Volume 3, Issue 3, March 2025 ISSN (E): 2938-3765

THE IMPACT OF METABOLIC SYNDROME ON THE MALE REPRODUCTIVE SYSTEM

Anvarov Nuriddin Rustam Ugli
Tashkent Pediatric Medical Institute
Department of Urology, Pediatric Urology

Abstract
Metabolic syndrome (MS) is a cluster of metabolic disorders, including obesity, insulin resistance, dyslipidemia, and arterial hypertension. It has been established that MS negatively affects the male reproductive system, leading to decreased testosterone levels, impaired spermatogenesis, and erectile dysfunction. This article examines the pathophysiological mechanisms underlying these disorders and discusses modern approaches to correcting reproductive dysfunction in men with MS.

INTRODUCTION

Metabolic syndrome is one of the most common metabolic disorders, affecting 20–25% of the adult population [1]. Men with MS are more likely to experience hypogonadism, infertility, and erectile dysfunction. The key pathophysiological mechanisms include endothelial dysfunction, oxidative stress, and chronic inflammation, all of which contribute to reproductive dysfunction [2].

1. Mechanisms of Metabolic Syndrome's Impact on the Male Reproductive System

1.1. Decreased Testosterone Levels

Excessive visceral fat accumulation is associated with increased aromatase activity, an enzyme that converts testosterone into estradiol. This leads to lower testosterone levels and the development of hypogonadism [3]. Insulin resistance also suppresses luteinizing hormone (LH) secretion, reducing Leydig cell stimulation and testosterone production [4].

1.2. Impaired Spermatogenesis

Men with MS exhibit changes in semen parameters, including decreased sperm concentration and motility, as well as an increased number of abnormal forms [5]. Oxidative stress caused by hyperglycemia and dyslipidemia damages sperm DNA, reducing fertility [6].

1.3. Erectile Dysfunction

Erectile dysfunction (ED) in MS is linked to endothelial dysfunction, reduced nitric oxide (NO) production, and impaired blood flow in the corpus cavernosum [7]. Insulin resistance disrupts the PI3K/Akt pathway, which regulates smooth muscle relaxation in blood vessels, further exacerbating ED [8].

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

2. Correction of Reproductive Disorders in Metabolic Syndrome

2.1. Lifestyle Modifications

A 5–10% weight reduction improves testosterone levels and semen quality [9]. Regular physical activity and a low-carbohydrate diet reduce insulin resistance and inflammation, contributing to hormonal balance restoration.

2.2. Pharmacotherapy

Metformin enhances tissue insulin sensitivity and helps normalize testosterone levels [10]. Phosphodiesterase type 5 (PDE5) inhibitors (sildenafil, tadalafil) improve erectile function by increasing nitric oxide levels [7]. Hormone therapy is prescribed for severe hypogonadism but requires monitoring due to cardiovascular risk [3].

Conclusion

Metabolic syndrome has a significant negative impact on the male reproductive system, leading to hypogonadism, infertility, and erectile dysfunction. The main mechanisms include chronic inflammation, insulin resistance, endothelial dysfunction, and oxidative stress. Correcting MS through lifestyle changes, pharmacotherapy, and emerging approaches such as exosomal therapy can improve reproductive outcomes in men.

References

1. Eckel R. H., Grundy S. M., Zimmet P. Z. The metabolic syndrome // The Lancet. – 2005. – Vol. 365, No. 9468. – P. 1415–1428.
2. Xu H., Barnes G. T., Yang Q. et al. Chronic inflammation in fat plays a crucial role in the development of obesity-related insulin resistance // Journal of Clinical Investigation. – 2003. – Vol. 112, No. 12. – P. 1821–1830.
3. Tsai E. C., Matsumoto A. M., Fujimoto W. Y., Boyko E. J. Association of bioavailable testosterone and visceral adiposity with insulin resistance in men // Diabetes Care. – 2004. – Vol. 27, No. 9. – P. 2295–2300.
4. Pitteloud N., Mootha V. K., Dwyer A. A. et al. Relationship between testosterone levels, insulin sensitivity, and mitochondrial function in men // Diabetes Care. – 2005. – Vol. 28, No. 7. – P. 1636–1642.
5. Sermondade N., Faure C., Fezeu L. et al. BMI in relation to sperm count: an updated systematic review and collaborative meta-analysis // Human Reproduction Update. – 2013. – Vol. 19, No. 3. – P. 221–231.
6. Alves M. G., Martins A. D., Rato L. et al. Molecular mechanisms beyond glucose transport in diabetes-related male infertility // Biochimica et Biophysica Acta (BBA) - Molecular Basis of Disease. – 2013. – Vol. 1832, No. 5. – P. 626–635.
7. Esposito K., Giugliano F., Di Palo C. et al. Effect of lifestyle changes on erectile dysfunction in obese men // JAMA. – 2004. – Vol. 291, No. 24. – P. 2978–2984.
8. Montorsi P., Ravagnani P. M., Galli S. et al. Common grounds for erectile dysfunction and cardiovascular disease // Current Vascular Pharmacology. – 2009. – Vol. 7, No. 6. – P. 723–733.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5

9. Corona G., Monami M., Rastrelli G. et al. Type 2 diabetes mellitus and testosterone: a meta-analysis study // International Journal of Andrology. – 2011. – Vol. 34, No. 6. – P. 528–540.
10. Traish A. M., Guay A., Feeley R., Saad F. The dark side of testosterone deficiency: metabolic syndrome and erectile dysfunction // Journal of Andrology. – 2009. – Vol. 30, No. 1. – P. 10–22.

Web of Medicine: Journal of Medicine, Practice and Nursing
webofjournals.com/index.php/5