



Diabetic autonomic neuropathy resulting in sexual dysfunction

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Abstract: Although diabetic autonomic neuropathy (DAN), a complication associated with diabetes, is increasing in prevalence, it is often overlooked. Knowledge regarding the development and progression of this complication is limited. NPs are in a key position to prevent complications of DAN, including sexual dysfunction, in this vulnerable population.

The incidence of diabetes mellitus in the US continues to rise, with an estimated 30.3 million individuals currently affected by this chronic illness.¹ Diabetes is the seventh leading cause of death in the country, making it a national health problem.² According to the American Diabetes Association (ADA), approximately 12 million adults over the age of 65 years are currently living with diabetes.³

In addition, the CDC reports that obesity rates are increasing more rapidly among adults between ages 40 and 59 years (40.2%) and older adults age 60 years and older (37%).¹ Diabetes is associated with damage to multiple systems, primarily the renal, nervous, and cardiovascular systems, affecting morbidity

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and mortality.⁴ The longer an individual lives with undiagnosed, untreated, or poorly managed diabetes, the greater the potential for serious health outcomes.⁴

Diabetes can affect numerous body systems, including the autonomic nervous system (ANS). The ANS is responsible for controlling many systems, including gastrointestinal, cardiovascular, and genitourinary.⁵ Damage to the ANS can result in a myriad of issues, including diabetic autonomic neuropathy (DAN) and sexual dysfunction (SD).⁵ In a hallmark position statement, Vinik described DAN as being one of the least recognized and understood complications of diabetes.^{6,7}

DAN has been described as a diverse disorder associated with the ANS and is considered a diagnosis of exclusion.³ This chronic illness can be defined as a combination of injuries or lesions to nerve fibers in the ANS that control both the sympathetic and parasympathetic systems.^{3,5,6} For patients with diabetes, risk factors for developing neuropathies are higher in those who have poor blood glucose control, longer duration of diabetes, and kidney disease, as well as in those who are obese, use alcohol, and who smoke.⁸ DAN typically coincides with other complications of diabetes, such as peripheral neuropathy, cardiovascular autonomic neuropathy, gastroparesis, and kidney disease.^{7,8} These complications have been associated with higher morbidity and mortality.^{3,5-7}

The detection of diabetic neuropathy may occur as early as 1 year after the diagnosis of type 2 diabetes mellitus (T2DM) and within 2 years of the diagnosis of type 1 diabetes mellitus (T1DM).^{6,7} Typically, clinical symptoms may be subtle and unrecognizable by the provider and patient.⁶ Subclinical presentation of DAN is related to hyperglycemia resulting in minute neurovascular changes.^{6,7} This timeline reflects the gradual progression of DAN.

This article discusses the detection and treatment of diabetes before the development of the microvascular complications that can ultimately lead to SD. NPs should conduct a thorough and accurate history and physical exam to improve the health of patients with diabetes. Patients should be counseled regarding healthy lifestyle choices, including controlling glucose levels, meal planning, and exercise. The diagnosis of DAN is often overlooked until later in the progression after microvascular changes have developed and when irreversible damage affecting quality of life (QoL) has already occurred.

■ Pathophysiology of autonomic neuropathy

The ANS is crucial in maintaining homeostasis throughout the body.^{9,10} Hyperglycemia causes damage to the ANS, resulting in an oxidative stress response.¹¹ Initially, hyperglycemia causes increased levels of intracellular glucose in nerves, which leads to oversaturation of the normal glycolytic pathway.¹¹ Glucose is shunted into the autonomic pathway and then converted to a sorbitol and fructose substance, which decreases the permeability of cellular membrane (sodium-potassium-adenosine triphosphate [Na⁺/K⁺-ATPase pump]), causing abnormal action potential of the cell.^{12,13} Oxidative stress deprives the nerve cells of oxygen, impedes growth, and leads to cellular apoptosis or cell death; this contributes to the development of progressive neuropathy or DAN.^{13,14}

SD is an often overlooked complication of DAN. Sexual arousal is controlled by the central nervous system (CNS), with the parasympathetic nerves governing the excitatory response.^{15,16} Excitatory signals originate in the brain based on visual or physical stimulation, which result in the release of nitric oxide and acetylcholine.^{16,17} This release of chemicals causes the arteries to relax and fill with blood, resulting in an erection or sexual response.^{15,16} When oversaturation of glucose occurs over time, the nerve cell response eventually slows and becomes damaged, which leads to an inadequate sexual response.¹⁵⁻¹⁷ This is the beginning of SD. Over time, this condition worsens, and the ability to respond is reduced.¹⁶

