

REVIEW

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to the article “Effect of metabolic syndrome on testosterone in patients with metastatic prostate cancer”

by the team of authors

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This publication presents the results of a single-center, limited-size retrospective study examining the influence of metabolic syndrome (MetS)-related factors on pre- and post-treatment testosterone levels and the timing of testosterone decline in patients with metastatic prostate cancer (mPC).

Relevance of the work. Prostate cancer (PCa) is the fourth most common type of cancer, accounting for 7.3% of new cases, and the second most common solid tumor in men worldwide. The incidence of prostate cancer varies between regions, with higher rates observed in Europe and the United States compared to Asia. In recent years, China has seen an increase in the incidence of prostate cancer. Metastatic prostate cancer (mPC) is a critical stage that significantly affects the patient's prognosis. Studies have shown that the 5-year survival rate for mPCa is only 31%. In addition, the incidence of mid- and late-stage PCa in China is higher than in Europe and the United States. Metabolic syndrome (MS) is a pathological condition characterized by abdominal obesity, insulin resistance, hypertension and hyperlipidemia as defined by the World Health Organization. Diagnostic criteria for MS may vary slightly between countries and medical organizations, but the basic features remain the same. In 2004, a connection between metabolic syndrome and the risk of developing prostate cancer was first discovered. Since then, several studies have confirmed that MetS has a negative impact on both the occurrence and prognosis of PCa. Juliana Porretti and her colleagues suggested an adverse effect of MetS on epigenetic regulation in prostate cancer.

Prostate tumor development is dependent on androgens for growth and development, with gonadal androgen deprivation therapy (ADT) serving as the cornerstone of treatment. Patients with metastatic disease are candidates for medical castration with first-line hormonal therapy, such as long-acting gonadotropin-releasing hormone (GnRH) agonists or GnRH antagonists. Patients treated with ADT achieve castration levels of circulating testosterone, reduced tumor burden, improved survival, and symptomatic relief.

The article was completed at a high scientific level. The structure of the article consistently reflects the logic of the study. The scientific novelty of the peer-reviewed article lies in the study of the effect of MS, its components and the total metabolic index on testosterone levels before and after treatment in patients with mPCa. Numerous studies have consistently demonstrated a strong association between low baseline serum testosterone levels, disease progression, and poor prognosis of PCa. PCa patients with lower testosterone concentrations during medical castration live longer. mPCa patients with MS had higher testosterone values after treatment and a longer time for testosterone values to fall to their lowest levels compared to the Non-MS group.

The reviewed work contributes to the theoretical side of the development of the issue, which has its practical application, since understanding the characteristics of cellular metabolism in prostate cancer and methods of influencing anaerobic glycolysis make it possible to understand the mechanisms of tumor progression and develop new therapeutic strategies aimed at cellular metabolism.

The presented scientific article meets all the requirements for publications in scientific journals, including the Scopus and Web of Science requirements for scientific publications.

The article is recommended for publication in the scientific journal PeerJ Life & Environment.

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