Electrophysiological Assessment of Pudendal Neuropathy in Diabetic Patients with Erectile Dysfunction in Baghdad

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Abstract
Erectile dysfunction (ED) may be caused by autonomic neuropathy, vasculogenic causes, endocrine deficit, drug-induced, psychosocial problems, or a combination of these. It has an impact on a patient's life that extends beyond its physical well-being. Aim: This study aimed to differentiate between neurogenic and non-neurogenic impotence (ED) by considering the Electrophysiological induced Bulbocavernosus Reflex in diabetic patients. Methodology: This analytic cross-sectional study included 84 diabetic subjects with an average age of 38.4 years presented with impotence (ED) to whom Electrophysiological induced Bulbocavernosus Reflex (BCR) was conducted on all patients depending on the methods described by Rushworth. Results: The results of assessing the pudendal never integrity and neurological functionality revealed that 40.5% had typical pudendal nerve latency of < 42 msec. Mean value (38.4±3.87). Although 4.8% of the subjects showed an absent response, the majority, 54.7%, showed prolonged pudendal nerve latency with > 42 msec. (48.81±2.56). Conclusion: Electrophysiological induced Bulbocaverneous reflex test is a practical test which can be helpful in a diabetic neurologic deficit of diabetic patients with erectile dysfunction.
Keywords: Erectile dysfunction, Electrophysiological induced Bulbocaverseouns reflex, Diabetes Mellitus.

1. Introduction

According to the National Institutes of Health, a persistent incapacity to perform and keep penile erection strong enough for fulfilling sexual interaction is known as erectile dysfunction (ED).[1][2] The condition is closely associated with becoming older. However, this does not mean that the condition might not occur in younger age groups. The global prevalence ranges from 1% - 10% for males less than 40 years, up to 15% for men 40 - 49 years, up to 30% for men 50 - 59 years, up to 40% men from 60 - 69 years, and 50% - 100% for men between 70 and 90 years.[3][4]

There have been many causes deemed to be associated with ED which could be categorised as organic comprising vascular, neurogenic, hormonal, or medication-related side effects, as well as non-organic reasons, which mainly contain psychogenic factors.[5] [6]

Diabetes is considered the frequent cause of erectile dysfunction among others. The incidence of erectile dysfunction is 52.5% among men aged 48-64 years with diabetes.[7][8] And the prevalence of ED seems to be increased with duration of diabetes, poor glucose management, and DM comorbidities such as vascular and microvascular illness, as well as neuropathies [7] [9].

Raised blood sugar (hyperglycaemia) induced molecular and cellular deterioration of vascular and neural structure and function primarily responsible for the development of chronic complications in diabetes. Another factor that significantly contributing to the development of long-term consequences of diabetes is oxidative stress. In addition, neuropathy and angiopathy caused by diabetes may result in malfunctioning of cells, tissues, and organs.[10] [11]

The elevated intracellular glucose levels in the neurons caused by the hyperglycemia oversaturate the usual glycolytic pathway.[12][13] When glucose enters the autonomic route, it is transformed into fructose and sorbitol, which leads to decrease in the cell membrane permeability of (the Na+/K+-ATPase pump) and results in aberrant action potentials in the cell. Oxidative stress depletes the oxygen supply to the nerve cells, inhibits their ability to proliferate, and causes cellular apoptosis, or cell death; this results in progressive neuropathy.[12][14][15]

Several previous studies showed that 43% of autonomic neuropathy was for those > 47 years old and 51.2% with peripheral somatic neuropathy for those > 40 years. [16] [17] [18] The studies showed how essential is the neuropathy and considered the main causative factor of ED in diabetic patients, both vascular and neural pathogenesis of ED are considered the leading causes. However, it is still unknown which one is the predominant cause.[19]

Moreover, the relationship between peripheral neuropathy and impotence, particularly in people with diabetes, is not well-defined and proven. In other words, diabetic individuals with ED are

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believed to have underestimated peripheral neuropathy.[16][19]

Thus, the main objectives of this present study were to differentiate between neurogenic and non-neurogenic causes of ED in diabetic subjects and to identify which is the earliest neuropathy between both peripheral and pudendal nerves.

