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ORIGINAL ARTICLE

A retrospective study on the prevalence of erectile dysfunction among Indians with T2DM using single-question self-reporting method and its association with diabetes complications and CV risk factors

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Abstract

Background Erectile dysfunction (ED) is a prevalent complication of Diabetes Mellitus (DM) globally. However, there are lack of comprehensive studies on the association of ED with DM comorbidities and complications, especially among the Indian population.

Objective To assess the prevalence of erectile dysfunction (ED) and its association with comorbidities and complications of T2DM.

Methods Data from 1423 cases of male T2DM patients with ED aged 18–85 (Group-A) were analyzed and compared to an equal number of T2DM patients without ED (Group-B).

Results ED was associated with CAD [1.5881;95%CI 1.236 to 2.041($p = 0.0003$)], diabetic autonomic neuropathy [4.1504;95%CI 2.894 to 5.953($p < 0.0001$)], retinopathy [1.5473;95%CI 1.266 to 1.891($p < 0.0001$)], hypothyroidism [1.3640;95%CI 1.0056 to 1.8502($p = 0.0460$)], smoking [2.2111;95% CI 1.7575 to 2.7816($p < 0.0001$)], alcohol dependence [2.0018;95%CI 1.6213 to 2.4716($p < 0.0001$)] and combined smoking and alcohol dependence [5.9178;95%CI 4.749 to 7.373($p < 0.0001$)]. Also, multivariable regression analysis revealed a significant association of ED with duration of DM ≥ 10 years ($p = 0.0023$), HbA1C ≥ 7 ($p < 0.0001$), BMI ≥ 23 ($p = 0.0211$), ESR ≥ 15 ($p < 0.0001$), dyslipidemia ($p < 0.05$) and CKD ($p = 0.0439$).

Conclusion Time constraints often force physicians to depend on self-reporting for diagnosing ED, leading to significant underreporting. This highlights the urgent need for efficient and time-saving diagnostic tools to aid Indian physicians in diagnosing ED accurately. Recognizing ED as a CV risk equivalent is crucial, as it is significantly associated with CVD. This suggests that ED could serve as an early marker of underlying CV risk, necessitating proactive vascular risk assessment and management.

Keywords Diabetes Mellitus, Type 2 · Erectile dysfunction · Self Report · Coronary Artery Disease · Prevalence

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Introduction

Diabetes mellitus (DM) is prevalent in nearly all countries and is associated with various sexual dysfunctions in men and women. A threefold increased risk of erectile dysfunction (ED) was found in men with DM compared to those without DM [1, 2]. In a meta-analysis, the general prevalence of ED in DM was found to be 52.5%, whereas the same was 66.3% among T2DM males [3]. In DM-induced ED, ischemic damage of the distal circulation occurs along with peripheral and autonomic neuropathy leading to impaired sensory impulses from the penis to the reflexogenic erectile center [4], and a lack of parasympathetic activity needed to relax the smooth muscle of the corpus cavernosum [5].

The proposed mechanisms of ED in DM patients include elevated Advanced glycation end-products (both soluble and tissue-bound forms), increased levels of oxygen free radicals and endothelin B receptor binding sites, impaired nitric oxide (NO) synthesis and cyclic guanosine monophosphate (cGMP)-dependent kinase-1 (PKG-1), ultrastructural changes, upregulated RhoA/Rho-kinase pathway and NO-dependent selective nitrergic nerve degeneration [6]. ED is characterized by a persistent inability to maintain or achieve an adequate erection and causes a decreased quality of life. The prevalence of ED ranges from 35 to 90% and occurs 10–15 years earlier in men with DM [7–9]. This extensive variation in the prevalence of ED has been attributed to the use of different definitions in distinct populations, varied study designs and the use of various diagnostic tools. In T2DM men, comorbidities such as hypertension, obesity, metabolic syndrome, atherogenic dyslipidemia, etc. are common and each of these factors could be an independent risk factor in the development of ED.

Sexual health inquiry is an important aspect of diabetes care. The management of DM and lifestyle changes have been associated with improvements in sexual function. However, there is a notable dearth of comprehensive studies examining the association between ED and its potential comorbidities and complications, particularly in the context of the Indian population. Despite the ample evidence that ED is among the major complications of T2DM its presence remains poorly evaluated in routine clinical practice. The discussion of sexual issues, especially those related to male potency may cause embarrassment and stress, and failure on the part of treating physicians to enquire about ED in routine diabetic care results in reporting bias leading to general underestimation of sexual problems. It may lead to an under recognition of ED, thereby negatively affecting the patient's quality of life. Taking into consideration the fact that patients with T2DM are at higher risk of ED, recently published guidelines have advised physicians to ask their patients with T2DM about their erectile function. Indeed, National Institute for Health and Care Excellence (NICE) guidelines have recommended men with T2DM to discuss ED with their physicians every year [10].

Generally, apart from self-reporting, standardized questionnaires are used to detect ED in male patients. The Brief Male Sexual Function Inventory (BMSFI), the International Index of Erectile Function (IIEF), and the Massachusetts Male Aging Study (MMAS) scales are three well-known questionnaire-based tools used extensively in research [11–14]. However, healthcare professionals in India often face significant time limitations that impede their ability to conduct comprehensive and detailed questionnaire-based assessments to diagnose ED. This challenge is particularly pronounced in clinical settings in developing countries like India, where busy daily schedules and a high volume of patients demand physicians' attention. Consequently, healthcare providers often resort to relying on patients' self-reported symptoms alone as a practical means of diagnosing ED. O'Donnell AB et al. have reported that the diagnosis of ED through self-reporting was significantly correlated with clinical urological examinations (Spearman $r = 0.80$) [15]. This suggests a strong concordance between self-reported symptoms and clinical evaluation. Moreover, several additional studies have provided further support for the efficacy of a single-question self-reporting approach in accurately capturing ED, aligning closely with the outcomes obtained from standard questionnaire-based assessments [16, 17]. We decided to put these findings to test in the Indian context. In our study, we aim to investigate the prevalence of ED diagnosed by single-question self-reporting and also to study its association with comorbidities and complications of DM.

