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Effect of Diabetes Mellitus on Sexual Arousal and Intercourse

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Abstract

Diabetes mellitus, when producing hyperglycemia, as well neuropathic angiopathic, vasculopathic, and complications, poses a threat to the function and viability of sexual arousal and intercourse at similar and different levels in males and females. Males are faced with hypogonadism, depression and anxiety, affecting their sexual arousal desire. Male intercourse may be impaired by erectile dysfunction, priapism, ejaculatory dysfunction, and/or benign prostatic hyperplasia. Female sexual arousal may be affected by depression, hormonal imbalance, and hypoactive sexual desire disorder. Female sexual intercourse may be disturbed by dyspareunia, vaginismus, and anorgasmia. Effects on sexual intercourse may also be seen at the gender neutral level due to cranial neuropathy and various autonomic neuropathies outside the genitourinary tract. Though specific treatments target most conditions, healthy diet and exercise are the best bets to avoid the long-term effects of diabetic complications on sexuality.

Keywords: Diabetes mellitus; Hypogonadism; Erectile dysfunction; Hypoactive sexual desire disorder; Cardiovascular autonomic neuropathy; Cranial nerve dysfunction

Abbreviations: Diabetes Mellitus (DM); Type 1 Diabetes Mellitus (T1DM); Type 2 Diabetes Mellitus (T2DM); Phosphodiesterase type 5 (PDE5); Cyclic Guanosine Monophosphate (cGMP)

Introduction

Diabetes mellitus (DM) is an increasingly common condition of chronic hyperglycemia and impaired carbohydrate, lipid, and protein metabolism, with over 550 million worldwide projected to be affected by 2030 [1]. Common risk factors for the development of DM include sedentary lifestyle, obesity, and increased caloric consumption. Type 1 (T1DM; insulindependent) and Type 2 (T2DM; noninsulin-dependent) DM can be major causes of system-wide micro and macrovascular

complications that ultimately impede the ability to sexually arouse males and females, so that they may be able to adequately perform sexual intercourse.

Long-term effects of DM on the cardiovascular system are marked by accelerated atherosclerosis and hyaline arteriosclerosis (i.e. diabetic macroangiopathy) as well as specific capillary lesions (i.e. diabetic microangiopathy). Diabetic macroangiopathy increases incidence and severity of coronary artery disease, cerebrovascular accidents, peripheral ischemic arterial disease, and hypertension. Microangiopathy may develop several years after diabetes onset. Capillary lesions are associated with accretion of capillary basement membrane fragments, ultimately leading to capillary occlusion. Hypertension in diabetics may aggravate the progression of these macro- and microvascular complications [2-4]. Meanwhile, metabolically damaged nerve fibers, as well as thickening, hyalinization, and perivascular denervation of the vasa nervorum may be critical factors behind somatic, autonomic, and cranial nerve dysfunction (i.e. diabetic neuropathy) [5,6].

This review utilizes a MEDLINE® database search to compile information on current pathogeneses and treatment options of sexual arousal and intercourse complications of DM. Most conditions below do not reflect a particular type of DM unless otherwise noted.

Male Sexual Arousal Complications of Diabetes Mellitus

Hypogonadism

Hypogonadism, or low levels of testosterone, may bring out symptoms such as decreased libido and energy. Depression and anxiety may also implicate the ability to attain sexual arousal [7]. The pathophysiological link between diabetes and hypogonadism is still poorly explained. Because obesity is often concomitant with diabetes, a possible hypothesis is that excessive estradiol secretion due to high aromatase activity in obese patients may inhibit gonadotropic releasing hormone secretion, thus decreasing testosterone secretion [8]. Another postulated cause is hypothalamic-pituitary-adrenal axis dysregulation-associated functional hypercortisolism [9].

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Testosterone supplementation is the best way to treat this complication, and it is available through intramuscular injections, transdermal creams/gels, buccal tablets, and subcutaneous depots [7].

