

APPRAISAL OF MALE ERECTILE DYSFUNCTION IN PATIENTS WITH DIABETES MELLITUSRajendra Prasad Suram¹, Prathima Raj Dara²¹Associate Professor, Department of General Medicine, Government Medical College, Nizamabad.²Associate Professor, Department of General Medicine, Government Medical College, Nizamabad.**ABSTRACT****BACKGROUND**

One of the most common adverse effects of diabetes mellitus, erectile dysfunction (ED) remains difficult to treat despite advances in pharmacotherapeutic approaches in the field. This unmet need has brought about a late re-focus on the pathophysiology, so as to comprehend the cellular and molecular mechanism prompting ED in diabetes. Diabetes-induced ED is a need to find focuses that may prompt novel approaches for a fruitful treatment. Present study was tried to understand role of vascular and neurogenic alterations in the pathophysiology of diabetic sexual dysfunction.

SUBJECTS AND METHODS

The study was carried out on 17 male impotent patients with Diabetes Mellitus: 5 patients (Aged 43-56 years; mean age 48.4) had insulin-dependent DM, and 13 patients (Aged 40-62; mean age 53.5) non-insulin-dependent DM. None of the patients were on medications known to interfere with male erectile sexual function. All patients were told to give informed consent. Every participant underwent a physical examination including complete medical and sexual history and routine laboratory tests. Penile vascular assessment and assessment of nocturnal penile erections investigation were assessed in the study.

RESULTS

As showed in Table I. alterations in the vascular system was found in 13 (61.5%) participants. Out of which moderate alteration noticed in 8 (47%) and severe alteration in 5 (16%) patients. Among the patients with non-insulin-dependent DM, 7 (41.1%) had showed Nocturnal Penile Erections abnormalities. According to the history and also to the normal responses to the Nocturnal penile tumescence monitoring (NPTM), patients were diagnosed as having impotence of psychogenic origin.

CONCLUSION

Initial involvement of both arterial supply and of the neurological pathways can be of some interest, suggesting the need for an early screening of neural and vascular status even in patients without penile erectile failure. As is the case for other degenerative ailments, the beginning of identification of injury could be the first step towards finding appropriate treatments to prevent more severe damage in near future.

KEYWORDS

Erectile Dysfunction, Nocturnal Penile Tumescence Monitoring, Doppler Ultrasound Examination, Nocturnal Penile Erections.

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INTRODUCTION: Sexual problems are most common among people who were diagnosed with diabetes, especially in elder age group men who had diabetes for years. In addition, suggestion from medical professionals believe that women diagnosed with diabetes also experience sexual difficulties. These complications were results from the disease. Normal physiology, male sexual capacity requires a perplexing cooperation of vascular, neurological, hormonal, and mental systems. The underlying mandatory occasion is procurement and upkeep of an erect penis, which is a vascular wonder. Typical erections require blood stream into the corpora cavernosae and corpus spongiosum. As the blood accelerates, the weight inside the intracavernosal space expands drastically to interfere with penile venous surge.

This mix of expanded intracavernosal blood stream and decreased venous surge permits a man to procure and keep up a firm erection. The role of nitric oxide (NO) is significant during penile erection. Smooth muscle unwinding is intervened medicated by NO, which is a vaporous and labile go between. NO is integrated by neuronal NO synthase (nNOS) in the autonomic postganglionic parasympathetic nerves.^[1,2] and by endothelial nitric oxide synthase in the endothelium coating the veins and cavernosal sinusoids. Nerve driving forces in light of sexual jolt are conveyed from the spinal string to the hypogastric plexus where the cell assemblages of the nitrergic nerves are found.

Once actuated, the nitrergic neurons inside the hypogastric plexus transmit activity potential through their axons to the penile vasculature. These nitrergic axons then discharge high amounts of NO on to the close-by smooth muscle cells. NO diffuses quickly into the smooth muscle cells, bringing about unwinding by expanding the intracellular centralisations of cGMP. The unwinding of the cavernosal and blood vessel, smooth muscle results in an expansion in blood stream into the penis.