To evaluate the prevalence of ED among Indian T2DM patients diagnosed using a single-question self-reporting method (Group A), and also to analyze ED's relationship with comorbidities and complications of diabetes and to compare them with T2DM patients without ED (Group B).

Materials and Methods

This is a retrospective data analysis. Out of the total 6525 males with T2DM who visited the diabetes clinic between 2018 and 2022, 1423 had single-question self-reported ED.

The sample size for the study was initially estimated using MINITAB Version 17 software. Based on a previous study reporting a self-reported ED prevalence of 80.5% [17], we aimed to achieve a margin of error of $\pm 5\%$ with a 95% confidence level, using a binomial probability distribution. The estimated sample size was 294 cases of ED. However,

the actual number of reported ED cases in our study population was significantly higher, at 1423. We included all these reported cases in our study analysis to capture a comprehensive representation of the population under study, thereby increasing the generalizability of the results. For the analysis, we compared the comorbidities and diabetes complications of these 1423 ED cases with an equal number of patients (1423) without ED selected at random from the same patient pool.

ED was diagnosed if the patient reports an "inability to achieve or maintain a satisfactory erection during attempted sexual intercourse" to a single pointed question asked by the physician during their OP visit. The study group (group A) included males with ED and T2DM between the ages of 18 to 85. Additionally, patients who did not have complaints of ED but were on medication for the same were also included in this group. The comparators (Group B) included people with T2DM without ED. Those under 18 and over 85, and those with a history of lower urinary tract, urethral or penile surgery, or pelvic and spine injury/surgery were excluded.

Categorical data were summarized as absolute numbers and percentages, whereas continuous data were summarized as mean and standard deviations (SD). Clinical characteristics were compared using the Student's t-test. Multivariable logistic regression analysis was used to examine the association between dependent and predictive variables with a statistical significance of $p < 0.05$. The presence of ED was taken as the dependent variable and the following as predictive variables: HbA1C (< 7 and ≥ 7 mg/dL), systolic blood pressure (< 140 and ≥ 140 mm Hg), duration of DM (< 10 and ≥ 10 years), BMI (< 23 and ≥ 23 kg/m^2), uric acid (< 7 and ≥ 7 mg/dL), serum creatinine (< 1.2 and ≥ 1.2 mg/dL), e-GFR (< 60 and ≥ 60 ml/min), total cholesterol (< 200 and ≥ 200 mg/dL), triglycerides (< 150 and ≥ 150 mg/dL), low-density lipoproteins (LDL) (< 100 and ≥ 100 mg/dL), high-density lipoproteins (HDL) (< 40 and ≥ 40 mg/dL), SGOT (< 40 and ≥ 40 mg/dL), SGPT(< 40 and ≥ 40 mg/dL), ESR (< 15 and ≥ 15 mm/hr), and urine albumin-creatinine ratio (< 30 and ≥ 30 mg/gm). The odds ratio is calculated to determine the association of comorbid conditions between two groups. The comorbid conditions and complications included in the study were CAD, stroke, peripheral artery disease (PAD), diabetic neuropathy (peripheral and autonomic), diabetic nephropathy, diabetic retinopathy, thyroid dysfunction [hypo (both overt and subclinical), and hyperthyroidism], psychiatric illness, smoking, and alcohol dependence.

Diabetic Retinopathy was diagnosed through direct ophthalmoscopy examination, after pupil dilation [18]. Diabetic Peripheral Neuropathy (DPN) was confirmed by the Vibration Perception Threshold test (VPT) after being diagnosed with the abnormal findings of the Monofilament Test and Ankle reflexes [19]. Diabetic Autonomic Neuropathy (DAN) was diagnosed using an automated R-R interval variation analysis of ECG during deep breathing and Valsalva maneuver, and postural blood pressure testing while lying supine and after 3 min of standing. CAD is diagnosed as having ECG changes suggestive of CAD validated against Minnesota code for Classification System for Electrocardiographic Findings [20], history of MI, abnormal stress test, Echocardiography showing regional wall motion abnormalities suggestive of past MI, presence of significant CAD on coronary angiography ($> 70\%$ stenosis of right coronary artery, left anterior descending artery, and left circumflex artery or $> 50\%$ stenosis of the left main coronary artery), and history of coronary revascularization including percutaneous intervention or coronary artery bypass grafting. Stroke was defined as a history suggestive of symptoms or treatments for stroke, radio imaging, CT or MRI suggestive of old ischemia or hemorrhage. PAD was defined as an ankle-brachial Index (ABI) of ≤ 0.9. Diabetic nephropathy was defined as persistent albuminuria 30–299 mg/24 h and albuminuria ≥ 300 mg/24 h, albumin-creatinine ratio > 30 mg/gm and/or estimated GFR < 60 ml/min in the absence of signs or symptoms of other primary causes of kidney damage. Psychiatric illness was defined as significant changes in thinking, emotion and/or behavior, distress and/or problems functioning in social, work or family activities. Thyroid dysfunction was defined as the presence of hypo or hyperthyroidism based on thyroid function tests. Smokers were defined as someone who has smoked more than 100 cigarettes (including hand-rolled cigarettes, cigars, cigarillos etc.) in their lifetime and has smoked in the last 28 days. Alcohol dependence was defined as consuming more than 3 standard drinks (90 ml) per day.

