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DIABETIC FOOT: CORRELATION BETWEEN CLINICAL ABNORMALITIES AND ELECTROPHYSIOLOGICAL STUDIES

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Abstract

Diabetic foot ulceration is a serious and expensive complication with considerable morbidity that affects up to 15% of diabetic patients during their lifetime and 80-85% of amputations are preceded by foot ulcers. The aim of this work is to study the correlation between severity of clinical abnormalities and electrophysiological studies in diabetic foot ulcers.

This study was a cross sectional evaluation of 44 patients with diabetic foot ulcers seen in 2 hospitals in Basrah (Al-Faiha General and Basrah Teaching) from October 2003 to July 2004. All patients were type 2 diabetes mellitus.

The sensitivity of numbness, burning feeling, pricking feeling and worse symptom at night was 84.6%, 69.2%, 61.5% and 51.5% respectively. While sensitivity of decreased pin prick sensation, absent vibration sense, absent ankle jerk, decreased temperature sensations and absent position sense was 100%, 87.2%, 71.8%, 56.5% and 12.8% respectively. Sensitivity of combined clinical symptoms was 66.6%, with specificity of 40%, and predictive value of 89.6% while that of clinical signs 48.7% and 60% respectively and predictive value of 90.4 %.

There was no significant difference in severity of electrophysiological abnormalities in the affected and non-affected feet. Clinical findings was correlated well with the severity of electrophysiological changes in patients with diabetic foot ulcers.

Introduction

Neuropathy is present in 80% of patients with foot ulcers; it promotes ulcer formation by decreasing pain sensation and perception of pressure, by causing muscle imbalance that can lead to anatomic deformities and by impairing the microcirculation and the integrity of the skin[1-5]. Even in the face of non-obstructed vessels, impaired micro vascular reactivity diminishes blood supply to the ulcerated areas.

About 20% of diabetic patients with foot ulcers will primarily have inadequate arterial blood flow, about 50% will primarily have diabetic neuropathy and about 30% will be afflicted with both conditions[6].

Diabetic foot ulceration is a serious and expensive complication with considerable morbidity that affects up to 15% of diabetic patients during their lifetime and 80-85% of amputations are preceded by non-healing foot ulcers[7-10] .

There is increasing evidence that measures of neuropathy, such as electrophysiology (including motor nerve conduction velocity) and quantitative tests, are predictors of not only end points, including foot ulceration, but also of mortality[11].

In Iraq, diabetic foot ulcers were reported in 17% of diabetics in a small

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series from Baghdad[12].

The aim of this work is to study the correlation between severity of clinical abnormalities and electrophysiological studies in diabetic foot ulcer.

Methods

This was a cross sectional study of patients with diabetic foot ulcer seen in 2 hospitals in Basrah (Al-Faiha general and Basrah Teaching) from October 2003 to July 2004. Patients from outpatient and inpatient clinics were included. All patients were type 2 diabetes mellitus (DM), they were 44.

Definitions

DM was diagnosed according to the American Diabetic Association recommendations in 2002[13]. Patient who were currently on drug treatment for diabetes and hypertension were considered hypertensive and diabetic respectively. For blood pressure, the average of second and third blood pressure measurements in the office were considered. Two blood pressure recordings were obtained from the right arm of patients in a sitting position after 30 min of rest at 5-min intervals and their mean value was calculated. Hypertension was considered if blood pressure is equal to 140/90 mmHg or above. Nephropathy was diagnosed on the basis of persistent frank proteinuria without erythrocytes or white blood cells in urine. Microalbuminuria detection was not feasible. An Ophthalmologist diagnosed retinopathy.

