Effect of tuberculostatic on antioxidant enzymes and thiobarbituric acid-reactive substance in patients with pulmonary tuberculosis

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It is known that patients with pulmonary tuberculosis produce enormous amount of free radicals, mainly as a consequence of phagocytic respiratory burst. This oxidative stress could also play an important role in the action mechanism of tuberculostatic drugs, which may induce hepatotoxicity. In this study the effects of tuberculostatic drugs (pyrazinamide, isoniazid and rifampicin) on erythrocyte antioxidant enzyme (superoxide dismutase - SOD, glutathione peroxidase – GPx and catalase) activities as well as on thiobarbituric acid-reactive substance (TBARS) concentration were investigated in two group of patients with pulmonary tuberculosis: group I with hepatotoxicity, (n=60; 34 males/26 females) and group II without hepatotoxicity (n=62; 39 males/23 females). All investigated parameters were determinate simultaneously before tuberculostatic therapy (T₀) and three-weeks (T₁) later. Obtained results were compared mutually and with control group (n=61). Before therapy we noted significant lower level of SOD (p<0.01) and GPx activity (p<0.001) in both groups with tuberculosis vs. control group, with the lowest level of SOD in group with hepatotoxicity (1149.5±235.7 vs. 1316.8±215.4 U/gHb; p<0.01). We showed significant increase in GPx (p<0.01) and catalase activity (p<0.05) during therapy with tuberculostatic vs. T₀ group and this rise was more pronounced in patients with tuberculosis without hepatotoxity. There was no significant fall in TBARS concentration during therapy. A significant correlation was observed between the activity of antioxidant enzymes, hepatotoxicity and severity of disease. So, our results indirectly suggest that tuberculostatic can reduce toxic tissue damage through the increase of antioxidant activity. An appropriate antioxidant therapy may improve to faster recovery, especially in patients with hepatotoxicity.