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Analysis of clinical variables associated with TNF-alpha and IL-6 expression in visceral adipose tissue

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Background: The obesity has high prevalence in many populations worldwide exhibiting high morbidities and premature mortality rates. Proinflammatory cytokine release from infiltrating immune cells in turn contributes to a chronic state of inflammation, with enhanced local secretion of proinflammatory cytokines, interleukin (IL)-1, IL-6, and tumor necrosis factor (TNF)-α, from the expanding adipose tissue mass. Extensive studies have demonstrated that obesity-related insulin resistance and type 2 diabetes are associated with inflammation in adipose tissue, in an obese state massively expanded adipose tissue secretes a variety of inflammatory markers, cytokines, and chemokines at elevated levels. The non-alcoholic fatty liver disease (NAFLD) is the hepatic manifestation of the metabolic syndrome and, frequently coexists with obesity, dyslipidemia and insulin resistance. Understanding the pathophysiology of hepatic steatosis is extremely important and some hypotheses have been considered to explain the pathogenesis of NAFLD especially the insulin resistance for accumulation of fatty acids in hepatocytes and the oxidative stress as the second stimulus for the development of inflammation and fibrosis.

Aim: To analyze dietary intake, clinical, biochemical, and anthropometric measures and correlation the expression of interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α) in tissue adipose and hepatic tissue in morbidly obese patients.

Material and Methods: Participants were morbidly obese adults individuals (n=21). Dietary survey by food recording was done for estimative of food intake. Assessmentswere made for clinical, anthropometric, hematological and biochemical variables. Samples of liver were submitted to morphological analysis for diagnosis of steatosis. Expression of IL-6 and TNF-α in liver samples and adipose abdominal tissue were performed by using quantitative real time-polymerase chain reactions. Groups were compared by using univariate statistical analysis and level of significance was set at 5% ($p<0.05$).

Results: We can observe that the waist circumference showed a tendency of static association with IL-6 expression in visceral adipose tissue. ($p=0.06$, $\beta=0.01$). There was a significant association between IL6 expression in visceral adipose tissue and total caloric intake ($p=0.02$). The expression of IL6 and TNF alfa in visceral adipose tissue was associated with levels of HDL ($p=0.04$ and $p=0.05$), ferritin ($p=0.02$ and $p=0.03$) and ALT ($p=0.03$ and $p=0.00$). The decreased levels of HDL relate to increased expression of interleukin in visceral adipose tissue (β-0.01). Although liver transaminase did not show significantly altered between the groups of patients with NAFL and NASH but ALT elevation was associated both a higher expression of IL6 (p=03) and TNF (p=00) in liver tissue.The expression of TNF invisceral adipose tissue also showed a positive association with elevated IL6 liver tissue ($p=0.00$ $\beta=0.04$) and IL6 visceral adipose tissue ($p=0.00$ $\beta=1.25$).

Conclusions: Our results suggest that imbalances in the rate of body weight and caloric intake may be associated with the modulation and expression of these cytokines. High expression of TNF-α and IL-6 may also be associated with changes in serum cholesterol and triglycerides. In this study, the expression of these cytokines in visceral adipose tissue showed no direct relationship with the severity of liver disease, but the expression of TNF in visceral adipose tissue showed a positive association with the overexpression of IL-6 in hepatic tissue and visceral adipose tissue. Thus it appears that these cytokine may be indirectly linked to liver damage by increasing expression of IL-6 in liver tissue, which in turn is directly related to elevated ALT, which is a marker of hepatocellular injury.