■ Sexual dysfunction

The World Health Organization defines SD as “the inability of an individual to participate in a sexual relationship.”⁴ SD prevalence is higher among individuals with diabetes, and the incidence increases with age.¹⁵ Men with diabetes are three times more likely to experience erectile dysfunction (ED) than men without diabetes.¹⁵ T2DM is becoming a major health problem worldwide.¹ As the number of individuals with T2DM increases, so will the number of individuals with SD. (See *Potential causes of SD.*)

The incidence rate of SD in women with diabetes is self-reported at between 20% and 80%.¹⁸ Therefore, women experience SD at a rate slightly lower than men.¹⁸ The broad range in reporting SD in women may be due to their hesitancy in reporting this problem due to social constraints and taboos. Women are often hesitant to discuss sexual issues with their healthcare providers (HCPs). Compounding this issue is that HCPs rarely assess SD in their patients unless it affects the patient’s QoL.¹⁹

SD in women. The effects of glycemic control on sexual health are poorly understood. Overtime, fluctuations in glycemic control have a detrimental effect on body tissues in women.¹⁶ Hyperglycemia results in dehydration, which affects vaginal mucosa hydration and causes uncomfortable sexual intercourse and dyspareunia.¹⁵ Pain during intercourse can lead to SD and places a strain on intimacy between partners.¹⁶

In order for individuals to experience arousal, the sensory nervous system and ANS must be intact for a sexual response to occur.^{15,17} An intact vascular system supported by the ANS is needed for the clitoral and vaginal tissue to become engorged past arousal.^{6,7} Smooth muscle relaxation must also occur to support blood flow to the vaginal area.⁶ The presence of hyperglycemia results in damage to the vascular area and extends to the neurologic system.⁶ There are few therapeutic options to improve SD after neurologic damage has occurred.

NPs need to be aware that women with T2DM have an increased prevalence of SD. Elyasi investigated women with T2DM and found that 78.7% of the 151 women in the study had one or more self-reported negative sexual outcomes.²⁰ This study revealed not only that the prevalence of SD was higher in women with T2DM but was linked to depression and/or anxiety.²⁰ Depression has been associated with SD and T2DM in women across multiple studies.^{19,21} This correlation has not been fully explored or addressed in clinical practice. As a result, sexual complications and low sexual satisfaction in women with T2DM continues to be a concern.

In a study by Copeland and colleagues of 2,270 ethnically diverse women ages 40 to 85 with T2DM, participants were more likely to report a decrease in sexual satisfaction compared with women who did not have diabetes.¹⁹ In addition, women with T2DM who required insulin to maintain glycemic control were found to be at higher risk for SD.¹⁹ This correlates with beta cell destruction and the development of latent neurologic damage due to the progression of T2DM.¹⁹ Therefore, as the duration of T2DM increases, the ability of the pancreas to produce adequate amounts of insulin decreases.³ This decline in insulin production may occur over time, causing gradual changes in health before clinical symptoms are detected.^{6,19} NPs need to remain vigilant and routinely screen for signs or symptoms of disease progression before symptoms of SD occur.⁷

Erectile dysfunction. ED is the inability of a male to achieve or maintain an erection that lasts long enough for satisfactory sexual activity.¹⁵ Males with T2DM

Potential causes of SD^{28,29}

Vascular	Atherosclerosis, uncontrolled hypertension, peripheral vascular disease, dyslipidemia, Peyronie disease
CNS	Neuropathy, radical pelvic surgery, spinal cord injury or tumor, stroke, multiple sclerosis, Alzheimer disease and Parkinson disease, temporal lobe epilepsy
Endocrine	Hypogonadism, hypothyroidism, hyperthyroidism, pituitary tumor, hyperprolactinemia secondary to alterations in circulating androgens
Chronic illnesses	Cancer, diabetes mellitus, heart disease, liver disease, kidney disease, chronic obstructive pulmonary disease, prostate cancer and the resultant treatments
Medications	Antihypertensives, antidepressants, CNS stimulants, diuretics, estrogens, antiandrogens, and digoxin
Lifestyle	Use of tobacco products, alcohol, marijuana, opioids, cocaine, or amphetamines.
Psychological	Depression, anxiety
Other	Menopause or pregnancy, sleep disorders

experience symptoms of ED 10 to 15 years earlier in life than those without this diagnosis.¹⁵ It is estimated that 10% of men with ED may have undiagnosed diabetes, making ED a potential marker for earlier diabetes screening.¹⁵ This chief complaint may heighten the NP's need for establishing baseline screening at an earlier age.

