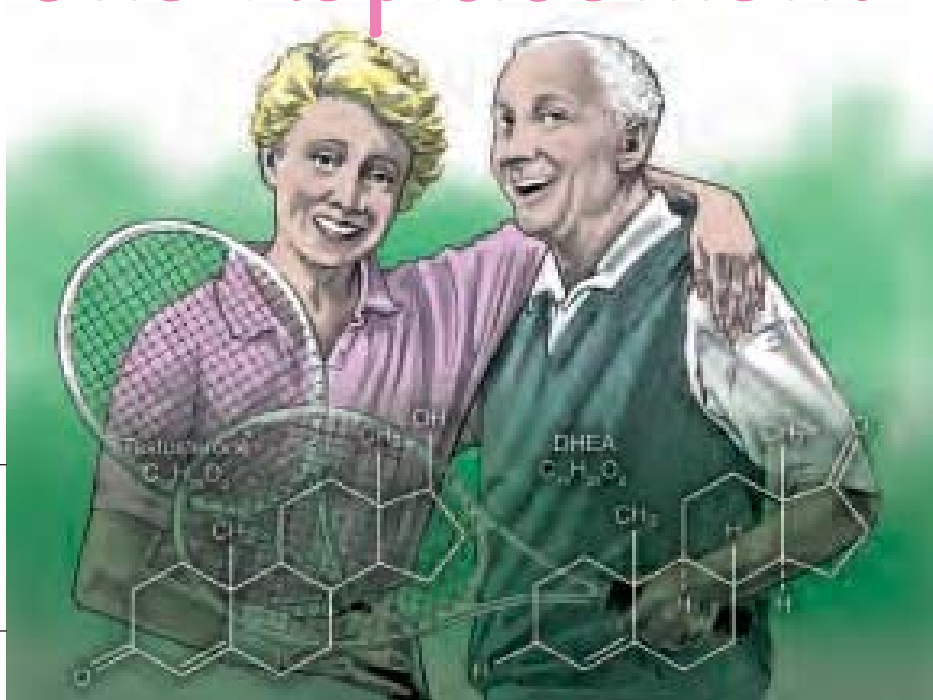


Testosterone Replacement Therapy

for Female Androgen Insufficiency Syndrome

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The two case reports highlighted in this article illustrate that, although its diagnosis can sometimes be challenging, the female androgen insufficiency (FAI) syndrome does indeed exist and can be easily treated; moreover, therapy with androgens can significantly improve a patient's quality of life.

Case Reports

Case 1

A 32-year-old woman presented with vague symptoms, among which was the inability to put on muscle. Despite being on a very rigorous diet and exercise program, which involved weights and aerobics, she had paradoxically put on weight (not muscle). Several other practitioners thought she was just vain and initially dismissed this patient. The possibility that the patient suffered from a psychiatric disorder was even considered. Her medical history was significant for a diagnosis of endometriosis, for which she was prescribed leuprolide acetate (Lupron). The medication was stopped about 6 months before my initial evaluation. She had also taken an oral contraceptive previously. Laboratory evaluation revealed a blood total testosterone level of 21 ng/dL (reference range, 20–76 ng/dL).

Coincidentally, her insulin growth factor 1 (IGF-1) level was 111 ng/dL (reference range, 114–492 ng/dL). Thyroid, hematocrit, and dehydroepiandrosterone sulfate (DHEAS) levels were normal. Symptoms reversed in response to treatment with a compounded preparation of 10 mg of testosterone in a vanishing cream base.

Case 2

A 67-year-old woman, who suffered from menopause-related symptoms, inquired about hormone replacement therapy. She had previously taken a combination of estrogen and progestin (Prempro), but discontinued that medication about a year before her inquiry because of adverse reports from the Women's Health Initiative studies. Among her symptoms were vaginal dryness, hot flashes, and decreased libido. Her medical history included hypertension, which was controlled with hydrochlorothiazide. Laboratory evaluation revealed a total testosterone level of 5 ng/dL. The patient's DHEAS and estradiol levels were subnormal as well. A topical estrogen cream, which resolved the vaginal dryness, was initially prescribed through a compounding pharmacy. An oral regimen including a combination of estrogen and testosterone (Estratest) and dehydroepiandrosterone (DHEA) was subsequently prescribed, as the patient preferred this route of administration. She reported that, after starting this therapy, her hot flashes ceased and that she had more energy and an improved quality of life.

