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INHIBITION OF HMGB1 RELEASE DECREASES BOTH APOPTOTIC AND AUTOPHAGIC ACTIVITY IN THE HEPATOCYTES AND REDUCES LIVER INJURY IN STEPTOZOTOCIN TREATED RATS

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Introduction: Hepatocellular death is the main trigger of liver disease. Since diabetic patients are very prone to liver diseases there is a urgent need to identify key regulators of cell death processes. High-mobility group box protein 1 (HMGB1) is a non-histone nuclear protein with a role in apoptotic and autophagic activation when it is present in cytosol and extracellular space. The aim of this study was to elucidate HMGB1 contribution to liver injury trough activation of apoptosis and autophagy in streptozotocin (STZ)-induced diabetic rats since the role of HMGB1 in hepatic cell death during diabetes is partially known.

Methods: Diabetes was induced with a single intraperitoneal (i.p.) injection of STZ (65 mg/kg). Inhibition of HMGB1 release was achieved by ethyl pyruvate (80 mg/kg/i.p./daily). We followed changes in expression of serum and cytosolic HMGB1 and its interaction with TLR4 and RAGE and how these changes affect on apoptotic and autophagic activity and liver morphology.

Results: In the serum of diabetic rats elevated levels of HMGB1 were accompanied by increased HMGB1 interactions with TLR4 and RAGE receptors. Enhancement in these interactions led to increased activity of both apoptotic and autophagic signaling pathways resulting in altered liver morphology and acummulation of autophagosomes in hepatocytes. Inhibition of HMGB1 release caused reduction in apoptotic and autophagic activity which resulted in preservation of normal liver architecture and decreased number of autophagosomes.

Conclusion: HMGB1 causes liver damage through activation of apoptosis and autophagy, therefore it's a suitable new target for prevention of liver diseases in diabetic patients.

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