The NP should consider advancing age and T2DM as risk factors for ED. The incidence rate of ED climbs to 95% in males age 70 years or older.²² Aging has been shown to increase the incidence of vascular issues in men. In older adults, vascular complications including both microvascular and macrovascular changes contribute to ED.⁷ Any mechanism or condition that decreases the blood supply to the penis or blocks the neurotransmitters can result in SD.²² This problem worsens over time without intervention, causing more systemic damage. As evidenced across multiple studies, the prevalence and severity of ED increases with age and becomes progressively worse over time without intervention.^{15,23}

Approximately 52.5% of males who have diabetes experience problems maintaining or sustaining an erection.²² Often, the first symptom is the inability to sustain an erection. Later in the disease progression, men can neither initiate nor sustain an erection.

Autonomic neuropathy and endothelial dysfunction induce smooth muscle relaxation, and are considered the primary causes leading to ED.²⁴

Uncontrolled hyperglycemia damages the endothelial lining, causing impaired vasodilation; this can impede the filling process.^{22,24} Erections require both filling and trapping of blood in the penile tissue.^{22,24} Males with DAN may have restrictions in the relaxation stage, impeding engorgement. Therefore, men who have DAN become unable to sustain engorgement and rigidity, which is needed for sexual performance.²⁴

Depression. Comorbid depression further complicates the relationship between DAN and SD.^{16,25} Low levels of neurotransmitters cause a decrease in sexual desire in those with depression.¹⁶ The brain is considered one of the body's most sensitive sex organs because it is responsible for initiating desire.¹⁶ Neurotransmitters, such as dopamine, serotonin, and acetylcholine, transmit signals to the sex organs to begin blood flow needed for arousal.¹⁶ However, many of the medications used to treat depression have been shown to block the neurotransmitters leading to SD.¹⁷ A study by Bajaj and colleagues demonstrated that individuals with depression experienced poor glycemic control, which in turn reduced the quality of sexual performance.²⁵

■ Diagnostics

Patients with DAN may be clinically symptomatic or asymptomatic. The NP should obtain a comprehensive history and perform a thorough physical exam including evaluating the patient's complaints of pain or burning in the lower extremities.^{7,8} In addition, referral to a neurologist can be useful in determining the degree of dysfunction and to help formulate a plan of care for the treatment of SD.⁸ The ADA recommends assessing the patient for neuropathy starting at the time of diagnosis of T2DM and 5 years after the diagnosis of T1DM and annually thereafter.^{3,7}

NPs should also consider screening patients with prediabetes who are asymptomatic for SD using a variety of tests or tools.⁷ The quantitative sensory test is a simple, noninvasive tool used to determine nerve cell response to vibrations, sensation, and temperature.^{7,8} Additional testing for autonomic dysfunction includes assessing the patient for orthostatic hypotension.⁸ Patients who are positive for orthostatic hypotension lack the ability to adjust BP levels when moving from a supine to an upright position, indicating a disturbance in autonomic system.⁸ Another indication of autonomic dysfunction

can be a notable decrease in heart rate variability (HRV).⁸ HRV refers to a variation in the time interval between heartbeats. This is detected by measuring the RR variability from one QRS complex to another in an ECG.⁸

This decrease in HRV is a signal that the ANS has affected vessel response in the cardiovascular system.⁸ Identifying changes in the ANS is an indicator of systemic dysfunction. To assess for systemic changes in men with ED, the nocturnal erection test can be performed at home to assess if symptoms of ED are physical or related to psychological or emotional issues.⁶ Men generally have three to five erections a night.⁶

To screen for psychological versus autonomic-related problems, a ring-like device can be placed on the penis at bedtime.²⁶ If the patient wakes up due to constriction of the band, he is probably experiencing ED related to psychological or emotional stress.²⁶ This is an easy-to-use tool that can be used to differentiate between psychological problems or DAN. Men with DAN may need a penile duplex ultrasound to assess blood flow to the penis.²⁷ During the ultrasound, medications may be injected into the penis to assess the ability to obtain an erection.²⁷