Physiology of Androgens in Women

Before discussing androgen insufficiency in women, it is prudent to review basic androgen physiology in women. In women, androgens are essentially prohormones for other steroids, including estrogens. The building block for androgens is cholesterol, which is obtained from in the diet. Androgens influence the development of both men and women. Androgens influence sexual desire, bone density, muscle strength and mass, mood, energy, and psychological well-being. In women, androgens are produced in the ovaries, as well as in the adrenal glands; approximately 40% of androgen biosynthesis takes place in the ovaries, 25% in the adrenal glands, and the remainder in peripheral tissues.¹ Androgens in women include androstenedione, DHEA, DHEAS, 5- α -dihydrotestosterone (DHT), and testosterone. Of the androgenic steroids, DHT and testosterone have the most biological activity. Circulating testosterone is converted to DHT and estradiol (E2). On the other hand, DHEAS is produced mainly in the adrenals and is converted to DHEA by steroid sulfatase. DHEA is produced by both the adrenals and the ovaries. Luteinizing hormone stimulates androgen secretion by the ovaries, whereas androgen secretion by the adrenals is controlled by adrenocorticotrophin.

Androgens work on multiple organ systems and receptors in women. The hypothalamus and limbic system, and thus memory and mood, can be influenced by androgens.² Furthermore, androgens can influence sweat glands (thus causing acne) and can affect other systems of the body, including bones, muscles, genitals, and the cardiovascular system. Some of the effects of androgens are direct, whereas others are the result of conversion to E2 or DHT.

Female Androgen Insufficiency Syndrome

The popular press, including Dr. Jennifer Berman and her sister, Dr. Laura Berman, creators of the Female Sexual Medicine Center at the University of California, Los Angeles, and the Berman Center in Chicago, Illinois, has in recent years brought attention to the fact that androgens can affect a woman's sexuality. Pending proper peer-reviewed evaluations in journals, however, mainstream medicine has been sitting on the fence regarding the existence of a clinical condition characterized by androgen insufficiency in women. A new definition of androgen insufficiency, along with consensus-based guidelines for clinical assessment and diagnosis known as the Princeton consensus statement, were developed after an evaluation of peer-reviewed literature and consensus statements of international experts.³ The consensus statement defines the FAI syndrome to be present in a woman who has the following symptoms:

- Blunted motivation
- Bone density changes
- Cognitive and memory changes
- Decreased libido, sexual receptivity, and pleasure
- Diminished psychological well-being
- Dysphoric changes
- Fat redistribution
- Low energy and persistent, unexplained fatigue
- Muscle mass and strength changes
- Sexual hair changes

In a sense, this is very similar to the androgen deficiency in aging males (ADAM) syndrome,⁴ except that the levels of testosterone considered low, which define the diagnosis, are different in women than in men. In a woman, a testosterone level less than 20 ng/dL is considered low (less than 10 ng/dL after oophorectomy). The low testosterone level in this syndrome exists in the presence of a normal estrogen level. A caveat is that women taking oral estrogen replacement tend to have higher levels of testosterone than other women. As such, it is preferable to measure the free or bioavailable testosterone level in women taking estrogen.⁵ In general, women produce about 10% of the testosterone that men produce. Although DHEA produced by the adrenals is one of the main sources of androgens in women, DHEA is not stable in the blood, and measuring DHEAS is much easier. A challenging area in medicine is the definition of normality. Typically, any value within two standard deviations of average is defined as normal. Unfortunately, receptor affinity cannot be measured, and the concept of relative hypogonadism has been forwarded by the author.⁶

Diagnostic Dilemmas

As in ADAM, there are significant challenges in making a diagnosis of FAI syndrome; symptoms can be very nonspecific, as exemplified by the first case reported in this article.⁷ Many clinical conditions can overlap or present with such symptomatology, and among the common conditions, the following must be excluded:

- Chronic fatigue syndrome
- Clinical depression
- Hypothyroidism or hyperthyroidism
- Immunologic diseases (eg, rheumatoid arthritis, systemic lupus erythematosus, HIV-AIDS)
- Major life stress or relationship problems
- Major metabolic or nutritional disorders (eg, anemia)
- Medications that suppress testosterone level, including antiandrogens (eg, leuprolide acetate), corticosteroids, oral contraceptives, or oral estrogen replacement therapies.