Results

We collected the data of a total of 2846 males with T2DM; 1423 with ED (group A) and 1423 without ED (group B). The mean age of patients in group A was 56.1 ± 9.39 and 57.6 ± 12.1 for group B and BMI was 26.65 ± 10.24 VS 25.67 ± 6.16. The main patient characteristics are presented in Table 1. The mean ED duration was 2.82 ± 3.55 years. Of the total population (6525), 9.4% of patients were under 40 years of age, 77.2% were between 40–70 years of age, and 13.4% were over 70 years of age and the prevalence of self-reported ED in those groups was found to be 12.6%, 25% and 9% respectively. The overall prevalence of ED was 22% only. 90% of ED cases were found in the 40–70 age group, 5% in the age group < 40 years, and the remaining 5% in the age group > 70 years (Fig. 1).

A multivariable logistic regression analysis was conducted to examine the association between ED and various factors. The results demonstrated that ED was significantly

Table 1 Patient characteristics of two groups

Parameters	Group A (n = 1423)	Group B (n = 1423)	*p* value
Age (years)	56.1 ± 9.39	57.6 ± 12.1	$p = 0.0001$
BMI (kg/m^2)	25.65 ± 10.24	25.67 ± 6.16	$p = 0.9535$
SBP (mm/Hg)	129.75 ± 17.77	130.73 ± 17.84	$p = 0.1426$
Total cholesterol (mg/dL)	191.45 ± 47.14	183.74 ± 43.92	$p < 0.0001$
TGL (mg/dL)	156.18 ± 93.43	150.46 ± 91.60	$p = 0.1530$
HDL (mg/dL)	46.99 ± 20.5	48.23 ± 25.62	$p = 0.2317$
LDL (mg/dL)	115.56 ± 38.51	109.39 ± 41.09	$p = 0.0005$
e-GFR (ml/min)	79.73 ± 23.30	82.48 ± 25.06	$p = 0.0105$
Uric acid (mg/dL)	5.47 ± 4.75	5.30 ± 3.755	$p = 0.3669$
HbA1C (%)	9.03 ± 4.76	8.47 ± 2.25	$p = 0.0001$
SGOT (Units/dL)	31.54 ± 23.38	32.53 ± 16.4	$p = 0.2599$
SGPT (Units/dL)	38.79 ± 25.25	42.57 ± 26.0	$p = 0.0011$
Hemoglobin (gm/dL)	14.31 ± 3.5	14.46 ± 6.3	$p = 0.5357$
ESR (mm/hr)	25.96 ± 19.27	23.79 ± 20.88	$p = 0.0211$
Duration of diabetes (years)	11.35 ± 7.71	13.81 ± 9.08	$p < 0.0001$
Urine albumin excretion (mg/24 h)	62.66 ± 92.92	52.22 ± 66.56	$p = 0.0180$
Albumin creatinine ratio (mg/g)	55.04 ± 66.07	53.78 ± 87.22	$p = 0.7790$

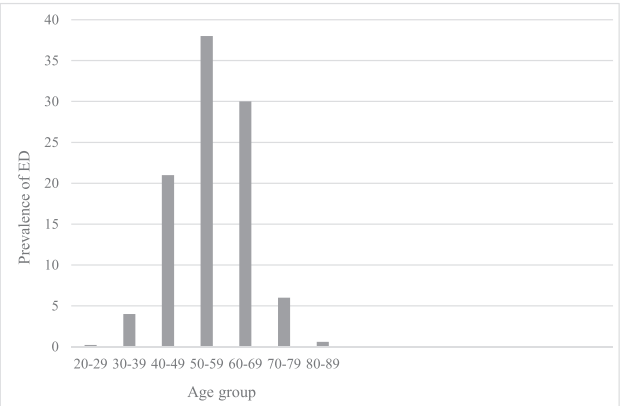

Fig. 1 Prevalence of ED (%) among men of different age groups with T2DM

associated with duration of DM ≥ 10 years [1.2621; 95% CI 1.0866 to 1.4659; $p = 0.0023$], BMI ≥ 23 kg/m^2 [1.2281; 95% CI 1.0313 to 1.4624; $p = 0.0211$], HbA1C ≥ 7% [1.8811; 95% CI 1.5359 to 2.3039; $p < 0.0001$], Total cholesterol ≥ 200 mg/dL [1.4754; 95% CI 1.2481 to 1.7440; $p < 0.0001$], TGL ≥ 150 mg/dL [1.2023; 95% CI 1.0106 to 1.4305; $p = 0.0376$], LDL ≥ 100 mg/dL [1.4134; 95%CI 1.1845 to 1.6866; $p = 0.0001$]; ESR ≥ 15 mm/hr [1.5490; 95% CI 1.2823 to 1.8711; $p < 0.0001$], e-GFR < 60 ml/min [1.2529; 95% CI 1.0062 to 1.5601; $p = 0.0439$] and urine albumin-creatinine ratio ≥ 30 mg/gm [1.3526; 95% CI 1.0790 to 1.6955; $p = 0.0088$] (Table 2).

When comorbid conditions were compared in Groups A and B, ED was found to be significantly associated with CAD [1.5881; 95% CI 1.236 to 2.041; ($p = 0.0003$)], DAN [4.1504; 95% CI 2.894 to 5.953; ($p < 0.0001$)], diabetic retinopathy [1.5473; 95% CI 1.266 to 1.891; ($p < 0.0001$)], hypothyroidism (both overt and subclinical) [1.3640; 95% CI 1.0056 to 1.8502 ($p = 0.0460$)], smoking 2.2111;95% CI 1.7575 to 2.7816 ($p < 0.0001$)], alcohol dependence [2.0018; 95% CI 1.6213 to 2.4716 ($p < 0.0001$)] and combined smoking and alcohol dependence [5.9178; 95% CI 4.749 to 7.373 ($p < 0.0001$)] (Table 3).