Diagnostic tests to rule out other causes of SD may include creatinine, microalbuminuria, lipid panel, urinalysis, thyroid panel, sex hormone-binding globulin test, parathyroid and luteinizing hormone, early morning testosterone level, prostate-specific antigen test, prolactin, and serum calcium levels.^{3,28,29}

■ Treatment of SD

Treatment of SD related to diabetes needs to be multifaceted and an ongoing process. Screening should be the first step in this process and continued at least annually.⁷ Conversations regarding sexual issues may be difficult for both the provider and the patient. However, developing a trusting relationship is an essential step in this process. In addition, critical conversations regarding sexual health will provide NPs with an opportunity to educate patients on sexual function in order to prevent complications associated with diabetes progression.²⁷⁻³⁰

According to the American College of Obstetricians and Gynecologists, between 5.4% and 13.6% of women between ages 40 and 60 years are affected by hypoactive sexual desire disorder.²⁹ This disorder consists of a persistent lack of sexual desire, and is usually associated with endocrine disorders, depression, or medications.²⁹ Current treatment guidelines include the need for annual health maintenance and preventive screenings.²⁹

Annual screenings are considered the minimum standard of care and may need to occur more frequently based on the needs of patients with DAN.³

NPs play an integral role in health maintenance and prevention. Women may present with complaints of depression due to changes in sexual function.²⁰ Women with DAN have a higher incidence of SD with depression.²⁰ There is a gap in the literature regarding whether SD precedes depression or depression contributes to SD.²⁰ NPs should also inquire about SD in patients who present with symptoms of depression.

In contrast, the most prevalent SD complaint in males with diabetes is ED, ejaculatory dysfunction (usually premature ejaculation), and decreased libido.²⁸ Symptoms can stem from a single issue or a combination of problems. Most men require a multifactorial approach to manage this issue.^{27,28}

Treatment should be aimed at not only improving libido but also at sustaining and maintaining an erection. Male patients need to be screened thoroughly prior to prescribing medications to treat ED.^{28,29,31}

Pharmacologic therapy. The first-line medications used to treat ED are the phosphodiesterase type 5 (PDE-5) inhibitors.³² The current list of FDA-approved oral PDE-5 inhibitors includes avanafil, sildenafil, tadalafil, and vardenafil hydrochloride.³³ The PDE-5 inhibitors are excellent first-line medications; however, some males have a poor response to these medications.

Patients should be warned that PDE-5 inhibitors are only effective if used in combination with sexual stimulation.³² Common adverse reactions that may be experienced include flushing, headache, rhinitis, visual disturbances, and dyspepsia.³² Because the PDE-5 inhibitors are vasodilators, they are contraindicated in all patients who are taking any form of organic nitrates for angina because of the potential of hypotension and syncope.³² Prescribers need to consult the prescribing label for each drug for specific information related to contraindications, precautions, warnings, and dosage adjustments related to kidney dysfunction. According to the American Urological Association, there is no evidence that any one PDE-5 inhibitor is more effective than the others.³²

Alprostadil injection, a prostaglandin E1 agonist, is approved for intracavernosal use by a trained provider for the treatment of ED. Alprostadil injection is contraindicated in men who have conditions that may predispose them to priapism, such as sickle cell anemia

or sickle cell trait, multiple myeloma, or leukemia, and in men who have fibrotic penile conditions, penile implants or a hypersensitivity to the drug. The most common adverse reaction is penile pain.³²

Libido decreases with age, causing erectile issues in men 60 years of age and older.³² Testosterone replacement therapy can be provided for those individuals with low levels (less than 300 ng/dL).^{31,32} Testosterone levels should be drawn early in the morning because the levels have a circadian variation, with the highest levels in the early morning and the lowest levels in late afternoon. The AUA reports that testosterone therapy

Both prescribed and over-the-counter medications can contribute to sexual issues in both genders.



has no value in men with normal testosterone levels.³² All testosterone replacement medications should be prescribed with careful consideration of both efficacy and adverse reactions.

