparable hepatic damage. In India, the infection seems to be more severe and its complications more frequent than in other countries, probably due to malnutrition.

Acute progressive hepatocellular necrosis is followed by rapid shrinkage and degeneration of liver tissue accompanied clinically by deepening of jaundice, encephalopathy and coma occurring after the onset of clinical symptoms[1]. The aetiology and pathogenesis of chronic liver disease is not known. In this disease, some morphological appearance and serological abnormalities suggest that immunological mechanism may be involved, which may determine liver cell damage and chronicity of the disease[2,3].

However, the problem of therapy of viral hepatitis demands an ideal drug with the essential requisites of quicker recovery and convalescence, without residual liver cell damage. The therapy and management of hepatocellular failure and acute fulminant failure, which occur in severe cases, naturally, is a major clinical and therapeutic problem.

Several studies in the past (Joglekar and Leevy, 1970;[4] Mukerjee and Dasgupta, 1971;[5] Deshpande, 1974;[6] Reddi, 1976;[7] and Patney and Kumar, 1973,[8] 1976[9]) have been published supporting and confirming the beneficial effect of Liv.52 in liver damage of varying degrees based on experimental background.

Liv.52 an indigenous drug, is claimed to have a protective and regenerative effect on the hepatic parenchyma, to be a stomachic and a choleretic, with a salutary effect on liver glycogen and serum proteins along with diuretic and anabolic actions. In view of these, its easy availability and economy of treatment, it was considered worth while to investigate Liv.52 in the management of various stages, severity and activity of infective hepatitis.

MATERIAL AND METHODS

Sixty-six clinically-diagnosed cases of infective hepatitis, chronic hepatitis (jaundice of more than three months' duration) and portal cirrhosis were picked from the general medical ward of S.N. Hospital, Agra during may 1974 to May 1976. A thorough clinical examination including history, features of liver cell insufficiency and portal hypertension was made in each case. The following investigations were carried out in each case.

1. Complete Blood Examination — which include haemoglobin level, total and differential leucocyte count, erythrocytic sedimentation rate (ESR), general blood picture and red blood cell (RBC) count.

2. Liver Function Tests — consisted of serum bilirubin, Van den Bergh reaction, alkaline phosphatase, zinc sulphate turbidity, thymol turbidity and flocculation, serum proteins and albumin/globulin ration (Varley, 1969)[10].

3. Enzymatic studies — consisted of:
   (i) Serum glutamate oxaloacetate transaminase (SGOT),
   (ii) Serum glutamate pyruvate ransaminase (SGPT) (Reitman and Frankel, 1957)[11],
   (iii) Lactate dehydrogenase (LDH) (King, 1965)[12] and
   (iv) Isocitrate dehydrogenase (ICDH) (Bell *et al,* 1960).[13]

By the above mentioned investigation and clinical examination, cases of infective hepatitis were categorised into *Mild* (serum bilirubin less than 5 mg%), *Moderate* (Jaundice + serum bilirubin 5 to 10 mg%), *Severe* (Jaundice ++, serum bilirubin 10 mg%, normal or marginally raised serum alkaline phosphatase) and *Cholestatic Hepatitis* (Jaundice +++, serum bilirubin 14 mg% and alkaline phosphatase markedly raised). A note was made if hepatitis had relapsed.

Allocation of trial treatment with Liv.52
After complete classification of the cases according to stages of severity and activity of the underlying liver disorders, they were randomly allotted one of the following treatment schedules:
(A) Liv.52 alone (2 tabs. t.i.d.)
(B) Liv.52 (2 tabs t.i.d.) + usual supportive treatment consisting of Vits. B & C, glucose and steroids.
(C) No Liv.52, only supportive treatment.
(D) No treatment (Liv.52 or supportive), only placebo treatment.

Cases in groups C and D were considered as controls, as in these two groups no Liv.52 therapy was employed. The treatment in all these 4 groups was continued for up to 6 weeks. Serial clinical and biochemical examination were done at the time of admission, and 6 weeks later.

OBSERVATIONS

The categorisation of the cases according to the stages of activity and severity of various aetiological groups as judged by clinical features and the biochemical investigations of the underlying disease process is shown in Table 1-A.

