Sexual Dysfunction Associated with Diabetes Mellitus

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Sexual dysfunction associated with diabetes mellitis has been reviewed. The prevalence of impotence among diabetic males ranges between 50 and 60 percent. Duration of diabetes does not correlate with the increasing incidence of impotence, but increasing age of the male is associated. The etiology of impotence is generally assumed to be autonomic neuropathy; the role of vascular pathology is moot. Retrograde ejaculation is present in one to two percent of the cases. Nocturnal penile tumescence monitoring during periods of rapid eye movement (REM) sleep will aid in the differential diagnosis of organic vs psychogenic etiology. Penile prostheses should be considered in the treatment of organic impotence, while sex counseling is indicated for psychogenic cases. The effect of diabetes on female sexual response is conflicting. Further research is needed.

There are at least four million known diabetics in the United States alone, and probably another three million that are undiagnosed. If roughly half of this population—3 1/2 million—is male, and approximately 50 percent of them will become impotent at some point, the magnitude of the problem speaks for itself.

The association between impotence and diabetes was first reported in 1797.[1] Naunyn,[2] in 1906, noted that impaired potency is more frequent among diabetic males and that it may be an early symptom of the disease. More recent studies[3-5] reported a prevalence of impotence among diabetic males ranging between 50 and 60 percent. These figures are considerably higher than those of the general population, as reported by Kinsey[6] in 1948.

Research data on the impairment of sexual function in the diabetic male have become available only within the past 20 years. This is "like yesterday" compared to the research on diabetes in general. It was only in August 1971 that the first statistical report on the impairment of sexual functioning in the diabetic female appeared.[7]

Impotence

Impotence may be the first symptom of diabetes, and may even appear before the diagnosis is made. It is not uncommon for patients to report that they have been impotent until their diabetes has been stabilized, and that their potency returned when the diabetic state was controlled. In

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Table 1. Incidence of Impotence by Age Among 198 Diabetic Men

Age (years)	Number of Subjects	Percent Impotent
15-19	2	0.9
20-24	6	16.7
25-29	4	0.0
30-34	4	25.0
35-39	8	25.0
40-44	16	37.5
45-49	13	38.5
50-54	28	53.6
55-59	30	66.7
60-64	23	73.9
65-69	28	67.9
70-74	21	57.1
75-79	10	70.0
80-84	4	100.0
85-90	1	0.0

those cases in which impotence developed while the diabetes was under control, it was usually more or less permanent. The physiological mechanism has not been clarified.

Renshaw[8] reported that for men who have had diabetes for over six years, up to 48 percent may be impotent. Rubin[9] reported on the incidence of impotency by age among 198 diabetic men; these data are summarized in Table 1.

Impotence does not seem to increase with increasing duration of diabetes mellitus, but does increase with the age of the patient. There is no apparent relationship between the severity of the diabetes, graded according to carbohydrate tolerance and insulin requirement,[10] and the incidence of impotence. Poor control of diabetes, with episodes of acidosis or hypoglycemia, was associated with transient periods of impotence.

The process of erection involves psychological and physiological factors.[11] In discussing the relationship of diabetes to impotence, the exact mechanism is unclear. Possibilities that have been considered include the following:

A. Atrophic changes in the testes of some diabetics have been reported. Warren and LeCompte[12] considered these changes to be due in all probability to malnutrition and attacks of acidosis in the poorly treated diabetic and that ". . . in the adequately treated and controlled diabetic patient there is no apparent alteration from the usual picture."

B. A hormonal or endocrine basis for impotence has long been espoused. Miller and Mason[13] reported that the urinary excretion of 17-ketosteroids, on the average, was lower in male diabetics. These low levels were unrelated to the duration and the severity of the diabetes, and were most pronounced in younger diabetics, ages 20 to 39 years. They attributed the low excretion to diminished output by the testes. More recently, Ellenberg[14] measured the plasma testosterone levels in male diabetics and found them to be within the normal range. Although considered moot, there is no real solid evidence that substantiates a relationship between low levels of 17-ketosteroids and impotence.[5,15,16] Cooper[17] has stated "In practical, clinical terms, this means that the reduction in the plasma testosterone level which may be found in chronically impotent men probably reflects sexual apathy rather than causes it." Simpson[18] has emphasized the importance of the balance of estrogens and androgens as distinct from an absolute decrease in androgens. There does not appear to be any role for androgen therapy.

