

EARLY SYPHILIS AND HEPATIC FUNCTION (Clinical, Biochemical and Pathological study)

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Summary

20 cases of secondary syphilis were studied clinically, biochemically and histopathologically for hepatic involvement. Most of the liver function tests were normal in our series. 25% cases showed nonspecific features with cellular infiltration and acute necrotic changes.

Recent reports and articles¹⁻⁴ have focussed the attention on the lacunal in hepatic involvement in early and secondary syphilis as against the well known luetic hepatic damage of congenital and tertiary syphilis. Baker et al¹, and Lee et al² described liver disease associated with secondary syphilis. They described a clinical picture simulating hepatitis which was accompanied by moderate rise of serum bilirubin, S.G.O.T. and disproportionately high values of serum alkaline phosphatase. While their articles opened new regions to explore, Sherlock's³ plea to venereologist and hepatologists to mount a joint campaign in this particular problem stimulated us to study the hepatic functions and structure in early syphilis as seen in V.D. Department, Gandhi Hospital, Secunderabad, A.P.

Material and Methods

Twenty patients with secondary syphilis (L II) admitted in Gandhi Hospital, V.D. Department, were studied in detail to evaluate their liver functions and architectures. The

patients who attended O.P. clinic with positive history of exposure, signs of secondary syphilis and positive V.D.R.L. test were admitted to the hospital. Detailed history was taken and clinical examination done. Liver function tests including serum bilirubin, serum proteins, vandenbergh test, thymol turbidity test, serum alkaline phosphatase, SGOT and SGPT were done. Liver biopsy was done taking the usual precautions and a comprehensive histological study made. The crushed liver tissue was examined under darkground illumination for spirochetes. Tests for Australian Antigen could not be done because of lack of facilities. However, possibility of associated infective hepatitis was excluded clinically and biochemically.

Results

Altogether 20 cases of secondary syphilis were studied in detail. The age group ranged between 16 years and 40 years. Females constituted 25% of cases (5 cases). All of them had history of exposure within a period ranging between 6 months to 1 year. Three cases (15%) had multiple exposures. None of these cases had jaundice, hepatomegaly or any past history suggestive of hepatitis. Almost

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all patients had significant titres of VDRL which ranged from 16 to 32 dil. Serum proteins were within normal limits and there was no change in NG ratio, S.G.O.T. or S.G.P.T. Serum bilirubin, Thymol turbidity test, urinary bile pigments were within normal range. Serum Alkaline phosphatase levels were normal. Histopathological study of liver revealed normal liver architecture in 15 (75%) cases. 5 cases (25%) showed non-specific features with lymphocytic infiltration. One case showed periportal inflammatory reaction and mononuclear cell infiltration. The other cases showed scattered areas of acute necrosis of liver and this patient also had acute nephrosis. In all these cases liver tissue was examined under darkground illumination but spirochetes could not be detected.

Discussion

Syphilis is a disease known to involve all the systems and organs of the body in its various stages. It is well known that hepatic damage occurs in congenital and tertiary syphilis as pericellular cirrhosis and Gummatous liver respectively. Liver function tests and sophisticated histological tests were not well developed when syphilis was in its heyday. With the advent of penicillin the uninhibited clinical and pathological picture of syphilis is modified. That is why very little was known regarding hepatic functions and structure in early syphilis until Baker et al¹ and Lee,² described single cases in which serological tests showed moderate rise in serum bilirubin and S.G.O.T. with disproportionately high alkaline phosphatase levels. Baker's case¹ showed necrotic areas extending from portal tract to central veins in which there was a total loss of parenchymal cells with moderate infiltration of lymphocytes. The portal tracts were oedematous and contained inflammatory cells. Lee's case² showed preservation of basic architecture of liver.

In our detailed study of twenty cases, there were no significant alteration in any of the biochemical liver function tests. The disproportionate rise of serum alkaline phosphatase reported by Baker¹ and Lee² could not be confirmed in our cases. In our series, the microscopic findings in liver tissues were variable and uncharacteristic. In all our cases, the basic architecture of liver was maintained. In one case there were scattered areas of necrosis with lymphocytic infiltration. In five cases, there was cellular infiltration mostly by lymphocytes. Thus this study indicates that histological changes in liver can occur without appreciable biochemical alterations. This type of picture probably occurs with inadvertant usage of penicillin. However by examining more patients and also with increasing availability of investigating tools, this problem can be further studied. We believe that our study raises more questions than it answers; but hope that we have stimulated our colleagues in carrying out studies in large number of patients with early syphilis to understand more fully the effect of the disease on liver architecture and or function.

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