



## Pattern of erectile dysfunction in diabetic patients

Essam-Elden Mohamed Mohamed<sup>1</sup>, Khaled Mohamed Tawfik<sup>2</sup>, Abdullah Hashim Ahmed<sup>3</sup>

<sup>1-3</sup> Department of Dermatology, Venereology and Andrology, Faculty of Medicine, Al-Azhar University, Assuit, Egypt

### Abstract

**Objective:** To evaluate pattern of erectile dysfunction (ED) in diabetic patients, common types and risk factors of it.

**Patient's and Methods:** 102 male diabetic patients complaining from ED were subjected to complete history taking including age and smoking and sexual history including the onset of ED and onset and duration of diabetes mellitus (DM), general examination including blood pressure and the body mass index (BMI), the genital examination including the Penis: (size, girth and peyronei's) and genital reflexes. Investigation including Hormonal assay of Serum Total and free testosterone and lipid profile. A Penile duplex was done to all patients.

**Results:** the mean age of patients studied was  $52.03 \pm 10.79$  years, mean duration of diabetes was  $8.45 \pm 6.45$  years, 24 patients with DM type 1 and 78 with type 2 DM. According to pattern of ED; Psychogenic ED are present in 16 patients and organic ED in 86, organic ED types are 34 of arterial nature, 28 patients of venous nature, and mixed vascular in 17 patients and a neurogenic in 7 patients. Relation between Hypertension (HTN), Hyperlipidemia and Pattern of ED showing statistical significance.

**Conclusion:** the main pattern of erectile dysfunction in DM is organic in nature and the cavernous arterial insufficiency is closely related to the DM. Hypertension and hyperlipidemia is strongly associated with erectile dysfunction in diabetics.

**Keywords:** erectile dysfunction, diabetic impotence, penile duplex

### Introduction

Erectile dysfunction (ED) is defined as the continuous inability to maintain or achieve a penile erection (PE) enough for satisfactory sexual performance <sup>[1]</sup>.

DM is considered the main risk factor for the development of erectile dysfunction and since the 1970s the association between diabetes and the development of erectile dysfunction has been documented both in animal models and humans <sup>[2]</sup>.

Although DM has been considered an independent risk factor for ED, the etiology of sexual complications in DM is multifactorial and associated with organic, psychological, and relational cause <sup>[3]</sup>.

Although erectile dysfunction is considered an age-related disease, it can be present across all the life-span from adolescence, especially when risk factors such as diabetes, metabolic syndrome or cardiovascular diseases coexist <sup>[4]</sup>.

Epidemiological studies suggest that both type 1 and type 2 diabetes are associated with an increased risk of ED, as compared with the general population <sup>[5]</sup>.

Type 2 diabetes is commonly associated with overweight and obesity, metabolic syndrome, hypertension, hyperlipidemia and sedentary lifestyle, which are themselves risk factors for ED <sup>[6, 7]</sup>.

The pathogenesis of ED in diabetic men is a multifactorial phenomenon in which vasculopathy, neuropathy, visceral adiposity, insulin resistance, and hypogonadism play roles <sup>[8]</sup>.

Chronic hyperglycemia can result in endothelial dysfunction <sup>[9]</sup> mainly because of the decreased bioavailability of nitric oxide, resulting in insufficient relaxation of the vascular smooth muscles of the corpora cavernosa leading to imbalance between the vasoconstrictor and vasorelaxant pathways,

favoring vasoconstriction <sup>[10]</sup>.

Somatic and autonomic neuropathies also contribute to diabetic ED owing to decreased sensory impulses from the penis to the reflexogenic erectile center <sup>[11]</sup>.

Hormonal factors also play role in the development of ED in diabetic men. Testosterone concentrations tend to decrease in association with low levels of luteinizing hormone and follicle stimulating hormone <sup>[12]</sup>.

### Patients and Methods

The study was approved by the local Ethics Committee of the Faculty of Medicine, Al-Azhar University, Assiut, Egypt. Informed consent was obtained from all cases.

### Patients

One hundred and two male diabetic patients complaining from erectile dysfunction who attended to the Andrology outpatient clinic of Al-Azhar University Hospital (Assiut) between May 2017 and May 2018 were included in this prospective study.