Male Sexual Intercourse Complications of Diabetes Mellitus

Erectile dysfunction

Erectile dysfunction is described as the inability to attain or sustain a normal erection despite appropriate sexual stimuli. Age and longer duration of diabetes are major risk factors [10]. It is reported to occur in more than half of diabetic men worldwide [11]. Vasculopathy, neuropathy, visceral adiposity, insulin resistance, and hypogonadism secondary to diabetes may be responsible for the pathogensis of erectile dysfunction [12]. Atherosclerosis and endothelial dysfunction may ultimately limit arterial blood flow necessary for erection to the penis [12]. Somatic neuropathy impairs sensory impulses from the penis to the reflexogenic erectile center, while autonomic neuropathy impairs parasympathetic relaxation of the corpus cavernosum smooth muscle [12]. Treatment for erectile dysfunction is primarily accomplished with oral phosphodiesterase type 5 (PDE5) inhibitors. Since the release of nitric oxide from cavernous nervous and endothelial cells is dwindled due to diabetic neuropathy and endothelial dysfunction, there is a lack of downstream upregulation of cyclic guanosine monophosphate (cGMP). cGMP relaxes penile smooth muscle for a proper erection. Because there is a shortage of cGMP, PDE5 inhibits the tendency of cGMP to break down and lose function [13]. Intracavernosal [14] or intraurethral [15] prostaglandin therapy, vacuum erection devices [16], and penile prostheses are some more involved solutions for sustaining erections. Diabetes may predispose penile implant recipients to infection [17]. Prothrombrotic factors secondary to diabetes may also be responsible for priapism, a chronic erection which may lead to painful sexual intercourse [18].

Ejaculatory dysfunction

Even though ejaculation is more of a concern for male fertility, it is also implicated in coitus as men report that the lack of ejaculation makes the act less pleasurable. Diabetic neuropathy may deteriorate the nerves controlling the external urethral sphincter. Because the sphincter remains open and urethral peristalsis is occurring to expel the ejaculate, the emission may end up in the bladder (i.e. retrograde ejaculation). Retrograde ejaculation may be treated with anticholinergics, antihistamines, and alpha-adrenergics. Premature ejaculation is the most prevalent of ejaculatory disorders. Anejaculation is the lack of ejaculation that may also present with the lack of orgasm, or anorgasmia [7].

Benign prostatic hyperplasia

Benign prostatic hyperplasia is described as the proliferative cellular growth of the glandular and stromal elements of the prostate. Diabetic vasculopathy and atherosclerosis may increase peripheral sympathetic nerve activity, causing hypoxia-related cell proliferation in the transitional zone of the prostate [19]. The resulting lower urinary tract symptoms are also concomitant with ejaculation difficulties and erectile dysfunction [20].

Female Sexual Arousal Complications of Diabetes Mellitus

Depression

Depression stemming from extensive diabetic complications may impair lifestyle, self-image, and relationship status, thus indirectly affecting sexual desire and arousal. In fact, psychosocial factors are likely the biggest reason women cannot achieve the proper sexual desire needed to enjoy intercourse [21].

Hypoactive sexual desire disorder

Hormonal imbalance regarding androgens, estrogens, and sex-hormone binding globulin [22] is a likely culprit for arousal disorder, which is defined by the lack of vasodilation and engorgement in the female external genetalia, namely the clitoris and vagina [23]. Diabetic neuropathy may be implicated in decreased innervation to the clitoris and vagina for blood flow, generating diffuse fibrosis of the clitoris and vaginal tissues, and thinning the musculature and epithelium of the vagina [12,24,25]. Endothelial dysfunction may be responsible for nitric oxide deficiency, which may be detrimental to clitoral smooth muscle [12]. Diabetic neuropathy may also be implicated in transducing sexual stimuli and the respective sexual response [12]. Atherosclerotic damage and endothelial dysfunction may also reduce vaginal lubrication and clitoral engorgement, causing decreased arousal [25].

