CONSIDERATIONS ON THE COMBINED VIRAL HEPATITIS AND TUBERCULOSIS AFFECTION

V. Zozikov, M. Radkov, V. Zheliazkova, D. Tzoncheva

Viral hepatitis is considered as a widely spread infectious disease, not infrequently affecting tuberculosis patients. Therefore, having in mind that hepatic lesions in the course of tuberculosis might be also caused by the specific infection itself, or by the administered drugs with an eventual toxo-allergic effect, a number of essential epidemiological, diagnostical and therapeutical problems arise, namely: is it a matter of a combination between viral hepatitis (VH) and tuberculosis, or of a hepatic lesion with other (specific or paraspecific) genesis, what therapeutical policy should be adopted (antituberculous means — hepatotoxic? glucocorticoids?), is there a possibility that clinico-laboratory changes occur in the course of either disease, are antiepidemic measures necessary, etc.

As a rule, tuberculous patients are often affected with VH — in the opinion of some authors — six times more frequently than the general population (4). This is attributed to their lowered bioresistance and to the frequent pareneteral (inoculation) infection (1, 6, 19).

A number of antituberculous drugs (PASA, streptomycin, isonicotinic acid derivatives, pyrazinamide) are capable to produce drug induced hepatitis of a toxic and/or allergic nature. The data concerning the incidence and severity of the latter are quite discordant (3, 4, 5, 13, 14, 17, 18, 20, 21). While Kalinowski (18), out of a total of 3148 tuberculous patients undergoing treatment with PASA, established a hepatic lesion in 0.18 per cent only, Ugryumov and co-authors accept a toxo-allergic hepatitis in 12 per cent out of a series of 600 patients, subjected to treatment with a variety of antituberculous agents. Moreover, there are reports describing hepatic dystrophy, caused by paraaminosalicylic acid (PASA) (20).

The opinions about the interference between VH and tuberculosis running a concomitant course are by no means convergent. Many authors fail to observe a mutual unfavourble influence (2, 6, 8, 12, 16, 22). Luksha (9) supports the statement that VH in tuberculosis patients runs a milder clinical course. Nevertheless, the reports on a unilateral or bilateral mutual negative influence are quite numerous (4, 7, 10, 15, 21).

Usually, VH in tuberculosis patients runs a heavier course. The latter fact is related as much to the unfavourable premorbid background (general chronic diseases, subclinical specific or iatrogenic hepatal lesion), as to the form of hepatitis itself (very often serum hepatitis which is usually heavier).

In the work submitted a series of 32 tuberculosis patients with concurrent VH affection are reviewed. They are of both sexes and various ages

(from 4—72, averaging 41.2 years). The tbc process disclosed the following localizations: lungs — 26 cases, bones and joints — 5, and peritoneum — one. Pulmonary tuberculosis was in the phase of suction — 13 patients, decomposition — 6, and fibrosis — seven. The affection dated back 1—15 years ago, or 5.2 years average. In four cases there was a past history of recent hemoptysis, and five were Koch-positive. Five patients were affected with a second chronic disease: cor pulmonale, ulcer and heart failure.

Ten of the patients, by the time of VH affection, have undergone treatment with streptomycin, PASA and rimiphon. The same scheme of therapy was proceeded with after their admission to the infectious clinic.

VH was diagnosed on the ground of a number of criteria, such as acute and cyclic course, comparatively severe (in 12 patients serum bilirubin amounted to 10—20 per cent) and prolonged icterus (mean duration 14.7 days), and considerable laboratory variations (substantial intensification of the activity of transaminases mainly). The diagnosis was confirmed epidemiologically in eleven patients: in 3 of them serum hepatitis was considered highly probable (hemotransfusions sustained in the last few months), whereas in 7 the same hepatitis form was merely suspected (frequent diagnostic-laboratory and therapeutic-medicamentous injections).

