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Activation of Macrophage and Its Impact on structural defects of rat Liver in experimental Diabetes

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Statement of the Problem: The Morphofunctional state of the liver in diabetes mellitus should be assessed since all metabolic processes occur in liver; hepatocytes have the insulin-dependent mechanism of glucose metabolism and 40-60% of insulin is inactivated in liver. The structure of damaged liver tissue and mechanisms of hyperglycemia which cause the damage were described in many investigations. The assessment of various methods of damaged liver correction was performed. It is known that hepatic macrophages play key role in tissue injury and repair. However, its effects on regenerative processes in diabetic liver are not investigated enough. We previously set that the macrophage modulator 3-aminophthalhydrazid (3-APH) activated anti-inflammatory and antioxidant functions in monocyte-macrophage cells.

The purpose of this study: is to evaluate the impact of macrophages activated by 3-APH on structural defects in diabetic rat liver.

Methodology: A damaged liver tissue and hepatocyte reparation were studied in alloxan diabetic rats after modulation of macrophage activity by 3-APH during 30 and 60 days.

Finding: The morphological study of diabetic liver tissue revealed degenerative changes in hepatocytes, inflammatory processes in the portal and intralobular stroma, microangiopathy and blood circulation disorders. Structural disorders of liver parenchyma increased at a prolongation of AD up to 60 days. It was found that the administration of 3-APH contributed to reduction of damage and restoration of normal liver histological structure due to activation of reparative processes.

Conclusion: Thus, macrophage activation in diabetic liver causes increase in hepatocyte activity and compensation of structural damage in liver tissue. 3-APH is likely to be a promising drug for correction of liver tissue in diabetes due to its hepato protective effect.

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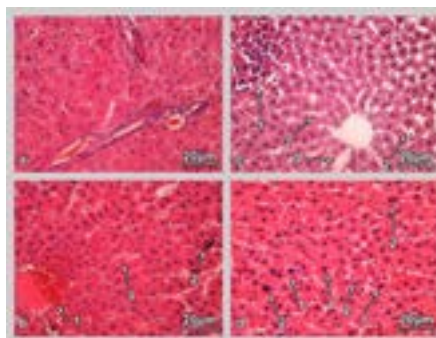


Figure 1: a - histological structure of intact rat liver tissue. b –diabetic liver tissue (30 days): 1- plethora in central venules, 2- lymphocytic infiltration of sinusoidal capillary, 3 - degeneration of hepatocytes. c – diabetic liver tissue (60 days): 1 - intralobular necrosis of hepatocytes, 2 - edema, vasodilatation, 3 - desquamation of endothelial of sinusoidal capillaries. d –diabetic liver tissue (60 days) treated by 3-APH: 1 - polypliod hepatocytes, 2 – dual-nucleus hepatocytes. Staining by hematoxylin-eosin. X400