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Review Article

Male sexual dysfunctions: A clinical review

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Abstract

Sexuality and sexual dysfunction have been described in various world literature since ancient times. With time advances, science and socio-cultural changes have evolved our understanding of sexuality and sexual disorders. Male sexual dysfunction is more commonly reported than female sexual dysfunction because of shyness, socio-cultural practices, and various associated stigma with sexual disorders. Male sexual disorders are a group of heterogeneous disorders involving multiple systems. Male sexual disorders have psychogenic as well as biogenic in origin. Commonly reported male sexual disorders are erectile dysfunctions, premature ejaculations, Dhat syndrome, and delayed ejaculations. The review focuses on highlighting the burden of illness and enhancing the understanding and approach to male sexual disorders. The review also highlights the available treatment options and appropriate referral services to address the problems adequately.

Keywords:
Male sexual dysfunction, Erectile dysfunction, Premature ejaculation, Dhat syndrome.

Introduction

The evidence for male sexual dysfunction (MSD) disorder is evident in ancient literature worldwide. In almost every civilization, MSD was well documented (Forth, 2008). According to the current classification system in ICD-11,the sexual disorder is classified under the section of sexual dysfunction & in DSM-5 under the chapter of sexual dysfunctions (APA, 2013).

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The worldwide prevalence of sexual dysfunction is 43% in women and 31% in men (Rosen, 2000) Similarly, in India, the burden of SD is 14% in females and 21% in males (Rao, 2015). Medical services available in India to manage SD are limited as well as the stigma and shyness associated with MSD hinder individuals from seeking medical help for vrious MSD. Worldwide premature ejaculations (PE) are the most common MSD. In Indian literature, the prevalence of erectile dysfunction at 15.77%, male hypoactive sexual desire disorder (HSDD) at 2.56%, and premature ejaculation at 8.76% was reported (Rao, 2015). This review discusses the common prevalent MSD, representing more than 90% of the entire MSD.

The advancement in the classification of disease, a better understanding of male sexual dysfunction in the purview of current scientific knowledge,

and changes in cultural practices and social values impact the approach and management of male sexual disorders. This review emphasizes the approach to MSD and its treatment in the current scenario.

1. Erectile dysfunction (difficulty in getting/keeping an erection): Erectile dysfunctions (ED) are described as difficulty in developing or maintaining an erection suitable for satisfactory intercourse (Muneer, 2014). In ICD-10 classification, erectile dysfunctions are classified as the failure of genital response (F.52.2) (WHO, 1993). In epidemiological studies, the prevalence of erectile dysfunction is 52% in the age group of 40-70 years.

ED has a multifactorial etiology and is associated with various risk factors. Increasing age is one of the important risk factors for ED (Feldman, 1994). The psychogenic factor was considered the primary cause of ED, but in the current scenario, most ED cases have organic etiology with co-morbid psychological factors for ED (Yafi, 2016).In routine clinical practices, subjects attending the sex clinic for ED have belonged to the 30 to 40 years of age with moderate to severe ED. In most of the cases, they also have medical comorbidities (e.g., Diabetes, Hypertension), psychiatric comorbidities (e.g., Depression, Substance use disorders), and other co-morbid sexual disorders (Table 1) (Tripathi, 2021). ED also indicates endothelial dysfunction and is often preceded before cardiac events, so it can be considered an early marker to identify the risk of cardiac events (Rao, 2015).

The approach to ED's case focused on assessing the cause, severity, co-morbid conditions, and management of ED. Initial assessment of ED started with focused history taking, including onset, course, and duration of symptom onset (Miller, 2000). Psychogenic ED is presented with sudden onset, situational, presence of nocturnal penile tumescence, and good response to phosphodiesterase-5 inhibitor. Most organic ED started with gradual onset and progressive, better erection in standing than lying down positions (Yafi et al., 2016). Evaluation and assessment of other co-morbid sexual disorders, mental health evaluation, and assessment of lower urinary tract symptoms (LUTS) are integral part of history-taking (Rosen, 2003). International Index of Erectile Function (IIEF-5) questionnaire is commonly used. IIEF-5 is a five-point Likert scale ranging from 5 to 25. IIEF score of 1-7 indicating severe, 8-11 moderate, 12-16 mild-moderate, 17-21 mild, and 22-25 no erectile dysfunction (Rosen, 1999). Evaluation of vascular risk factors for cardiac events and arteriovenous malformation in ED is also important. Laboratory tests include blood sugar, lipid profile, thyroid profile, serum testosterone, prolactin, and penile color Doppler. Nocturnal Penile Tumescence Rigidity (NPTR) Tests, penile electromyography, cavernography, and assessment of structural abnormality with USG are additional investigations in the assessments of ED(WHO, 1993). Detailed assessment of ED revealed the type, severity, and cause of ED with associated comorbidity (Awasthi, 2017).