C. A diminished excretion of gonadotropins was reported by Berquist.[19] In other situations, however, alternation of gonadotropin excretion usually shows no relationship to potency, unless there is total destruction of the pituitary gland.

D. Vascular or neurologic causes have been considered etiologically significant. Simpson[18] reported that "Possible organic causes of chronic impotence in diabetes may be endarteritis affecting the vascular mechanism of erections, or peripheral neuritis involving the presacral and other nerves." Learmouth,[20] in his classic paper, reported that the autonomic nervous system pathways involved in micturition and erection are identical. Since there has been no direct method of objectively measuring the integrity of the nerves supplying the penis, studies of the urinary bladder were done[14] based on the assumption that involvement of these nerves would be reflected simultaneously by abnormalities in both areas. Ellenberg performed neurogenic bladder studies in 45 impotent diabetics with an average age of 43.2

years. Thirty-seven had neurogenic vesical abnormalities, and 38 had neuropathy. Of 30 potent diabetics, with an average age of 42.7 years, three had bladder involvement, and six neuropathy. A random survey of 200 diabetic males showed 59 percent to be impotent, and 82 percent of them had neuropathy. Among the 41 percent of the male diabetics that were potent, only 12 percent of them had neuropathy. While it is generally assumed that autonomic neuropathy is directly related to the impotence, vascular pathology (eg, atherosclerosis, arteriosclerosis, and arterial calcification) is also common in diabetic males. At present, the exact etiology is still debatable.

Ejaculation

Ejaculation is a complex reflex act, consisting of the emission of semen into the prostatic urethra, followed by the antegrade forceful propulsion of this fluid through the anterior urethra. Mitsuya[21] and colleagues, using cineradiographic techniques, demonstrated that contractions and peristalsis of the seminal vesicles, and the up-and-down movements of the ejaculatory ducts, injected the contents of these structures into the prostatic urethra. Contractions of the smooth muscles of the prostate gland occurred several times, emptying the glandular contents into the prostatic urethra. The presence of semen in the prostatic urethra results in several reflex actions: closure of the vesical neck, relaxation of the external sphincter, and rhythmic contraction of the constrictor urethrae, ischiocavernosus and bulbocavernosus muscles, and other associated perineal muscles. Rieser[22] stated that these reflex activities are mediated by sensory nerves from the prostatic urethra. Sympathetic stimulation results in contraction of the vesical neck and prevents retrograde passage of semen, while the external sphincter is actively relaxed by parasympathetic activity.

Retrograde ejaculation in the diabetic male had been unappreciated, unrecognized, and clinically unknown until reported by Greene and Ellenberg.[23,24] In one instance, retrograde ejaculation was the presenting problem and antedated the diagnosis of diabetes by one year. With retrograde ejaculation, the semen reaches the prostatic urethra and then passes in a retrograde fashion into the urinary bladder. The male experiences an orgasm, but the "quality" of the orgasm in retrograde vs antegrade ejaculation has yet to be reported.

Although the incidence of retrograde ejaculation is low, its exact occurrence is not known. The mechanism of action is believed to be diabetic sympathetic neuropathy. Odel[25] demonstrated that the sympathetic nervous system could be affected by diabetes; and since sweat glands have sympathetic innervation, dysfunction of the sympathetic nerves results in anhidrosis. Greene[26] did sweat tests on four diabetic males with retrograde ejaculation and demonstrated anhidrosis involving both lower extremities, indicating neuropathic involvement of the second and third lumbar sympathetic ganglia or their peripheral fibers. Roth[27] reported that 68 percent of 248 diabetics had areas of sweating deficits, and a similar proportion had mild and asymptomatic orthostatic hypotension. Klebanow and McLeod[28] reported on nine diabetic males who did not ejaculate externally. In six of the cases, "occasionally too many spermatozoa were found in the urine immediately after orgasm, but not in the numbers that one would expect with true retrograde ejaculation." Most of those men had noted a gradually decreasing ejaculatory volume over a period of years. With retrograde ejaculation, the male will be infertile unless the spermatozoa can be retrieved from the urinary bladder for insemination. Many healthy children have been born by this technique.[29]

Differential Diagnosis

Since there is no direct correlation between the duration of diabetes and the presence of impotence, but there is an apparent increase in the incidence of impotence in relation to age, among both diabetics and nondiabetics, some serious questions need to be asked about the exact cause of the impotence that is associated with diabetes. As Kolodny[30] appropriately states, ". . .most other complications of diabetes are related to the duration of the illness and *not* the age of the patient."

