THE ROLE OF INSULIN-LIKE GROWTH FACTOR-1 ON STEATOHEPATITIS

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Purpose: Recent experimental studies have revealed that growth hormone and its transcription factor STAT5 were related to hepatosteatosis in mice. Loss of signal transducer and activator of transcription factor-5 leads to hepatosteatosis and impaired liver regeneration. We aimed to investigate the role of IGF-1 in hepatosteatosis and steatohepatitis in humans. Method: We included 272 NAFLD patients and 110 age, sex and body mass index (BMI)-matched healthy controls. We measured routine blood biochemistry and complete blood count, as well as IGF-1, insulin, c-peptide, ferritin, hsCRP and ESR. We also calculated HOMA-IR to measure insulin resistance. We subdivided NAFLD patients into hepatosteatosis and steatohepatitis. Results: Age, sex and BMI were similar between NAFLD and controls. IGF-1 levels were significantly lower in NAFLD patients (120,6±48,2) than controls (148,9±53,8), (p0,0001). IGF-1 levels were also lower in steatohepatitis subgroup (93,4±27,8) than hepatosteatosis subgroup (123,1±49,0), (p:0,032). Waist circumference, fasting blood glucose, HbA1c, uric acid, hsCRP, AST, ALT, GGT, WBC, hemoglobin, hematocrit, ferritin, insulin, c-peptid and HOMA-IR measurements were significantly higher in NAFLD patients than controls (for all values: p0,0001). Total cholesterol (p:0,026), triglycerides (p0,0001), ESR (p:0,006) were significantly higher in NAFLD patients than controls. HDL-chelesterol levels were significantly lower (p:0,002) in NAFLD patients than controls. Conclusion: This study supported previous findings of experimental studies in that, IGF-1 levels were lower in hepatosteatosis and steatohepatitis. Growth hormone-IGF-1 system may be involved in the pathogenesis of NAFLD.