The oral PDE-5 inhibitor, testosterone supplement, and intra-urethral and intracavernosalinjections of prostaglandin E (alprostadil) are commonly available treatment options for ED. External vacuum device, penile prosthesis, penile revascularization surgery, and venous ligation surgery are also used as a treatment option for ED (Sooriyamoorthy, 2022).

2. Premature ejaculation (reaching orgasm too quickly): Premature ejaculation (PE) is the most common sexual dysfunction in males. Worldwide, 30% of males reported PE across all age groups (Montorsi, 2005). PE is defined as a condition of short ejaculation that occurs sooner than desired (intravaginal ejaculatory latency time (IELT) is less than 3 min), either before or shortly after penetration and one or both partners experience distress (Montague, 2004). PE is a self-reported problem and in most cases, it depends upon the subject's expectations and beliefs about the sexual acts. A person who has a reasonably good time for ejaculation still reported PE due to unrealistic expectations of ejaculatory time during sexual activities.

PE is classified as lifelong PE (LPE), acquired PE (APE), variable and subjective PE (Table 2) (Waldinger, 2006). Genetic and neurological etiology are behind most primary PE, but it can

be triggered by psychological impulses like the traumatic sexual experiences before puberty, conditioning, and rearing. Diabetes, hypertension, hyperthyroidism, substance use disorder, medications, a psychological issue like performance anxiety, depression, stress, and poor sleep are the common etiological factors behind the acquired PE (Raveendran, 2021).

PE hampered the sexual satisfaction between couples, which led to low self-esteem and confidence among males, further aggravating the PE due to anxiety. PE is a self-reported condition, but people usually take a long time to consult with a physician due to shame or low confidence and start with self-medication or herbal preparation to manage it.

The advancement of sexual medicine enhanced the understanding of PE, and now PE is considered not a purely psychological disorder but also has an underlying organic basis too. Understanding the pathophysiology of ejaculation is essential to understanding the approach and management of PE (Table 3) (Martin et al., 2017). The lumbo-sacral spinal cord and cerebral and spinal areas are interconnected to regulate ejaculations. Under the neuronal control of pelvic floor muscle rhythmic contraction, the bladder and urethra muscles function in a coordinated manner during the ejaculations and prevent retrograde ejaculation of sperm in the bladder. The central and spinal neuronal impulses integrated and coordinated by the spinal ejaculatory generator work during ejaculation. Serotonin, dopamine, and acetylcholine are important neurotransmitters during ejaculations. An increase in postsynaptic serotonergic activities increases the ejaculatory latency. Post-synoptically lower concentration of serotonin or hyposensitivity of serotonin receptor leads to PE, and medicinesthat increase the level of serotonin are useful in the treatment of PE. Disorder affected the pelvic floor muscle, e.g.,ererectile dysfunction also affects the ejaculations (Raveendran, 2021).

Assessment and management of PE case started with a detailed history to establish the diagnosis, estimation of IELT, ejaculatory control, type of PE, severity, and medical, psychological, substance/medication history (Chung, 2015). Evaluation of other co-morbid sexual disorders and genitourinary problems is also needed. Most of the time, the examination of PE subjectsare unremarkable but still detailed neurological examination, spinal cord assessment, lower abdomen, and genitourinary system examination is a crucial part of PE evaluation. Primary PE (LPE) is managed with pharmacotherapy (selective serotonin reuptake inhibitors, tricyclic antidepressant Trazodone, etc.), behavioral therapy, and psychotherapy, in secondary PE (APE) primarily focus on the management of comorbidities, precipitating, and maintaining factor for PE followed by behavioral therapy and pharmacotherapeutic approach is considered. Variable and subjective PE is not considered pathological but they are considered variants of the normal ejaculatory process and managed with psycho-education, reassurance, and behavioral therapy (Pereira-Lourenco, 2019).

SSRI is commonly used as an off-label medication for PE. TCA is also used as off-label in PE, and the most commonly used TCA is clomipramine. Dapoxetine, tramadol, and PDE-5i, the topical local anesthetic agents, are also used for PE.

Behavioral therapy approach included the stop-start technique by H. Semans and the squeeze technique by Masters and Johnson. Pre-coital masturbation, use of a condom, and local anesthetic cream over the glans are some techniques used for sensory abstractions to manage the PE.

3. Delayed or inhibited ejaculation (reaching orgasm too slowly or not at all): Delayed ejaculation (DE) is a less common form of the male sexual disorder (MSD). Population-based epidemiologic studies reported that the prevalence is 1-4% (Chen, 2016). Aging, pro-erectile medication use, and over-diagnosis of erectile dysfunctionlead to an increasing prevalence of DE (Perelman, 2011). DE is classified in DSM-5 in sexual dysfunction and defined as a marked delay in ejaculation and marked infrequency or absence of ejaculation on almost all the occasions for 6 months during partnered sexual activity and without the

individual desiring delay (APA, 2013). Intravaginal ejaculation latency time (IELT) between 4 to 10 min is considered a normal ejaculatory time during sexual activities, so more than 10 min IELT is the objective measurement of DE.