### Methods

All patients were subjected to the following

#### 1. Complete history taking

Included patients' personal history: name, age, marital status (single, married, divorced, widow), residence, Occupation, Number of Children and Smoking index (non-smoker, light/moderate or heavy smoker)

Sexual history: Impotence (onset, course and duration), Libido, Morning erection, Frequency of intercourse, Pain during erection, Deviation during erection and Ejaculation (premature, normal or delayed)

History of medical diseases (hypertension, Undescended testis, hypothyroidism, hyperthyroidism, Epididymorchitis, fever), drug intake (diuretics, anti-depressant, anti-psychotics, anti-thyroid agents, anticonvulsants and anti-androgen as Gn RH analogues and antagonists), trauma or surgical operation. Diabetic history: onset, course, duration, family history of the disease, type of diabetes, medication for diabetes and presence or absence of complication.

**2. Examination**

**General Examination included**

- Vital signs
- Gynecomastia (absent or present)
- Hair distribution
- Body mass index: [BMI = weight (kg) /height (m)2]: normal (BMI between 18.5 and 25), overweight(BMI between 25and 29.9), and obese (BMI >29.9)
- Frontal baldness

**Local Examination included:**

- Penis: (size, girth and peyronei's)
- Presence and distribution of hair
- Genital reflexes
- Testes: (size, tenderness and varicosity)
- Epididymis (tenderness and varicosity)
- Spermatic cord and scrotum

**3. Investigation**

**Hormonal and Biochemical assay**

A Three ml of blood is collected and we measure:

- **Serum Total and free testosterone**  
Serum total testosterone (TT) was measured using the electrochemiluminescence immunoassay (ELISA). As there is a circadian variation in testosterone release, men with ED should have serum testosterone measured on a blood fasting sample taken in the morning between 8-11 AM, total serum testosterone above 12 nmol/l is considered normal and for free testosterone: lower limit of 225 pmol/l is accepted as normal
- **Serum Estradiol**
- **Serum Prolactin**  
Serum prolactin was measured after a rest of at least 20 min; hyperprolactinaemia considered with prolactin level above 20 ng/ml
- **Lipid profile (Triglycerides, total cholesterol, HDL and LDL)**  
Patients had to fast for 9–12 hours before the test. Normal values of triglycerides (TG) are less than 150mg/dL. The reference range of high-density lipoprotein (HDL) is 40-50 mg/dL. Total cholesterol < 190mg % and low-density lipoprotein < 115mg % was taken as standard.
- **HBA1C and Fasting blood glucose and Post Prandial blood glucose.**  
Fasting (FBS) and post-prandial (PPBS) blood sugar were measured using the colorimetric. Glycated haemoglobin (HbA1c) was quantitatively assessed using latex immunoagglutination inhibition methodology

**Penile duplex**

All the examinations were performed on GE Voluson S6 and

GE Logiq P5 devices (GE Medical Systems, Milwaukee, WI, USA), with a high-frequency probe and the availability of color Doppler ultrasonography. Grayscale ultrasonography was performed in the transverse and the longitudinal sections to look for any abnormality. Following this baseline, the velocities of the right and the left cavernosal arteries were recorded before the injection. Spectral waveforms from the cavernosal arteries were measured at the base of the penis because it is the location with the highest velocities and the optimum angle correction.

Patients with a peak systolic velocity of less than 25 cm/s were considered to have arterial insufficiency. An end diastolic velocity of more than 5 cm/s was considered to indicate venous incompetence.

**Statistical analysis**

SPSS version 22.0 program for windows was used for data processing. Data are presented and expressed in tables as mean ± standard deviation (SD), frequency, and percentage. P-values less than 0.05 were considered significant.

**Results**

All the 102 patients included in the study were above the age of 28 years and less than 75 years. Among them the majority was in the age of 46-60 years (47.1%). followed by the age of 28-45 years (27.5%). The duration of the disease was from less than 1year to more than 19 years

**Table 1:** Demographic data of the studied patients (n=102).

Age (Years)	
(Range) Mean ± SD	(28-75) 52.03± 10.79
Age groups: n (%)	
I (28- 45)	28 (27.5)
II (46 – 60)	48 (47.1)
III (61 – 75)	26 (25.5)
Duration (Years):	
(Range) Mean ± SD	(0.1-20) 2.22± 3.31
Duration groups: n (%)	
Less than 1 year	59 (57.8)
More than 1 year	43 (42.2)

Characteristics of DM among studied patients show mean duration of 8.45 ± 6.45 years (range 1 - 27 years), 24(23.5%) patients with diabetes mellitus type 1 and 78(76.5%) with type 2 diabetes and there were 85 (83.3%) with controlled diabetes while 17 (16.7%) of non-controlled diabetes.

