Biochemical profile of cholestasis and oxidative stress markers in chronic hepatic disorders

Received for publication, november, 15, 2013.
Accepted, december, 1, 2013

Mihaela BASA¹, Natalia Rosoiu², Adrian Sever RUS³
¹ Clinical Laboratory, Emergency Military Hospital, Constanța, Romania
² "Ovidius" University Faculty of Medicine, Biochemistry Department, Constanța, Romania; Academy of Romanian Scientists 54 Splaiul Independentei 050094, Bucharest,
³ Emergency Military Hospital, Constanța, Romania

Abstract

Background: Due to the essential role in the metabolization of exo- and endogenous compounds, the liver is exposed to numerous oxidative stresses and, when the production of reactive species outruns the activity of purifying enzymes, hepatic lesions occur. Oxygen reactive species may affect reversibly or irreversibly the biochemical substances thus influencing the fluidity and the membranous function, the cellular metabolism and even the genes expression.

Methods: in this study were included 194 patients, from which: 64 patients with C-type hepatic disease (viral C hepatitis – 41, viral C cirrhosis - 23), 44 patients with B-type hepatic disease (viral B hepatitis – 27, viral B cirrhosis - 17), 71 patients with alcoholic etiology hepatic disease (chronic alcoholic hepatitis – 37, chronic alcoholic cirrhosis – 34) and 15 healthy patients as a control group. For these patients we determined the markers for the cholestasis syndrome and the antioxidant system (both enzymatic and nonenzymatic)

The objective of this study is to provide information regarding the estimation of the disease severity through appreciation methods of the cholestasis and oxidative stress.

Results: between the parameters means from hepatitis and cirrhosis with the same etiology there are statistical significant differences for the following analytes: total bilirubin, conjugated bilirubin, unconjugated bilirubin (not valid for the C-type viral diseases), gamma glutamyl transpeptidase (valid only for the B-type viral chronic hepatic diseases), alkaline phosphatase, total cholesterol, glutathione reductase (valid only for the C-type viral diseases), super oxide dismutase (valid only for male patients infected eith B-type viral chronic hepatic diseases), serum albumin.

Conclusions: When the aggression over the hepatic cell is caused only by the alcohol metabolites, the cholestasis extension, compared to the injury of the hepatocyte membrane, is smaller than in the cases with double determination (viral and alcoholic). The extension of the cholestasis syndrome and the permeability increase of the hepatocyte membrane lead to an accentuated imbalance between antioxidants and the reactive species of oxygen by the decrease of total antioxidant status and the increase of antioxidant enzymes activity (glutathione reductase, super oxide dismutase). The great efficiency of antioxidants is represented by their synergy in action, each of them acting on different levels of the free radicals evolution chain; this efficiency is demonstrated by the statistical significant correlations between them.