Sexual Dysfunction in Diabetic Men

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SUMMARY

Sexual function was studied in 175 diabetic male outpatients randomly selected. Data collected included medical and sexual histories, physical examination, complete blood count, urinalysis, T4, 12 channel chemistry screening profile and plasma testosterone determination. Eighty-five of the 175 men were impotent (49 per cent), four reported premature ejaculation (2 per cent) and two subjects had retrograde ejaculation (1 per cent). In the group with impotence, the mean age of the subjects and duration of diabetes were respectively fifty-three and six years. In patients without impotence, the comparable values were forty-five and five years.

Typically, the onset of impotence was gradual, usually progressing over a period of six months to a year with an interval during which firmness of erection was decreased. Levels of sexual interest were sustained in almost all subjects. Over 90 per cent of the males studied gave a pattern of impotence compatible with an organic rather than a psychologic etiology.

Occurrence of impotence was not definitely correlated with duration of diabetes or with insulin or oral agents. Incidence of peripheral neuropathy was increased in the group with sexual dysfunction.

Plasma testosterone levels were within normal limits in impotent diabetics or in the group without impotence, respectively (mean ± S.E.M.) 627 ± 15 and 637 ± 14 ng per cent. It is apparent from these findings that androgen deficiency was not an etiologic factor in this group of diabetic men with impotence.
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Sexual dysfunction is a common complication of diabetes mellitus in both men[1-6] and women.[7] Despite previous investigators generally agreeing that the prevalence of impotence in diabetic men approximates 50 per cent, there is controversy surrounding the etiology of this problem. Endocrine causes of the impotence associated with diabetes have been suggested[2,3,6] although not verified. Vascular complications of diabetes may produce impotence on the basis of large vessel disease (e.g., the Leriche Syndrome) or, theoretically, secondary to microvascular changes. The neurologic factor, long felt to be important in the pathogenesis of erective difficulties in diabetic males, has been recently re-emphasized by the demonstration of a high association of neurogenic vesical abnormalities in a group of impotent diabetics.[5] Other systemic diseases, patterns of drug use, and psychological factors[8] may all contribute to difficulty in the clinical evaluation of the etiology of impotence in the diabetic male, and may confuse the choice of treatment modality.

In addition to impotence, other types of sexual dysfunction may complicate diabetes, including retrograde ejaculation[4] and premature ejaculation. This study was undertaken to obtain further information concerning sexual dysfunction in male diabetics.

METHOD

One hundred and eighty-six men between the ages of eighteen and eighty-three with previously diagnosed diabetes mellitus were interviewed during outpatient visits to Joslin Clinic during 1971. Of this group, 175 men completed all phases of this study. For each subject, a sexual and medical history was obtained, a physical examination was performed and the following laboratory studies were done: complete blood count, urinalysis, T4, 12 channel chemistry screening profile and plasma testosterone determination.[9]

The interview included specific questions regarding sexual distress; the mode of onset; duration, frequency and degree of impotence; the temporal relationship of impotence to the clinical onset of diabetes; and the current existence of such signs of intact neurovascular function as nocturnal or morning erections, or the ability to obtain an erection during masturbation. A review of the patient's medical records was also conducted to supplement the clinical material obtained in

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this study, including an itemization of current medications; assessment of the control of diabetes, and the presence of retinopathy, nephropathy, peripheral vascular disease, neuropathy or other diabetic complications.

RESULTS

Eighty-five of 175 diabetic men (48.6 per cent) were impotent. The age distribution among impotent diabetics and the control diabetics is shown in table 1. In the group of men with impotence, the mean age was 53.2 years; in the control group, the mean age was 45.4 years.

TABLE 1

Age distribution of study subjects

Age	Number of subjects	Number of impotent
18-24	11	0
25-31	14	4
32-38	13	5
39-45	21	11
46-52	26	12
53-59	33	17
60-66	31	18
67-73	16	11
74-80	8	6
81 plus	2	1
Totals	175	85

There was no apparent correlation between duration of diabetes and presence or absence of impotence (See table 2). In fourteen men, impotence had been an initial manifestation of diabetes and preceded the establishment of the diagnosis; eight of these men regained normal potency following the initiation of diabetic therapy.