2. Methods

This quantitative cross-sectional analytical study was conducted prospectively in Ghazi Al-Harri Surgical Teaching hospital, linked to Baghdad Medical College, from October 2021 to June 2022. It included a sample of 84 male diabetic subjects aged 18 – 70 years old.

All males with impotence (ED) due to both types of diabetes, whether with peripheral neuropathy or without, were enrolled in the study. Accepted cases were subjected to the same clinical, biochemical and electrophysiological evaluations for pudendal and peripheral nerves but excluded autonomic nervous system and cranial nerves.

Those with neurological dysfunction not explicitly related to diabetes were ruled out. The standard history, general physical, and neurological evaluations, as well as the standard laboratory assessment, were performed.[20] Two standard scoring system questionnaires were used, the Toronto Clinical Scoring System (TCSS) and the International Index of Erectile Dysfunction-5 (IIED-5), for clinical categorisation of peripheral neuropathy and erectile dysfunction, respectively. Electrophysiological induced Bulbocavernosus Reflex (BCR) was performed on all subjects at the electrophysiological department considering the methods described by Rushworth.[21]

A ring stimulating electrode is placed to the penile shaft to measure the BCR., with a distance of 2-3 cm apart between both active and reference electrodes, setting the former more distal than the latter, respectively.[22][23] The BCR is recorded via concentric electrode is inserted into the bulbocarvenosus (BC) muscles, the insertion site is 2-3 cm anterior to the anal orifice in the midway between the scrotum and the anus with few millimetres lateral to the perineal raphe line on both sides. The depth of the insertion is around 1-2 cm which depends on the body built of the subject, the proper needle position was confirmed by the pelvic floor voluntary contraction, which was, elicited by squeezing the anal sphincter or coughing many times throughout the session, as well as squeezing the glans penis while observing the EMG wave on the monitor.[24][25]

Stepwise rectangular pulses with durations of 0.1 and/or 0.2 msec and intensities starting at "0" up to 300 v were used to stimulate the penis glans. The sweep speed for recording was ranging from 5 to 10 msec. while the stimulation sensitivity was set on 200 µV.[26] The psych sensory threshold for feeling in the penis mainly the glans was identified by a stepwise stimulation increment per l sec.[26] The latency was assessed by determining the distance from stimulus to the first upward wave direction (negative peak), and is considered abnormal when it was more than 42 msec or absent. Figure 2 shows the reflex single CMAP.[26][27] [28]

lastly, highest stimulation reaching up to 300V was applied. In all subjects, stimulation was given with duration of 0.2 msec. and intensity was 150-240 v were able to produce stable response.[26]

Furthermore, lower limb peripheral nerves were assessed by electro diagnostic studies mainly motor and sensory nerves (Common Peroneal, Sural and Tibial nerves).

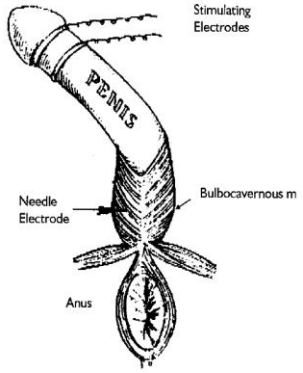

Figure 1 – drawing of BCR study technique [28]

Table 1 – BCR latency results		
Group	Latency	
	Mean±SD msec.	(Min-Max) msec.
Normal	38.4±3.87	31.12 - 42
Prolonged	48.81±2.56	43 - 52.8
Absent	0	0

