Histopathological changes and oxidative damage in hepatic tissue of rats experimentally infected with *Babesia bigemina*

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Abstract

The present study was aimed to investigate oxidative stress, DNA damage, and histopathological alterations in hepatic tissues of splenectomized Wistar rats experimentally infected with *Babesia bigemina*. Rats were challenged with 5x10⁶ infected erythrocytes. *Babesia* infection was confirmed both with Giemsa’s staining blood smears and nested-PCR amplified region of apical membrane antigen-1 (AMA-1) gene. Parasitemia reached approximately 10% at day 5 post-infection. Livers of infected rats were enlarged and darker in color, became extremely brittle with marked congestion. Microscopic evaluation showed cytoplasmic clearing of hepatocytes and severe hydropic changes with significantly dilated sinusoids containing macrophages and also intrasinusoidal parasitized erythrocytes. Severe infiltration of lymphoplasma cells was also present throughout the liver parenchyma. Furthermore, Kupffer cells were enlarged and, occasionally, containing *Babesia*-parasitized erythrocytes. The activity of Glutathione (GSH) and catalase (CAT), and total antioxidant capacity (TAC) were also significantly decreased (p < 0.05) after infection of rats with *B. bigemina*. *B. bigemina* infection also induced a significant increase (p < 0.05) in hepatic malondialdehyde (MDA) and nitric oxide-derived products (NOx) concentrations as well as amount of endogenous hepatocytes DNA damage. Hepatic damage was also reflected through the measurement of lactic acid dehydrogenase (LDH) and protein carbonyl content (PCO) in liver cells. These two indices of liver injury were also significantly elevated (p < 0.05) during *B. bigemina* infection. Evaluation of correlation between assayed variables in infected rats revealed that MDA levels were positively correlated with PCO, NOx, LDH and DNA damage in the infected group and negatively correlated with GSH, CAT and TAC. There was also an inverse relationship between the antioxidant enzymes activities of GSH, CAT and TAC with PCO, NOx and DNA damage in infected rats. However, NOx showed positive correlation with PCO and DNA damage in infected rats. On the basis of the above results it can be concluded that the *Babesia* infection increases oxidative stress markers, protein carbonyl content and DNA damage and decreases antioxidant enzymes activities in the liver. These results suggest that *B. bigemina* infection could alter the liver histopathology and causes DNA damage following oxidative stress in hepatic tissue. Further studies are needed to precisely define how hepatic tissue damage takes place in *B. bigemina* infection.

Key Words: liver, oxidative damage, rats, antioxidants, *Babesia bigemina*

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