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PATHOGENETIC ROLE OF OMENTIN IN THE DEVELOPMENT OF CHRONIC DIABETIC COMPLICATIONS

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Introduction. The impact of diverse adipokines, as byproducts of adipose tissue metabolism, on the progression of angiopathy in individuals diagnosed with type 2 diabetes mellitus (T2DM) underscores the significance of addressing vascular complications in diabetes. The modified release of these compounds can mimic the mechanisms underlying angiopathy development in these individuals. Augmentation in the thickness of the intimo-medial segment (TIMS) correlates with cardiovascular risk factors and vascular emergencies. A correlation between increased TIMS and the onset of metabolic syndrome has been identified. The connection between arterial structural alterations and the secretion of specific adipokines in obese patients with type 2 DM remains ambiguous.

Aim: The objective is to ascertain the omentin-1 levels in obese individuals with T2DM and to evaluate the presence and character of the correlation between its levels and TIMS of the carotid arteries in these subjects.

Materials and Methods: A comprehensive examination was conducted on 90 diabetes patients receiving treatment under hospital conditions. Group 1 comprised 57 participants (30 females and 27 males) with a body mass index exceeding 25 kg/m2, with an average age of 56,5 ± 10,28 years and an average diabetes duration of 7,45 ± 3,23 years. Group 2 consisted of 31 patients (17 females and 14 males) with a body mass index of 25 kg/m2 or less, with an average age of 55,61 ± 6,24 years and

an average diabetes duration of 6,31 ± 2,71 years. The control group comprised 27 apparently healthy individuals, matched to groups 1 and 2 in terms of gender and age. All participants provided written consent to participate in the study, adhering to ethical standards outlined in the Declaration of Helsinki.

Omentin-1 levels were assessed through enzyme immunoassay utilizing test systems provided by Bender MedSystems GmbH (Austria). Carotid artery ultrasound was conducted employing a MyLab50X device, TIMS measurements were taken in B-mode approximately 1.0–1.5 cm proximal to the bifurcation of the common carotid artery, following widely accepted guidelines.

Correlation analysis, utilizing the Spearman correlation coefficient, was employed to ascertain the direction and nature of relationships. Intergroup differences were evaluated using the Mann-Whitney method. Statistical analysis was conducted using "Statistica 6.1" software (StatSoft Inc., USA, serial number RGXR412D674002FWC7). Statistically significant distinctions were acknowledged at $p<0,05$.

Results and Discussion:

Patients diagnosed with T2DM and obesity exhibited notably lower omentin-1 levels compared to individuals in group 2, with a marked difference of 121,16% ($p<0,05$). Further examination of omentin-1 levels among subjects, categorized based on the degree of diabetes compensation, revealed that in group 1, consisting of patients with HbA1c values ≥ 8%, omentin-1 levels were lower compared to those with compensated diabetes, showing a reduction of 27,22% ($p<0,05$). Conversely, subjects in the control group exhibited omentin-1 levels 47,38% ($p<0,05$) higher than those in group 1 with compensated diabetes and 63,24% ($p<0,05$) higher than patients with HbA1c ≥ 8%. Notably, in patients with T2DM and normal body weight, the level of omentin-1 remained unaffected by the state of carbohydrate metabolism compensation.

In patients diagnosed with T2DM alongside obesity, with a diabetes duration exceeding 5 years, omentin-1 levels were significantly lower by 36,27% ($p<0,05$) compared to those with a shorter disease history. This disparity was slightly lower in

group 2, where the difference stood at 24,81% ($p<0,05$). Individuals in group 1 with a diabetes duration of up to 5 years exhibited omentin-1 levels 62,74% lower ($p<0,05$) than the control group. With a prolonged diabetes duration in group 1 patients, omentin-1 levels were lower by 76,27% ($p<0,05$) compared to the control group. Consequently, the duration of T2DM appeared to correlate with a gradual reduction in omentin-1 levels, particularly in the presence of increased body weight.

Analysis of TIMS registration of carotid arteries in patients with T2DM revealed that in group 1, the indicator was significantly higher on both sides compared to the corresponding indicators in individuals in group 2, by 15,81% ($p <0,05$) and 20,83% ($p <0,05$) respectively. Between group 1 and practically healthy individuals, the TIMS difference was 37,51% ($p<0,05$) and 26,92% ($p<0,05$) on the right and left sides, respectively. However, in group 2, the corresponding indicators did not differ from those of the control group.

The compensation state of carbohydrate metabolism had a notable impact on pathological changes in arterial vessels in the context of T2DM and obesity. In group 1, the TIMS indicator in individuals with diabetes decompensation on the left was 15,81% ($p<0,05$) higher than the corresponding indicator in individuals with an HbA1c level <8%, while the difference on the right was 16,71% ($p<0,05$).

In individuals within group 1, diabetes duration exceeding 5 years correlated with higher TIMS values on the right by 15,97% ($p<0,05$) compared to those with a shorter history of diabetes, with a similar difference observed on the left at 17,19% ($p<0,05$). In the control group, TIMS indicators were lower on the left by 21,21% ($p<0,05$) and 32,01% ($p<0,05$) compared to patients in group 1 with diabetes duration under 5 years and over 5 years, respectively. On the right, the disparity was 30,32% ($p<0,05$) and 46,59% ($p<0,05$), respectively. However, in group 2, the variation in TIMS values between individuals with different durations of DM was not significant.

Chronic diabetic complications in patients of group 1 influenced the condition of the carotid arteries. In the presence of complications, the TIMS indicator compared to patients without complications was higher by 11,28% ($p<0,05$) on the left and by 12,26% ($p<0,05$) on the right. Moreover, the discrepancy between the data of the

control group and group 1 was 25,43% ($p<0,05$) on the right and 37,43% ($p<0,05$) on the left. Conversely, in patients of group 2, the TIMS indicator did not significantly differ with or without chronic complications of diabetes.

Correlation analysis aimed to evaluate the presence and directionality of the relationship between the studied indicators. A negative correlation was identified between TIMS (particularly on the right) and the blood level of omentin-1 ($Rs = -0,54$, $p<0,05$).

Our study data underscore the significance of decreased blood levels of omentin-1 in the development of pathological changes in arterial vessels, as indicated by an increase in the TIMS index of the carotid arteries, particularly in patients with T2DM in combination with obesity.

Conclusions:

1. The co-occurrence of obesity and T2DM is associated with reduced omentin-1 levels compared to patients solely diagnosed with T2DM and normal body weight. These dynamics are influenced by the decompensation of diabetes and the duration of the disease.

2. The TIMS rate of carotid arteries in patients with T2DM alongside obesity was notably elevated, particularly in cases of diabetes decompensation, long-term diabetes, and the presence of chronic diabetic complications.

3. A negative correlation was identified between the level of omentin-1 and the TIMS indicator of carotid arteries in patients with T2DM combined with obesity.