When considering the etiology of a low testosterone level, the Princeton consensus statement has suggested the following five categories:⁵

1. **Adrenal:** As mentioned previously, a large proportion of androgens in women come from the adrenals, and an adrenalectomy or adrenal insufficiency can result in a low androgen level. Adrenal insufficiency can result from Addison's disease or a related immunologic disorder.
2. **Drugs:** Androgen insufficiency can be secondary to certain drugs (ie, antiandrogens, corticosteroids, oral contraceptives, or oral estrogen replacement therapies).
3. **Hypothalamic-pituitary:** Any insult to the hypothalamic-pituitary axis can result in a hypoandrogenic state. Insults could include a tumor or vascular lesion, and these have to be excluded.
4. **Idiopathic:** No cause is identifiable.
5. **Ovarian:** Chemotherapy or radiation therapy can destroy a patient's ovarian cells. Alternatively, a surgical operation such as an oophorectomy can remove the source of androgens.

Testing for Androgen Insufficiency

There is a need for accurate and reliable biological measurements of androgens in women. Laboratories can differ in their accuracy, and it is important to identify good laboratories, especially if therapeutic decisions have to be made.

In general, the Princeton consensus statement suggests that blood total testosterone level is a sufficient measure.³ There is also agreement that the dialysis equilibrium method of measuring free testosterone is most accurate and the reference standard, but this test is not readily available. If dialysis equilibrium is not available, free-calculated testosterone as measured by radioimmunoassay is an alternative. This is sometimes referred to as the free testosterone index, which takes into account the level of sex hormone-binding globulin (SHBG). Measurement of SHBG can be important, as an abnormal level can influence testosterone level. Oral contraceptives can alter SHBG level, which in turn influences testosterone level. Testosterone level should be taken in the morning and in the middle third of the menstrual cycle in premenopausal women.

Measurement of DHEAS level in the blood is the most useful method of determining adrenal androgen levels in women. The current methods for measurement of DHEAS are robust and reliable, and they are not influenced by diurnal variation. Low DHEAS

level is distinct from the low testosterone syndrome in women, and they can coexist. Treatment strategies should be targeted to maintaining normal levels in the presence of symptoms.

Saliva testosterone analysis is very contemporary because of its ease of use. (It spares the patient a needle stick.) The use of saliva testing for measurement of adrenal androgens was addressed during the Princeton consensus meeting, and the panel of experts concluded that salivary testosterone measurement was not sufficiently accurate or reliable to be recommended for clinical use, and that further research should be conducted before it can be recommended.³ Personal experience with salivary testosterone testing has been that its utility may be limited to screening selected patients who have never received exogenous testosterone, but there is no role for saliva testing in monitoring a patient on testosterone therapy.

Androgen Replacement Therapies

Compounding allows many novel methods of delivery of androgens to women who are symptomatic and have low levels of androgens. It must be stated at the onset that the US Food and Drug Administration (FDA) has not yet approved androgen replacement for the treatment of FAI syndrome, and the practice at this time is considered off-label. As such, replacement has to be done on a

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case-by-case basis with informed consent, bearing in mind that no long-term safety data are available at this time. Suggested routes of androgen replacement include implants, oral dosage forms, and transdermal dosage forms.

Implants

Implants are available but not very popular in the United States. Davis et al evaluated the efficacy of the 50-mg implant in a clinical trial. They primarily investigated the role of androgens in increasing bone density and improving libido in postmenopausal women. Postmenopausal volunteers were randomly selected for treatment with an implant of either estradiol 50 mg or a combination of estradiol 50 mg plus testosterone 50 mg, administered every 3 months for 2 years. Cyclical oral progestins were prescribed for the participants who had an intact uterus. As determined by dual energy X-ray absorptiometry (DEXA), bone mineral densities (BMD) of the entire body, the lumbar vertebrae (L1-L4), and the hip area increased significantly in both treatment groups. Bone mineral densities increased more rapidly in the testosterone-treated group than in the estrogen-only group at all sites: total body ($P < 0.008$), vertebrae L1-L4 ($P < 0.001$), and trochanter ($P < 0.005$). All sexual parameters, determined by the Sabbatsberg Sexual Self-Rating Scale, improved significantly in both groups. Addition of testosterone resulted in significantly greater improvements in sexual activity ($P < 0.03$), satisfaction ($P < 0.03$), pleasure ($P < 0.01$), orgasm ($P < 0.035$), and relevancy ($P < 0.05$), verifying the therapeutic value of testosterone implants for diminished libido in postmenopausal women. Total cholesterol and low-density lipoprotein cholesterol declined in both groups, as did total body fat. Total body fat-free mass, determined by DEXA, anthropometry, or impedance, increased in the estradiol plus testosterone group only. These findings show that implants are effective for hormone replacement, and that combined estradiol and testosterone in this form was more effective than estradiol alone in increasing BMD in the hip and lumbar spine and improving quality of sexual life. The favorable effects of estrogens on blood lipids were preserved in women treated with testosterone, and were associated with beneficial changes in body composition.⁸