Twenty-nine men reported impotence of less than one year's duration. Although these men, taken as a group, gave the impression of poorer control than a group of randomly selected, age-matched diabetic men (by review of the preceding year's medical records and laboratory tests), formal criteria assessing control had not been planned prospectively and thus no definite conclusions could be drawn.

Typically, the onset of impotence was gradual, usually progressing over a period of six months to a year with a period of time during which there was a decreased firmness of the erection but not total absence of the erectile response. However, in men with impotence of more than one year's duration, total loss of erective function was described by fifty-four of fifty-six men. Seven men (8.2 per cent) reported the relatively sudden onset of impotence. These men provided information that strongly suggested that their sexual distress was psychogenic: morning or nocturnal erections occurred frequently, the ability to masturbate was unimpaired, or a selective pattern of impotence was seen.

TABLE 2

Characteristics of study subjects

Characteristic	Impotent (N = 85)	Not impotent (N = 90)
Mean age (years)	53.2	45.4
Duration of diabetes (years)	6.4	5.9
Treatment:		
Diet alone	1	3
Oral agents	38	43
Insulin	46	44
Complications:		
Retinopathy	19	20
Neuropathy	32	19
Nephropathy	11	10
Hypertension	10	8
Weekly coital frequency		
At present	0.1	2.0
Five years ago	1.9	2.3
Plasma testosterone	627 ± 15 (S.E.M.) ng./100 ml.	637 ± 14 (S.E.M.) ng./100 ml.

Table 2 presents additional characteristics of the impotent and nonimpotent diabetic men. Mode of treatment of diabetes did not correlate with the presence or absence of impotence; insulin-requiring men with impotence were using an average daily dose of 40.4 U. while nonimpotent insulin-requiring men were using an average daily dose of 42.3 U. The incidence of vascular disease, as evidenced by retinopathy and/or nephropathy, and of hypertension was similar in the two groups. The incidence of neuropathy was significantly greater in the group of men with impotence (37.6 per cent) than in nonimpotent diabetics (21.1 per cent) ($> .05$ using the chi-square test).[10]

TABLE 3

Differential Diagnosis

Types	Libido	Erection loss	A.M. Erections	Masturbation ability
Organic (most causes)	N↓	Abrupt	Absent	Absent
Diabetes	N↑	Gradual	Absent	Partially/totally
Psychological (including aging male)	N↓	Abrupt	Present	Unimpaired

The means for plasma testosterone in the impotent diabetic men (627 ± 15 ng./100 ml.) and the group without impotence (637 ± 14 ng./100 ml.) (mean ± S.E.M.) were remarkably similar. Plasma testosterone levels of the nonimpotent diabetics and all but three of the eighty-five impotent diabetics were in the normal range (400 to 1,000 ng./100 ml.). A subject with documented hemochromatosis had a plasma testosterone of 256 ng./100 ml.; a subject who was receiving estrogen therapy for carcinoma of the prostate had a plasma testosterone of 313 ng./100 ml. (in this case, impotence antedated the prostatic disease by six years); and a man with uremia (BUN = 85 mg. per cent, Hgb = 8 gm.) had a plasma testosterone of 334 ng./100 ml. The latter three subjects reported markedly depressed libido, in contrast to the normal libido described by the remainder of the men, and had small testes. Two other subjects also had small testes and impotence, but their libido and plasma testosterone levels were normal. Five men had previously been treated with testosterone with transient improvement.

Two subjects (1.1 per cent) had retrograde ejaculation and four subjects (2.3 per cent) reported premature ejaculation. No case of ejaculatory incompetence was discovered.

DISCUSSION

The frequent occurrence of impotence with diabetes mellitus described in this study (eighty-five of 175 men or 48.6 per cent) is in agreement with previously reports.[2,3,5,6] Despite the frequency of this disorder, the pathophysiologic mechanism resulting in loss of erective function in diabetes is unclear.