Table 1-A: Distribution of cases studied showing degree of liver damage and activity of disease

Aetiological group			Distribution of cases	Total No. of cases
(A)	**Infective hepatitis**	Mild	20	33
		Moderate	10	
		Severe	3	
	Indeterminate (Chronic hepatitis)	Mild active hepatitis	4	10
		Moderately active hepatitis	–	
		Severely active hepatitis	6	
	Precholemia	Infective hepatitis with precholemia	14	14
(B)	**Portal cirrhosis**	Uncomplicated portal cirrhosis	6	9
		Precholemia with portal cirrhosis	3	
		Total cases		66

Table 1-B: Showing the cases of cholestatic hepatitis			
Aetiological group		Distribution of cases	Total No. of cases
Cholestatic hepatitis	Infective hepatitis	8	27
	Infective hepatitis with precholemia	9	
	Indeterminate	10	

Ten cases of chronic hepatitis, in which the nature of the underlying aetiological condition could not be determined, were labelled as the indeterminate group. Four cases showed mildly active hepatitis and 6 cases showed severely active hepatitis.

A. Infective Hepatitis

Out of 57 cases of infective hepatitis 27 case showed some evidence or the other of cholestatis - hence were subclassified as cholestatic hepatitis. The group included cases belonging to all groups.

Cholestatic hepatitis was observed in 8 cases of uncomplicated infective hepatitis, 9 cases of infective hepatitis with precholemia and 10 cases of indeterminate groups.

B. Portal Cirrhosis

As regards 9 cases of portal cirrhosis, 4 cases showed mild liver damage, 3 moderate and the remaining 2, severe degree of liver damage. Further classifying them according to the activity of the cirrhotic process, 6 cases showed severely active disease, two had moderately active disease and only one mildly active disease.

Age: Out of these 66 cases studied, two were under 15 years of age, 25 between 15-25 years of age, 22 between 26-35 years of age, 12 between 36-45 years of age and 5 cases over 45 years of age.
The categorisation of cases according to various treatment schedules is given in Table 2-A.

Table 2-A: Distribution of cases according to the treatment schedule given						
Sl. No.	Aetiological group	A	B	C	D	Total
1.	Infective hepatitis	12	6	7	8	33
2.	Indeterminate (chronic hepatitis)	—	6	2	2	10
3.	Precholemia with infective hepatitis	3	8	1	2	14
4.	Portal cirrhosis	1	2	1	2	6
5.	Precholemia with portal cirrhosis	—	1	2	—	3
	Total	16	23	13	14	66

Results of haematology and liver function tests

In cases of uncomplicated infective hepatitis, the maximum rise/fall in different parameters were in therapeutic groups A and B (Liv.52 group) as compared to groups C and D (without Liv.52) Table 3-A.

Table 3-A: Showing the mean results and rise/fall of haematology and liver function tests in cases of infective hepatitis before and after therapy

Gro-up	Haemoglobin (g%)			E.S.R. (Fall in 1st hr. Wintrove)			Serum bilirubin (mg%)			S. albumin (g%)			S. Alkaline Phosphatase (K.A. units)		
	On ad.	After 6 wks	Diff.	On ad.	After 6 wks	Diff.	On ad.	After 6 wks.	Diff.	On ad.	After 6 wks	Diff.	On ad.	After 6 wks.	Diff.
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
A	10.3 ±1.8	11.8 ±2.6	1.5	44 ±11	18 ±7	26	6.6 ±2.0	1.1 ±0.2	5.5	3.1 ±0.6	3.7 ±0.6	0.6	25 ±6	10 ±2	15
B	9.0 ±2.0	11.4 ±2.3	2.4	53 ±17	18 ±8	35	3.3 ±1.2	0.8 ±0.1	2.5	2.8 ±0.4	3.9 ±0.7	1.1	20 ±4	11 ±2	9
C	8.7 ±2.3	10.7 ±3.0	2.0	47 ±14	20 ±9	27	4.9 ±1.4	1.2 ±0.3	3.7	2.9 ±0.8	3.6 ±0.4	0.7	11 ±3	16 ±3	5
D	11.0 ±1.6	12.4 ±1.9	1.4	36 ±10	15 ±6	21	4.6 ±1.7	1.9 ±0.2	2.7	2.9 ±0.5	3.5 ±0.3	0.6	22 ±8	15 ±5	7

In cases of indeterminate aetiology, the patients on therapy B (Liv.52) showed better recovery as compared to therapy groups C & D (Table 3-B).