Discussion

Sexual dysfunction is a well-known complication of DM and a significant number of males with DM suffer from ED. In our retrospective study, we utilized self-reported cases of ED to analyze cardiovascular risk factors, diabetic complications, and comorbidities. This approach was necessitated by the practical constraints faced in clinical settings in India, where extensive questionnaire-based assessments are not commonly conducted due to time limitations. Moreover, numerous studies have demonstrated that single-question self-reported ED correlates accurately with clinical diagnoses and questionnaire-based evaluations [15–17]. Nonetheless, a significant disparity emerged in the prevalence of self-reported ED when compared to studies utilizing standard questionnaires. Our study identified an overall

Table 2 Multivariable logistic regression analysis of risk factors for ED

Variables	Odds ratio	*p* value	95% Confidence Interval for Odds-Ratio	
			Lower	Upper
HbA1C (%)	1.8811	**< 0.0001***	1.5359	2.3039
Systolic Blood Pressure (mmHg)	1.1579	0.0675	0.9895	1.355
Duration of DM (years)	1.2621	**0.0023***	1.0866	1.4659
BMI (kg/m2)	1.2281	**0.0211***	1.0313	1.4624
Total cholesterol (mg/dL)	1.4754	**< 0.0001***	1.2481	1.7440
TGL (mg/dL)	1.2023	**0.0376***	1.0106	1.4305
HDL (mg/dL)	1.0076	0.9425	0.8207	1.2370
LDL (mg/dL)	1.4134	**0.0001***	1.1845	1.6866
Uric acid (mg/dL)	0.9979	0.7321	1.3601	0.9893
SGOT (mg/dL)	0.6676	**0.0005***	0.5309	0.8396
SGPT (mg/dL)	0.7484	**0.0019***	0.6235	0.8923
ESR (mm/hr)	1.5490	**< 0.0001***	1.2823	1.8711
e-GFR (mL/min)	1.2529	**0.0439***	1.0062	1.5601
Urine albumin-creatinine ratio (mg/gm)	1.3526	**0.0088***	1.0790	1.6955

* = p value < 0.05

Table 3 Comorbidities showing significant association with ED

Comorbid factors	Group A (n)	Group B (n)	Odds ratio; 95% CI [p value]
CAD	170	112	1.5881; 1.236 to 2.041 [**$p = 0.0003$***]
Stroke	15	10	1.5053; 0.674 to 3.362 [$p = 0.3185$]
Peripheral Vascular Disease	42	30	1.4122; 0.879 to 2.269 [$p = 0.1539$]
Diabetic Peripheral Neuropathy	669	696	0.9268; 0.800 to 1.074 [$p = 0.3111$]
Diabetic Autonomic neuropathy	149	39	4.1504; 2.894 to 5.953 [**$p < 0.0001$***]
Diabetic Retinopathy	278	193	1.5473; 1.266 to 1.891 [**$p < 0.0001$***]
Diabetic Nephropathy	415	588	0.5847; 0.5004 to 0.6831 [**$p < 0.0001$***]
Psychiatric illness	70	93	0.7415; 0.5389 to 1.0204 [$p = 0.0664$]
Hypothyroidism	103	77	1.3640; 1.0056 to 1.8502 [**$p = 0.0460$***]
Hyperthyroidism	10	3	3.3498; 0.9200 to 12.1977 [$p = 0.0667$]
Smoking	248	124	2.2111; 1.7575 to 2.7816 [**$p < 0.0001$***]
Alcohol dependence	283	157	2.0018; 1.6213 to 2.4716 [**$p < 0.0001$***]
Combined smoking and alcohol dependence	487	115	5.9178; 4.749 to 7.373 [**$p < 0.0001$***]

* = p-value < 0.05

prevalence of 22%, which is notably lower than the general prevalence (35 to 90%) reported in studies relying on standard questionnaire-based assessments [7–9]. This discrepancy can be attributed to variations in definitions, study designs, diagnostic methods and sociocultural factors such as reluctance to openly discuss sexual matters.

The incidence of sexual problems increases with age, driven primarily by comorbid conditions associated with aging. The influence of age on the prevalence of ED is well-established in men with and without T2DM [21, 22]. For instance, the Massachusetts Male Aging Study revealed that over half of men aged 40–70 with diabetes experience ED, with prevalence rising with age and DM duration [1]. Psychosocial factors play a more significant role in younger men with ED, while systemic factors become more predominant as age increases. Consequently, a psychosocial approach is warranted for younger individuals with ED, whereas older individuals with ED should receive attention focused on chronic diseases [23].

In our study, we found that the single-question self-reported prevalence of ED among the DM population was 12.6% in men under 40, 25% in those aged 40–70, and 9% in those over 70 years old. Though previous studies have reported higher prevalence among aging population [7, 24], the lower prevalence in that age group in our study is most likely a result of lower self-reporting of ED due to socio-cultural inhibitions inherent in Indian society for openly discussing matters related to sex.

Our study showed the duration of DM of ten or more years is significantly associated with ED which was in concordance with earlier reports [25–27]. ED is very prevalent in men with DM and obesity [28]. A pro-inflammatory state associated with insulin resistance and visceral adiposity leads to a decreased availability and activity of NO in overweight and obese diabetic men, resulting in ED [29]. In the present study, BMI ≥ 23 was found to be significantly associated with ED. The association of high BMI and ED suggests that there is a potential for reversal of ED with significant weight loss. We found a significant association between elevated HbA1C (≥ 7%) and ED, which is consistent with findings from some previously reported studies [2, 30]. No relationship was found between systemic hypertension and ED in our study whereas others had reported significant association between these two. It was found that every 10 mmHg increase in systolic blood pressure above normal significantly increases the risk of ED [31]. But few studies showed contradictory results [31, 32]. An abnormal increase in LDL and TGL promotes endothelial dysfunction and atherosclerosis and correlates with the severity of arteriogenic ED [33, 34]. Our study found that ED was positively associated with total cholesterol ≥ 200 mg/dL, TGL ≥ 150 mg/dL and LDL ≥ 100 mg/dL. This was supported by earlier findings that dyslipidemia was an independent risk factor for ED and conversely that people with ED and DM had a 2.3-fold higher risk for dyslipidemia [35, 36]. Research has shown that men with elevated levels of inflammatory markers are at an increased risk of developing ED [37]. Consistent with previous findings, our study also demonstrated a significant association between ED and ESR levels ≥ 15 mm/hr.