Body mass index was calculated according to the formula: wt (kg)/ht^2 (m^2)[14]. The women were non pregnant. Autonomic function tests were not done because they were final–consuming and add nothing to our context. Diabetic foot ulcer was defined as any full-thickness skin lesion distal to the ankle that required treatment in hospital, excluding minor abrasions or blisters and/or the presence of any other cause of diffuse peripheral neuropathy (malignancy, alcohol abuse, drug abuse, anemia, known vitamin B12 deficiency, or untreated hypothyroidism). Vibration sensation was measured on the plantar hallux using a 128-Hz tuning fork and was graded as absent if the subject reported no vibration while the examiner could still sense vibration. Achilles tendon reflex was elicited with the subject in a seated position. Neuropathy screening instruction questionnaire was done for all (appendix –1)[15].

Quantitative assessment of the clinical findings was done by the same examiner according to general practice protocols (appendix–2)[1]. Nerve conduction studies were performed using standard protocols[16]. Nerve conduction abnormalities were classified into normal and abnormal according to the common peroneal nerve conduction of each leg separately (normal >44.4ml/second, mild 40-44.3ml/second, moderate 36-39.9ml/second and severe <36 ml/second). Using electrophysiological study as gold standard for the neuropathy, we calculated measures of validity, namely sensitivity and specificity. The results were expressed as percent. For statistical analysis, Chi–square test was used as appropriate. Level of significance was set to be <0.05 throughout analysis.

Results

Major characteristics of patients are present in table I and feet findings in table II. Fifty percent of ulcers were from Wagner grade one.

The sensitivity of numbness, burning feeling, pricking feeling and worse symptoms at night was 84.6%, 69.2%, 61.5% and 51.5% respectively (table III). While sensitivity of decreased pin prick sensation, absent vibration sense, absent ankle jerk, decrease temperature sensations and absent position sense

was 100%, 87.2%, 71.8%, 56.5% and 12.8% respectively (table III). All patients had abnormal motor nerve conduction velocities (table IV).

Sensitivity of combined clinical symptoms was 66.6%, with specificity of 40%, and predictive value of 89.6% while that of clinical signs was 48.7% and 60% respectively with predictive value of 90.4 % (table IV).

No significant difference in severity of electrophysiological abnormalities between the affected and non-affected foot was abscond (table V).

Twenty five percent of those with optimal diabetes control had severe electrophysiological changes versus 63.6% in those with non-optimal control (table VI).

Discussion

It is generally agreed that diabetic neuropathy should not be diagnosed on the basis of one symptom, sign or test alone: a minimum of two abnormalities (symptoms, signs, nerve conduction abnormalities, quantitative sensory tests or quantitative autonomic tests) is recommended (Dyck)[17]. In our study, the sensitivity of clinical symptoms in predicting severe electrophysiological changes in patients with diabetic foot ulcer was 66.6% and that of clinical signs 48.7%. In some other studies the prevalence of diabetic neuropathy has been estimated to be as high as 62% of diabetic based on subjective complaints, 55% by signs and 100% by nerve conduction studies[18].

Of our patients 22.7% were smoker, 38.6% hypertensive, 63.6 % having non-optimal control of diabetes and most of them were with low education level. In univariate analyses, diabetic foot problems were characterized by older age, male preponderance, longer duration of diabetes, smoking, poorer glycemic control, more insulin users, hypertension, hyperlipidemia, higher diastolic and systolic blood pressure, lower education level and living in rural areas[19].

Retinopathy was seen in 63.6%, nephropathy in 45.4%, and absent pulsation of the feet in 13.6%. Only 34.1% used insulin with or without oral hyperglycemic agents. Theories of ulcer development other than the roles for neuropathy include diminished vascular perfusion, foot deformity and higher foot pressure, diabetes severity (reflected by type of treatment) and preexisting diabetic complications[20].

This study showed muscle atrophy in 75% with pes cavus in 50%. Motor neuropathy is commonly believed to lead to weakness in the intrinsic muscles of the foot, thus upsetting the delicate balance between flexors and extensors of the toes. Atrophy of the small muscles responsible for metatarsophalangeal plantar flexion is thought to lead to the development of hammer toes, claw toes, prominent metatarsal heads and pes cavus[21].