Erection difficulty in a diabetic patient may be caused by psychogenic factors, organic factors, or

a combination of the two. The hallmark of psychogenic impotence is that it is selective in nature, and occurs under one set of circumstances, but not under another. In most, but not all cases, the male will have an erection occasionally on waking in the morning, and with masturbation. If the impotence is psychogenically based, the male will experience erections at night, nocturnal penile tumescence (NPT), that occur during periods of REM sleep. Varying degrees of penile erection begin and end in rhythm with REM sleep. During an average night's sleep, four to five such periods will occur, at intervals of about 90 minutes. They usually average about 25 percent of total sleep time. The amount of nocturnal penile tumescence experienced by an individual sleeper is directly related to his age. Fisher[31] reports that in adolesence, NPT constitutes 32 percent of total sleep. It remains at this level until about age 40 years then slowly declines until it reaches about 20 percent when a man is in his 60s. The degree of NPT is also age related. During adolesence there are about 90 minutes of full erection; in middle age full erection is present for about 45 minutes; and in old age, the 70s, for about 20 to 25 minutes. The number of maximum erections per night declines from about four in the young, to two or less after 40 years.

Nocturnal erections can be recorded by using a N-7600 Nocturnal Penile Tumescence Monitor (American Medical Systems, Minneapolis). Separate mercury-filled strain gauges are attached to the base and glans of the penis. Changes in penile circumference are continuously monitored and recorded. The presence of normal nocturnal tumescence rules out an organic etiology to the impotence, while partial, or absent nocturnal penile tumescence, in the presence of REM sleep, indicates organic pathology.[32]

Female Sexual Functioning

The impact of diabetes on female sexual functioning has received remarkably little attention. Prior to 1970, the literature dealt exclusively with the effect of diabetes on a female's reproductive capacity, the complications of pregnancy, and the possibility of congenital defects.[8,33]

For years, physicians rarely asked about the sexual concomitants of chronic disease in their patients, particularly females. To date, only two studies on sexual functioning in the diabetic female have been reported. The first published report, by Kolodny,[7] was in the early 1970s. One hundred twenty-five coitally active females between the ages of 18 and 42 years, with previously diagnosed diabetes, were interviewed. For controls, a group of 100 coitally active nondiabetic hospitalized females in the same age group were used. There was a "close similarity in the two groups of women; regarding age, religion, education, marital status, age at menarche, incidence of dysmenorrhea, parity, frequency of coital activity, sexual interest by self-stimulation, and history of psychiatric care." A marked difference in the incidence of sexual dysfunction was found in the two groups: 44 of the 125 diabetic females (35.2 percent) reported complete absence of orgasmic response during the previous year, whereas only six out of 100 nondiabetic females (six percent) reported complete absence of orgasm during the same interval. Past history revealed that none of these six females had ever experienced orgasm; whereas of the 44 diabetics who were now nonorgasmic, 40 had been orgasmic in the past. The onset of orgasmic dysfunction among the diabetic females was gradual, usually over a period of six to twelve months, and, in all cases, followed the onset of diabetes. Unlike their male counterparts, there was a striking correlation between the duration of diabetes and the frequency of orgasmic dysfunction. Similar to their male counterparts, the severity of diabetes was not a relevant factor.

Ellenberg[34] recently reported on 100 diabetic females, 54 of whom had clear evidence of diabetic neuropathy and 46 of whom did not. The age range was 20 to 74 years, encompassing an older population than Kolodny's study. Both groups were questioned concerning their interest in sex and the presence or absence of orgasm. Among the 54 diabetic females with neuropathy, 44 (81 percent) had "normal libido and orgasmic reaction." Seven out of 44 had a decrease in libido and orgasm, and three females reported no sexual interest or response. Among the 46 diabetic females without neuropathy, 38 (82 percent) reported a normal interest in orgasmic response, six noted a decrease, and two experienced no sexual interest or orgasm.