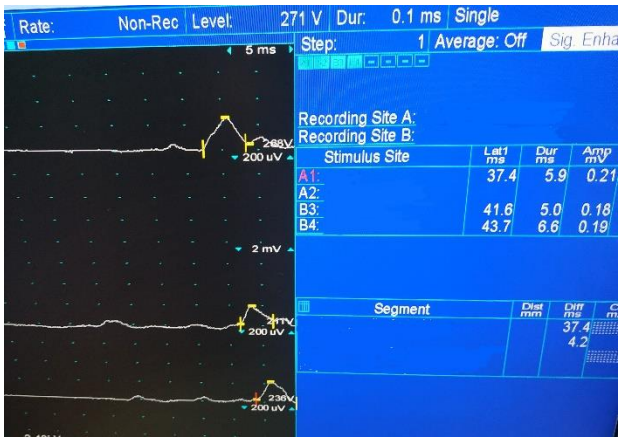

Figure 2 – BCR response of one of the included cases

3. Statistical Analysis

The data was processed by comparing any difference between electrophysiological recording results of BCR between those impotence diabetic subjects with and without peripheral neuropathy. The results were analysed using SPSS version 25, and the means ± SD were compared using an independent T-test. Correlation, and the percentage using Fisher's exact and Pearson Chi-square tests whenever is applicable to compare these two groups. A difference in P-value between the groups of <0.05 is considered statistically significant.

4. Result

84 diabetic male subjects with impotence (ED) were enrolled in the study, after clinical assessment was performed, there was 73.8% ED with peripheral neuropathy and 26.2% without peripheral neuropathy.

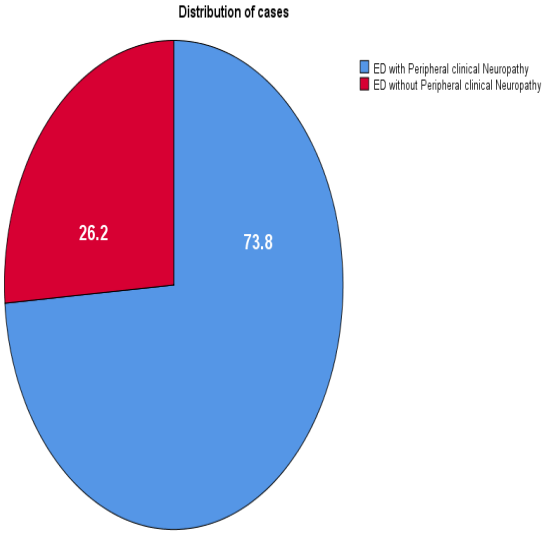

Figure 3 – the percentage of the two groups

The mean age was (45.38 ±6.604) ranging from 32 to 62 years, 52.3% of the cases were aged between 41-50 years old, and the mean duration of diabetes was (8.38 ± 2.81) with a minimum of 4 and a maximum of 15 years, while the mean duration of ED was (2.6±1.9) ranging from 1 to 8 years.

HBA1c was performed for all subjects with a mean value (9.2±1.94) ranging from 6.5 to 14. In addition, BMI mean value was (27.5±2.67) ranging from 22 to 32.

Through the clinical evaluations conducted on all involved subjects, they were asked about any morning erection, 61.9 % had no morning erection, and only 38.1% had a morning erection. As well as clinical scoring was performed to assess ED and peripheral neuropathy by following IIED-5 and TCSS, respectively. The results revealed 45.2% complaint from moderate degree of clinical ED, and 31% suffered from moderate degree of clinical peripheral neuropathy.

All 84 diabetic male subjects were evaluated depending on BCR method to assess the pudendal nerve integrity and neurological functionality, the results revealed that 40.5% revealed normal latency of pudendal nerve of < 42 msec. mean value (38.4±3.87). Although 4.8% showed absent response, the majority of the subjects 54.7% revealed prolonged latency of > 42 msec. (48.81±2.56) as mentioned in Table 1.