Etiopathology of DE is associated with psychosocial and biological factors. Ailment such as insufficient sexual stimulation (mental and physical), preferences for unusual methods of masturbation, and conflicts between sexual partners is considered psychosocial factors (Abdel-Hamid, 2018). On the other hand, biological factors included aging, neurological disorders, genitourinary tract disorders, anatomical malformations in pelvic floor muscles, hormonal disturbance, and use of psychotropic medications (Corona, 2011).

No confirmatory test is available to establish the diagnosis of DE, detailed sexual history and focused history for DE, physical examination including detailed neurological and genitourinary examination is included for assessments of DE. The investigation included in DE are lower abdomen ultrasonography, first void of urine after masturbation to assess retrograde ejaculations, assessment of vas deference patency, lower urinary tract patency, PSA, urine routine, and microscopic examination, hormonal test, and investigation focused on comorbidities (Abdel-Hamid, 2018). Currently, no drug is approved for DE, but bupropion and cabergoline are commonly used off label drugs for DE (Abdel-Hamid, 2016).

4. Low libido (reduced interest in sex): Decreased interest in sexual activities isclassified in ICD-10 under the lack or loss of sexual interest disorder (F.52.0) and in DSM-5 classified as Male Hypoactive Sexual Desire Disorder (MHSDD) (APA, 2013). Low sexual desire and distress is the key feature of MHSDD. In India, limited epidemiological studies are on MHSDD, but the prevalence of MHSDD worldwide is estimated to be 1 5% (Rosen, 2000). Sexual desire consists of sexual drive, sexual motivation, and sexual wishes. Sexual drive is regulated by the hypothalamus, the preoptic area of the anteromedial hypothalamus (Montgomery, 2008). Drive is also influenced by hormones, especially testosterone, and neurotransmitters, mainly dopamine. Other neurochemicals, prolactin, melanocortin, and serotonin, are also linked with sexual desire. Pathogenesis of MHSDD is multifactorial; androgen deficiency, medications (antihypertensive, psychotropic, anti androgens, etc.), chronic systemic disease (diabetes, cancers, hypothyroidism, Addison's disease, HIV, coronary artery disease, strokes, etc.), substance use disorders, other sexual disorders, depression, anxiety, conflicts between sexual partners, a relationship issue, adjustment disorders, are important factors to contribute pathogenesis of MHSDD (Abdallah, 2007).

Evaluation and management of MHSDD start with routine screening during visits to a sex clinic. Decrease sexual desire screener is a 5-item questionnaire tool widely used as a screening tool for hypoactive sexual desire disorder (Clayton, 2009). The questionnaire-based assessment findings suggestive of MHSDD are further examined/assessed with detailed sexual history and comorbidities such as diabetes and hypothyroidism. The assessment is extended with psychological examination for depression, anxiety, relationship issues, etc. Hormonal assay for testosterone, prolactin, and gonadotropin hormones and investigation for co-morbid conditions like diabetes and hypothyroidism are included in the assessment of MHSDD (Clayton, 2018).

Treatment of MHSDD primarily focuses on managing the underlying cause and associated co-morbid conditions. The pharmacotherapeutic approach includes the supplementation of testosterone hormones in the case of prolactinoma, pramipexol, and bupropion, but none of the agents are approved for MHSSD. Filbensaerin is FDA approved drug for female hypoactive sexual desire disorder and not for MHSSD. Psychotherapeutic approaches like dual sex therapy by Masters and Johnson (Master, 1996), cognitive behavior therapy, and psychoanalytical psychodynamic therapy are included in the management of MHSSD (Abdallah, 2007).

5. Dhat syndrome: The term Dhat syndrome was given by the late Prof N.N. Wig in 1960. Dhat

syndrome is a culture-bound syndrome. Classification system ICD-10 considers Dhat syndrome inanother nonpsychotic mental disorder (F48) category, and DSM-5 mentions Dhat syndrome in the appendix section (Prakash, 2019). The prevalence rate of Dhat syndrome is varied from 7-to 64 % in special sex clinics (Kendurkar, 2008; Kar, 2021). Dhat syndrome is usually presented by young unmarried or newly married males, who belong to low-middle socioeconomic status, have rural backgrounds, and have a conservative attitude toward sex.

