

CONGENITAL SYPHILITIC HEPATITIS

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ABSTRACT

Hepatitis is a known manifestation of congenital syphilis, however hepatitis developing during penicillin therapy is unknown. Ten patients of congenital syphilis were studied and serial liver enzymes were done before and after starting penicillin therapy. Eight of the ten patients developed hepatitis after initiating penicillin therapy. Whether hepatitis in these cases was secondary to toxic reaction to the products of treponemal lysis or an autoimmune reaction needs to be investigated.

Key words: Congenital syphilis, Hepatitis, Penicillin therapy.

Congenital syphilis has become a rare disease with the advent of penicillin and routine antenatal screening, but the entity still continues to plague the developing world. Hepatitis as a manifestation of syphilis is reported(1), but not much is reported on hepatitis developing after starting penicillin therapy. We report a series of congenital syphilitic patients who manifested clinical and biochemical features suggestive of hepatitis following the initiation of penicillin therapy.

Material and Methods

Ten patients were admitted to the Neonatal Division, Department of Pediatrics of Safdarjang Hospital, New Delhi, between September, 1987 and October, 1989 with a diagnosis of congenital syphilis. The diagnosis was based on clinical and radiological features,* supported with positive serology both in blood as well as in CSF. Serum bilirubin and liver enzyme (SGOT and SGPT) were estimated before and after instituting crystalline penicillin therapy. Penicillin therapy was given for 10 to 21 days in the usual doses depending on presence and/or absence of positive CSF serology(2). Serum bilirubin and liver enzymes were done serially till they showed the decline. Liver biopsy was done in a single case at post mortem.

Results

Table I depicts the clinical manifestations along with their investigations in the congenital syphilitic patients. Of the 10 infants with symptomatic congenital syphilis, 8 developed clinical and biochemical features of liver dysfunction. The peak serum bilirubin levels ranged from 7.2 to 18.0 mg/dl with conjugate fraction being 3-12

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TABLE I—Clinical Parameters in Congenital Syphilitic Infants

S. No.	SGA	Skin lesion	Hepato-splenomegaly	Pseudo-paral-ysis	Radio-logical skeletal lesion	VDRL		S. Bil. (mg/dl) T/Conj.	SGOT/SGPT (KA units)	
						Blood	CSF		Pre R	Post R
1.	+	+	-	-	-	+	+	5.4/3.2	20/30	140/82
2.	+	-	+	-	+	+	-	11.4/6.2	18/20	200/102
3.	+	-	-	+	+	+	+	6.3/3.0	16/18	130/62
4.	+	-	+	-	-	+	+	10.2/4.0	40/32	210/110
5.	+	+	-	-	+	+	-	5.2/1.2	18/21	30/18
6.	+	+	+	+	+	+	+	18.0/12.0	70.0/100.0	350/500
7.	+	-	-	-	+	-	-	7.2/5.4	30/18	160/87
8.	+	-	+	-	+	+	+	6.0/2.1	18/20	32/22
9.	+	-	+	-	+	+	-	16/3.2	26/18	350/180
10.	+	-	+	-	+	+	-	8.0/3.1	18/20	450/380

SGA - Small for gestational age:

mg/dl. Peak transaminase levels ranged from 6 to 15 times the normal values. Liver enzymes were within normal limits in two cases. Increased levels were seen in the remaining 8 patients. Liver dysfunction persisted for more than 5 weeks in single case. All the babies had uneventful recovery and serum bilirubin and transaminases levels returned to normal within three to five weeks. One case was severely growth retarded (term SGA 1100 g). Post mortem liver biopsy in this infant showed non-specific features of neonatal hepatitis (giant cells with foci of extramedullary hematopoiesis).

Discussion

Hepatitis associated with congenital syphilis has been reported in approximately 30% of cases(1). Whether penicillin therapy for congenital syphilis potentiates liver cell dysfunction was not known till Long *et al.*(3), in their series of 7 patients

observed features of hepatitis following penicillin therapy in four of five cases in whom serial liver enzymes were done. We in our series of 10 cases observed liver dysfunction in 8 of the 10 cases in whom liver enzymes were done serially before and after penicillin therapy. Liver biopsy done in a single case at post mortem showed features of giant cell hepatitis.

The question regarding hepatitis following penicillin therapy is still unresolved. The pathogenesis as postulated by Long *et al.* may be a toxic reaction to the products of treponemal lysis or an autoimmune reaction or whether hepatitis is secondary to viable treponemes is still controversial(3). However, the fact that liver dysfunction continues long after eradication of the organism refute the postulation that hepatitis following penicillin therapy is secondary to viable treponemes.

The above observation suggests that pathogenesis of hepatitis following therapy can only be ascertained if immunological

markers/antibodies to treponemal antigen are studied in liver biopsy specimen. Hepatitis following penicillin therapy is a benign and self limiting disease, liver biopsy is not routinely indicated. However, if the disease is progressive, liver biopsy may be done.

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NOTES AND NEWS

XVI ANNUAL CONFERENCE OF THE GUJARAT STATE BRANCH OF INDIAN ACADEMY OF PEDIATRICS

The XVI Annual Conference of the Gujarat State Branch of the Indian Academy of Pediatrics is to be held at Himatnagar on March 3, 1991, Sunday.

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