Table 3-B: Showing the mean results and rise/fall of haematology and liver function test in cases of indeterminate aetiology before and after treatment

Gro-up	Haemoglobin (g%)			E.S.R.			Serum bilirubin (mg%)			S. albumin (g%)			S. Alkaline Phosphatase (K.A. units)		
	On ad.	After 6 wks	Diff.	On ad.	After 6 wks	Diff.	On ad.	After 6 wks.	Diff.	On ad.	After 6 wks	Diff.	On ad.	After 6 wks.	Diff.
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
B	11.0 ±1.8	12.1 ±1.9	2.5	34 ±10	16 ±6	18	16.0 ±4	1.3 ±0.3	14.7	2.7 ±0.6	3.9 ±0.8	1.2	49 ±9	15 ±3	34
C	11.0 ±1.6	13.0 ±1.8	2.0	52 ±15	20 ±9	32	18.0 ±5.3	4.4 ±0.9	13.6	3.0 ±0.8	3.9 ±0.7	0.9	45 ±9	18 ±3	27
D	8.7 ±2.1	10.5 ±1.9	1.8	36 ±11	17 ±8	19	3.7 ±1.1	1.6 ±0.7	2.1	3.5 ±0.5	3.8 ±0.4	0.3	26 ±5	16 ±2	10

The mean results in cases of precholemia due to infective hepatitis (Table 3-C) show the maximum mean rise/fall recorded in therapy group A (Table 3-C).

Table 3-C: Showing the mean results and rise/fall of haematology and liver function test in cases of precholemia due to infective hepatitis before and after therapy

Gro-up	Haemoglobin (g%)			E.S.R.			Serum bilirubin (mg%)			S. albumin (g%)			S. Alkaline Phosphatase (K.A. units)		
	On ad.	After 6 wks	Diff.	On ad.	After 6 wks	Diff.	On ad.	After 6 wks.	Diff.	On ad.	After 6 wks	Diff.	On ad.	After 6 wks.	Diff.
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
A	11.0 ±1.8	12.1 ±1.9	1.1	35 ±10	16 ±8	19	10.5 ±3.7	1.7 ±0.3	8.8	3.1 ±0.7	3.9 ±0.4	0.8	20 ±5	11 ±2	9
B	11.5 ±1.3	13.4 ±1.5	1.9	41 ±12	14 ±6	27	12.1 ±4.3	1.3 ±0.2	10.8	3.2 ±0.5	4.0 ±0.7	0.8	23 ±6	12 ±3	11
C	10.0 ±1.9	10.8 ±1.8	0.8	28 ±8	18	10	16.2 ±5.1	3.8 ±0.9	12.4	2.0 ±0.3	3.6 ±0.3	1.6	26 ±8	18 ±5	18
D	8.9 ±2.0	9.7 ±1.9	0.8	45 ±11	25 ±7	20	8.9 ±2.7	5.5 ±1.7	3.4	3.2 ±4.0	3.7 ±0.6	0.5	26 ±5	16 ±5	10

Similarly the mean results (rise/fall) in cases of portal cirrhosis and precholemia are shown in Table 3-D.

Table 3-D: Showing the mean results and rise/fall of haematology and liver function tests in cases of portal cirrhosis and precholemia before and after therapy

Gro-up	Haemoglobin (g%)			E.S.R.			Serum bilirubin (mg%)			S. albumin (g%)			S. Alkaline Phosphatase (K.A. units)		
	On ad.	After 6 wks	Diff.	On ad.	After 6 wks	Diff.	On ad.	After 6 wks.	Diff.	On ad.	After 6 wks	Diff.	On ad.	After 6 wks.	Diff.
1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Portal cirrhosis															
A	10.4 ±1.5	11.3 ±2.0	0.9	38 ±11	14 ±8	24	1.2 ±0.6	1.1 ±0.2	0.1	2.9 ±0.3	3.8 ±0.8	0.9	12 ±3	9	3
B	10.2 ±1.9	12.2 ±1.8	2.0	52 ±15	19 ±6	33	1.3 ±0.3	0.8 ±0.1	0.5	2.5 ±0.5	4.0 ±0.5	1.5	21 ±6	15 ±2	6
C	11.1 ±1.6	11.8 ±1.9	0.7	45 ±13	20 ±8	25	3.3 ±0.9	0.8 ±0.4	2.5	2.3 ±0.6	3.8 ±0.6	1.5	22 ±7	15 ±2	7
D	11.6 ±1.0	11.7 ±1.4	0.1	44 ±9	29 ±10	15	10.9 ±3.5	1.6 ±0.7	9.3	2.8 ±0.8	3.8 ±0.4	1.0	27 ±7	14 ±4	13
Precholemia															
B	10.5 ±1.3	12.0 ±1.6	1.5	85 ±19	34 ±11	51	3.8 ±1.4	1.1 ±0.2	2.7	3.2 ±0.6	2.8 ±0.5	0.4	16 ±4	11 ±2	5
C	11.5 ±1.4	12.5 ±1.9	1.0	52 ±12	16	36	8.6 ±2.3	2.3 ±0.6	6.3	2.4 ±0.8	3.9 ±0.4	1.5	17 ±3	26 ±8	9