Error! Reference source not found. illustrates the results of the BCR of the Pudendal nerve after categorizing the sample in two main groups, those with peripheral neuropathy and without peripheral neuropathy by considering the Nerve Conduction Study (NCS), the mean latency of the Pudendal nerve within the first group was (36.44±3.58), while the second group was (46.14±3.85) which showed significant (p-value of 0.0001).Through following the similar categorization but this time including the three main parameters of the NCS (Latency, Amplitude and Conduction Velocity) to explore any significant differences of lower limb nerves between two aforementioned main groups. The results showed statistically significant P-value for all nerves and involved parameters.

Most the patients revealed abnormal NCS findings with 59.5% lower motor nerves results whether prolonged latency, conduction velocity, low amplitude and/or absent; with 40.5% showed normal results.

Together both lower Motor and Sensory NCS revealed 69% abnormal findings with 31% normal findings.

Table 2 – Distribution of the sample based on NCS of peripheral lower limb nerves

Independent sample T-test Different NCS parameters	ED + Normal NCS of Lower Limb Nerves (mean±SD)	ED + Abnormal NCS of Lower Limb Nerves (mean±SD)	P-value
Pudendal Nerve NCS Latency	36.44±3.58	46.14±3.85	0.0001
Peroneal Nerve Latency	3.75 ±0.7	6.46 ±1.15	0.0001
Tibial Nerve Latency	3.86 ±0.7	4.77 ±0.8	0.0001
Sural Nerve Latency	2.74 ±0.17	4.23 ±1.38	0.002
Peroneal Nerve Amplitude	7.01 ±2.03	2.57 ±1.67	0.0001
Tibial Nerve Amplitude	8.67 ±2.0	4.14 ±2.4	0.0001
Sural Nerve Amplitude	10.23 ±2.96	4.8 ±2.37	0.0001
Peroneal Nerve Conduction Velocity	47.9 ±2.7	33.25 ±7.24	0.0001
Tibial Nerve Conduction Velocity	46.83 ±3.05	34.91 ±7.89	0.0001
Sural Nerve Conduction Velocity	51.0 ±3.2	34.93 ±9.94	0.0001

There was an obvious difference between clinical evaluation and electro diagnostic study as it is explained in

Figure 4 where it revealed all the presented cases have a variety degree of ED according to IIED-5 assessment. However, electro diagnostic study of the pudendal nerve exploring BCR showed only 54.9% have abnormal finding. From the other hand, the same steps were conducted on peripheral lower limbs nerves, through completing clinical evaluation depending on TCSS which showed 73.8% which means the majority of the subjects have a specific degree of peripheral neuropathy. While the electro diagnostic study confirmed only 69% of the patient with significant either motor or sensory or both.

The last figure shows the correlation between diabetes mellitus, peripheral neuropathy and erectile dysfunction duration, almost all of the cases revealed

peripheral neuropathy symptoms earlier than erectile dysfunction.