Decreased pinprick sensation was observed in all patients (100%), absent ankle reflex in 70.4% and decreased vibration in 84.1% in this study. In prospective studies, the three main independent predictors for foot ulceration has been shown to be absent Achilles tendon reflex, impaired mono-filament pressure sensation and impaired vibration sensation[22]. Most of our patients had low educational level, nevertheless, high incidence of foot ulceration has been reported in a population of diabetic patients with established peripheral neuropathy, despite the patients receiving a high level of education[23].

In conclusion clinical findings correlated with the severity of electrophysiological changes in patients with diabetic foot ulcers.

Appendix-1-

Neuropathy screening instruction questionnaire (yes or no)[15]

Are your leg or feet numb?( ) Do you ever have any burning pain in your legs and/or feet?( ) Are you feet

too sensitive to touch? ( ) Do you get muscle cramp in your legs and/or feet?( ) Any pricking feeling in your legs and/or feet?( ) Does it hurt when the bed covers touch your skin?( ) Can you tell in the bathroom the cold from hot water?( ) Any ever seen open sore in the feet?( ) Any doctor told patients that he is having diabetic neuropathy?( ) Do you feel weak all over most of time?( ) Are you symptoms worse at night?( ) Do your legs hurt when you walk?( ) Are you able to sense your feet hen you walk?( ) Is the skin on your feet so dry that it cracks open?( ) Have you ever had amputation?( )

Appendix-2-

Quantitative assessment of symptoms:[1]

*What is the sensation felt – burning, numbness, or tingling (2 points); fatigue, cramping, or aching (1 point). Maximum is 2 points.

- What is the location of symptoms – feet (2 points); calves (1 points); elsewhere (no points). Maximum is 2 points.
- Have the symptoms ever awaken you at night – yes (1 point).
- What is the timing of symptoms – worse at night (2 points); present day and night (1 points); present only during the day (no points). Maximum is 2 points.
- How are symptoms relieved – walking around (2 points); standing (1 point); sitting or lying or no relief (no points). Maximum is 2 points.

The total symptoms score can then be determined:

- 0 to 2 – normal
- 3 to 4 – mild
- 5 to 6 – moderate
- 7 to 9 – severe

Quantitative assessment of physical findings:

- What is the Achilles tendon reflex – absent (2 points for each foot); present with reinforcement (1 point for each foot).
- What is the vibration sense – absent or reduced (1 point for each foot).
- What is the pin prick sensation – absent or reduced (1 point for each foot).
- What is the temperature sensation – reduced (1 point for each foot).

The neurologic signs score can then be determined:

- 0 to 2 – normal
- 3 to 5 – mild
- 6 to 8 – moderate
- 9 to 10 – severe

Table -1: Patients characteristics

Variables	***No.* (%)**
No.	**44(100)**
Age(years) mean (range)	**58.7±8.7(31-75)**
Sex male	**20(45.4)**
Females	**24(54)**
Qualification	**3.6 ±3.7**
Duration of diabetes mellitus	**12.25±7.8**
BMI	**24.1±4.12**
Smoker	**10(22.7)**
Drinker of alcohol	**3(6.8)**
Lines of treatment	
Diet alone	**1(2.2)**
***Oral hypoglycemic agents**	**28(63.6)**
****Insulin with oral hypoglycemic drugs**	**7(15.9)**
Insulin alone	**8(18.2)**
Degree of control of DM	
Poor	**24(54.5)**
Acceptable	**8(18.2)**
Optimal	**12(27.3)**
Associated vascular disease	
Hypertension	**17(38.6)**
CVA	**5(11.4)**
HF	**4(9.1)**
IHD	**5(11.4)**
Social class	
Low	**36(81.8)**
Intermediate	**7(15.9)**
High	**1(2.3)**
Nephropathy	**20(45.4)**
****Retinopathy**	**28(63.6)**

***2 of them on combined sulfonylurea and metformin**
****1 of them on combined sulfonylurea, metformin**
*****3 patients had mature cataract and 2 glaucoma**