The incidence of ED in people with DM is found to increase with the number and severity of complications and co-morbidities [1]. Both microvascular and macrovascular DM complications increase the risk of ED in men with DM. An association between overt CAD and ED was shown in various studies [38–40]. The presence of atherosclerotic plaques can lead to arterial ED, which can be a marker of CAD. Since the penile arteries are smaller in diameter than the coronary arteries, arteriosclerosis will first show its effects in the small arteries like penile arteries earlier than larger coronary arteries and before clinical manifestations of ischemic heart disease. There is an eightfold greater prevalence of ED among patients with silent myocardial ischemia than those without [41] and ED can precede the onset of CAD by 2–5 years [42]. Our study revealed a strong association between ED and the presence of CAD in people with T2DM. This suggests that individuals with ED and T2DM may be more susceptible to developing cardiovascular diseases (CVD). Consequently, addressing ED in individuals with T2DM could contribute to reducing the risk of atherovascular diseases as well. It also suggests that diagnosis of ED can be used as a strong clinical tool for improving the early detection and prevention of atherosclerotic vascular disease which can aid in the aggressive management of CVD and associated risk factors.

In our study, we found an association between ED and Diabetic Retinopathy. This could be due to generalized endothelial dysfunction characterized by reduced vasodilatation in response to stimuli, pro-coagulation, inflammation, and arterial stiffness. ED is a well-established marker of diffuse endotheliopathy, a condition common to DM and the pathophysiological pathway for the development of diabetic retinopathy [43]. Henis O et al. also reported that ED is associated with severe retinopathy in men with diabetes regardless of DM duration, macrovascular comorbidities, or CV risk factors [44]. The frequency of sexual dysfunction increases as renal function deteriorates [45]. In our study, the presence of diabetic nephropathy was significantly associated with ED. Also, ED was found to be independently associated with e-GFR < 60 ml/min, and albumin creatinine ratio ≥ 30 mg/gm. Studies showed that the severity of ED is directly proportional to that of diabetic nephropathy [46]. In the later stage of CKD uremia also contributes to ED through multiple mechanisms. Studies have reported that DAN was very frequent in DM men with ED [27, 47]. Our study also revealed a four-fold increased Odds of having ED in patients with DAN. No association was found between ED and DPN without DAN in our study. However, some studies demonstrated a relationship between ED and DPN [48].

Our study found a significant correlation between ED and hypothyroidism (both overt and subclinical). However, statistical significance was not found for hyperthyroidism, despite the higher odds, which might have been due to a

lower number of cases. Previous research by Chen D et al. [49] also reported a positive association between subclinical hypothyroidism and ED, while Krassas GE et al. noted that ED was highly prevalent in men affected by both hyper and hypothyroidism [50]. Furthermore, our study revealed higher odds for stroke and PAD, akin to hyperthyroidism, although statistical significance was not reached, probably due to the lower number of cases. Previous research has provided evidence of a higher prevalence of ED in post-stroke patients [51]. Additionally, ED has been identified as an independent predictor of PAD, with the severity of ED correlating with an increased prevalence of PAD [52].

No correlation was observed between psychiatric illness and ED in our study even though various previous studies have reported a significant association between psychiatric disorders like depression and ED in people with DM [53]. Among lifestyle factors, a two-fold increase in the Odds of having ED was found in men with either cigarette smoking or alcohol dependence in our study. These results are well supported by Al Hunayan et al. [54], Mirone V et al. [55] and Bortolotti A et al. [56]. At the same time, the Odds were five-fold higher in those with both cigarette smoking and alcohol dependence which is more than the additive values of the individual ones which shows that smoking and alcohol work synergistically in the causation of ED.

Our study has reaffirmed the significant associations between ED and a range of factors, including the duration of T2DM, obesity, CAD, atherovascular risk factors, microvascular complications such as retinopathy and nephropathy, and also with autonomic neuropathy, smoking, alcohol dependence, and hypothyroidism. Notably, all these factors are already known to be correlated with increased CV morbidity and mortality. This convergence of ED and CV risk factors suggests that ED may serve as an important marker of underlying CV risk. Consequently, when an individual is diagnosed with ED, whether through patient self-reporting or questionnaire-based assessments, it becomes imperative to take a proactive approach. This includes subjecting the individual to a comprehensive vascular risk analysis and appropriate management.

Indeed, it is a well-established fact that hyperglycemia, dyslipidemia, and hypertension are notable risk factors for atherosclerotic vascular disease. However, even among patients receiving treatment for these conditions, only about a third manage to reach the recommended target levels, leaving a substantial residual risk. Particularly, when lipid or blood pressure levels are only mildly elevated and patients are asymptomatic, they may resist medication, and physicians may underestimate its necessity. This oversight results in unaddressed residual CV risk in DM patients. In such circumstances, a clinical condition suggesting significant CV risk, like ED, which is known to precede clinical CAD by 2 to 5 years, can be valuable in making decisions regarding whether intensive management is required.

However, Indian physicians confront substantial time constraints that hinder their ability to conduct extensive questionnaire-based evaluations in the diagnosis of ED. Consequently, they often rely on self-reporting by patients as a practical approach. However, this reliance on self-reporting carries the risk of underdiagnosis, potentially leading to the oversight of ED and its associated CV consequences. Additionally, depending solely on self-reporting may lead to the underdetection of mild cases of ED. Certain patients may consider mild ED less significant and may not deem it necessary to report until it becomes more severe. As our study is based on retrospective data analysis, only a well-designed prospective study can address many of the ambiguities in our findings, such as the lower reporting of ED in the elderly.