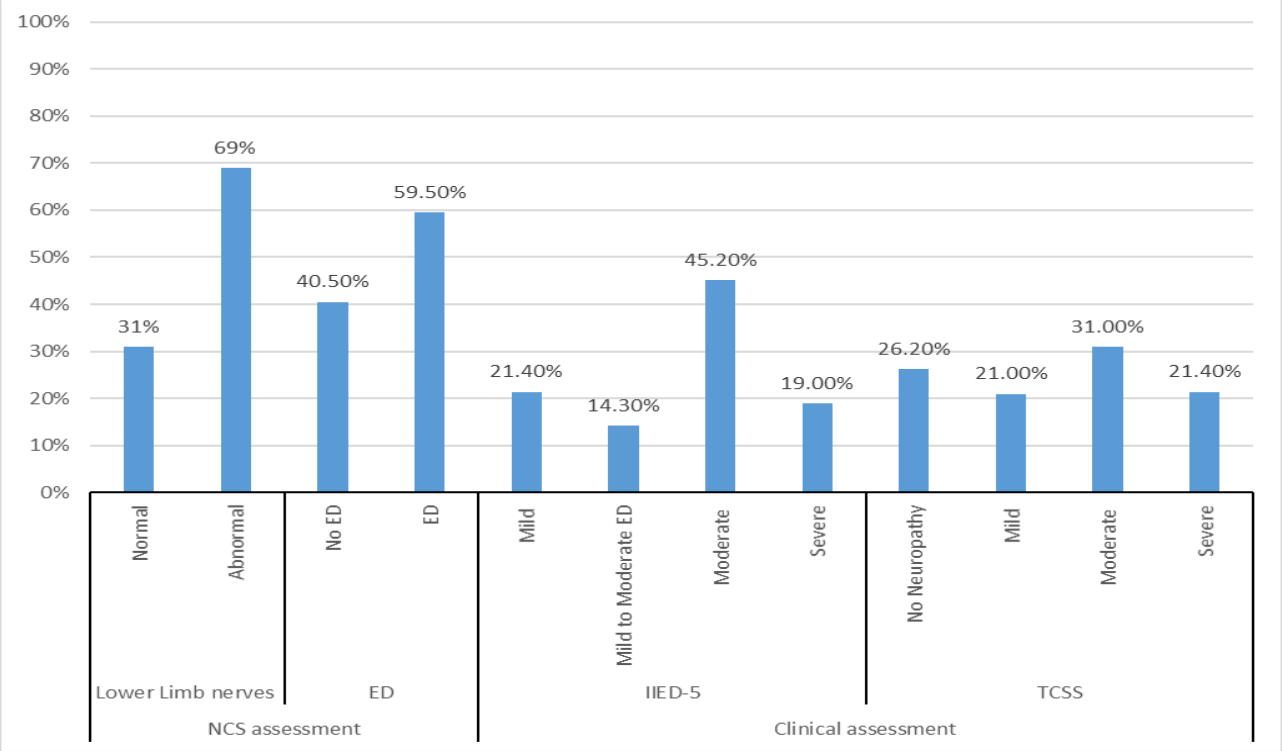

Figure 4 – distribution of the sample based on NCS and clinical assessments

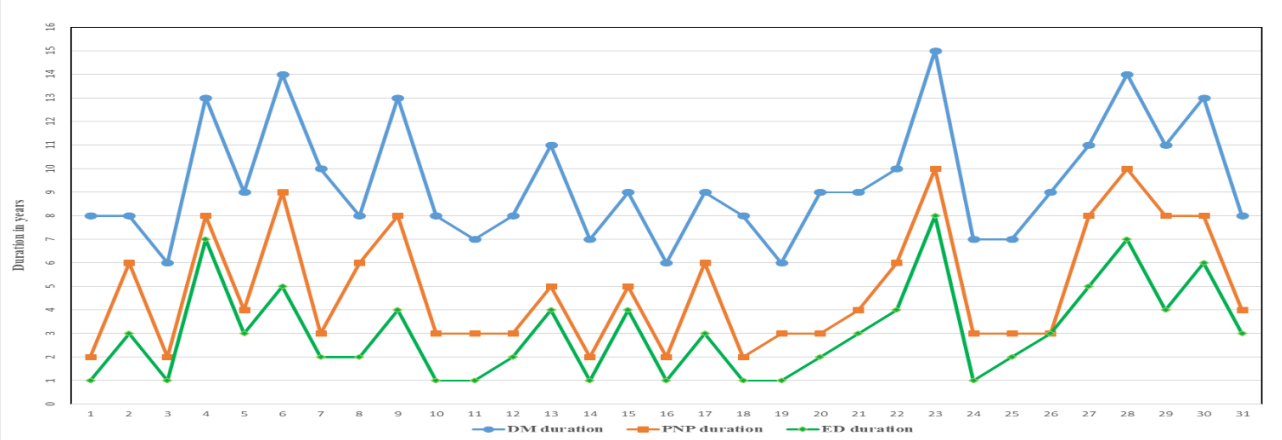

Figure 5 – duration of Diabetes, Peripheral neuropathy and Erectile dysfunction

5. Discussion

The erection process is mainly a parasympathetic autonomic activity. However, there is a histochemical indication of a sympathetic involvement as well.[29] The penile dorsal nerve is the origin of the somatic reflex route that is measured by the electro physiologically induced BCR. This route includes the afferent limb of the pudendal nerve (sensory), S2-S4 (sacral spine), and the efferent limb of the pudendal nerve (motor).[25] When the BCR arcs are damaged, no reaction is elicited, and when the afferent and efferent limbs are partially damaged, the BCR is prolonged.[30]

The current study indicates that nerve conduction abnormality of pelvic nerves is evident in 59.5% of diabetic men with erectile dysfunction.[29] This refers to 46 impotent diabetic subjects out of 84 patients; the rest, which represents 40.5%, had a BCR latency of less than < 42 msec, that are mentioned in previous studies.[26][27][28] Abnormal findings of prolonged or absent BCR latency refer to the neurological deficit, which indeed has an essential role in explaining the impotent status of the diabetic presented cases. These findings are compatible with the results found in the previous studies. [31] At the same time, electrodiagnostic study of the peripheral nerves of the lower limbs demonstrated that 69% of the patients have confirmed peripheral neuropathy, in terms of sensory, motor or both, which is less than 73.8% of the peripheral neuropathy that was identified by TCSS before. Additionally, IIED-5 was not specific sufficient to prove the link between clinical complaints of ED with the results of an electrodiagnostic study of BCR.

There is a significant statistical correlation between BCR latency and those with clinical and electrophysiological peripheral neuropathy (p-value=0.0001), which proves the major contribution of peripheral neuropathy effect in those diabetic patients with ED, as well as it is consistent with the previous study regarding underestimation of

peripheral neuropathy in ED. [16] [18][19]

