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# RELATIONSHIP BETWEEN TESTOSTERONE LEVELS AND METABOLIC DISORDERS IN MEN WITH TYPE 2 DIABETES MELLITUS

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Abstract. Androgen deficiency is a common comorbidity in men with type 2 diabetes mellitus (T2DM) and influences metabolic processes and disease progression. The aim of this study was to evaluate the clinical and metabolic characteristics of men with T2DM depending on their testosterone levels. Forty patients were examined and divided into groups with normal and reduced testosterone levels. It was found that lower testosterone levels are associated with higher body mass index (BMI), increased HOMA-IR values, and pronounced lipid metabolism disturbances. Negative correlations were observed between testosterone levels, insulin resistance, and triglycerides. These findings highlight the need for early diagnosis of androgen deficiency to improve prognosis and correct metabolic disorders in men with T2DM.

**Keywords:** Testosterone; Type 2 diabetes mellitus; Insulin resistance; HOMA-IR; Dyslipidemia; Metabolic syndrome; Men's health; Endocrinology.

**Introduction.** The incidence of androgen deficiency in men with type 2 diabetes mellitus (T2DM) is increasingly recognized as an important factor influencing metabolic homeostasis. Reduced testosterone levels are associated with insulin resistance, visceral obesity, impaired lipid metabolism and a higher cardiometabolic risk [1, 2]. The present study aimed to evaluate the metabolic and biochemical characteristics of men with T2DM depending on their testosterone levels. Understanding this relationship may contribute to the improvement of diagnostic algorithms and individualized therapeutic approaches in endocrinology [3].

**Materials and methods.** A total of 40 men aged 40–65 years with confirmed T2DM were examined. All participants were divided into two groups according to serum total testosterone levels:

- 1. Normal testosterone (n = 18),
- 2. Low testosterone (n = 22; <12 nmol/L).

Anthropometric measurements (body mass index, waist circumference) and fasting blood samples were obtained to determine glucose, insulin, HOMA-IR index, total cholesterol, HDL-cholesterol, and triglycerides. Serum testosterone was measured using immunoassay methods.

Statistical analysis included mean  $\pm$  standard deviation, intergroup comparison, and Pearson correlation analysis to evaluate associations between testosterone levels and metabolic parameters. A p-value <0.05 was considered statistically significant.

**Results.** A decreased testosterone level was identified in 55% of the examined men.

Participants with low testosterone demonstrated significantly higher values of BMI, fasting glucose, fasting insulin, and HOMA-IR index compared with men with normal androgen status.

Lipid abnormalities were also more pronounced in the low-testosterone group, particularly manifested by decreased HDL-cholesterol and elevated triglycerides.

Correlation analysis showed a moderate negative relationship between serum total testosterone and HOMA-IR (r = -0.48; p < 0.05), indicating that a reduction in testosterone is

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associated with increased insulin resistance. Additionally, testosterone levels negatively correlated with triglycerides (r = -0.41; p < 0.05), suggesting the contribution of androgen deficiency to dyslipidemia in patients with T2DM.

Overall, the findings demonstrate that metabolic alterations in men with T2DM are more severe when testosterone levels are reduced. These results highlight the interplay between hormonal imbalance and metabolic dysfunction and point to the need for a comprehensive evaluation of androgen status in the management of diabetic patients [4].

**Conclusion.** Low testosterone levels in men with type 2 diabetes mellitus are associated with a more adverse metabolic profile, including increased insulin resistance and more pronounced lipid disturbances. Screening for androgen deficiency may serve as an important component of risk assessment and help optimize treatment strategies aimed at improving metabolic control and reducing cardiovascular risk in this patient population.

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