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Thus, this causes shear weight on the endothelial covering, which advances phosphorylation and delayed initiation of endothelial nitric oxide synthase prompting long-lasting arrival of NO from the endothelium to keep up the smooth muscle unwinding.^[3] As the intracavernosal weight achieves the level of the systemic blood vessel pulse, the subtunical venules are compacted, which results in an unbending erection. Diabetes mellitus is one of the transcendent risk factor of erectile dysfunction (ED); furthermore, a standout amongst the most hard to treat.^[4,5] Around 50% of diabetic men will experience the ill effects of ED within 10 years of the diagnosis.^[6] Earlier studies demonstrate that pervasiveness of ED in men with diabetes ranges from 35–75% versus 26% all in all population.^[7] ED displaying in these men is broadly viewed as a sign of more systemic vascular sickness and is prone to go before a coronary occasion by 5 years.^[8,9]

Combined with the pervasiveness of weight and metabolic disorder in these men, it is evident that sickness movement is prone to decrease the adequacy of traditional pharmacotherapeutic choices for the treatment of ED in diabetic men. This has implied that diabetic men frame a large gathering of patients experiencing end-stage treatment as penile prosthesis surgery. The present study was embraced to research the part of neurologic and vascular pathologies in the pathophysiology of diabetic ED. Therefore, we examined a gathering of male feeble diabetic patients so as to determine the recurrence of disconnected or consolidated vascular and neurologic irregularities, and to research conceivable connections between the observed variations from the norm and the term and sort (insulin-dependent and non-insulin dependent) of the diseases.

MATERIAL AND METHODS: The study was carried out on 17 male impotent diabetic patients with DM: 5 patients (aged 43-56 years; mean age 48.4) had insulin-dependent DM, and 13 patients (aged 40-62; mean age 53.5) non-insulin-dependent DM. None of the patients were on medications known to interfere with male erectile sexual function. All patients were told to give informed consent. Every participant underwent a physical examination including complete medical and sexual history and routine laboratory tests. The following investigations were assessed.

Penile Vascular Assessment: Doppler ultrasound examination (DUE) of penile supply routes was performed by strategy portrayed by Jevtich. By method, for a 9-MHz test, four conduits were studied: The two dorsal supply routes and the two enormous courses of the penis. DUE was considered seriously adjusted when no less than one of the accompanying conditions was met: (1) not stable in both dorsal penile conduits; (2) not stable in one dorsal vein and in both enormous corridors; and (3) poor sound in both dorsal penile supply routes with not stable in both huge courses. DUE was considered tolerably adjusted when changes were not as extreme as in the aforementioned cases.

Assessment of Nocturnal Penile Erections: Nocturnal penile tumescence observing (NPTM)^[10] was utilised to confirm the nearness of Nocturnal penile erections with a specific end goal to bar psychogenic weakness. The test was rehashed on no less than three progressive evenings, to determine the consistency of the reactions. It was performed at home in everything except seven patients, who were not agreeable for mental and social reasons. All things considered, these seven patients have been incorporated into this study, as both sexual history and the aforementioned examinations unmistakably represented a natural inception of their weakness.

RESULTS:

Patient Number	Age	Type of the DM	Vascular Assessment by Doppler Method	Nocturnal Penile Erection
1	40	II	+	Absent
2	55	II	++	Present
3	56	I	+++	Absent
4	45	II	++	Present
5	48	I	++	Absent
6	61	II	+	Absent
7	52	II	+++	Absent
8	49	I	+	Absent
9	48	II	++	Present
10	54	II	++	Absent
11	46	I	+++	Present
12	56	II	++	Absent
13	60	II	++	Absent
14	55	II	+++	Present
15	62	II	++	Present
16	43	I	+	Absent
17	48	II	+++	Absent

Table 1: Patient Showing Penile Erectile Failure during Diabetes

- DM=Diabetes mellitus.
- I=Insulin dependent.
- II= Non-insulin dependent.
- + = Normal.
- ++ = moderately altered + + += severely altered.