**Table 2:** Characteristics of DM among studied patients (n=102).

Duration (Years)	
(Range) Mean ± SD	(1-27) 8.45± 6.45
Type of DM: n (%)	
Type I	24 (23.5)
Type II	78 (76.5)
Complications of DM: n (%)	
Controlled	85 (83.3)
Non controlled	17 (16.7)

According to smoking index: heavy smokers were 35(34.3%) and light/moderate smokers were 10(9.8%) of the studied patients. Hypertensive patients were 31(30.4%). Peyronei's disease associated with 20 (19.6%) patients. Body mass index

of the studied patients is 39 (38.2) of average, 27 (26.5%) overweight and 36 (35.3%) of obese type. Laboratory investigation showed that 30 (29.4%) were hyperlipidemic. Testosterone level is low in 18 (17.6%) of patients and normal in 84 (82.4%) patients.

**Table 3:** Comorbidities and risk factors of the studied patients (n=102).

Smoking index: n (%)	
Non smoker	57 (55.9)
Light smoker	10 (9.8)
Heavy smoker	35 (34.3)
HTN: n (%)	
Not hypertensive	71 (69.6)
Hypertensive	31 (30.4)
Hyperlipidemia: n (%)	
Normal	72 (70.6)
Hyperlipidemic	30 (29.4)
Testosterone level: n (%)	
Normal Testosterone level	84 (82.4)
Low Testosterone level	18 (17.6)
BMI: n (%)	
Average	39 (38.2)
Overweight	27 (26.5)
Obese	36 (35.3)
Pyronis disease: n (%)	
Absent	82 (82.4)
resent	20 (19.6)

According to pattern of erectile dysfunction Psychogenic ED were present in 16 (15.7%) of the studied patients and Organic ED in 86 (84.3%), organic ED types are 34 (33.4%) of arterial nature, 28 (27.5%) venous, mixed vascular in 17 (16.7%) patients, neurogenic in 7 (6.9%) patients and mixed vascular and neurogenic ED in 9 (8.8%) of studied patients.

**Table 4:** Pattern of ED of the studied patients. N (%)

Psychogenic ED	16 (15.7)
Organic ED	86 (84.3)
Arterial ED	34 (33.4)
Venous ED	28 (27.5)
Mixed vascular ED	17 (16.7)
Neurogenic ED	7 (6.9)

There is statistical significance (p value less than 0.05) in the relation between patients' demographic characteristics and Pattern of ED.

**Table 5:** Relation between patients' demographic characteristics and Pattern of ED.

	Psychogenic ED (16)	Organic ED (86)	p-value
Age (Years)			
(Range) Mean ± SD	(30-58) 40.38± 9.68	(28-75) 54.20± 9.57	0.001*
Age groups: n (%)			
I (28)	12 (75)	16 (18.6)	0.001*
II (48)	4 (25)	44 (51.2)	
III (26)	0 (0)	26 (30.2)	
Duration (Years)			
(Range) Mean ± SD	(0.1-2) 0.77± 0.59	(2-20) 2.49± 2.49	0.001*
Duration groups: n (%)			
Less than 1 year(59)	14 (87.5)	45 (52.3)	0.001*
More than 1 year (43)	2 (12.5)	41 (47.7)	

Chi-square and Independent – sample T test were used

\* P. value<0.05 is significant

Relation between Characteristics of DM among studied patients with Pattern of ED was statistically non-significant.