Schoffling and co-workers[3] reported in 1963 that two thirds of their patients with impotence and diabetes had decreased urinary excretion of pituitary gonadotropin (determined by bioassay), yet urinary excretion of 17-ketosteroids was increased. According to these workers, the elevated 17-ketosteroid levels were due to an increase in adrenal androgens with low potency, while the metabolites of testosterone were decreased. In addition, they found that one-third of their group had low sperm counts. They reported that all but one of their patients aged forty or under responded significantly to therapy with a combination of chorionic gonadotropin and testosterone; most patients over age forty responded significantly to testosterone therapy alone.

In more recent work by Schoffling, thirteen of forty diabetic men with sexual disturbances were found to have low plasma testosterone levels, whereas only one of thirteen diabetic men without sexual disturbances had subnormal levels.[6] However, the normal range described in this report is extremely narrow; using values more commonly accepted as the normal male range would show only six of these men to have diminished plasma testosterone. Although Schoffling states that "endocrine dysfunction is the main cause" of impotence in diabetes, our results weigh heavily against this view. Further documentation of the normal plasma testosterone levels of impotent diabetic men is provided by others.[5,11]

The results of testosterone therapy, alone or in common with chronic gonadotropin, have not been successful in the treatment of impotent diabetics in our experience or in the experience of Ellenberg.[5] Indeed, Schoffling's group is the only one to advocate its usefulness under these circumstances. In addition to possible adverse effects resulting from testosterone therapy such as sodium retention, hepatic dysfunction and prostatic hypertrophy, the administration of exogenous androgen may increase libido while pathologic conditions resulting in erective dysfunction are unchanged, thus creating a far less comfortable and more frustrating situation for the patient.

Ellenberg's work presents careful documentation of the importance of neuropathy in the pathogenesis of impotence in diabetic men.[5] Judging by the clinical examination alone, we detected significantly greater incidence of neuropathy in diabetic men with impotence than in nonimpotent diabetics, thus supporting the contention that impotence in diabetes is most likely due to neurogenic factors. Additionally, Weiss[12] in a review of the physiology of penile erection discusses the various anatomical sites of pathology that could account for the problem. Valve-like structures called "polsters" containing smooth muscle have been described near the corpora cavernosa and are under the control of the autonomic nervous system. Poor neural transmission most likely would disturb the steady state of increased inflow of blood into the erectile tissues. This site of pathology seems logical as an area for further consideration in the diabetic male, especially with the recent work of Faerman et al.[13] who found histological evidence of autonomic neuropathy in the neural fibers of the corpora cavernosa.

Since there is no known effective therapy for such sexual dysfunction, it is most important that the physician be certain that he is not dealing with impotence due to correctible causes. Assessment of drug

therapy to watch for erective difficulties arising from the use of tranquilizers, antidepressants, antihypertensives or estrogens will be rewarded with an occasionally simple solution to what might otherwise appear to be a complex problem. Alcohol consumption, statistically, is a frequent factor in the appearance of impotence.

The differential diagnosis of impotence in the diabetic male resolves around other organic causes and the psychogenic etiology including the aging male variant described by Masters and Johnson.[14] A useful analysis of this differential is shown in table 3 along the lines of presentation by Cooper.[15] Characteristically, diabetic men with organic impotence have continued sexual interest despite the slow onset of erectile dysfunction, often described as "50 per cent firm." They generally have an inability to masturbate or stimulate erection in any way.

The organically impotent male due to other causes such as impotence following prostatectomy usually has much more abrupt onset of impotence and a complete lack of ability to obtain morning erections or masturbate. Psychogenic impotence is generally abrupt in onset; libido is low and masturbation ability is maintained. A change of partner often alleviates the disorder. This variation of impotence is frequently reversible.[9] Depression and anxiety often accompany organic impotence, leading to a psychologic overlay.

The low percentage of impotence due to psychologic causes in this series of patients was most likely due to the nature of the patient population; the subjects came for medical treatment of diabetes on a regular basis, as contrasted with patients who seek a specialist, like a urologist, for a specific sexual problem. In most physicians' experience, strong psychologic concerns are more often the main factor in the latter group.

A study of sexual dysfunction in diabetes would be incomplete without mention of the importance of appropriate counseling for both spouses in such instances. The physician, although unable to offer a cure for the disorder can often alleviate a tense home situation by adequate explanation. This allows for misinformation and cultural bias to be corrected and provides an opportunity for each spouse to attain a greater degree of comfort in living with a difficult problem.

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