Female Sexual Intercourse Complications of Diabetes Mellitus

Dyspareunia

Dyspareunia is described as painful sexual intercourse in women. Hyperglycemia from diabetes may reduce hydration levels of the mucous membranes of the vagina [12]. This lack of mucus lubrication can make it difficult and painful for the penis to slide effortlessly in during intercourse. The hyperglycemic state may also predispose the female to genitourinary infections- another possible cause for vaginal irritation-induced dyspareunia [12]. Atherosclerotic damage and endothelial dysfunction may also reduce vaginal lubrication and clitoral engorgement, inciting dyspareunia [25].

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Anorgasmia

Dyspareunia is accompanied with a state of discomfort that makes it very difficult for the woman to enjoy sexual intercourse, thus putting sexual climax and the associated orgasm out of reach (i.e., anorgasmia).

Gender Neutral Sexual Intercourse Complications of Diabetes Mellitus

In addition to the gender-specific diabetic manifestations affecting optimal sexual arousal and performance, several factors may implicate the beginnings and the general course of coitus. Psychosocial factors such as adjustment to the diagnosis of DM, the burden of living with a chronic disease, and depression may impair sexual function, particularly in females [26]. Significant sensory, motor, and autonomic neuropathies of different physiological systems may affect proper sexual intercourse. Though extremely rare, diabetic cranial neuropathy involving cranial nerves III, IV, and VI may induce ophthalmoparesis, potentially implicating initial stages of sexual activity; retinopathy, optic neuropathy, and retinal vascular conditions may pose comparable threats [27].

DM may also present with cardiovascular autonomic neuropathy (CAN), giving rise to abnormalities in heart rate control and vascular dynamics [28]. This may elicit cardiac stress in strenuous physical exercise in this instance, sexual intercourse. CAN contributes to exercise intolerance by hindering response to change in heart rate, blood pressure, and cardiac output [29,30]. Dysfunction in parasympathetic and sympathetic responses results in exercise tolerance, which would otherwise improve cardiac output and increase perfusion to skeletal muscles [30]. Reduced ejection fraction, systolic dysfunction, decreased rate of diastolic filling, and blunted response to catecholamines may also limit exercise tolerance [30,31]. In cardiovascular disease-free diabetics exhibiting asymptomatic vagal CAN, exercise capacity (i.e., greatest tolerable workload and maximal oxygen uptake), heart rate, blood pressure, cardiac stroke volume, and hepatosplanchnic vascular resistance may all be diminished [30,32]. QT-interval prolongation, as well as cardio-autonomic nervous dysfunction, may contribute to the pathogenic mechanism of sudden unexpected death in diabetics [33]. The severity of CAN and heart rate are inversely proportional to one another during exercise [32].

The Health Professional's Guide to Diabetes and Exercise by the American Diabetes Association lists several more exercise risks secondary to diabetic autonomic neuropathy. Cardiorespiratory instability may pair with CAN. Defective sympathetic regulation and pseudomotor activity may lead to heat intolerance. Gastroparesis may delay delivery of nutrients to the bloodstream. Finally, there may be unawareness for hypoglycemic episodes [31]. Besides autonomic neuropathy, excess oxidative stress through the production of reactive oxidative species (e.g. superoxide anion, peroxide, and hydroxyl radicals) may also contribute to metabolic cardiac and

skeletal muscle dysfunction, jeopardizing adequate sexual performance [34,35].

Conclusions

There has been a surge in patients exhibiting sexual intercourse and arousal complications as a reflection of the worldwide diabetes mellitus epidemic. Neuropathy, macroangiopathy, and microangiopathy due to increased oxidative stress and an immunodeficient state are the main malefactors behind the secondary conditions. Male diabetics erectile demonstrate dysfunction, dysfunction, and hypogonadism. Females may exhibit hypogonadism, hypoactive sexual desire disorder, and dyspareunia. Female sexual dysfunction is still not as welldefined or pathophysiologically explained as male sexual dysfunction, although we assume there are many parallels. Cranial neuropathy and cardiovascular autonomic neuropathy may be implicated in sexual performance in either gender. Extensive study isolating the roles of gender, age, onset duration of diabetes, and diabetes as the individual risk factor (i.e. without the full aspects of metabolic syndrome) in the development of sexual complications remains to be adequately seen.

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Conflicts of interest

The authors declare no conflict of interest.

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