Role of APN and TNF-α in type 2 diabetes mellitus complicated by nonalcoholic fatty liver disease

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ABSTRACT. Non-alcoholic fatty liver disease (NAFLD) is a chronic liver disease caused by non-excessive alcohol consumption and is the most common cause of elevated levels of serum liver enzymes. We examined changes in adiponectin (APN) and tumor necrosis factor-α (TNF-α) in type 2 diabetes mellitus (T2DM) complicated by NAFLD and their relationships with insulin resistance (IR). Forty-two T2DM, 39 NAFLD, and 45 T2DM complicated with NAFLD (complicated group) patients were enrolled in this study. Body mass index, fasting blood plasma glucose (FPG), fasting insulin, triglyceride (TG), alanine aminotransferase, gamma-glutamyl transpeptidase, APN, TNF-α, and homeostasis model of assessment (HOMA)-IR were determined. The degree of fatty liver was graded according to liver/spleen computed tomography ratio and intrahepatic vessel manifestations. Compared with the T2DM and NAFLD groups, fasting blood plasma glucose, alanine aminotransferase, gamma-glutamyl transpeptidase, TG,

TNF-α, and HOMA-IR in the complicated group were significantly increased, while APN was significantly reduced. Body mass index in the complicated group was significantly higher than in the T2DM group. The complicated group was prone to severe fatty liver compared with the NAFLD group. APN was negatively correlated with body mass index, fasting blood plasma glucose, TG, TNF-α, and HOMA-IR. TNF-α was negatively correlated with APN, but positively correlated with FPG, fasting insulin, TG, and HOMA-IR. The complicated group had clear IR. A more severe degree of fatty liver was associated with higher HOMA-IR and TNF-α and lower APN. APN was an important factor for antagonizing inflammation and mitigating IR.