In Dhat syndrome, individuals report loss of semen in urine, during sleep, masturbation, and suffer from various nonspecific somatic symptoms, e.g., fatigue, anxiety, headache, asthenia, and depressive symptoms. Depression (40-42%), anxiety (21-38%), somatoform / hypochondriasis (32-40%), erectile dysfunction (22-62), premature ejaculation (22-44%) are associate comorbidities with Dhat syndrome (Prakash, 2007). Ayurved and ancient literature from India mentioned that semen is a precious body fluid and that loss of semen leads to poor health (Deb, 2013).

Management of Dhat syndrome encompasses detailed history taking to confirm the diagnosis, evaluation of comorbidities associated with Dhat syndrome, assessment for venereal disease, and detailed urine analysis. Psycho-education, culture-informed cognitive behavioral therapy, relaxation therapy, low dose antianxiety, and antidepressants are commonly used (Awasthi, 2017).

Conclusion: Male sexual disorder is a group of heterogeneous disorders with multifactorial etiopathology. Although MSD has the organic and psychosocial origin of an illness that belongs to multispecialty, e.g., endocrinal, genitourinary, neurological, and psychiatry, most MSD cases in the community are primarily attended by primary care physicians. A basic understanding of presenting symptoms and approach to MSD can enhance the skill and competency of primary care physicians to evaluate, treat, and appropriately refer MSD cases for a better outcome.

Table 1: Risk factors of ED

Organic (80%)	**Psychogenic (20%)**
1. Non-endocrine i. Vasculogenic (most common) a. Arterial inflow disorders b. Venous outflow disorders (corporeal veno-occlusion) ii. Neurogenic iii. Iatrogenic 2. Endocrine i. Reduced serum testosterone levels ii. Increase serum prolactin 3. Medications 4. Lower urinary tract symptoms (LUTS), e.g., benign prostatic hyperplasia	1. Generalized type i. Generalized unresponsiveness a. Primary lack of sexual arousability b. Aging-related decline in sexual arousability ii. Generalized inhibition a. Chronic disorder of sexual intimacy 2. Situational type i. Partner related a. Lack of arousability in specific relationship b. Lack of arousability owing to sexual object c. High central inhibition owing to partner preference conflict or threat ii. Performance related a. Associated with other sexual dysfunctions b. Situational performance anxiety e.g., fear of failure like rapid ejaculation 3. Psychological distress or adjustment related a. Associated with negative mood state (e.g., depression) or major life stress (e.g., death of a partner)

Table 2: Classification of premature ejaculation (PE)

	Lifelong	**Acquired**	**Variable**	**Subjective**
Prevalence	2.3%-3%	3.9%-4.8%	8.5-11%	5.1%-7%
IELT	<1 minute	<3 minute	Short/Normal	Prolong/Normal
Onset	Early	Anytime of sexual life	Anytime of sexual life	Anytime of sexual life
Risk factor	Genetic	Genitourinary ds. Hormonal disturbance Psychological	Normal variances	Psychological stressors
Ejaculation control	No control	Diminish	Diminish	Diminish
Prognosis	Poor	Variable	Good	Good

Table 3: Structure involved in normal ejaculation process

	Organ involved	**Neuronal control**	**Event**
Emission	Epididymis Vas deferens. Seminal vesicles. Prostate gland & urethra. Bladder neck.	Pelvic plexus (sympathetic and parasympathetic innervations)	Sympathetic supply from thoracolumber lead to closure of bladder neck via alpha adrenergic to prevent retrograde ejaculation.
Expulsion (Ejaculation)	Bladder neck Urethra Pelvic floor muscles.	Spinal cord Pudendal nerve Motor neurons located in the nucleus of Onuf (ON).	Upper lumbar cord (L2/L4): emission phase Upper sacral cord (probably in S1/S2): expulsion phase
Orgasm			

Table 4: Spinal ejaculatory generator

Spinal ejaculatory generator	**Peripheral structure**	**Central structure**	**Pathway**	**Neurotransmitters**
Moderator for emission and ejaculation. (Central & peripheral structure)	Pudendal nerve Nucleus of Onuf (ON). Sacral parasympathetic motor neuron. Neurons in the area of lamina X. Lumbo-sacral region of spinal cord	Paragigantocellularis, Paraventricular nucleus of the hypothalamus Medial preoptic area. Nucleus paragigantocellularis (nPGi)	The projection from the central structure terminates in the lumbar preganglionic motor neurons, nucleus of ON, sacral parasympathetic motor neurons, and neurons in the area of lamina X, where ejaculation generators are supposed to be located. Serotonergic projections from the nucleus paragigantocellularis (nPGi) in the brain stem exert tonic inhibition of ejaculation via the motor nucleus in the lumbosacral spinal cord, influencing the spinal ejaculation generator in the spinal control center located in the lumbosacral spinal cord.	serotonin (5HT), dopamine, oxytocin, gama-aminobutyric acid (GABA), adrenaline, acetylcholine, and NO

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