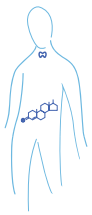
71



Men's Health Topics

David James Osborn, Douglas F. Milam, and Joseph A. Smith, Jr.

INTRODUCTION

This chapter addresses disorders that are unique to men because they involve the male genitalia and reproductive system. It incorporates commonly encountered and clinically important aspects of voiding, oncology, reproductive function, and endocrinology.

A. Androgen Deficiency in Adult Men

DEFINITION AND EPIDEMIOLOGY

Several clinical guidelines address the syndrome of symptomatic low testosterone levels in men. The two most referenced are from the Endocrine Society and from a collaboration of five societies, including the International Society for the Study of the Aging Male (ISSAM), International Society of Andrology (ISA), European Association of Urology (EAU), European Academy of Andrology (EAA), and American Society of Andrology (ASA). One guideline uses the term "androgen deficiency in adult men" and the other uses the term "late-onset hypogonadism" to refer to a disease process characterized by low serum testosterone levels and clinical symptoms.

The syndrome of androgen deficiency in adult men (late-onset hypogonadism) can be defined as a low level of serum testosterone (total or free) combined with three symptoms of low testosterone such as erectile dysfunction (ED), decreased libido, and lethargy or sleep disturbance. Using this general definition, the incidence of androgen deficiency ranges from 2.1% to 6% in the literature. On the other hand, the incidence of low serum testosterone (so-called low T) in men older than 40 years of age is much higher, between 17% and 38.7% in multiple studies. The clinical guidelines require only one symptom for the diagnosis of androgen deficiency, so the true incidence of androgen deficiency is probably closer to that of low T.

Over the last 10 years, there has been increased coverage of low T in the mainstream press and media. In addition, clinics specializing in testosterone replacement have proliferated. As a result, androgen use among men has increased threefold, from 0.8% in 2001 to 2.91% in 2011. This increase in the prescribing of testosterone is probably the result of both a heightened awareness about the problem and overutilization in men who have been improperly diagnosed. The diagnosis and treatment of androgen deficiency is challenging and full of potential pitfalls. Therefore, it is imperative for physicians to have a thorough understanding of this disease process, particularly in these times when information in the media and on the Internet creates an eager and motivated patient population.

PATHOPHYSIOLOGY

Normal aging sometimes results in malfunctions of the hypothalamic-pituitary-gonadal axis that lead to decreased serum testosterone levels. One of these errors is termed *primary hypogonadism* (testicular failure). In this disease process, the testicles produce an inadequate amount of testosterone (and sperm) even though there is adequate stimulation from the anterior pituitary gland in the form of elevated or normal release of luteinizing hormone (LH). Low serum testosterone also can result from failure of the pituitary gland to secrete an adequate amount of LH. When this occurs, the Leydig cells of the testicle that produce testosterone are not adequately stimulated and hence do not make a normal amount of testosterone. This form of testosterone deficiency is termed *secondary hypogonadism*. It can result from disease processes such as pituitary tumors, hemochromatosis, and obstructive sleep apnea.

Measurements of total testosterone (TT) include the concentrations of both unbound (free) and bound testosterone in the serum. Only 2% of testosterone is unbound; the other 98% is bound to proteins such as albumin or sex hormone binding globulin (SHBG). Because it is not bound to another substance, free testosterone (FT) is the more biochemically active form of testosterone. If a man has a low normal TT level but a low FT level and has clinical manifestations of this hormone deficiency, he is considered to have androgen deficiency syndrome.

Increased levels of SHBG in the serum can decrease the level of FT. The level of SHBG increases with smoking, coffee consumption, age, and disease processes such as hepatitis and hyperthyroidism. Therefore, increased levels of SHBG can contribute to androgen deficiency. Obesity decreases levels of SHBG, but it can cause androgen deficiency through the peripheral conversion of testosterone into estrogen in adipose cells. Conditions such as extreme exercise, recreational drug use, nutritional deficiency, stress, use of certain medications, and acute illness can transiently lower serum testosterone.