**Table 6:** Relation between patients' Characteristics of DM and Pattern of ED.

	Psychogenic ED (16)	Organic ED (86)	p-value
Duration (Years):			
(Range) Mean ± SD	(1-20) 7.94± 6.03	(2-27) 8.55± 6.56	0.487
Type of DM: n (%)			
Type I	7 (43.8)	17 (19.8)	0.054
Type II	9 (56.2)	69 (80.2)	
Complications of DM: n (%)			
Controlled	16 (100)	69 (80.2)	0.067
Non controlled	0 (0)	17 (19.8)	

Chi-square and Independent – sample T test were used

\* P. value<0.05 is significant

Relation between HTN and Pattern of ED showing statistical significance as HTN associated mainly in organic ED patients in the study 30 (34.9%) compared with 1 (6.2%) in Psychogenic ED. also, the relation between Hyperlipidemia and Pattern of ED showing statistical significance as Hyperlipidemia associated mainly in organic ED patients, other comorbidities and risk factors was statistically non-significant.

**Table 7:** Relation between comorbidities and risk factors of the patients and Pattern of ED.

	Psychogenic ED (16)	Organic ED (86)	p-value
Smoking index: n (%)			
Non smoker	11 (68.8)	46 (53.5)	0.360
Light smoker	2 (12.5)	8 (9.3)	
Heavy smoker	3 (18.7)	32 (37.2)	
HTN: n (%)			
Not hypertensive	15 (93.8)	56 (65.1)	0.022*
Hypertensive	1 (6.2)	30 (34.9)	
Hyperlipidemia: n (%)			
Normal	13 (81.3)	59 (68.6)	0.038*
Hyperlipidemic	3 (18.7)	27 (31.4)	
Testosterone level: n (%)			
Normal Testosterone level	12 (75)	72 (83.7)	0.401
Low Testosterone level	4 (25)	14 (16.3)	
BMI: n (%)			
Average	8 (50)	31 (36.1)	0.529
Overweight	4 (25)	23 (26.7)	
Obese	4 (25)	32 (37.2)	
Peyronie's disease: n (%)			
Absent	14 (87.5)	68 (79.1)	0.435
Present	2 (12.5)	18 (20.9)	

Chi-square test was used

\* P. value<0.05 is significant

### Discussion

An estimated 20% to 71% of men with type 1 or 2 DM report erectile dysfunction, with an increasing incidence with older age [13, 14]. Cho *et al.*, 2006 [15] observed that advancing age has been consistently shown to increase the risk of ED in diabetics. And this in agreement in our study There is statistical significance (p value less than 0.05) in the relation between patients' age, duration of erectile dysfunction and Pattern of ED.

Although organic factors play the major role, psychological and relationship issues often coexist [16].

De Berardis *et al.*, 2007 [17] demonstrated in a longitudinal

study the strong predictive value of depressive symptoms in the incidence of ED among diabetic men.

In our study the pattern of erectile dysfunction mainly of organic type but Psychogenic ED are also present in 15.7% of the studied patients.

The most common organic ED type in our study was of arterial nature, followed by venous, mixed vascular and neurogenic pattern.

And this in agreement with wang *et al.*, 1993<sup>[18]</sup> which found that Sixty-eight patients (87.2%) had moderate or severe cavernous arterial insufficiency, Older patients and those having a longer duration of diabetes had a higher incidence of cavernous arterial insufficiency and Cigarette smoking, hypertension, were also related to cavernous arterial insufficiency.

Several studies have demonstrated a significant association between low testosterone levels and ED in men with DM<sup>[19]</sup>. Hamdan and Al-Matubsi, 2008<sup>[20]</sup> found no significant difference in testosterone levels in the diabetic-ED patients as compared to the healthy control subjects.

In our study, we also found no clinical significance in testosterone levels in relation to pattern of diabetic-ED. Contrary to our findings, Bodie *et al.*, 2003<sup>[21]</sup>. Observed decreased testosterone (in 18.7% out of 3547) levels in the diabetic-ED patients. Moreover, Longcope *et al.*, 2000<sup>[22]</sup> stated that a slight decrease in bioavailable testosterone could result in a decline in sexual function and a reduced ability to achieve spontaneous erection. In contradiction to these two studies, Miller, 2000<sup>[23]</sup> found that testosterone replacement may increase libido without improving erectile function.

This discrepancy in the results of our study as compared to the literatures abroad could be ascribed to that (1) testosterone is predominately protein-bound and is influenced by a variety of clinical conditions and normal testosterone levels gradually decline with advancing age<sup>[24]</sup>. (2) These studies were conducted on populations of different ages and the number of patients studied by some researchers was very large<sup>[21]</sup>.