Table-II: Foot examination

		No.(%)
Side of foot ulcer	Right	16(36.5)
	Left	21(47.7)
	Both	7(15.9)
Site of the ulcers	Big toe	18(40.9)
	Other toes	12(27.3)
	Big toe and other toe	5(11.3)
	Foot and toe	2(4.5)
	Heel	1(2.3)
	Malleolus	1(2.3)
No. of ulcers	Single	35(79.5)
	Multiple	9(20.4)
Wagner grade	1	22(50)
	2	12(27.3)
	3	6(13.6)
	4	4(9.1)
	5	0(0.0)
Nails changes		24(54.5)
Fissures in the skin		18(40.9)
Callosities		9(20.4)
Pes cavus		22(50)
Muscle wasting		35(79.5)
*Absents pulsation		6(13.6)
Dermopathy		16(36.3)

***Absents dorsalis pedis and/or posterior tibial artery.**

Table-III: Clinical finding in patients with diabetic foot

	Clinical finding	No.	%	Sensitivity (%)	Specificity (%)	Positive predictive value (%)
Symptoms	Numbness	38	86.3	84.6	0.0	86.8
	Burning feet	32	52.2	69.2	20	87
	Pricking feeling	27	61.3	61.5	20	85.9
	Symptoms worse at night	23	52.2	51.2	40	86.9
Signs	Decrease pin prick sensation	44	100	100	0.0	88.6
	Absent vibration	37	84.1	87.2	0.0	87.1
	Ankle jerk absent	31	70.4	71.7	60	90.3
	Decrease temperature sensation	24	54.5	56.4	60	91.6
	Absent position sense	6	13.6	12.8	80	83.3

Table –IV: Correlation between clinical finding and electrophysiological study

Clinical finding		Electrophysiological study				Sensitivity (%)	Specificity (%)	Positive predictive value (%)
		Severe	Moderate	Mild	Total			
Clinical symptoms	Severe	26	3	0	29	66.6	40	89.6
	Moderate	7	0	1	8			
	Mild	6	1	0	7			
Total		39	4	1	44			
Clinical signs	Severe	19	2	0	21	48.7	60	90.4
	Moderate	15	1	0	16			
	Mild	5	1	1	7			
Total		39	4	1	44			

Table –V: Correlation between electrophysiological study in the affected and non- affected foot. Each lower limb was considered a subject in the analysis.

Electrophysiological study	Affected foot no. (%)	Non-affected foot No. (%)	Total	P value
Severe	*39(88.6)	35(79.5)	73	***NS
Moderate	4(9)	5(11.3)	9	
Mild	1(2.2)	4(9)	5	
**Non severe total	5(11.3)	9(20.4)	14	
Total	44(100)	44(100)		

***7 patients have both feet affected and we took foot with higher Wagner grade as the affected.**
****Non-severe includes moderate and mild.**
*****NS denote non-significant.**

Table –VI- correlation between severity of electrophysiological study and degree of diabetic control.

Electrophysiological study	Optimal	Acceptable	Poor	*Non- optimal	Total
Severe	11(25%)	6	22	28(63.6%)	39
Moderate	1	2	1	3	4
Mild	1	0	0	0	1
Non severe total	2	2	1	3	5
Total	13	8	23	31	44

***Non-optimal includes acceptable and poor control**
P value =NS between optimal and non-optimal.

References

1-Young MJ, Boulton AJM, Macleod AF,Williams DRR, Sonksen PH. A multicenter study of the prevalence of diabetic peripheral neuropathy in the United Kingdom hospital clinic population. Diabetologia 1993;36:150–154.

2-Caputo GM, Cavanagh PR, Ulbrecht JS, Gibbons GW, Karchmer AW.Assessment and management of foot disease in patients with diabetes . N Engl J Med 1994;331:854–860.

3-Lavery LA, Armstrong DG, Vela SA, Quebedeaux TL, Fleischli JG. Practical criteria for screening patients at high risk for diabetic foot ulceration. Arch Intern Med 1998; 158:157–162.