During the HV affection no detectable subjective or objective changes were noted in the course of tuberculosis. In general, febrility, sweating and coughing were the same as before the occurrence of icterus. The X-ray controls likewise failed to reveal a deterioration of the basic condition.

To clarify the question of the eventual influence exerted by the tuberculous background on the clinico-laboratory picture of VH, a comparison was made with a control group of 120 VH patients, free of preceding or concomitant diseases. From the comparative table appended, it becomes evident that no one-way differences exist between the two groups of patients which would allow to accept with certainty a heavier or slighter course of the viral hepatitis in tuberculous patients.

A number of rather important indicators (such as a higher incidence of middle and heavy clinical forms, longer hospitalization and bilirubinuria, more frequent hyperbilirubinemia, prolonged Weltmann test at discharge) point to a slightly heavier and rather protracted course of hepatitis among tuberculous patients. However, the impression upon comparative study of the remaining criteria is to the contrary (duration of icterus, hepatomegalia, hypertransaminasemia, positive Maclagan test upon discharge). Obviously, all differences referred to are by no means substantial, and most of them are statistically unreliable. Nevertheless, the predomination of slight clinical forms with ensuing considerably shorter hospitalization among the control cases is statistically reliable, and most probably points to a slightly heavier course of hepatitis in tuberculous patients.

The tuberculous patients were subjected to conventional treatment in compliance with the clinical form and severity of VH — mainly bed rest and dietary regimen, vitamins and glucose (per os or intravenously). In four patients glucocorticoids were also prescribed, and no side effects whatsoever were recorded.

Clinical and Laboratory Characteristics of the Patients Reviewed

Indicators	Group of patients	Tuberculosis + viral hepatitis n=32	Viral hepatitis n=120	Statistical reliability
Clinical forms — %	Slight	40.58	60.00	$p<0.05$
	Middle heavy	40.58	29.18	$p>0.2$
	Heavy	12.56	4.16	$p>0.1$
	Miscellaneous	6.28	7.66	$p>0.8$
Duration in days	Hospitalization	33.93 24.04	24.03±7.69	$p=0.02$
	Hyperurobilinogenuria	13.80	13.95	
	Bilirubinugenuria	12.30	8.90	
	Icterus	14.70	15.60	
% at discharge	Hepatomegalia	43.75	46.67	$p>0.8$
	Mild form of hyperbilirubinemia	50.0	36.66	$p>0.1$
	Increased GPT	25.00	59.16	$p<0.01$
	Prolonged Weltmann test	59.37	45.16	$p>0.1$
	Positive MacLagan test	65.62	85.83	$p<0.01$

Insofar VH affection is concerned, the patients were dismissed with completely or almost completely subsided clinical manifestations and with normal or close to normal laboratory results. No lethal cases were recorded.

Conclusions

1. There are quite reliable clinical and laboratory criteria for viral hepatitis diagnosis in tuberculosis patients.
2. The basic affection — tuberculosis — is not influenced perceptibly by the concurrent hepatitis.
3. Virus hepatitis among tuberculosis patients does not show substantial one-way clinico-laboratory variations, but nevertheless, it runs up to a certain degree a slightly heavier and protracted course.
4. The conventional hepatitis therapy leads to a good or satisfactory therapeutical outcome.

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О СОЧЕТАНИИ ВИРУСНОГО ГЕПАТИТА С ТУБЕРКУЛЕЗОМ

В. Зозиков, М. Радков, В. Желязкова, Д. Цончева

РЕЗЮМЕ

Прослежены 32 больных туберкулезом, заболевших вирусным гепатитом. Проведено сравнение с другими 120 больными вирусным гепатитом, без сопровождающих других заболеваний. Вирусный гепатит у больных туберкулезом не показал значительных клинико-лабораторных отклонений в одном направлении, но до известной степени протекает немного тяжелее и длительнее. Основное заболевание — туберкулез. не влияется доловимо присоединившимся гепатитом.