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Viral Hepatitis in Children

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INTRODUCTION

During the past few decades, particularly since World War II, viral hepatitis has become an increasingly important public health problem throughout the world. Viral hepatitis is one of the commonest causes of jaundice in children and is one of the diseases still unconquered even in the West. In recent years, there has been a better understanding of the epidemiology and clinical picture of the disease which has helped in the formulation of control measures. However, in the absence of isolation of the specific virus and specific serological diagnosis, many lacunae in our knowledge still persist. Infective hepatitis is endemic in most of the urban areas in India. Large number of epidemics have been investigated and reported in recent years. (Mehta and Acharya, 1973). Aurangabad is an area of endemic hepatitis. Dhamahere and Nadkarni (1962) reported water-borne epidemic of infective hepatitis in part of Aurangabad city. As a general rule, infective hepatitis is a mild disease with low mortality. However, the commonest cause of death in this disease is acute liver failure leading to coma and death. In children, its course is usually mild but can lead to post-hepatic cirrhosis, chronic cholestasis, subacute necrosis and hepatic failure. The chances of such complications are high, particularly in our country, where malnutrition is extremely rampant (Rao *et al*., 1975). Viral hepatitis in children has many interesting facets. Of particular interest is its possible relationship to Indian childhood cirrhosis (Madhavan *et al.*, 1973); a theory of viral aetiology (Achar *et al.*, 1960, Chandra, 1970) is based on a frequent occurrence of jaundice at the onset, history of jaundice and hepatitis - like illness in other family members and clinical resemblance of the acute fulminant variety to submassive necrosis of the liver seen in infective hepatitis.

There is no specific therapy for viral hepatitis and evaluation of any drug in the treatment of infective hepatitis is difficult as it is a variable disease. There are many clinical types having a variable course and prognosis. However, a drug which can restore liver function quickly without producing harmful effects and is reasonably inexpensive is most welcome. Various reports of corticoid therapy are available (Evans *et al.,* 1953; Huber and Willey, 1956; Vakil *et al.,* 1965; Vakil, 1973). However of late, there have been number of reports about the efficacy of Liv.52 in this disease (Arora, 1961. Ramalingam *et al.,* 1971, Sule *et al.,* 1961, Mitra *et al.,* 1974, Rao *et al.,* 1975).

We present a clinical profile of viral hepatitis in children from Aurangabad.

MATERIAL AND METHODS

One hundred and thirty cases of viral hepatitis admitted to the Medical College Hospital, Aurangabad, during the period January 1972 to July 1975, were studied. The majority of cases were admitted in the summer months. A detailed clinical history was recorded, physical examination and routine laboratory investigations such as complete blood picture, urinalysis for bile salts, bile pigments, urobilinogen and liver function tests, which included serum bilirubin, serum transaminases

etc. were done. Liver biopsy could be done only in 40% of cases on admission and it could not be repeated after treatment in any of the cases.

OBSERVATIONS

The ages of these children ranged from 5 months to 12 years: the highest incidence being in the age group 4-7 years. (Table 1). Sixty-five percent belonged to the poor socio-economic group, 20% to the middle income and the rest to the high income group.

Table 1: Age distribution of infective hepatitis cases

Age	No. of cases	Percentage
Below 1 year	10	7.7
1 - 3 years	35	27.0
4 - 7 years	45	34.6
8 - 12 years	40	30.7

Jaundice lasted for less than 10 days prior to admission in 50% of cases and 10-20 days in 30.7% of cases (Table 2).

Table 2: Duration of jaundice prior to admission

Duration	No. of cases	Percentage
Less than 10 days	65	50.0
10 - 20 days	40	30.7
21 - 30 days	15	11.6
More than 30 days	10	7.7
Total	130	100.0

Table 3: Complaints on admission

Complaints	No. of cases	Percentage
Lack of appetite	130	100.0
Fever	100	77.0
Yellow urine	125	96.1
Jaundice	80	61.5
Clay coloured stool	65	50.0
Nausea or vomiting	40	30.7
Pain in abdomen	50	42.3
Insomnia	15	11.6
Chills and rigors	10	7.7
Bleeding tendencies	5	3.8

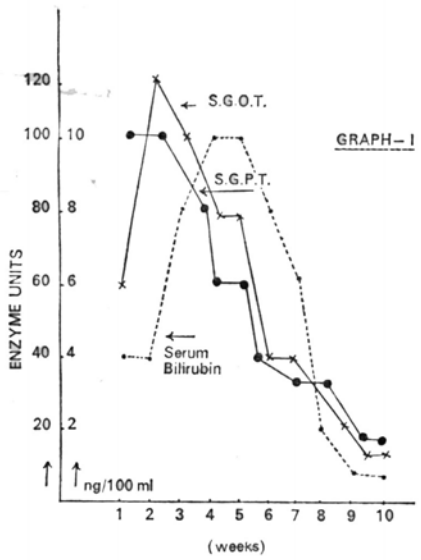
Sudden onset of loss of appetite was the most frequent complaint, and jaundice was noticed by mothers in 61.5 percent cases, the time of appearance of jaundice varying from 2-6 days after loss of appetite. Fever was present in 77 percent and yellow urine was noticed by the mothers in 96.1 percent cases. Ten cases (7.7%) presented with fever, chills and rigors and were mistaken for malaria on admission. None of the cases presented with coma.

Liver was enlarged in 97 percent of cases, the hepatomegaly ranging from 1-6 cm. below the costal margin in the midclavicular line. Tenderness was present in 70 percent of cases although it was difficult to assess tenderness in infants below 1 year. Lymph nodes were enlarged in 22% of cases. Ascites was observed in 3% of cases and bradycardia was detected in 1.5 percent of cases.

