

Hepatic Stem Cells and Adipocytokines in Nonalcoholic Fatty Liver Disease pediatric patients after Laparoscopic Sleeve Gastrectomy

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Hepatic stem/progenitor cells (HpSCs) are facultative bipotential stem cells [1], located in Canals of Hering and surrounded by a specialized niche [2]. We aimed to investigate the modulation of HpSC niche and the modification of adipocytokine expression induced by laparoscopic sleeve gastrectomy (LSG) in adolescents with nonalcoholic fatty liver disease (NAFLD). Twenty obese adolescents who underwent LSG and with biopsy-proven NAFLD were included. At baseline (T0) and 1 year after treatment (T1), patients underwent clinical evaluation, blood tests, and liver biopsy. HpSCs, hepatic stellate cells (HSCs), macrophages, and adipocytokines were evaluated by immunohistochemistry and immunofluorescence. Liver biopsies after LSG demonstrated a significant improvement of NAFLD Activity Score and fibrosis. Immunohistochemistry indicated a significant reduction of hepatocyte cell cycle arrest, HpSC activation, activated HSC, and macrophage number after LSG compared with T0. Hepatocyte expression of adiponectin was significant higher after LSG than at T0. Moreover, LSG caused decreased resistin expression in Sox9+ HpSCs compared to T0. The number of S100A9+ macrophages was also reduced by LSG correlating with resistin expression in HpSC. Finally, serum levels of pro-inflammatory cytokines significantly correlated with macrophages and activated HSC numbers. The histologic improvement induced by LSG is associated with the reduced activation of local cellular cross-talks, thus, strengthening the role of stem cell niche and hepatic adipocytokine production in the pathogenesis of NAFLD.

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References

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Key words

Liver, Stem Cell, NAFLD, adiponectin.