Oral Therapy

DHEA level in postmenopausal women is frequently decreased, which may influence libido. The oral or transdermal delivery of DHEA requires more study, as past trials have been small in size.⁹ A small study by Hackbert et al investigated DHEA effects on 16 sexually functional postmenopausal women who participated in a randomized, double-blind, crossover protocol in which DHEA (300 mg) or placebo was administered 60 minutes before presentation of an erotic video segment. Blood DHEAS changes, subjective and physiological sexual responses, and affective responses were measured in response to neutral and erotic video segments. The concentration of DHEAS increased two- to five-fold following DHEA administration in all 16 women. Subjective ratings across DHEA and placebo conditions showed significantly greater mental ($P < 0.016$) and physical ($P < 0.036$) sexual arousal by the erotic video after treatment with DHEA than after treatment with placebo.

Positive affect also increased during the erotic video across drug conditions. Vaginal pulse amplitude and vaginal blood volume increased significantly ($P < 0.001$) between neutral and erotic film segments within both conditions (DHEA and placebo) but did not differentiate drug conditions. This small study showed that mental and physical sexual arousal ratings increased significantly in response to an acute dose of DHEA in postmenopausal women. The long-term application of this treatment is unclear.¹⁰

Methyltestosterone, given orally, has androgenic effects like those of testosterone and can reduce SHBG, which in theory can result in more free testosterone. Lobo et al recently evaluated methyltestosterone's effects on women who reported a decline in sexual interest after menopause. Hormonal effects of the combination of oral esterified estrogens and methyltestosterone were characterized. In a double-blind randomized trial, the authors investigated whether the regimen improved hypoactive sexual desire. Patients received treatment for 4 months with either 0.625 mg of esterified estrogens or a combination of 0.625 mg of esterified estrogens and 1.25 mg of methyltestosterone. Baseline and end-of-study measurements of total and bioavailable testosterone and SHBG were collected, and changes in level of sexual interest or desire were rated on a sexual interest questionnaire. Treatment with the combination of esterified estrogens and methyltestosterone significantly increased the concentration of bioavailable testosterone and suppressed SHBG. Scores that measured sexual interest or desire and frequency of desire increased from baseline with combination treatment and were significantly greater than those achieved with esterified estrogens alone. Treatment with the combination was well tolerated. Increased circulating levels of unbound testosterone and suppression of SHBG provided a plausible hormonal explanation for the significantly improved sexual function in women who received the combination of esterified estrogen and methyltestosterone.^{11,12}

Transdermal Treatment

Transdermal testosterone therapy offers the most convenient delivery system for women. Transdermal application generally is preferred over injection because it is less painful, more physiological, and requires no nurse or doctor visit.

Shifren et al investigated the transdermal delivery system in premenopausal women who had oophorectomies that resulted in androgen loss. As already stated, the ovaries provide about 40% of the circulating testosterone in premenopausal women, and many women reported impaired sexual functioning after bilateral oophorectomy despite estrogen replacement. Premenopausal women who had undergone oophorectomy and hysterectomy received conjugated equine estrogens (at least 0.625 mg per day orally) and, in random order, placebo, testosterone 150 µg, or testosterone 300 µg per day transdermally for 12 weeks each. The outcome measures included scores on the Brief Index of Sexual Functioning for Women, the Psychological General Well-Being Index, and a sexual-function diary completed over the telephone. The mean (\pm standard deviation) serum-free testosterone concentration increased from 1.2 ± 0.8 pg/mL (4.2 ± 2.8 pmol/L) during