Selective serotonin reuptake inhibitors and 5-alpha-reductase inhibitors are medications that can contribute to decreased libido.³² Identifying common medications that can cause issues with libido are essential in the care of those with SD. Both prescribed and over-the-counter medications can contribute to sexual issues in both genders.³² Patients need to be informed of the risks and benefits of any medications they are taking including antidepressants, antipsychotics, anticholinergics, antihypertensive, and diuretics that can impact sexual desire and arousal.²⁰

When prescribing medications for depression, NPs should prescribe medications that have low to no effect on sexual function. Medications that have low to no impact on sexual function include bupropion, mirtazapine, and vilazodone.³³

Newer treatments for ED. Stem cell transplant has been reported as a new emerging therapy in the treatment of ED. The transfer of stem cells into target areas is the main design behind this concept.³⁴ Stem cells play a role in increasing nitric oxide synthesis as well as in neuroregeneration.³⁴ This may be the most promising form of treatment in men who have undergone a prostatectomy due to the ability to regenerate and repair damage to the cavernous nerves in the penis.³⁴ While this work is still in progress, research in rodents has shown promising results.

Lifestyle management. The NP's approach to management and treatment of SD should focus on prevention, identification of risk factors, and early detection of modifiable symptoms and complications.⁷ In addition to modifiable interventions, all aspects of management need to stress the importance of maintaining the A1C at a level of less than 7% for most nonpregnant adults with diabetes.^{3,6,7} An individualized plan of care should be developed to assess and control T2DM complications. This plan of care needs to include a focus on weight loss, meal planning, exercise, and BP and cholesterol management.^{3,6,7} The ADA recommends nutrition therapy for patients with diabetes.³ (See *Nutrition therapy for the promotion of sexual health.*)

Exercise and sexual health. Exercise for patients with T2DM is recommended to achieve optimal glycemic control.³ NPs may be met with resistance from patients when advising integrating a cardiovascular or muscle-strengthening program. Alternative options for supporting glycemic control that can simultaneously treat SD should be provided. In one study, yoga was found to be significantly effective ($P < .00001$) in improving six domains of SD including desire, arousal, lubrication, orgasm, satisfaction, and pain in women ages 45 years and older.³⁶ Yoga is an alternative exercise that may appeal to both women and men who have previously had a sedentary lifestyle.³⁶

Health maintenance. HCPs strive to provide health maintenance to achieve goals established by Healthy People 2020.² Routine maintenance includes promotion of a healthy lifestyle. Obstacles that can impede meeting

the national goals include substance use disorder, tobacco use, alcohol, and sedentary lifestyle. Over time, substance use can cause permanent damage to the microvascular and vascular system, contributing to DAN and leading to SD. Health maintenance should include an evaluation of risk factors for SD, including smoking, obesity, drug use, hypertension, and hyperglycemia. Many long-term conditions such as SD can be prevented or improved by supporting patients in making lifestyle changes that will improve their overall health.

Conclusion

DAN and its influence on SD has resulted in a new direction for the understanding of hyperglycemic effects on sexual health. The role of the NP is to promote health and prevent complications from chronic conditions such as diabetes. SD is a complex, multifaceted problem that differs between and within individuals. Part of the NP's role is to change attitudes toward issues concerning sexual health. Embracing a holistic perspective and addressing sexual concerns early in the illness may provide individuals the freedom to discuss sexual issues in a nonjudgmental clinical environment.

Given the clinical and economic impact of DAN, preventive interventions such as maintaining a healthy lifestyle and promoting diabetes self-management need to be a primary focus for preventing complications. NPs need to be proactive and explore patients' thoughts and complaints regarding sexual issues by routinely initiating conversations to screen for SD. Based on what has been learned in this review, patients will usually not volunteer information on sexual health. Providing patient-focused care encourages conversations between the provider and can potentially improve the patient's QoL. 

Nutrition therapy for the promotion of sexual health^{3,7,35}

Nutrition therapy has been recommended for the treatment of diabetes for many years. However, most of the recommendations do not completely target the inflammatory component of diabetes and its effects on the body.

Nowlin and colleagues discussed dietary programs that promoted an anti-inflammatory effect for optimal glycemic control. Of the 16 studies reviewed, the Mediterranean and DASH diets and several low-fat diets were associated with lower inflammatory markers ($P < .05$). A low-fat Mediterranean diet was shown to improve risk factors associated with diabetes control.

A diet low in fat but high in whole grains, fruits, and vegetables aids in preventing and controlling inflammation in the body contributing to diabetic neuropathy. Increasing fiber has been shown to be highly effective in reducing glycemic levels, resulting in lower insulin resistance. Nutrition support from a dietitian can be an essential component in maintaining glycemic control.

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