Erectile Dysfunction: The Hard Truth

Erectile dysfunction (ED), or impotence, is a condition defined by difficulty getting or keeping an erection firm enough for sexual intercourse. Intermittent difficulty with erectile function does not necessarily warrant thorough evaluation and treatment. However, prolonged issues with ED can indicate the presence of more serious medical conditions, such as atherosclerotic heart disease, poorly controlled diabetes, or hypogonadism, and should be evaluated.

Etiology:
The etiology of ED is usually classified as organic vs. psychogenic.

Organic causes can include:
- Heart disease or Atherosclerosis
- Diabetes
- Metabolic Syndrome
- Parkinson’s Disease
- Multiple Sclerosis
- Hypogonadism
- Tobacco, alcohol, drug abuse
- Medication side effects (antidepressants, narcotics, anti-androgen medications used to treat prostate cancer, etc.)
- Post-prostatectomy and in some cases, following surgery for treatment of benign prostatic hyperplasia (TURP)
- Surgeries or injuries affecting the pelvis or spinal cord

Psychogenic causes could include performance anxiety, history of abuse, or generalized anxiety and depression. Some patients may present with a combination of any of these causes.

Diagnosis:
The most important components of diagnosis of erectile dysfunction include detailed medical, surgical, psychological, and sexual history and a thorough physical examination. This should include defining the problem and clearly distinguishing ED from complaints regarding ejaculation and/or orgasm. The patient and partners expectations should be established during this phase of the evaluation process, as well. Also important in the evaluation process is checking basic labs, including serum testosterone, PSA, and a blood count. These labs can contribute to identifying the source of the patients ED, and can also act as a pre-treatment baseline in patients who are found to be hypogonadic.
Treatment:

The management of erectile dysfunction begins with identification and treatment of any treatable organic or psychogenic sources. In some cases, treating these comorbidities may be enough to correct the erectile function. If the patient is still experiencing impotence, therapeutic options include, oral phosphodiesterase inhibitors (PDE-5), Alprostadil intra-urethral suppositories, intracavernosal injection therapy with Alprostadil, Papaverine, Phentolamine, or a combination of these medications, vacuum erectile devices, and the most invasive option of penile prosthesis. The main contraindication regarding these treatment options is nitrate use with PDE-5 medications, as this combination of medications can cause severe hypotension. Any of the pharmacological treatment options also carry the risk of priapism, and patients must be educated on this possibility prior to use.

Hypogonadism

Male hypogonadism is the condition in which the body does not produce sufficient amounts of testosterone. This process can occur during fetal development or later in life. Congenital hypogonadism involves poor testosterone production during development resulting in impaired growth of the external genitalia. This could present with a genetically male child presenting with female genitalia, ambiguous genital development, or underdeveloped male genitalia. Childhood hypogonadism can result in delayed puberty or abnormal development, including decreased muscle mass, impaired growth of body hair, disproportionate growth of the limbs in relation to the trunk, and even gynecomastia. Adult males suffering from hypogonadism may present with symptoms of fatigue, erectile dysfunction, decreased muscle/bone mass, low libido, depression, or infertility.

Diagnosis:

Male hypogonadism is characterized as either primary or secondary. Primary hypogonadism results from a problem in the testicles. Common causes of primary hypogonadism include, Klinefelter syndrome, uncorrected undescended testicles, Mumps Orchitis, injury to the testicles, or chemotherapy or radiation treatment in the area.

Secondary hypogonadism is caused when there is an issue with hormone production at the level of the hypothalamus or pituitary gland, which works to stimulate the testicles to produce testosterone. The hypothalamus produces gonadotropin-releasing hormone (GNRH), which triggers the pituitary gland to make follicle stimulating hormone (FSH) and luteinizing hormone (LH). LH then triggers the testes to produce testosterone. Causes of secondary hypogonadism can include pituitary disorders, HIV/AIDS, inflammatory disorders that affect pituitary function, such as Tuberculosis, medications (opiates, in particular), obesity, and normal aging.
Initial diagnosis of hypogonadism includes a thorough history and physical exam, as well as presence of low serum testosterone level on one or more blood test. Testosterone levels vary throughout the day and are generally highest first thing in the morning, so blood testing is recommended early in the day. Once it has been determined that a patient is hypogonadic, further evaluation with serum hormone testing, pituitary imaging, and semen analysis may be carried out by either the urologist or endocrinologist to determine whether the hypogonadism is primary or secondary in nature.

**Treatment:**

Treatment of male hypogonadism is dependent on whether it is of primary or secondary origin. Hypogonadism caused by testicular failure is generally treated by hormone replacement in the form of topical gels, depot testosterone, medicated patches, or even implantable pellets. This type of treatment can improve sexual function, energy level, libido, muscle strength, and can help prevent bone loss. Risks of testosterone replacement therapy include accidental skin to skin transfer when using the topical gels, sleep apnea, limiting sperm production, stimulating growth of existing prostate cancer, Polycythemia Vera, and blood clots. Labs should be carefully monitored in anyone undergoing hormone replacement therapy to detect the presence of any of these conditions early. Abnormal rise in PSA (prostate specific antigen) should trigger immediate referral to urology for further evaluation of possible undiagnosed prostate cancer. Complete blood count should also be checked routinely to evaluate for rise in hemoglobin or hematocrit, potentially indicating early Polycythemia Vera. Testosterone level should be checked at least every three months until dosing has been adjusted to keep level in the therapeutic range. Once this is obtained, testosterone can be checked every six months. Testosterone repletion is not appropriate for all men. Further investigation is ongoing concerning the relative safety of testosterone, but there is a clear benefit for some men.

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