placebo treatment to 3.9 ± 2.4 pg/mL (13.5 ± 8.3 pmol/L) during treatment with 150 µg per day, and to 5.9 ± 4.8 pg/mL (20.5 ± 16.6 pmol/L) during treatment with 300 µg of testosterone per day; the normal range is 1.3 to 6.8 pg/mL (4.5 to 23.6 pmol/L). Despite an appreciable placebo response, the higher testosterone dose resulted in further increased scores for frequency of sexual activity and pleasure-orgasm on the Brief Index of Sexual Functioning for Women ($P = 0.03$ for both comparisons with placebo). At the higher dose of testosterone, the percentage of women who had sexual fantasies, masturbated, or engaged in sexual intercourse at least once a week increased two to three times from baseline. The positive-well-being, depressed-mood, and composite scores of the Psychological General Well-Being Index also improved at the higher dose ($P = 0.04$, $P = 0.03$, and $P = 0.04$, respectively, for comparison with placebo). Scores on the telephone-based diary did not increase significantly. In women who had undergone oophorectomy and hysterectomy, transdermal testosterone therapy improved sexual function and psychological well-being.¹³

Androgen Therapy in Treatment of Hot Flashes

The physiological basis for excess sweating is increased norepinephrine in the hypothalamus. Menopausal hot flashes in women with androgen insufficiency symptoms may be triggered by small elevations in body temperature acting within a reduced thermoneutral zone.¹⁴ Androgens are estrogen pro-hormones and, as such, may influence hot flashes through the estrogenic pathways and affect serotonin level. Androgens such as methyltestosterone work on both estrogen receptors and androgen receptors. Methyltestosterone also lowers SHBG and thus increases the level of testosterone, which crosses the blood-brain barrier. Testosterone increases serotonin, which decreases norepinephrine and thus sweating. Preparations that include both an estrogen and an androgen may help with decreasing the hot flashes and sweating that occur in the perimenopausal period.

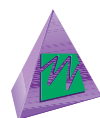
Hot flashes can be a prominent problem in women with a history of breast cancer. Given concerns regarding the use of hormonal therapies in patients with hot flashes and a history of breast cancer, other nonhormonal means for treating hot flashes are required. On the basis of anecdotal information regarding the efficacy of fluoxetine and other newer antidepressants in treating hot flashes, Loprinzi et al evaluated selective serotonin reuptake inhibitors (SSRIs) such as fluoxetine, and they concluded that SSRIs reduced hot flashes to some degree.¹⁵

Conclusion

In the short term, androgens can positively influence the quality of life in postmenopausal women. There have been reports of improved libido and mood, and reduced hot flashes. The impact of androgens in the long term seems positive as well. Observational studies have correlated testosterone to greater BMD in women,¹⁶ while SHBG sex hormone-binding globulin level has been inversely correlated with BMD.¹⁷ An interventional study demonstrated that, in postmenopausal women, testosterone replacement improved BMD.^{18,19} There is even a suggestion that DHEA may influence BMD.²⁰

Female sexual dysfunction or low libido tends to be very common; surveys have suggested prevalence as high as 42%.²¹ The role of androgens in treating female sexual dysfunction is less clear, but it is promising. Interventional studies with methyltestosterone have been conducted.¹¹ Female sexual dysfunction tends to be multifactorial, and the presence of risk factors for the condition (ie, chronic disease, partners, psychological states) influence and confound the diagnosis. Female sexual dysfunction should be considered a part of the larger entity of FAI syndrome.

The first case reported in this article illustrates that the presentation of FAI syndrome can be subtle, and often these patients are classified as depressed or vain individuals. Menopause can be a part of natural aging or iatrogenic in nature. Surgery (eg, oophorectomy), chemotherapy, or even antiandrogen therapy may cause a well woman to suffer the effects of testosterone insufficiency. On the other hand, the second case demonstrates that androgen replacement can mean tremendous improvement in the quality of life for an older woman; hot flashes were reduced, libido improved, and osteoporosis possibly prevented. Hormonal replacement in women should not be solely estrogens but should include androgens in some cases. While the FDA has yet to approve the use of androgens for the FAI syndrome, these hormone have great potential for helping our patients.



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