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THE CORRELATION BETWEEN SERUM OMENTIN-1 LEVELS AND INSULIN RESISTANCE IN TYPE 2 DIABETIC WOMEN

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Adipose tissue has been known as a source of a variety of bioactive peptides called adipokines. Recently, a new protein omentin-1 (also named intelectin-1, endothelial lectin and intestinal lactoferrin receptor) has been identified as a major visceral (omental) fat secretory adipokine. Omentin is highly and selectively expressed in visceral adipose tissue relative to subcutaneous adipose tissue and together with visceral obesity play important roles in carbohydrate and lipid metabolism, homeostasis, insulin resistance, diabetes and cardiovascular function. Insulin resistance links nutrition, glucose, insulin and adipokines in various metabolic important tissues. While omentin is highly expressed in human visceral fat tissue, circulating omentin levels are reduced in obese subjects. Omentin is also down regulated in association with obesity- linked metabolic disorders including insulin resistance, glucose intolerance and type 2 diabetes. The aim from our study was to increase our knowledge about omentin-1 and its relationship with type 2 diabetes mellitus, insulin resistance and obesity. The study included 60 female patients with type 2 diabetes mellitus with their age group (40-60). 30 aged matched female subjects formed the control group. All subjects were subjected to full clinical examination, body weight, height, BMI, waist and hip circumference, fasting plasma glucose, fasting insulin, fasting serum lipid profile, HbA1c and fasting omentin-1 levels. Insulin resistance was calculated as HOMA-IR. We found the serum Omentin-1 significantly lower in cases as compared to control group (p value <0.001). We also found that plasma omentin-1 is inversely related to obesity (negatively correlated to BMI, weight, waist and hip circumference). A statistically significant positive correlation between weight, BMI, waist, hip circumference, fasting glucose, HbA1c, insulin and insulin resistance within cases were detected in our study. In our study, the ROC curve analysis showed that the cut off value of serum omentin-1 levels was 22.2 pg/mL (yielding sensitivity and specificity values of 100.0% for both). These results emphasize the usefulness of the discriminant ability of plasma omentin -1 to differentiate between cases (who were obese and having high insulin resistance) & controls. Our study showed that omentin-1 levels are low in type 2 diabetic and insulin resistant females. We also found that plasma omentin-1 is inversely related to obesity (BMI, weight, waist and hip circumference).

IMPACT OF GLYCOXIDATION ON STRUCTURAL AND IMMUNOLOGICAL CHARACTERISTICS OF IGG ISOLATED FROM DIABETES TYPE II PATIENTS: A CLINICAL STUDY

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Immunoglobulin G (IgG), a 150 kDa molecule and the most abundant serum protein has been described as sensitive to glycation, nitration, oxidation and other modifications. Amongst these post translational modifications, glycation and oxidation being most common, deserves special attention. Increasing evidences suggest the role of glycoxidation in the onset and progression of diabetes type II (T2DM). This study was designed to elaborate the cumulative effect of glycation (using Methylglyoxal) and oxidation (using Hydroxyl Radical) on IgG with reference to T2DM. We found appreciable binding of T2D auto-antibodies towards epitopes in hydroxyl radical modified methylglyoxal glycated IgG (OH•-MG-IgG). Furthermore, spectroscopic characterization of IgG isolated from T2D patients (T2D-IgG) revealed structural changes in comparison to IgG from healthy human subjects (NH-IgG); with hyperchromicity in UV absorbance spectroscopy, quenching in fluorescence spectroscopy, decreased β sheet content in far-UV CD spectroscopic analysis and shifting of amide I and II bands in FTIR spectroscopy. OH•-MG induced damage in T2D-IgG was evaluated by anti-OH•-MG antibodies (generated in female rabbits) using competitive binding immunoassay. Compared to NH-IgG, T2D-IgG was observed to be more specific towards the immunogen (OH•-MG-IgG). Our results confirm that IgG in T2D patients is prone to glycoxidation induced structural damage leading to the generation of neo-epitopes that renders the protein immunogenic.