Results of enzymatic studies

The mean fall of enzymatic levels after scheduled therapy in various aetiological group is shown in Table 4-A. The group of uncomplicated infective hepatitis showed a better and quicker recovery of

enzymatic levels in therapies A & B where Liv.52 was given, than groups C & D as clear from the mean fall of SGOT, SGPT, LDH and ICDH.

Table 4-A: Showing mean results and fall in enzymatic levels in cases of infective hepatitis before and after therapy

Group	SGOT (IU/L)			SGPT (IU/L)			LDH (IU/L)			ICDH (IU/L)		
	On Ad.	After 6 wks.	Diff.	On Ad.	After 6 wks.	Diff.	On Ad.	After 6 wks.	Diff.	On Ad.	After 6 wks.	Diff.
1	2	3	4	5	6	7	8	9	10	11	12	13
A	54 ± 20	10±2	44	96±27	16±6	80	664±107	227±53	387	138±32	15±4	123
B	77±25	16±3	61	124±40	16±10	108	736±132	275±68	461	137±40	14±3	123
C	70±18	29±5	41	107±36	28±13	79	744±69	370±43	374	140±26	37±11	103
D	60±24	31±8	29	109±28	37±21	72	693±142	391±39	302	123±48	36±17	87

In cases of indeterminate aetiology i.e. chronic hepatitis Table 4-B showed a better improvement in mean fall of SGOT, SGPT, LDH and ICDH in group B, groups C and D respectively. While in cases of precholemia (Table 4-C) group B showed a significant mean fall of enzymatic levels in group B as compared to group A, group C and group D.

Table 4-B: Showing mean results and fall in enzymatic levels in cases of indeterminate aetiology before and after therapy

Group	SGOT (IU/L)			SGPT (IU/L)			LDH (IU/L)			ICDH (IU/L)		
	On Ad.	After 6 wks.	Diff.	On Ad.	After 6 wks.	Diff.	On Ad.	After 6 wks.	Diff.	On Ad.	After 6 wks.	Diff.
1	2	3	4	5	6	7	8	9	10	11	12	13
B	43±8	19±3	24	53±11	13±4	40	635±138	278±72	357	57±8	10±2	47
C	40±12	18±6	22	60±25	27±3	33	619±210	300±49	319	63±11	43±17	20
D	37±6	22±8	15	64±23	25±7	39	565±200	330±53	235	118±29	60±11	58

Table 4-C: Showing mean results and fall in enzymatic levels in cases of precholemia due to infective hepatitis before and after therapy

Group	SGOT (IU/L)			SGPT (IU/L)			LDH (IU/L)			ICDH (IU/L)		
	On Ad.	After 6 wks.	Diff.	On Ad.	After 6 wks.	Diff.	On Ad.	After 6 wks.	Diff.	On Ad.	After 6 wks.	Diff.
1	2	3	4	5	6	7	8	9	10	11	12	13
A	60±20	19±3	41	99±29	18±4	81	729±149	307±103	422	63±26	16±4	47
B	73±13	16±4	57	147±62	16±2	131	746±82	264±87	482	142±48	20±8	122
C	90±30	46±6	44	126±84	50±20	76	740±135	320±46	420	100±51	44±20	56
D	101±40	50±17	51	143±57	65±32	78	713±310	467±67	246	137±37	80±36	57

In cases of portal cirrhosis (Table 4-D) a nice recovery of mean fall of enzymatic levels namely SGOT, SGPT, LDH, ICDH was observed in group A and group B as compared to group C and group D respectively. Similarly the cases of precholemia, showed a better recovery in group B as compared to group C.