Penile Vascular Assessment: Results are shown in Table I. Alterations in the vascular system was found in 13 (61.5%) participants. Out of which moderately altered in 8 (47%) and severely altered in 5 (16%) patients. The two dorsal veins and the two enormous courses ended up being required with the same recurrence. All patients with insulin-dependent DM had confirmation of penile natural vascular and/or neurogenic association to represent their ineptitude.

Nocturnal Penile Erections: Nocturnal penile tumescence checking (NPTM) was utilised to confirm the nearness of night-time penile erections keeping in mind the end goal to bar psychogenic ineptitude.

Among the patients with non-insulin-subordinate DM, 7 (41.1%) had indicated Nocturnal Penile Erections variations from the norm. As per the history, furthermore to the typical reactions to the NPTM, were analysed as having ineptitude of psychogenic beginning.

DISCUSSION: Microvascular and macrovascular weakening of the blood vessel supply to the penis has been depicted in both after death^{[11,12][13]} hence giving proof of vasculogenic association in the pathophysiology of the sexual dysfunction in male diabetic patients. As of late, the presentation of non-obtrusive vascular testing,^[14,15] has permitted the pervasiveness of penile vascular modifications in diabetic patients to be concentrated all the more precisely. The estimation of DUE has been as of late scrutinised particularly with respect to the assessment of the huge courses.^[16]

In any case, as we would see it, the non-intrusive Doppler method still remains a significant screening test for distinguishing penile blood vessel block in feeble patients, its unwavering quality having been appraised by past studies.^[17] that demonstrated a relationship between 'DUE of penile corridors and angiographic discoveries; further investigation of the vascular status by method of angiography, which are more point by point yet obtrusive, ought to be saved for those cases in which revascularisation surgery is viewed as doable. Present results affirm past perceptions.^[18] that vascular inclusion is a noteworthy reason for ineptitude in male diabetics and shows encouragement that bright and serious harm of penile blood vessel supply might be observed even in the early phases of the illness. Peripheral somatic neuropathy has been found consistently related to sexual dysfunction in diabetic patients.

Present results concur with past reports.^[19] that stretch the part of psychogenic variables in diabetic ineptitude. Truth be told, in our arrangement four patients did not indicate vascular or neurogenic variations from the norm to represent their grumblings and reported ordinary night-time penile erections with NPTM. The last perception might be clarified by considering the way that pudendal and pelvic afferent and efferent strands might be brought out by sacral reaction, are not quite the same as those really required in the innervation of penile erectile instrument. Inside this connection, as diabetic neuropathic injuries happen indiscriminately.^[20] modifications of the strands' incitement evoked, might be sacral reaction, as a matter of course, infer that the adjustment of the filaments is required in the penile erectile system.

By differentiation, it might likewise be derived that psychogenic elements can assume a conclusive part in erectile disappointment.^[21] notwithstanding when serious vascular and neurogenic changes can be illustrated, as is by all accounts demonstrated by the adequacy of psychotherapeutic systems even in patients with natural irregularities.^[22]

CONCLUSIONS: Present results show that vascular obstacle is a more successive component than NPTM in diabetic ED. To determine the nearness of modifications of the vascular obstacle, present and past perceptions propose that in a far reaching indicative convention of diabetic ineptitude. The investigation of vascular obstacle is demonstrated, generally speaking, just in patients with adjusted NPTM contemplates. From the clinical perspective, our study shows the requirement for a complete screening assessment of patients with DM compiling of penile erectile disappointment for conceivable reasons for their unsettling influences, including vascular and neurological variables. Besides, the perceptions of early contributions both of blood vessel supply and of the neurological pathways can be of some interest, proposing the requirement for an early screening of neural and vascular status even in patients without penile erectile disappointment. Similar to the case for other degenerative illnesses, the early location of moment sores could be the initial move towards finding proper medications to counteract more extreme harm of the included structures in longer periods.