The relationship between ED and cardiovascular disease is well established<sup>[25, 26]</sup>.

In our study Hypertensive patients are 30.4% and non-hypertensive patients are 69.6%.

Wang *et al.*, 1993<sup>[18]</sup> concluded that there was no significant difference in cavernous arterial insufficiency between the insulin-dependent and the insulin-nondependent groups. In addition, the prevalence of cavernous arterial insufficiency increases with age, duration of diabetes, cigarette smoking and hypertension but it is not definitely correlated with the type of diabetes management.

And this in agreement in our study Relation between HTN and Pattern of ED showing statistical significance as HTN associated mainly in organic ED patients in the study 34.9% compared with 6.2% in Psychogenic ED while 93.8% of Psychogenic ED patient are normotensive, other comorbidities and risk factors was statistically non-significant.

Nikoobakht *et al.*, 2005<sup>[27]</sup> concluded that there was a significant difference between mean plasma cholesterol and LDL levels in the individuals suffering from ED and the control group and this in agreement in our study Relation between Hyperlipidemia and Pattern of ED showing statistical significance as Hyperlipidemia associated mainly in organic

ED patients.

Body mass index of the studied patients showed that 26.5% were overweight and 35.3% of obese type.

In 2000 Fedele *et al.*<sup>[28]</sup> concluded that there is no association emerged between BMI and ED risk in type 2 diabetes.

And this in agreement in our study There is no statistical significance (p value less than 0.05) in the relation between patients' BMI and Pattern of ED.

Smoking doubled the chance of ED developing over an 8-year follow-up period<sup>[29]</sup>. Smoking is well known as a risk factor for endothelial damage and vascular disease<sup>[30]</sup>.

In our study patients with smoking index of heavy smoker type are 34.3%, light/moderate smokers are 9.8% of the studied patients while 55.9% are non-smokers.

## Conclusion

We concluded from this study the main pattern of erectile dysfunction in diabetics is organic in nature and the cavernous arterial insufficiency is closely related to the diabetic impotence and erectile dysfunction increase with age and duration of the complaint. Hypertension and hyperlipidemia is strongly associated with erectile dysfunction in diabetics

## References

1. Adefegha S, Oboh G, Okeke B, Oyeleye SI. Comparative effects of alkaloid extracts from aframomummelegueta (alligator pepper) and aframomumdanielli (basteredmelegueta) on enzymes relevant to erectile dysfunction. *Journal of Dietary Supplements*. 2017; 14(5):542-552.
2. Gur S, Peak T, Kadowitz P, Sikka S and Hellstrom W. Review of erectile dysfunction in diabetic animal models. *Curr Diabetes Rev*. 2014; 10:61-73.
3. Corona G, Giorda C, Cucinotta D, Guida P, Nada E. Sexual dysfunction at the onset of type 2 diabetes: the interplay of depression, hormonal and cardiovascular factors. *J Sex Med*. 2014; 11:2065-2073.
4. Cohen S. The challenge of erectile dysfunction management in the young man. *Curr Urol Opin*. 2015; 16:84.
5. Lindau ST, Tang H, Gomero A, Vable A, Huang E, Drum ML, *et al.* Sexuality among middle-aged and older adults with diagnosed and undiagnosed diabetes: a national, population-based study. *Diabetes Care*. 2010; 10:2202-2210.
6. 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; 22:204-209.
7. Esposito K, Giugliano F, Maiorino M and Giugliano D. Dietary factors, Mediterranean diet and erectile dysfunction. *J Sex Med*. 2010; 7:2338-2345.
8. Castela A, Costa C. Molecular mechanisms associated with diabetic endothelial-erectile dysfunction. *Nat Rev Urol*. 2016; 13:266-274.
9. Guay A. erectile dysfunction = endothelial dysfunction. *Endocrinol Metab Clin North Am*. 2007; 36:453-463.
10. Thorve VS, Kshirsagar AD, Vyawahare NS, Joshi VS, Ingale KG, Mohite RJ. Diabetesinduced erectile dysfunction: epidemiology, pathophysiology and