Onuf's nucleus, located in the sacral ventral horn, is where most of the motoneurones (MNs) that control the pudendal nerve is found (Onuf 1900). Although the anterior horn cells assigned for pudendal nerve motor fibres are smaller than other motoneurones of the lower limb motor fibres, the conduction velocity of motor fibres of the pudendal nerve is similar to motor fibres of the lower limb nerves.[32] This confirms the correlation of abnormal BCR latency with peripheral lower limbs nerves latency.

The study explored the earlier onset and longer duration of peripheral neuropathy (3.62±3.1) than ED (2.6±1.9). However, ED was sufficient to bring the patients to seek medical attention as early as possible. With one year in most patients and a maximum of eight years, while for peripheral neuropathy alone, ranging from 2-10 years.

In our study, several limitations could be summarised as follows: the first point is the limited sample of patients, which might not be representative, and the second point is regarding the fixed period of the study to perform other relevant electrodiagnostic studies like Sympathetic Skin Response test (SSR) that could add a positive value in evaluating the autonomic nervous functionality. Another point is the unavailability of penile vascular assessment tools or instruments like penile Doppler to assess the vessels' vascular competency. This could exclude other reasons for ED in diabetic patients who showed normal BCR latency.

6. Conclusion

The test performed distinguished neurogenic causes of ED from non-neurogenic causes. Other tests include nerve conduction studies and clinical indicators of polyneuropathy. Moreover, the study revealed that the percentage of neuropathy in ED is higher in those with diabetes for a more extended period than those for a shorter duration. This means peripheral neuropathy has a significant role in diabetic subjects with ED. Diabetic patients with erectile dysfunction should have a comprehensive neurological work-up that includes a sensory-stimulation response (SSR) test to study the functioning of autonomic nerve fibres and a neurophysiological examination of myelinated and unmyelinated nerve fibres.

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