

HEPATOBIILIARY SCINTIGRAPHY WITH ^{99m}Tc-DISIDA OF PULMONARY TUBERCULOSIS PATIENTS IN TREATMENT WITH ISONAZID, RIFAMPICIN AND PYRAZINAMIDE – RELATION WITH THE NUTRITIONAL STATE AND THE ACETYLATOR PHENOTYPE

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ABSTRACT: This study aimed to evaluate 13 pulmonary tuberculosis patients (10 men and 3 women) by hepatobiliary imaging immediately before (M1) and two months (M2) after the beginning of a continuous oral treatment with isoniazid (INH), rifampicin (RFP) and pyrazinamide (PRZ). The patients (mean age of 43 +/- 12 years) had their acetylase phenotypes determined after the diagnosis of pulmonary tuberculosis. At M1 and M2, they received standard doses of ^{99m}Tc-DISIDA, endovenously. Images of this radiopharmaceutical kinetics were successively obtained, from the heart through the intestinal lumen, with the aid of an Orbiter scintillation camera. The radiopharmaceutical hepatic uptake and the excretion velocities were calculated with the aid of computerized software. The serum biochemical nutritional parameter and the liver integrity were determined at M1 and M2. The patients were from urban areas, of low economic class, of low literacy, undernourished, alcoholics and/or smokers. None of them presented clinical or laboratory history of hepatobiliary diseases. At M1, there were no hepatobiliary alterations, including the uptake and excretion of ^{99m}Tc-DISIDA. At M2, there was a significant increase in the aspartate aminotransferase mean level and a reduction in the velocity of ^{99m}Tc-DISIDA excretion. Other liver enzymes showed a non-significant increase compared to the mean values recorded at M1 and to the normal reference

values. In general, the patients presented better nutritional conditions at M2 than at M1. The hypothesis raised was that the triple treatment acts on the liver inducing the microsomal P450 enzyme system, mainly by INH and RFP. The present data suggest that the triple treatment for pulmonary tuberculosis does have some influence on the liver, apparently more adaptive than toxic, since the recorded laboratory alterations were mild and none of them was clinically manifested. It is possible that the active metabolites resultant from the biotransformation of the anti-tuberculosis drugs stimulated the liver function and increased the excretion of the radiopharmaceutical, which runs through the same route as bilirubin.

KEY WORDS: pulmonary tuberculosis, ^{99m}Tc -DISIDA, isoniazid, rifampicin, pyrazinamide.

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