Table 4-D: Showing the mean results and fall in enzymatic levels in cases of portal cirrhosis and precholemia before and after therapy

Group	SGOT (IU/L)			SGPT (IU/L)			LDH (IU/L)			ICDH (IU/L)		
	On Ad.	After 6 wks.	Diff.	On Ad.	After 6 wks.	Diff.	On Ad.	After 6 wks.	Diff.	On Ad.	After 6 wks.	Diff.
1	2	3	4	5	6	7	8	9	10	11	12	13
Portal Cirrhosis												
A	80±27	15±3	65	117±32	15±3	102	606±163	284±82	322	125±63	16±8	109
B	171±32	40±10	131	156±60	25±5	131	657±109	251±26	406	116±75	20±6	96
C	127±40	31±6	96	141±38	38±11	103	640±207	323±103	317	122±93	33±12	89
D	77±17	31±8	46	100±26	37±18	63	515±140	402±146	113	85±17	36±14	49
Precholemia												
B	68±26	24±5	44	72±16	11±3	61	780±69	321±68	459	125±35	20±7	105
C	83±32	40±12	43	76±17	32±11	44	735±192	369±97	366	104±40	32±6	72

DISCUSSION

Viral hepatitis is often a self limiting disease, but can have high morality and morbidity during epidemics (Melnick[14]) particularly where malnutrition is endemic. In the past and even today the major interest regarding the therapy of viral hepatitis has centred around the use of corticosteroid. Corticosteroids did produce clinical and biochemical remission as observed by Ducci,[15] although Sule *et al*[16] did not find much change in the histopathological lesions. Mitra[17] and Libov[18] reported

in a study that addition of corticosteroids to Liv.52 did not seem to have any extra advantage in the treatment and routine management of cases. All cases on corticosteroids developed moon face and oedema of the feet.

Therefore, the present study was undertaken to evaluate the role of Liv.52 in the management of varrious stages, severity and activity of infective hepatitis, and portal cirrhosis. Several experimental and clinical studies have clearly demonstrated its beneficial role on the hepatic parenchyma in cases of infective hepatitis. Joglekar,[4] proved the beneficial effect of Liv.52 in liver damage of varying degree, using different methods including the latest method of Indocyanine green clearance and autoradiographic patterns. Sule[16] *et al.,* using different parameters, confirmed that Liv.52 accelerates clinical and biochemical recovery. Mukerjee *et al.,* using different parameters, confirmed that Liv.52 accelerates clinical and biochemical recovery. Mukerjee *et al*[5] observed that in viral hepatitis Liv.52 brought about reduction in the period of illness, residual liver damage and consequent gain in weight.

Toxicity studies in animals had also revealed that Liv.52 has no acute or chronic toxicity, teratogenic or carcinogenic effect and no effect on fertility and does not cause malfunction of any organs or affect the growth of animals.[6]

Liv.52 stimulates mitotic activity (Prasad, 1974)[19] of the cells and so stimulates the liver cell regeneration and would thus correct all the secondary abnormalities consequent to liver parenchymal necrosis and degeneration.

The treatment of this complication of the liver used to be unsatisfactory but with the advent of Liv.52 therapy there is some enthusiasm in its treatment in view of its therapeutic action as mentioned above.

The present study comprises of 66 cases in all, including infective hepatitis (33 cases), hepatitis of undetermined cause (chronic hepatitis) 10 cases, precholemia (14 cases) and portal cirrhosis (9 cases). Liv.52 tablets (2 t.i.d.) was given for 6 weeks to these patients either alone (group A) or in combination with supportive treatment (group B). The two other groups on supportive treatment (group C) or placebo capsule (group D), served as controls.

It is evident from Tables 3-A to 3-D that Liv.52 therapy may also improve the blood picture to some extent but the degree of improvement is not very much significant when compared with groups C & D who did not get any Liv.52 therapy. There is no doubt that the blood picture improves but this effect is rather a non-specific one and is probably because of an improvement in the functional status of hepatic parenchyma.

In cases of uncomplicated infective hepatitis, the results clearly showed that Liv.52 when given alone (group A) gave significant improvements in liver function tests and enzymatic studies as compared to groups B, C and D. The mean rise in haemoglobin level in group A and fall in ESR is the maximum improvement as compared to groups B, C and D in these cases. This proved that the patients of uncomplicated infective hepatitis showed a nice response with Tab. Liv.52 alone.

In cases of indeterminate aetiology and precholemia, Liv.52 was not given alone (group A). When the results of liver function test and enzymatic studies of group B are compared with C & D, the improvement in group B is more than in group C because patients in group B are also getting other drugs e.g. steroids, vitamin B complex and C glucose etc. which all are said to have a protective action on liver parenchyma. Hence there is a possibility that this drug could have potentiated the action of other drugs and this potentiating synergistic action of all the drugs on the liver parenchyma

brought about a quicker and better recovery. In fact Liv.52 stays as the main factor behind the quicker and faster recovery in patients in group B judged clinically and biochemically.

