

## HEPATIC LESIONS IN SCARLET FEVER

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It is well established that in scarlet fever, along with ordinary and characteristic symptomatics, phenomena also may be observed, occasionally indicating the involvement of the liver in the pathological process: subicterus, urobilinogenuria, hyperbilirubinemia, slight hepatomegalia. On the other hand, cutaneous alterations may be present in the pre-icteric stage of epidemic hepatitis, strongly resembling in some instances the scarlet fever exanthema. Doubtful scarlet fever in these cases is rendered much more justified when the disease displays an onset with angina and vomiting accompanied by high temperature ("pseudogrippe" form of the pre-jaundice period). The difficulties encountered in establishing the diagnosis were further increased, recently in particular, due firstly, to the higher incidence of cases with epidemic hepatitis and secondly, to the very often slight, atypical course of present day scarlet fever. Concomitant hepatic disorders in scarlet fever have been long since known (1, 2, 3, 4, 6, 8, 9, 10, 11). In order to solve the problem of differential diagnosis between scarlet fever and hepatitis, we should before all, answer the question concerning the degree of widespreading of the "hepatic-subicteric" syndrome in scarlet fever, and wherefrom do characteristic for epidemic hepatitis phenomena start.

With particular regard to the question thus posed for solution, our attention was mainly attracted by the patients with scarlet fever and epidemic hepatitis among the clinical material available. Our analysis was chiefly based on a series comprising 302 patients with scarlet fever; in addition to usual paraclinical investigations, 127 patients were also subjected to a number of tests routinely carried out for hepatic function. The patients with scarlet fever under observation were aged from 4—32 years, 10.88 years in the average. All of them sustained a slight form of scarlet fever. Complications were noted in five patients merely: polyarthritides — 4 (1.32%) and glomerulonephritis — 1 (0.33%).

Subicteric tinge of the skin, especially after transitory pressure exerted with fingers, was observed in 142 patients (47.02%). The latter sign was established during the climax of the disease and was invariably slightly manifested and transitory.

Hepatomegaliae were found in 26 patients (8.61%), and in ten of them the enlargement of the organ was associated to palpatory painfulness in the right subcostal area. The enlargement of the liver was usually moderate and without changes in consistency and surface. Hepatomegaly was frequently accompanied by additional "hepatic" symptoms as subicterus, elevated urobilinogenuria, extended Weltmann ribbon, positive thymol test and increased serum bilirubin.

In 50.80% of the affected by scarlet fever, the MacLagan test exhibited values exceeding 40 PU up to 97 PU. The comparative assessment of the latter finding, however, with the remainder of clinical and laboratory indices in the same patients did not warrant its assumption as a phenomenon invariably displaying a parallel course with the rest of the data for hepatic damage. The identical test performed in 22 healthy individuals of the same age group resulted in a mean value ranging from  $33 \sigma \pm 13$  PU.

The Weltmann reaction was normal in 79.13% of the patients, prolonged coagulation band — in 11.20% and reduction — in 9.67%.

Interesting and rather indicative results were obtained during determination of bilirubin in the serum of our patients (according to the method of Jendrassik, Cleghorn and Graf). It is true that the mean value obtained —  $0.52 \sigma \pm 0.33$  mg% approximates the normal one —  $0.41 \sigma \pm 0.18$  mg%. Found during investigation of 22 healthy subjects, but the analysis of the results received in different patients demonstrates not very few cases of borderline values, as well as cases with obvious hyperbilirubinemia. Thus, in 25.26% of the patients, the serum bilirubin was within the 0.70—0.99 mg% range, and in 7.36% — between 1.00 and 1.50 mg%. In one patient with scarlet fever the bilirubinemia amounted to 2.61 mg%. We believe that the increase of serum bilirubin is an expression almost exclusively of a hepatic lesion, and the least of hemolysis. In support of this concept, the fact is mainly emphasized that the quantitative changes in the biliary pigments of our series exhibit a course, parallel to the remaining clinical and paraclinical phenomena, indicative of involvement of the liver in the morbid process. The determination of both bilirubin fractions show that the increase of total bilirubin in the serum is effected at the expense of its direct fraction mainly. In 15 of the 18 patients studied purposefully, the concentration of direct bilirubin is higher than indirect values. The mean value of the former in the same group of patients amounts to  $0.41 \sigma \pm 0.18$  mg%, whereas in the group of healthy individuals investigated, it is  $0.17 \sigma \pm 0.11$  mg%. The very slight impairment of biliary pigments metabolism may reveal a course characterized merely by increase of direct bilirubin without accounting for placement of the total bilirubin beyond its normal quantitative limits (7).

The investigation of urine for biliary pigments revealed an increase of urobilinogen merely in 8.27% of the patients, whereas bilirubinuria was established in none of patients. The urobilinogenuria did not exhibit pronounced casual relationship with the indices hitherto described.

The activity of the enzymatic alkaline phosphatase in the group of patients studied proved to be within the normal limits.

With a view to ascertaining the degree and duration of the hepatic lesion discussed, a catamnestic follow-up of 21 individuals with past history of scarlet fever was carried out; they were called for check-up examinations one to six months after dismissal from the clinic. Subjective complaints were not recorded. The paraclinical investigations did not provide data for hyperbilirubinemia. The thymol test displayed a mean value of  $34 \sigma \pm 13.5$  PU, i. e. nearly equal to the value of healthy individuals ( $33 \sigma \pm 13$  PU). The Weltmann test likewise exhibited no deviations whatever from normal values.

Against the background of clinical and laboratory follow-up of 302 patients with scarlet fever, the following conclusions were reached:



1. In the course of scarlet fever data are established almost by rule, proving the presence of hepatic lesion.

2. Such lesions are slight and transitory.

3. The latter lesion could be proved mainly by positivation of the Mac-Lagan test, presence of subicterus, increase of bilirubin in the serum and some other indices; however, none of the indices revealed parallelity related to occurrence and intensity.

4. The hyperbilirubinemia is characterized by hepatic genesis and is not caused by the hemolytic properties of the etiological agent of scarlet fever.

5. The indices observed do not exhibit qualitative characteristic features, specific for scarlet fever. The differences between the latter disease and epidemic hepatitis relevant to the indices listed are mainly quantitative.

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#### ПОРАЖЕНИЯ ПЕЧЕНИ ПРИ СКАРЛАТИНЕ

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#### РЕЗЮМЕ

При клиническом и лабораторном прослеживании 302 больных скарлатиной часто и почти закономерно обнаруживаются данные о поражении печени, которое является легким и быстро переходным. Это поражение доказывается главным образом позитивированием пробы Мак Лагана (у 50,8% больных), субиктером (47,02%) и граничными (0,70—0,99 мг% у 25,26% больных) или слегка повышенными (1,00—1,50 мг% — у 7,36% больных) значениями билирубина в сыворотке крови, главным образом за счет его директной фракции. Наблюдаемые изменения не показывают присущих только скарлатине особенностей.