Our study indicates that relying solely on self-reporting of ED may significantly under-represent its prevalence among people with diabetes. This can in turn lead to missed diagnoses, overlooking potential cardiovascular risk factors associated with ED, and missed opportunities to address the issue effectively. Therefore, there is an urgent need for an intermediate diagnostic approach- a combination of a time-saver and an efficient tool that can assist Indian physicians in identifying as many ED cases as possible. We suggest that screening of all male DM patients in the reproductive age group at least once a year using one of the standard questionnaires like BMSFI, IIEF or MMAS should become a routine clinical practice in diabetes care. This approach can save physicians time while ensuring that no one is overlooked in the evaluation for ED.

The limitations of our study include its retrospective single-center design and the reliance on single-question self-reporting to identify cases of ED. Despite these limitations, our study stands out due to the inclusion of a larger number of cases and the comprehensive analysis of all possible comorbidities and complications of DM.

Conclusion

Our study underscores ED as a potential marker of underlying CV risk, signifying associations with various factors. However, reliance solely on self-reporting may lead to underdiagnosis and thus overlook cardiovascular consequences. Annual screenings with standardized questionnaires like BMSFI, IIEF, or MMAS are recommended for efficient diagnosis and treatment.

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Data Availability The data that support the findings of this study are available from the corresponding author upon reasonable request.

Declarations

Ethical approval The study protocol was approved by the Institutional Ethics Committee of Dr. Suresh's DiabcareIndia. The study was performed in accordance with the ethical standards as laid down in the 1964 Declaration of Helsinki and its later amendments or comparable ethical standards.

Conflicts of interest The authors certify that they have no affiliations with or involvement in any organization or entity with any financial interest in the subject matter or materials discussed in this manuscript.

References

1. Levy J. Impotence and its medical and psychosocial correlates: results of the Massachusetts Male Aging Study. Br J Diabetes Vasc Dis. 2002;2(4):278–80. https://doi.org/10.1177/14746514020020040801.
2. Giugliano F, Maiorino M, Bellastella G, Gicchino M, Giugliano D, Esposito K. Determinants of erectile dysfunction in type 2 diabetes. Int J Impot Res. 2010;22(3):204–9.
3. Kouidrat Y, Pizzol D, Cosco T, Thompson T, Carnaghi M, Bertoldo A, Solmi M, Stubbs B, Veronese N. High prevalence of erectile dysfunction in diabetes: a systematic review and meta-analysis of 145 studies. Diabet Med. 2017;34(9):1185–92. https://doi.org/10.1111/dme.13403.
4. Nehra A, Moreland RB. Neurologic erectile dysfunction. Urol Clin North Am. 2001;28(2):289–308.
5. Sáenz de Tejada I, Angulo J, Cellek S, et al. Pathophysiology of erectile dysfunction. J Sex Med. 2005;2(1):26–39.
6. Moore CR, Wang R. Pathophysiology and treatment of diabetic erectile dysfunction. Asian J Androl. 2006;8(6):675–84. https://doi.org/10.1111/j.1745-7262.2006.00223.x.
7. Sharifi F, Asghari M, Jaberi Y, Salehi O, Mirzamohammadi F. Independent predictors of erectile dysfunction in type 2 Diabetes mellitus: Is it true what they say about risk factors? ISRN Endocrinol. 2012;2012:502353. https://doi.org/10.5402/2012/502353.
8. Díaz-Díaz E, Cárdenas LM, Olivares Arzuaga N, Timossi CM, Gómez Díaz RA, Aguilar Salinas C, et al. Erectile dysfunction: a chronic complication of the diabetes mellitus. In: Nunes KP, editor. Erectile dysfunction - disease-associated mechanisms and novel insights into therapy. Rijeka: IntechOpen; 2012. https://doi.org/10.5772/31005.
9. Nicolosi A, Moreira ED, Shirai M, Bin MI, Glasser DB. Epidemiology of erectile dysfunction in four countries:Cross-national study of the prevalence and correlates of erectile dysfunction. Urology. 2003;61:201–6.