As we have reported in our communications,[7,8] Liv.52 displays a marvellous beneficial effect clinically in the form of lessening of jaundice and itching, marked subjective feeling of well-being, recovery in appetite, gain in weight and lessening of dyspeptic flatulence, nausea etc.

Liv.52 has a definite beneficial effect, though not highly significant when given alone, in cases of precholemia and cholemia. It can restore the functional status of liver parenchyma to almost normal in the majority of cases particularly when combined with others (group B). The same is true for cholestatic hepatitis, drug hepatitis. Thus it can be aid that Liv.52 is not only helpful in uncomplicated infective hepatitis but also in many other groups including drug hepatitis, serum hepatitis, jaundice of obscure origin and also the cases showing cholestasis and jaundice.

The drug can safely be used even in very high doses and for prolonged duration without any hazards and side-effects.

SUMMARY

A total 66 cases (infective hepatitis 33, chronic hepatitis with cholestatic hepatitis 10, precholemia 14 and portal cirrhosis 9) were studied to evaluate the role of Liv.52 on various stages severity and activity of portal cirrhosis and infective hepatitis.

All the cases were allotted into four regimens group Liv.52 (2 tabs, t.i.d. 6 weeks) was given to these patients either alone (group A) or in combination with other drugs (group B). The other two groups on supportive treatment (group C) or placebo capsule (group D), served as control.

In cases of uncomplicated infective hepatitis a better improvement in liver function test and enzymes are observed in group A (Mean rise in Hb. 1.5 g%, serum bilirubin 5.5 mg%, Alkaline phosphatase 15 KA Units, SGPT 80 and LDH 387 IU/L) and in group B (Hb. 2.4 g%, serum bilirubin 2.5 mg% and serum alkaline phosphatase 9 KA Units, SGPT 108 and LDH 461 IU/L) as compared to group C (Hb 2.0 g%, serum bilirubin 3.7 mg%, alkaline phosphatase 5 KA Units, SGPT 79 and LDH 374 IU/L) and D (Hb. 1.4 g%, serum bilirubin 2.7 mg% and serum alkaline phosphatase 7 KA Units, SGPT 72 and LDH 302 IU/L). The improvement in group B is somewhat more than in group A while comparing the group B with Group C, group B showed a better and quicker recovery in clinical and biochemical parameters. This is all due to the effect of Liv.52 tablets because group B in comparison to group C got Liv.52 along with steroids etc.

In cases of chronic hepatitis and precholemia, a better response of the therapy is shown by group B (Hb. 2.5 g%, serum albumin 1.2 g%, serum bilirubin 14.7 mg%, SGPT 40 and LDH 357 IU/L) as compared to C (Hb. 2.0 g%, serum albumin 0.9 g%, bilirubin 13.6 g%, SGPT 33, and LDH 319 IU/L) and group D (Hb 1.8 g%, serum albumin 0.3 g%, serum bilirubin 2.1 g%, SGPT 39 and LDH 235 IU/L).

In cases of precholemia due to portal cirrhosis also a better recovery in serum enzymes observed by therapeutic group A (SGPT 102, & LDH 322 IU/L) and B (SGPT 131 and LDH 406 IU/L) as compared to group C (SGPt 103 and LDH 317 IU/L) and D (SGPT 63 and LDH 113 IU/L).

Therefore, on the basis of this clinical study we can say that Liv.52 possesses a definite beneficial role in the cases showing clinical and biochemical evidence of precholemia, hepatitis of indeterminate aetiology (chronic hepatitis) besides the uncomplicated cases of portal cirrhosis and infective hepatitis.

CONCLUSION

Liv.52 is a safe and effective drug in the treatment of uncomplicated cases of infective hepatitis and portal cirrhosis as well as cases of these hepatic disorders precholemia and cholestasis.

Liv.52 not only helped in improving the liver function tests, it expedited the recovery. Liv.52 therapy showed satisfactory response when used alone in the treatment of uncomplicated infective hepatitis, in other complicated disorders including hepatic cirrhosis and precholemia it expedited the recovery when used in cirrhotic with steroids and has been shown as a useful adjunct in the treatment of these conditions.

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