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Conclusions: Serum uric acid is independently associated with elevated ALT, as a surrogate for NAFLD, and thus may serve as a serum marker and should be further investigated as a risk factor for NAFLD.

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THE INTERACTION BETWEEN VISCERAL ADIPOSE AND HEPATIC TISSUE AFFECTS LIVER INJURY IN NAFL AND NASH PATIENTS

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Background and Aims: Non-alcoholic fatty liver disease (NAFLD) and the more severe form non-alcoholic steatohepatitis (NASH) are the most common liver diseases in western countries. The interaction between liver and adipose tissue is fundamental for the development of NAFLD and NASH. Aim of this study was to investigate the interaction between adipose tissue and liver at the time of bariatric surgery.
Methods: Blood, visceral adipose tissue, and liver tissue samples were obtained from 40 (median age 46±8.6 y; 29 w/11 m; BMI 51.3±8.1 kg/m^2) morbidly obese patients undergoing bariatric surgery. Histopathological assessment of the biopsies was done according to the NAFLD activity score (NAS). Blood samples taken before surgery were analyzed for parameters of liver injury (M30, M65). Hepatic and adipose tissue mRNA levels of one gene for triacylglycerol regulation (CGI-58) and a central regulating gene for fatty acid storage and glucose metabolism (PPARγ2) were assessed by qrtPCR.
Results: CGI-58 mRNA expression was increased in adipose tissue of morbidly obese patients and showed a strong correlation to low density lipoprotein (LDL) in sera and with the NAS. In morbidly obese individuals mRNA of PPARγ2 was significantly upregulated in hepatic and adipose tissue. Moreover, PPARγ2 mRNA levels in adipose tissue were correlated significantly with hepatic PPARγ2 mRNA levels. The ratio of markers for apoptosis and necrosis (M30, M65) in serum also correlated significantly with mRNA levels of metabolic regulators in adipose tissue.
Conclusions: The presented data shows that adipose CGI-58 and PPARγ2 mRNA expression is associated on the one hand with mRNA expression in the liver and on the other hand with the grade of liver injury.

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INTERLEUKIN 15 IN NONALCOHOLIC FATTY LIVER DISEASE AND OBESITY

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Background and Aims: *Background:* Non-alcoholic fatty liver disease (NAFLD) and obesity represent widespread pathologies associated with low-grade inflammatory processes. Experimental data suggest crucial role of anabolic cytokine interleukin 15 (IL-15) in NAFLD development. Y. Cepero-Donates et al. showed that increased secretion of IL-15 promotes fat accumulation in the liver stimulating hepatic inflammatory response in mice.
The aim was to investigate IL-15 concentration in patients with NAFLD associated with obesity depending on steatosis degree and anthropological parameters. The study included 32 patients with NAFLD associated with obesity, 31 normal weight patients with NAFLD and 26 normal weight volunteers without hepatic steatosis.
Methods: NAFLD diagnosis and assessment were performed by abdominal ultrasound examination. Steatosis degree was evaluated using hepatorenal index (HRI). For all NAFLD patients other causes of hepatic steatosis were excluded. Obesity was measured using body mass index (BMI) and waist circumference (WC). Concentration of IL-15 were measured using enzyme-linked immunosorbent assay kit.
Results: The results showed that IL-15 concentration was significantly increased in patients with NAFLD comparing to control group ($p < 0.05$). Concentrations of IL-15 observed in NAFLD patients with concomitant obesity were significantly higher than those measured in NAFLD patients with normal weight ($p < 0.05$). In all NAFLD patients IL-15 correlated with HRI supporting its role in hepatic fat accumulation. Furthermore, in patients with NAFLD there was significant correlations of IL-15 concentration with BMI ($p < 0.05$) and WC ($p < 0.05$).
Conclusions: Patients with NAFLD and concomitant obesity might have more significant proinflamatory status that could be caused by adipose tissue dysfunction and cytokine synthesis abnormalities. Increased synthesis of IL-15 observed in NAFLD obese patients supports its role in hepatic lipid accumulation found in experimental studies. Further investigations are needed to explore correlations of IL-15 with histological findings in NAFLD obese patients.

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NO EVIDENCE FOR PLATELET HYPERACTIVITY IN PATIENTS WITH NON-ALCOHOLIC FATTY LIVER DISEASE

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Background and Aims: The mechanism of increased prevalence of cardiovascular disease in patients with non-alcoholic fatty liver disease (NAFLD) is unknown. Platelet activation has been implicated as a contributor of the increased risk of cardiovascular disease in the metabolic syndrome, but its role in NAFLD is unclear. We, therefore, assessed the platelet activation status in lean and obese individuals in comparison to patients with various histological severities of NAFLD.
Methods: Blood was drawn from 68 patients with biopsy-proven NAFLD (simple steatosis n = 24, NASH n = 22, and NASH cirrhosis n = 22), 20 lean controls (BMI <25 kg/m^2), 20 overweight controls (BMI >25 kg/m^2), and 15 patients with alcoholic (ASH) cirrhosis. Subjects with congenital coagulation disorders, recent infection (<2 weeks), anticoagulant or anti-platelet therapy, and recent transfusion with blood products were excluded. We studied basal and agonist-induced platelet activation using flow cytometry. In addition, we studied plasma levels of von Willebrand factor (VWF), the VWF-cleaving protease ADAMTS13, and the platelet activation marker soluble P-selectin.
Results: Basal platelet activation was comparable between lean and overweight controls, patients with NASH, and patients with NASH or ASH-related cirrhosis. There was only a slight increase in basal platelet activation in patients with simple steatosis compared to overweight controls. Agonist-induced platelet activation was decreased in patients with cirrhosis, most notably in patients with ASH-related cirrhosis, but similar between patients with non-cirrhotic NAFLD and controls. Plasma levels of VWF were increased in patients with NASH or ASH cirrhosis compared to all other groups; however levels were comparable between lean and overweight controls, patients with simple steatosis, and patients with NASH. ADAMTS13 levels were comparable between all patients and controls. Soluble P-selectin levels were mildly elevated in plasma from patients with NASH, NASH cirrhosis, or ASH cirrhosis compared to lean and overweight controls.