REFERENCES

1. Moncada S, Higgs A, Furchgott R. International union of pharmacology nomenclature in nitric oxide research. *Pharmacol Rev* 1997;49(2):137-142.
2. Celtek S. Let's make NO mistake!. *Int J Impot Res* 2005;17:388-389.
3. Hurt KJ, Musicki B, Palese MA, et al. Akt dependent phosphorylation of endothelial nitric oxide synthase mediates penile erection. *Proc Natl Acad Sci USA* 2002;99(6):4061-4066.
4. Cameron NE, Cotter MA. Erectile dysfunction and diabetes mellitus: mechanistic considerations from studies in experimental models. *Curr Diab Rev* 2007;3:149-158.
5. Chitale K, Kupelian V, Subak L, et al. Diabetes, obesity and erectile dysfunction: field overview and research priorities. *J Urol* 2009;182(Suppl 6):S45-S50.
6. Vinik AI, Erbas T. Recognizing and treating diabetic autonomic neuropathy. *Cleve Clin J Med* 2001;68(11):928-944.
7. Martin-Morales A, Sanchez-Cruz JJ, de Tejada SI, et al. Prevalence and independent risk factors for erectile dysfunction in Spain: results of the epidemiologia de la disfuncion erectil masculina study. *J Urol* 2001;166(2):569-574.
8. Jackson G. Erectile dysfunction: a marker of silent coronary artery disease. *Eur Heart J* 2006;27:2613-2614.
9. Celtek S. Nitrogenic noradrenergic interaction in penile erection: a new insight into erectile dysfunction. *Drugs Today (B arc)* 2000;36(2-3):135-146.
10. Barry JM, Blank B, Boileau M. Nocturnal penile tumescence monitoring with stamps. *Urology* 1980;15(2):171-172.

11. Faerman I, Glocer L, Fox D, et al. Impotence and diabetes: histological studies of the autonomic nervous fibers of the corpora cavernosa in impotent diabetic males. *Diabetes* 1974;23(12):971-976.
12. Ruzbarsky V, Michal V. Morphologic changes in the arterial bed of the penis with aging. Relationship to the pathogenesis of impotence. *Invest Urol* 1977;15(3):194-199.
13. Michal V, Pospichal J. Phalloarteriography in the diagnosis of erectile impotence. *World J Surg* 1978;2(2):239-248.
14. Abelson D. Diagnostic value of the penile pulse and blood pressure: a Doppler study of impotence in diabetics. *J Urol* 1975;113(5):636-639.
15. Jevtich MJ. Importance of penile arterial pulse sound examination in impotence. *J Urol* 1980;124(6):820-824.
16. Sharlip ID. Further evidence that Doppler auscultation of the penis does not detect the central penile artery. *J Urol* 1986;135:234A.
17. Jevtich MJ, Edson M, Jarman WD, et al. Vascular factor in erectile failure among diabetics. *Urology* 1982;19(2):163-168.
18. Lehman TP, Jacobs JA. Etiology of diabetic impotence. *J Urol* 1983;129(2):291-294.
19. Waxman SG. Pathophysiology of nerve conduction: relation to diabetic neuropathy. *Ann Intern Med* 1980;92(2):297-301.
20. Schiavi RC. Psychological treatment of erectile disorders in diabetic patients. *Ann Intern Med* 1980;92(p 2):337-339.
21. Kaplan HS. *The new sex therapy*. New York: Brunner and Masel. 1974
22. Benvenuti F, Tosto A, Boncinelli L, et al. Electrophysiologic tests of bladder innervation as indicators of autonomic neuropathy in diabetes mellitus. *Neurourol Urodynam* 1987;6(1):21-28.