

Clinical and Biological Insights of Hypogonadism in Obese and Ageing Men

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DESCRIPTION

Male hypogonadism is a clinical condition characterized by inadequate testosterone production by the testes, leading to a range of metabolic, physiological, and reproductive consequences. The condition is increasingly recognized in obese males and aging men, where testosterone levels decline due to multifactorial causes, including hormonal dysregulation, metabolic dysfunction, and testicular failure. This commentary explores the pathophysiology, clinical presentation, and diagnostic approaches for male hypogonadism in these two populations.

Pathophysiology of male hypogonadism

Hypogonadism can be classified into primary (testicular failure), secondary (hypothalamic-pituitary dysfunction), or mixed types. In obese males, increased adiposity leads to elevated aromatase activity, converting testosterone to estradiol, which suppresses the Hypothalamic-Pituitary-Gonadal (HPG) axis through negative feedback. Additionally, insulin resistance, chronic inflammation, and leptin dysregulation contribute to the suppression of Gonadotropin-Releasing Hormone (GnRH), further exacerbating testosterone deficiency.

In older men, a natural decline in testosterone, termed Late-Onset Hypogonadism (LOH), occurs due to reduced Leydig cell function, decreased gonadotropin sensitivity, and changes in hypothalamic function. Moreover, the accumulation of comorbid conditions such as diabetes, cardiovascular disease, and metabolic syndrome accelerates testosterone decline.

Clinical manifestations

Male hypogonadism presents with a spectrum of symptoms that vary based on severity and duration. Common manifestations in both obese and aging men include:

Sexual dysfunction: Reduced libido, erectile dysfunction, and decreased morning erections.

Metabolic disturbances: Increased visceral fat, insulin resistance, and unfavorable lipid profiles.

Musculoskeletal effects: Reduced muscle mass, strength loss, and osteoporosis.

Neuropsychiatric symptoms: Fatigue, depression, cognitive decline, and irritability.

Reproductive consequences: Reduced sperm production and fertility issues.

Obese men often exhibit a phenotype similar to functional hypogonadotropic hypogonadism, where lifestyle modifications can improve testosterone levels. In contrast, aging men experience a progressive decline in testosterone with irreversible testicular dysfunction.

Diagnosis of male hypogonadism

The diagnosis of hypogonadism requires a comprehensive evaluation, including clinical assessment and laboratory investigations.

Clinical assessment: A thorough history and physical examination are essential. Symptoms such as fatigue, erectile dysfunction, and decreased libido should raise suspicion. Measurement of testicular volume and assessment of secondary sexual characteristics can provide further clues.

Biochemical testing

Serum total testosterone: Morning testosterone levels (between 7-10 AM) should be measured on at least two separate occasions to confirm deficiency. A level below 300 ng/dL is commonly used as a diagnostic threshold.

Free testosterone: In conditions with altered sex hormone-binding globulin (SHBG), such as obesity, measuring free testosterone is recommended for accurate assessment.

Luteinizing Hormone (LH) and Follicle-Stimulating Hormone (FSH): Differentiates primary (high LH/FSH) from secondary (low or normal LH/FSH) hypogonadism.

Prolactin and estradiol: Helps identify secondary causes, such as hyperprolactinemia or estrogen excess.

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Glycemic and lipid profiles: Evaluating metabolic parameters is crucial, especially in obese and aging individuals.

Hemoglobin and hematocrit: To assess for anemia, which is common in testosterone deficiency.

Clinical implications and management considerations

Identifying and managing male hypogonadism in obese and older men is essential to improve quality of life and mitigate metabolic and cardiovascular risks.

Lifestyle modifications: Weight loss through diet and exercise can improve testosterone levels in obese men by reducing insulin resistance and inflammation.

Testosterone Replacement Therapy (TRT): Recommended for symptomatic men with confirmed hypogonadism. However, careful risk-benefit assessment is necessary, particularly in older men with cardiovascular concerns.

Adjunct therapies: Clomiphene citrate or human Chorionic Gonadotropin (hCG) may be considered in younger men to stimulate endogenous testosterone production.

Monitoring and follow-up: Regular monitoring of testosterone levels, hematocrit, prostate health, and cardiovascular parameters is crucial to ensuring safe treatment outcomes.

CONCLUSION

Male hypogonadism in obese and aging men is a significant yet often underdiagnosed condition with wide-ranging metabolic, psychological, and reproductive implications. A systematic diagnostic approach, including clinical evaluation and biochemical testing, is crucial for accurate diagnosis and management. Addressing underlying risk factors, optimizing lifestyle interventions, and judicious use of TRT can help improve patient outcomes. Increased awareness and early intervention are key to mitigating the long-term consequences of testosterone deficiency in these populations.