4-McNeely MJ, Boyko EJ, Ahroni JH, Stensel VL, Reiber GE, Smith DG, Pecoraro RF. The independent contributions of diabetic neuropathy and vasculopathy in foot ulceration: how great are the risks? Diabetes Care 1995;18:216–219.

5-Veves A, Murray HJ, Young Mj, Boulton AJM. The risk of foot ulceration in diabetic patients with high foot pressure: a prospective study. Diabetologia 1992; 35:660–663.

6-Reiber GE, Vileikyte L, Boyko EJ, del Aguila M, Smith DG, Lavery LA, Boulton AJ. Causal pathways for incident lower extremity ulcers in patients with diabetes from two settings. Diabetes Care 1999; 22:157–162.

7-Mayfield JA, Reiber GE, Sanders LJ, Janise D, Pogach LM: Preventive foot care in people with diabetes. Diabetes Care 1998; 21: 2161–2177.

8-Palumbo PJ, Melton LJ: Peripheral Vascular disease and diabetes. In Diabetes in America. Washington, DC, publ. no. 85-1468). U.S. Govt. Printing Office, 1985 (NIH

9-Pecoraro RE, Reiber GE, Burgess EM:Pathways to diabetic limb amputation:basis for prevention. Diabetes Care 1990 13:513–521.

10-Boyko EJ, Ahroni JH, Smith DG, Davignon D: Increased mortality associated with diabetic foot ulcer. Diabet Med 1996; 13:967–972.

11-Carrington AL, Abbott CA, Shaw JE, Vileikyte L, Van Schie CHM, Boulton AJM:Can motor nerve conduction velocity predict foot problems in diabetic neuropathy over a 6-year outcome period?Diabetes Care 2002; 25:2010–2015.

12-Ali SM.Skin manifestation of diabetes mellitus.Iraqi J Comm. Med 2001;14:164-166.

13-Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. The expert committee on the diagnosis and classification of diabetes mellitus. Diabetes Care 2002; 25:S5-S20.

14-Billewics WZ,Kemsley WFF,Thompson AM.Indices of adiposity.Br J Prevent Soc Med 1962;6:183-188.

15-Brown MB, Canal N, Greene DA: Practical two-step quantitative clinical and electrophysiological assessment for the diagnosis and staging of diabetic neuropathy. Diabetes Care 1994 17:1281–1289.

16-Kimura J.Principles of nerve conduction studies.In Electrodiagnosis in disease of nerve and muscle:Pricnciple and practice.Kimura J,editor.Philadelphia:Davis;1989;p.83-104.

17- Dyck PJ: Severity and staging of diabetic polyneuropathy. In Textbook of Diabetic Neuropathy. Gries FA, Cameron NE,Low PA, Ziegler D, Eds. Stuttgart, Thieme,2003, p. 170–175

18-Melton JL,Dyck PJ.Epidemiology .In Dyck PJ,Thomas PK,Asbury AK,Winergrade AI,Porte Jr. D,editors.Diabeetic neuropathy .Philadelphia:WB Saunders ,1987:27-35.

19-Tseng CH. Prevalence and risk factors of diabetic foot problems in taiwan. Diabetes Care 2003; 26: 3351.

20-Boyko EJ, Ahroni JH , Stensel V , Forsberg RC, Davignon DR , Smith DG. A Prospective Study of Risk Factors for Diabetic Foot Ulcer. Diabetes Care 1999;22: 1036-1042.

21-Murray HJ, Young MJ, Hollis S, Boulton AJ: The association between callus formation,high pressures and neuropathy in diabetic foot ulceration. Diabet Med 1996;13:979–982.

22-McNeely MJ, Boyko EJ, Ahroni JH, Stensel VL, Reiber GE, Smith DG, Pecoraro RE:The independent contributions of diabetic neuropathy and vasculopathy in foot ulceration:how great are the risks? Diabetes Care 1995;18:216–219 .

23-Abbott CA, Vileikyte L, Williamson S, Carrington AL , Boulton AJM . Multicenter study of the incidence of and predictive risk factors for diabetic neuropathic foot ulceration. Diabetes Care 1998; 21:1071-1075