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Studies on 222 cases of Acute Infective Hepatitis During an Epidemic

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Infective hepatitis is a well recognised communicable disease caused by a virus. It produces clinical, symptomatic and general effects. The liver, the largest metabolic gland in the body having diverse functions, bears the brunt of the disease. Diseases of the liver, by producing various disturbances, throw the entire system out of gear. In the diagnosis and the study of progress of this disease, evaluation of the hepatic functions is of major importance. Biochemical and histopathological studies are of great value in confirming the diagnosis and assessing the progress of the disease process.

Infective hepatitis can be mild, moderate or severe; and may cause acute or subacute hepatic necrosis, leading to the clinical condition of precoma or coma. There are inflammatory changes in the hepatic parenchyma and interstitial tissue with clinical and histopathological evidence of hepatocellular degeneration in various stages of the disease process. Yeogt in Germany was the first to demonstrate infectivity of the disease in volunteers. In U.S.A. there were 72000 cases in 1961, the highest incidence for any year since the disease was reported as a common one and also as occurring in moderate to severe epidemics.

This study was conducted on 222 cases of viral hepatitis in an epidemic when the morbidity and mortality rate was fairly high. The clinical features, their progress, laboratory findings and their weekly progress were studied. Laboratory studies routine blood and urine examination, estimation of total and direct bilirubin, thymol turbidity test, serum alkaline phosphatase tests, plasma protein estimate, blood group assessment were done in all cases and histopathological studies of the liver were carried out wherever possible. The progress of the clinical and laboratory findings was noted every week for four weeks on a previously planned and prepared proforma. 78 patients were treated with Liv.52 and 144 cases served as control, other treatment and general management being the same in both the groups. The age distribution in years is given in Table I.

Table I: Age in years, with Liv.52 and without Liv.52 — 222 cases

Age in years	No. of cases	%	No. of cases	%
0 - 5	4	5.1%	12	8.3%
6 - 10	1	1.3%	1	0.7%
11 - 20	19	24.4%	22	15.3%
21 - 30	31	39.7%	61	42.4%
31 - 40	12	15.4%	30	20.8%
41 - 50	5	6.4%	9	6.3%
51 - 60	5	6.4%	4	2.8%
61 - 70	—	—	3	2.1%
71 - 80	1	1.3%	2	1.3%
Total	78	100%	144	100%

The highest incidence was in the age group between 21-30 years and the largest number of patients were between the ages of 11 and 40; 62 in the first group of 78 cases and 113 in the second group of 144 cases.

The patients mostly came from the low and poor socio-economic group. The intensity of the disease varied from case to case. A large number of cases were in the precoma or coma state. Clinical evaluation of symptomatology in these cases revealed the data as shown in Tables II and III.

Table II: Sex distribution

Sex	Liv.52 group		Without Liv.52	
	No. of cases	%	No. of cases	%
Males	47	60.3%	77	53.5%
Females	31	39.7%	67	46.5%
Total	78	100%	144	100%

Table III: Socio-economic group

Income group	Liv.52 group		Without Liv.52	
	No. of cases	%	No. of cases	%
Upper	7	9.0%	11	7.6%
Middle	67	85.9%	128	88.9%
Lower	4	5.1%	5	3.5%
Total	78	100%	144	100%

A study of the clinical symptomatology showed that a large number of patients were in the acute fulminating stage of the disease as this study was during the period of an epidemic as could be seen by 11 cases in confused mental state, 9 semiconsciousness and 4 in coma i.e.. 24 cases out of 78 in the Liv.52 group and 44 cases in confused mental state, 49 semiconsciousness and 43 in coma in the control group. Some of the cases might have lapsed from one stage of consciousness to another and there may be some degree of overlapping but these symptoms bring out that the group of cases were acute and severe with a fair degree of presumed hepatic damage.

Table IV: Showing signs and symptoms

Symptoms & signs	Liv.52 group		Without Liv.52	
	No. of cases	%	No. of cases	%
Fever	50	64.1%	112	77.8
Yellow urine	57	73.1%	121	84.0%
Jaundice	66	84.6%	123	85.4%
Pain in abdomen	34	43.6%	64	44.4%
General bodyache	10	12.8%	14	9.7%
Pruritus	7	9.0%	5	3.5%
Anorexia	47	60.3%	53	36.8%
Nausea	28	35.9%	39	27.1%
Vomiting	33	42.3%	40	27.8%
Confused mental state	11	14.1%	44	30.6%
Semiconsciousness	9	11.5%	49	34.0%
Coma	4	5.1%	43	29.9%
Concomitant pregnancy	4	5.1%	27	18.7%
Enlarged and palpable liver	37	47.4%	56	38.9%
Enlarged and palpable spleen	5	6.4%	12	8.3%
Ascites	16	20.5%	22	15.3%
Clinical discernible cirrhosis of liver	8	10.2%	17	11.8%
Portal hypertension	3	3.8%	6	4.2%

Table V: Laboratory studies showed the following on urine examination

Urine	Liv.52 group		Without Liv.52	
	No. of cases	%	No. of cases	%
Urine bile salts present	31	39.7%	36	25.0%

Urine bile pigments present	40	51.3%	47	32.6%
Urine albumin present	62	79.5%	15	10.4%
Sugar in urine	—	—	—	2.8%

RESULTS

Results	Liv.52 group		Without Liv.52	
	No. of cases	%	No. of cases	%
Good	35	44.9%	20	13.9%
Fair	18	23.1%	37	25.7%
Died	15	19.2%	68	47.2%
Otherwise dropped from study	10	12.8%	19	13.2%
Total	78	100%	144	100%

In the Liv.52 group initial levels of total serum bilirubin varied from 22.8mg to 2.3 mg and gradually returned to normal. Direct serum bilirubin levels also followed to the total serum bilirubin. Serum alkaline phosphatase and thymol turbidity tests also showed significant improvement. Initial SGPT levels ranged from 1660 to 250 and gradually came down. Total serum proteins were not affected significantly.

In the group without Liv.52, out of 144 cases, initial level of serum total bilirubin varied from 25.8 mg to 2.4 mg and direct serum bilirubin from 15.8 k to 1.5 mgm%. Serum alkaline phosphatase and thymol turbidity tests were initially high and in many cases did not return to normal soon. SGPT levels varied from 1870 to 350 and remained high in acute, precoma and coma cases. Total serum proteins were not affected significantly.

The results were considered very good when the clinical response was good, laboratory findings showed improvement and returned to normal almost within a week and the patient was cured; fair when there was slow but definite clinical and biochemical progress towards normality. It took two, three and even four weeks in serious cases to come to normal. Results were considered poor when there was no response—clinical or laboratory.

In the Liv.52 group, out of 78 cases, the results were good in 35, fair in 18, and 15 patients expired, while out of the 144 control cases, the results were good in 20, fair in 37 and 68 patients expired.

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