10. Seid A, Gerensea H, Tarko S, Zenebe Y, Mezemir R. Prevalence and determinants of erectile dysfunction among diabetic patients attending in hospitals of central and northwestern zone of Tigray, northern Ethiopia: A cross-sectional study. BMC Endoc Disord. 2017;17(1):16. https://doi.org/10.1186/s12902-017-0167-5.
11. Chung CM, Lu MZ, Wong CY, Goh SG, Azhar MI, Lim YM, et al. The SAD–MEN questionnaire: A new and reliable questionnaire for assessing sexual dysfunction in Asians with diabetes. Diab Med. 2016;33(5):674–80. https://doi.org/10.1111/dme.12864.
12. O'Leary MP, Fowler FJ, Lenderking WR, Barber B, Sagnier PP, Guess HA, Barry MJ. A brief male sexual function inventory for urology. Urology. 1995;46(5):697–706. https://doi.org/10.1016/S0090-4295(99)80304-5.
13. Rosen RC, Riley A, Wagner G, Osterloh IH, Kirkpatrick J, Mishra A. The International Index of Erectile Function (IIEF): A multidimensional scale for assessment of erectile dysfunction. Urology. 1997;49(6):822–30. https://doi.org/10.1016/S0090-4295(97)00238-0.
14. Kleinman KP, Feldman HA, Johannes CB, Derby CA, McKinlay JB. A new surrogate variable for erectile dysfunction status in the Massachusetts Male Aging Study. J Clin Epidemiol. 2000;53(1):71–8. https://doi.org/10.1016/S0895-4356(99)00150-X.
15 O'donnell AB, Araujo AB, Goldstein I, McKinlay JB. The validity of a single-question self-report of erectile dysfunction: Results from the Massachusetts Male Aging Study. J Gen Intern Med. 2005;20(6):515–9. https://doi.org/10.1111/j.1525-1497.2005.0076.x.
16. Derby CA, Araujo A, Johannes C, Feldman HA, McKinlay JB. Measurement of erectile dysfunction in population-based studies: The use of a single question self-assessment in the Massachusetts Male Aging Study. Int J Impotence Res. 2000;12(4):197. https://doi.org/10.1038/sj.ijir.3900542.
17. Batais MA, Mujammami MH, ldossari KK, et al. The validity of using a single-question self-report of erectile dysfunction as a screening tool among men with type 2 diabetes mellitus. J Mens Health. 2020;16(4):e18–27. https://doi.org/10.31083/jomh.v16i4.286.
18 Ansari P, Tabasumma N, Snigdha NN, Siam NH, Panduru RVNRS, Azam S, Hannan JMA, Abdel-Wahab YHA. Diabetic retinopathy: an overview on mechanisms, pathophysiology and pharmacotherapy. Diabetology. 2022;3:159–75. https://doi.org/10.3390/diabetology3010011.
19. Kasznicki J. Advances in the diagnosis and management of diabetic distal symmetric polyneuropathy. Arch Med Sci. 2014. https://doi.org/10.5114/aoms.2014.42588.
20. Prineas RJ, Crow RS, Zhang Z-M. The Minnesota code manual of electrocardiographic findings. London: Springer; 2010. XIII, 328 pp. https://doi.org/10.1007/978-1-84882-778-3.
21. Shiferaw WS, Akalu TY, Petrucka PM, Areri HA, Aynalem YA. Risk factors of erectile dysfunction among diabetes patients in Africa: A systematic review and meta-analysis. J Clin Transl Endocrinol. 2020;3(21):100232. https://doi.org/10.1016/j.jcte.2020.100232.
22. Kumar R, Kumar U, Trivedi S. Comparison of risk factors for erectile dysfunction (ED) in type 2 diabetics and nondiabetics: a retrospective observational study. Cureus. 2023;15(9):e44576. https://doi.org/10.7759/cureus.44576.
23. Song WH, Park J, Yoo S, Oh S, Cho SY, Cho MC, et al. Changes in the prevalence and risk factors of erectile dysfunction during a decade: The Korean Internet Sexuality Survey (KISS), a 10-year-interval web-based survey. World J Mens Health. 2019;37(2):199–209. https://doi.org/10.5534/wjmh.180054.
24. Shi MD, Chao JK, Ma MC, Chiang SK, Chao IC. The connection between type 2 diabetes and erectile dysfunction in Taiwanese aboriginal males. Int J Impot Res. 2014;26:235–40. https://doi.org/10.1038/ijir.2014.26.
25. Walle B, Lebeta KR, Fita YD, Abdissa HG. Prevalence of erectile dysfunction and associated factors among diabetic men attending the diabetic clinic at Felege Hiwot Referral Hospital, Bahir Dar, North West Ethiopia, 2016. BMC Res Notes. 2018;11(1):130. https://doi.org/10.1186/s13104-018-3211-2.
26. Wing RR, Rosen RC, Fava JL, Bahnson J, Brancati F, Gendrano IN III, et al. Effects of weight loss intervention on erectile function in older men with type 2 diabetes in the look AHEAD trial. J Sex Med. 2009;6:1414–22.