Future research is obviously needed to clarify

the many questions concerning the effects of diabetes on female sexual response and to resolve conflicting data. Perhaps objective studies of the clitoris and vagina, similar to the nocturnal penile plethysmograph, will assist in the elucidation.

Discussion

Whenever diabetes mellitus is diagnosed, it is important that a sexual history be taken by an experienced person.

The hallmark of psychogenic impotence is its selective nature; the male may, and usually does, wake up with morning erections and also has erections at other times of the day or night, or with masturbation. The difficulty in either obtaining or maintaining an erection occurs *only* when he tries to have intercourse. When organic impotence is associated with diabetes mellitus, the sexual history reveals a slow, steady, usually *progressive* decrease in erectile ability. The full, turgid erections occur very rarely, if at all, and usually the maximum erection the male is able to get is 50 to 70 percent. Karacan[35] reported on nocturnal penile tumescence (NPT) characteristics of 35 impotent diabetic males, and 35 age-matched, healthy control subjects. The mean minutes of sleep per night were 401 in the diabetic group and 398 in the control. The minutes of full nocturnal penile tumescence per night was 5.8 in the diabetic group, and 81.9 in the control group; while the minutes of partial NPT per night were 20.7 in the diabetic group and 24.2 in the control group. The diabetic males had approximately the same duration of partial NPT but a significant reduction ($P>0.001$) in the duration of full NPT. Confirmatory studies need to be done.

Even though diabetes is a common cause of organic erectile dysfunction, it is premature to assume that all diabetic males with erection problems have the sexual difficulty secondary to the diabetes. There is no reason to believe that the diabetic male is less vulnerable to psychogenic impotence. If there is doubt about the etiology of the impotence, monitoring of NPT and REM sleep is indicated. If the impotence is on an organic basis, then the patient and the partner should be made aware of the possibility of a penile implant.

Three types of penile prostheses are available. The rigid prosthesis, of which the Small-Carrion[36] is an example, is a simple rodlike device with a silicone sponge interior encased in a medical-grade silicone exterior. It is implanted in pairs, within the crura and the corpora cavernosa of the penis via a perineal or penile incision, with minimal risk and few complications.[37] After surgery, the male has a normal appearing, sustained erection. Wearing a broad banded athletic supporter or jockey-type shorts will allow the erect penis to remain undetected.

In 1973, Scott[38] reported on a prosthesis composed of inflatable silastic rods placed in the corpora. A small pump, placed in the upper scrotum, could be squeezed and radiopaque fluid would be transferred from a reservoir to the penile prosthesis, producing an erection. Detumescence is achieved by squeezing a deflating valve. The major advantage of the inflatable prosthesis is that the penis is not constantly erect, and not detectable by the partner. Malloy[39] reported that major complications developed in 23 percent of the cases.

Furlow[40] found major complications in seven of 36 patients, but the end result of implantation with the inflatable prosthesis has been "quite satisfactory." Thirty-five out of 36 patients have normally functioning prostheses and patient-partner acceptance has been excellent. His contention was "that implantation of the inflatable penile prosthesis is a highly acceptable method of treating organic impotence."

More recently, Finney,[41] in 1977, described a hinged silicone penile implant. The soft hinge permits the penis to hang down in a normal anatomical position and yet provide the necessary stability for coitus when desired. Twenty of the new hinged implants have been inserted. The results were "quite satisfactory in all patients except the first, in whom a hinge that was too short was used."

If the impotence is on a psychogenic basis, sexual counseling is the treatment of choice.

There is, to date, no effective treatment of retrograde ejaculation secondary to diabetic sympathetic neuropathy, although Abrahams[42] reported on two cases of retrograde ejaculation subsequent to Y-V plasty of the bladder neck which were corrected surgically. It is important, though, that the male and the spouse understand that the

retrograde ejaculation is a complication of the disease. In a number of instances the female has assumed that the absence of external ejaculation represented a sexual problem and often left her feeling insecure about herself, thinking that she was the cause of the problem.

When a previously orgasmic woman becomes nonorgasmic, one organic possibility that should be considered is whether this represents early diabetes. If a marital-sexual history does not reveal any apparent cause for the decrease in orgasmic response, then a glucose tolerance test is indicated.

Sexual health may be more important to the patient than to the physician. As members of a helping profession, physicians can alleviate a considerable amount of pain and suffering in the marital and sexual relationships of their patients. To do less minimizes their effectiveness.

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