- management. *J Diabetes Complications*. 2011; 25:129-136.
11. Nehra A, Moreland R. Neurologic erectile dysfunction. *UrolClin North Am*. 2001; 28:289-308.
  12. Dhindsa S, Prabhakar S, Sethi M, Bandyopadhyay A, Chaudhuri A, Dandona P. Frequent occurrence of hypogonadotropic hypogonadism in type 2 diabetes. *J Clin Endocrinol Metab*. 2004; 89:5462-5468.
  13. Matfin G, Jawa A, Fonseca V. Erectile dysfunction: interrelationship with the metabolic syndrome. *Curr Diabetes Rep*. 2005; 5:64-69.
  14. Kaya E, Sikka S, Gur S. A comprehensive review of metabolic syndrome affecting erectile dysfunction. *J Sex Med*. 2015; 12:856-875.
  15. Cho N, Ahn C, Park J, Ahn T, Lee H, Park T *et al*. Prevalence of erectile dysfunction in Korean men with Type 2 diabetes mellitus. *Diabet Med*. 2006; 23:198-203.
  16. Maiorino M, Bellastella G, Esposito K. Diabetes and sexual dysfunction: current perspectives. *Diabetes Metab Syndr Obes*. 2014; 7:95-105.
  17. De Berardis G, Pellegrini F, Franciosi M, Belfiglio M, DiNardo B, Greenfield S *et al*. Clinical and psychological predictors of incidence of self-reported erectile dysfunction in patients with type 2 diabetes. *J Urol*. 2007; 177:252-7.
  18. Wang C, Shen S, Wu C, Huang C, Chiang C. Penile blood flow study in diabetic impotence. *Urol Int*. 1993; 50:209-212.
  19. Kapoor D, Clarke S, Channer K, Jones T. Erectile dysfunction is associated with low bioactive testosterone levels and visceral adiposity in men with type 2 diabetes. *Int J Androl*. 2007; 30:500-507.
  20. Hamdan F, Al-Matubsi H. Assessment of erectile dysfunction in diabetic patients. *International Journal of Andrology*. 2008; 32:176-185.
  21. Bodie J, Lewis J, Schow D, Monga M. Laboratory evaluations of erectile dysfunction: an evidence based approach. *Journal of Urology*. 2003; 169:2262-2264.
  22. Longcope C, Feldman H, McKinlay J, Araujo A. Diet and sex hormone-binding globulin. *The Journal of Clinical Endocrinology and Metabolism*. 2000; 85, 293-296.
  23. Miller T. Diagnostic evaluation of erectile dysfunction. *American Family Physician*. 2000; 61:95-104.
  24. Nicolosi A, Moreira E, Shirai M, Bin Mohd Tambi M, Glasser D. Epidemiology of erectile dysfunction in four countries: cross-national study of the prevalence and correlates of erectile dysfunction. *Urology*. 2003; 61:201-206.
  25. Gandaglia G, Briganti A, Jackson G, Kloner R, Montosori P, Vlachopoulos C. A systematic review of the association between erectile dysfunction and cardiovascular disease. *EurUrol*. 2014; 65:968-78.
  26. Dattatrya K, Vedpalsingh T, Gorakhnath W, Kiran P. Can erectile dysfunction in young patients serve as a surrogate marker for coronary artery disease? *J Clin Diagn Res*. 2015; 9:PC01-3.
  27. Nikoobakht M, Nasseh H, Pourkasmaee M. The relationship between lipid profile and erectile dysfunction. *International Journal of Impotence Research*. 2005; 17:523-526.
  28. Fedele D, Bortolotti A, Coscelli C, Santeusano F, Chatenoud L, Colli E, *et al*. erectile dysfunction in Type 1 and Type 2 diabetics in Italy. *International journal of epidemiology*. 2000; 29:524-531.
  29. Jackson G, Montorsi P, Adams MA, Anis T, El-Sakka A, Miner M, *et al*. Cardiovascular aspects of sexual medicine. *J Sex Med*. 2010; 7:1608-26.
  30. Rosen RC, Fisher W, Eardley I, Neiderberger C, Nadel A, Sand M. The multinational Men's Attitudes to Life Events and Sexuality (MALES) study: I.Prevalence of erectile dysfunction and related health concerns in the general population. *Curr Med Res Opin*. 2004; 20:607-617.