27. Cho NH, Ahn CW, Park JY, Ahn TY, Lee HW, Park TS, et al. Prevalence of erectile dysfunction in Korean men with type 2 diabetes mellitus. Diabet Med. 2006;23:198–203.
28. Tamler R. Diabetes, obesity, and erectile dysfunction. Gend Med. 2009;6(Suppl 1):4–16. https://doi.org/10.1016/j.genm.2008.12.003.
29. Esposito K, Giugliano D. Obesity, the metabolic syndrome, and sexual dysfunction in men. Clin Pharmacol Ther. 2011;90(1):169–73.
30. Penson DF, Latini DM, Lubeck DP, Wallace KL, Henning JM, Lue TF. Comprehensive Evaluation of Erectile Dysfunction (ExCEED) database Do impotent men with diabetes have more severe erectile dysfunction and worse quality of life than the general population of impotent patients? Results from the Exploratory Comprehensive Evaluation of Erectile Dysfunction (ExCEED) database. Diabetes Care. 2003;26(4):1093–9.
31. Sarma AV, Hotaling JM, de Boer IH, et al. Blood pressure, antihypertensive medication use, and risk of erectile dysfunction in men with type I diabetes. J Hypertens. 2019;37(5):1070–6.
32. Joseph P, Lonn E, Bosch J, et al. Long-term effects of statins, blood pressure-lowering, and both on erectile function in persons at intermediate risk for cardiovascular disease: a substudy of the heart outcomes prevention evaluation-3 (HOPE-3) randomized controlled trial. Can J Cardiol. 2018;34(1):38–44.
33 Silverman MG, Ference BA, Im K, et al. Association between lowering LDL-C and cardiovascular risk reduction among different therapeutic interventions: a systematic review and meta-analysis. JAMA. 2016;316(12):1289–97.
34. Gu X, Yang X, Li Y, et al. Usefulness of low-density lipoprotein cholesterol and non-high-density lipoprotein cholesterol as predictors of cardiovascular disease in Chinese. Am J Cardiol. 2015;116(7):1063–70.
35. Ishikawa K, Sasaki H, Tanifuji S, et al. Lipid abnormality, current diabetes and age affect erectile hardness: an analysis of data from complete medical checkups carried out at a single hospital. Int J Urol. 2020;27(4):339–43.
36. Azad AK, Setunge S, Selim S, et al. Dyslipidaemia as a risk factor for erectile dysfunction in type 2 diabetes mellitus patients. Diabetes Metab Syndr. 2019;13(1):748–53.
37. Esposito K, Giugliano F, Martedì E, Feola G, Marfella R, D'Armiento M, Giugliano D. High proportions of erectile dysfunction in men with the metabolic syndrome. Diabetes Care. 2005;28(5):1201–3. https://doi.org/10.2337/diacare.28.5.1201.
38. Fedele D, Bortolotti A, Coscelli C, et al. on behalf of Gruppo Italiano Studio Deficit Erettile nei Diabetici. Erectile dysfunction in Type 1 and Type 2 diabetics in Italy. Int J Epidemiol. 2000;29:524–31.
39. Yannas D, Frizza F, Vignozzi L, Corona G, Maggi M, Rastrelli G. Erectile dysfunction is a hallmark of cardiovascular disease: unavoidable matter of fact or opportunity to improve men's health? J Clin Med. 2021;10(10):2221. https://doi.org/10.3390/jcm10102221.
40. Montorsi F, Briganti A, Salonia A, Rigatti P, Margonato A, Macchi A, Galli S, Ravagnani PM, Montorsi P. Erectile dysfunction prevalence, time of onset and association with risk factors in 300 consecutive patients with acute chest pain and angiographically documented coronary artery disease. Eur Urol. 2003;44:360–5. https://doi.org/10.1016/S0302-2838(03)00305-1.
41. Gazzaruso C, Giordanetti S, De Amici E, Bertone G, Falcone C, Geroldi D, Fratino P, Solerte SB, Garzaniti A. Relationship between erectile dysfunction and silent myocardial ischemia in apparently uncomplicated type 2 diabetic patients. Circulation. 2004;110(1):22–6. https://doi.org/10.1161/01.CIR.0000133278.81226.C9.
42. Foresta C, Ferlin A, Lenzi A, Montorsi P, Italian Study Group on Cardiometabolic A. The great opportunity of the andrological patient: cardiovascular and metabolic risk assessment and prevention. Andrologia. 2017;5(3):408–13.
43. Chew SK, Taouk Y, Xie J, Nicolaou TE, Wang JJ, Wong TY, Lamoureux EL. Relationship between diabetic retinopathy, diabetic macular oedema and erectile dysfunction in type 2 diabetics. Clin Exp Ophthalmol. 2013;41(7):683–9. https://doi.org/10.1111/ceo.12099.
44. Henis O, Shahar Y, Steinvil A, et al. Erectile dysfunction is associated with severe retinopathy in diabetic men. Urology. 2011;77:1133–6.
45. Bellinghieri G, Santoro D, Mallamace A, Savica V. Sexual dysfunction in chronic renal failure. J Nephrol. 2008;21(Suppl. 13):S113–7.
46. Costa MR, Ponciano VC, Costa TR, de Oliveira AM, Gomes CP, de Oliveira EC. Prevalence and factors associated with erectile dysfunction in patients with chronic kidney disease on conservative treatment. Int J Impot Res. 2017;29(6):219–24.
47. Vinik AI, Maser RE, Mitchell BD, Freeman R. Diabetic autonomic neuropathy. Diabetes Care. 2003;26(5):1553–79.
48. Defeudis G, Mazzilli R, Tenuta M, Rossini G, Zamponi V, Olana S, Faggiano A, Pozzilli P, Isidori AM, Gianfrilli D. Erectile dysfunction and diabetes: A melting pot of circumstances and treatments. Diabetes Metab Res Rev. 2022;38(2):e3494. https://doi.org/10.1002/dmrr.3494.
49. Chen D, Yan Y, Huang H, Dong Q, Tian H. The association between subclinical hypothyroidism and erectile dysfunction. Pak J Med Sci. 2018;34(3):621–5. https://doi.org/10.12669/pjms.343.14330.
50. Krassas GE, Tziomalos K, Papadopoulou F, Pontikides N, Perros P. Erectile dysfunction in patients with hyper- and hypothyroidism: how common and should we treat? J Clin Endocrinol Metab. 2008;93(5):1815–9. https://doi.org/10.1210/jc.2007-2259.
51. Zhao S, Wu W, Wu P, Ding C, Xiao B, Xu Z, Hu Y, Shen M, Feng L. Significant increase of erectile dysfunction in men with post-stroke: a comprehensive review. Front Neurol. 2021;28(12):671738. https://doi.org/10.3389/fneur.2021.671738.
52. Polonsky TS, Taillon LA, Sheth H, Min JK, Archer SL, Ward RP. The association between erectile dysfunction and peripheral arterial disease as determined by screening ankle-brachial index testing. Atherosclerosis. 2009;207(2):440–4. https://doi.org/10.1016/j.atherosclerosis.2009.05.005.
53. Wang X, Yang X, Cai Y, Wang S, Weng W. High prevalence of erectile dysfunction in diabetic men with depressive symptoms: a meta-analysis. J Sex Med. 2018;15(7):935–41.
54. Al-Hunayan A, Al-Mutar M, Kehinde EO, Thalib L, Al-Ghorory M. The prevalence and predictors of erectile dysfunction in men with newly diagnosed type 2 diabetes mellitus. BIU Int. 2007;99:130–4.
55. Mirone V, Ricci E, Gentile V, BasileFasolo C, Parazzini F. Determinants of erectile dysfunction risk in a large series of Italian men attending andrology clinics. Eur Urol. 2004;45:87–91.
56. Bortolotti A, Fedele D, Chatenoud L, et al. Cigarette smoking: a risk factor for erectile dysfunction in diabetics. Eur Urol. 2001;40(4):392–6 discussion 397.