ABSTRACT:

Background: It is known that metabolic syndrome characterized by diabetes, hypertension, dyslipidemia, and central obesity is associated with the syndrome of early vessels aging, characterized by a change in elasticity of the vessel wall. The early manifestation of the metabolic syndrome in younger people in the modern society, leads to earlier manifestation of the complications of early vessels aging, and the combination of several risk factors is crucial and leads to acceleration of the vessels aging. Elastin is one of the main building blocks of the vessel wall. Its main characteristic is its elasticity, allowing the vessel to restore its shape after stretching or shrinking. Loss of elasticity is a key component in the pathogenesis of cardiovascular complications.

Materials and methods: A study is conducted on 62 subjects with metabolic syndrome without vascular complications and 42 controls. The main objective of the study was to compare the immunological markers of elastin degradation in both groups and to assess their relationship with the risk factors characterizing the metabolic syndrome.

Results: When comparing the mean value of AEAb IgG in the control group and subject group with metabolic syndrome (respectively 0.45 / - 0.11 and 0.54 / - 0.29) statistically significant higher mean value of AEAb IgG in the group with metabolic syndrome, t = -1,85, p = 0.03 is observed. When comparing the mean value of ATEAb IgG in the control group and subject group with metabolic syndrome (respectively 0.45 / - 0.13 and 0.55 / - 0.43) statistically significant higher mean value of ATAb IgG in the group with metabolic syndrome, F = 6,83, p = 0.01 is observed. There isn’t a statistically significant difference in AEAb IGM and ATropoEAb IgM in both groups. In the whole sample AEAb IgG showed positive correlation with total cholesterol with a Pearson correlation coefficient r = 0.33, and p = 0.001. The Correlations are described by regression analysis and the relationship is linear.

Conclusion: It is proved that the AEAb IgG and ATropoEAb IgG are significantly elevated in the subjects with metabolic syndrome without manifested cardiovascular complications compared with the control group, whereas no difference in AEAb IGM and ATropoEAb IgM has been observed in the both groups.

Key words: Metabolic syndrome, AEAb IgG, ATEAb IgG, risk factor

BACKGROUND:

It is known that metabolic syndrome characterized by diabetes, hypertension, dyslipidemia, and central obesity is associated with the syndrome of early vessels aging, characterized by a change in elasticity of the vessel wall. The early manifestation of the metabolic syndrome in younger people in the modern society, leads to earlier manifestation of the complications of early vessels aging, and the combination of several risk factors is crucial and leads to acceleration of the vessels aging. Elastin is one of the main building blocks of the vessel wall. Its main characteristic is its elasticity, allowing the vessel to restore its shape after stretching or shrinking. Loss of elasticity is a key component in the pathogenesis of cardiovascular complications.

Number of studies have shown that the immune system reflects the physiological changes in the elastin metabolism in the vascular wall. In the recent years, using immunological methods, changes in elastin metabolism is examined, as a sign of vascular aging in high-risk subjects with cardiovascular complications.

There isn’t sufficient information about the nature of the immune response against tropoelastin and elastin metabolism in patients with metabolic syndrome without cardiovascular complications. The data on the prognostic value in the evaluation process of early vascular aging is scarce. Therefore, the attempt to determine the changes in elastin and tropoelastin metabolism in metabolic syndrome...
is one of the first steps to seek early immunological rather than instrumental criteria to evaluate changes of the vascular wall.

**MATERIALS AND METHODS:**

A study is conducted on 62 subjects with metabolic syndrome without vascular complications and 42 controls.

The main objective of the study was to compare the immunological markers of elastin degradation in both groups and to assess their relationship with the risk factors characterizing the metabolic syndrome.

**RESULTS:**

When comparing the average AEAb IgG in the control group and subjects with metabolic syndrome (respectively 0.45 +/- 0.11 and 0.54 +/- 0.29) was determined statistically significantly higher mean values of AEAb IgG in the group with metabolic syndrome, t = -1.85, p = 0.03, Figure.1.

![Fig.1. Comparison between the mean AEAb IgG value in healthy subjects and those with metabolic syndrome](image)

The distribution of AEAb IgM in the group of controls and subjects with metabolic syndrome is different from normal. When comparing the medians of the two groups (0.34 and 0.32, respectively) no statistically significant difference (Mann-Whitney (Wilcoxon) W test = 729.5, p = 0.06)

When comparing the average ATEAb IgG in the group of controls and subjects with metabolic syndrome (respectively 0.45 +/- 0.13 and 0.55 +/- 0.43) was statistically significantly higher values ATEAb IgG in the group with metabolic syndrome, t = -2.638, p = 0.005, Fig. 2

![Fig.2. Comparison between the mean ATEAb IgG value in healthy subjects and those with metabolic syndrome](image)

No significant difference was found between the median IgM ATEAb value in healthy subjects and those with metabolic syndrome, respectively 0.28 and 0.25, p > 0.05.

Weak correlation was determined between the AEAb IgG levels in the total group and total cholesterol with a correlation coefficient of Spearman c = 0.25, p = 0.02. Regression analysis best describes this dependence by a linear model, c = 0.25, p = 0.02, Fig.3.

![Fig.3. Correlation analysis between AEAb IgG value and total cholesterol in the total group](image)

When evaluating the correlation between AEAb IgG levels in the whole group and triglyceride levels, a moderate correlation was found with a Pearson correlation coefficient r = 0.35, and p = 0.001. Regression analysis best describes this relationship by a linear model, r = 0.35, p = 0.001, Fig.4.
In addition to total cholesterol and triglycerides the AEAb IgG levels show weak positive correlation with the LDL levels, Spearman correlation coefficient $c = 0.29$, and $p = 0.006$. Regression analysis best describes this relationship by a linear model, $c = 0.29$, $p = 0.006$, fig.5.

Weak but statistically significant positive correlation was determined between the levels of ATEAb IgG in the whole group with total cholesterol levels with Pearson’s $r = 0.25$, and $p = 0.02$. Regression analysis best describes this relationship which is linear, $r = 0.25$, $p = 0.02$, fig.6.

Statistically significant, moderate, positive correlation was determined when assessing the correlation between ATEAb IgG and total cholesterol levels in the general group with Pearson correlation coefficient $r = 0.33$, and $p = 0.001$. Regression analysis best describes this relationship which is linear, $r = 0.33$, $p = 0.001$, fig.7.

CONCLUSION:

The changes in the immunological markers against elastin and tropoelastin, showed that antibodies from class IgG, characterising the secondary immune response, are elevated in the long-term chronic inflammation characterising the metabolic syndrome and atherosclerosis, respectively, the vascular aging process. Therefore, the constellation of elevated AEAb IgG, ATEAb IgG, can be used as an early marker of vascular aging.

The following conclusion can be made: dyslipidemia and its inherent hypertriglyceridemia is one of the laboratory criteria for metabolic syndrome, Characterising the increased insulin resistancy. Increased insulin resistancy affects the turnover of elastin and tropoelastin. The correlation between elevated levels of AEAb IgG, ATEAb IgG, and triglycerides adequately reflects the process of vascular aging, apparently orchestrated by insulin resistance and therefore can be discussed as a constellation, which is a sure sign of early vascular aging.
REFERENCES:

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