Serum bilirubin levels ranged from 2-10 mg% and alkaline phosphatase levels from 10-25 Kingsley Armstrong units. Elevated levels of serum glutamic oxaloacetic and pyruvic transaminases (SGOT and SGPT) were found in 90 percent cases. Graph I shows the mean levels of serum bilirubin, SGOT, SGPT in these children at weekly intervals.

The histopathological changes in the liver included vacuolation of hepatocytes in varying degrees with scanty and granular cytoplasm. The portal tracts were infiltrated with mononuclear cells and segmented leucocytes. Focal areas of necrosis were observed in most of the cases studied. Cholestasis with bile pigment in hepatocytes and biliary canaliculi was found in a few cases.

Mean Serum Bilirubin S.G.O.T., S.G.P.T. Levels at Weekly Intervals



The majority of the cases were treated with Liv.52 (2 teaspoonfuls twice a day upto 2 years of age, 2 teaspoonfuls thrice a day from 2-10 years of age and 2 tablets thrice a day to cases between 10-12 years). Only a few cases were treated with prednisone 1-2 mg/kg for 1-2 weeks. There were 5 cases who could not survive due to development of hepatic coma.

DISCUSSION

Infective hepatitis is a systemic viral disease with predominant involvement of the liver. It is a disease of varying severity ranging from mild anicteric hepatitis to acute fulminating hepatitis leading to precoma and coma. The disease is widely prevalent in India. In the Marathwada region it is endemic with periodic outbreaks of epidemics, especially in the summer and early rainy season.

Infective hepatitis is caused by hepatitis virus 'A'. It occurs endemically and more characteristically in epidemics in institutions, schools and in military camps. The virus is excreted in the faeces and infection follows the ingestion of contaminated food; under conditions of community living, spread is facilitated and epidemics may occur. Viral hepatitis may be caused by hepatitis virus 'B' (serum hepatitis) in hospitals and clinics where syringes and needles are used without sterilisation before each inoculation. It has been well proved that serum hepatitis can also be transmitted by the orofaecal route and hepatitis 'A' infection by transfusion. Both the types of hepatitis have indistinguishable clinical manifestations which may vary from severe to mild and silent forms. In both, the virus has been shown to be present in the blood in the presymptomatic period. It is therefore apparent that blood from supposedly normal donors who have never had hepatitis may yet contain virus A or virus B or both.

There is strong circumstantial evidence that Australia (Au) antigen is a surface antigen of serum hepatitis (SH) virus which is mildly infectious and which can be transmitted by contagion (Krugman *et al.*, 1967). At present detection of Au (Hepatitis-Associated) antigen is the only simple means available for detecting the SH virus. In the United States, urban endemic hepatitis, particularly in adults, is largely caused by the SH virus (Prince *et al.,* 1970). Data are meagre (Hills *et al.,* 1970); Sehgal and Aikat, 1970; Sama *et al.,* 1971; Sundaravalli, *et al.,* 1971; Kelkar *et al.,* 1973, a, b).

Laboratory acquired infection (Epigastric hepatitis)—endemic hepatitis can occur in laboratory personnel working with materials containing the hepatitis B virus (Sutnick *et al.*, 1971). In India, hepatitis B infection was noted by Baxi (1973) and Mahajan *et al.,* (1974).

Viral hepatitis is often a self-limiting disease, but can have high mortality and morbidity during an epidemic (Melnick, 1957), particularly where malnutrition is endemic. Therapy with an inexpensive and efficient drug is, therefore, welcome. Peak incidence in the present study was in the pre-school age group, an observation made by other workers (Dave *et al.,* 1972; Rao *et al.,* 1975). The higher

frequency noted in our study during the summer months also agrees with the observation of other workers (Viswanathan, 1957; Rao *et al.,* 1975; Mitra *et al.,* 1974). Lack of appetite with irritability and fever should alert the physician for the development of jaundice, in a few days and urine examination for bile salts, bile pigments and urobilinogen should be a routine in such cases. Early detection is of importance, to avoid administration of hepatotoxic drugs, such a chlorpromazine, etc. for vomiting, or drugs such as cyprohepatadine hydrochloride for appetite. Abdominal pain was noticed in 42.3 percent; this is in agreement with the observations of Sule *et al.,* (1968). It is attributed to perihepatitis, distension of Glisson's capsule, phlegmonous enteritis or sudden shrinkage of liver in acute hepatic necrosis (Litchman, 1953).

In the present study Liv.52 was given in the majority of cases in view of our previous experience (Mitra *et al.,* 1974). However, corticosteroids were given a few cases including 5 cases of coma. No control trial of Liv.52 was done in the present study as we have recently reported our experiences from this institution. (Mitra *et al.,* 1974). The efficacy of Liv.52 was observed by several other workers (Arora, 1961, Ramalingam *et al.,* 1971, Dave *et al,* 1972, Deshpande and Sheth, 1972). We feel from our experiences that the absence of untoward side-effects with even prolonged administration makes this drug safe and effective in the management of viral hepatitis in children.

SUMMARY

In the Marathwada region of Maharashtra viral hepatitis is endemic with periodic outbreaks of epidemics, especially in the summer and early rainy season.

One hundred and thirty cases of viral hepatitis in children admitted to Medical College Hospital, Aurangabad were studied. The ages of these children ranged from 5 months to 12 years the highest incidence was in the age group 4-7 years the majority of them (65%) belonged to the poor socio-economic group. Jaundice had lasted for less than 10 days prior to admission in 50% of cases. Mortality rate was 3.8%. Complete biochemical recovery took 8-10 weeks in the majority of the cases.

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