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Foot Bones And Joints Changes In Diabetic Patients-A Case-Control Study.

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ABSTRACT

This work aims to study the feet bone and joint changes in diabetic patient and to identify the causes, relationships and risk factors. To achieve the objectives of this work, two groups of patients were chosen. The first group (case group) includes 60 diabetic patients.46 of them were females and 14 were males who have feet pain, their mean age were 55.56± 9.60 years and mean duration of diabetes were 9.60± 5.56 years . The second group (control group) includes 60 diabetic patients,36 of them were females and 24 were males who have no feet pain, their mean age were 53.00 ± 13.69 years and mean duration of diabetes were 9.02± 5.13 years. The study was conducted between February 2004 and July 2004 at AL-Wafaa diabetes outpatient clinic in Mosul. A detailed history was taken from each patient with clinical examination, investigation and radiological examination of both feet were done. patients older than 65 years, females, housewives ,non compliant with treatment, family history of diabetes, prolonged duration of > 10 years, autonomic neueropathy, high FBG and impaired renal function are more liable to have feet joint changes in diabetes, while there is no association with BMI, uric acid and ESR. The main findings were sensory neuropathy. Deformity, callus, ulcer, muscle wasting and limitation of movement of the mid tarsal and subtalar joints. The main radiographic findings were osteopenia, calcanial spur, hyperostosis of the shafts of the metatarsals and resorption of the Tufts of the distal phalanges. Feet joint changes in diabetes are mainly the result of peripheral neuropathy , which in turn due to prolonged duration and poor control of diadetes. The main feet joint changes in diabetes were swelling, tenderness, deforminty and limitation of movements. Therefore, good control of diabetes is the most important factor in reducing the feet joint changes in diabetics. In addition, every diabetic patient should have examination of the feet at routine consultation for the diabetic statue.

Keywords: Feet, Joint, Changes, Diabetes.

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INTRODUCTION

Diabetes mellitus is associated with a wide variety of musculo skeletal problems(1). Diabetes mellitus can lead to foot problems because it may predispose to peripheral vascular atheroselerosis with associated ischemia, or it may cause peripheral neuropathy with altered proprioception, touch or pain sensation ,and secondary atrophy of skeletal muscles in the legs and feet(2). The likelihood of involvement of the nervous system by diabetes mellitus increases with the duration of disease and is influenced by the degree of glycemic control(3). Diabetic neuropathy currently is the most common cause of neuropathic arthropathy(1,4,5). Neurouparthic arthropathy (Charcot joint, diabetic osteoarthropathy) is adestructive bone and joint condition that is the consequence of peripheral neuropathy ; most commonly affects the foot (4). The incidence of arthropathy was estimated to be between 0.1% and 0.5% with an approximately equal sex ratio (6). The presence of sensory neuropathy is the only established risk factor for neuropathic arthropathy (4). Two major theories have been proposed to explain the development of neuropathic arthropathy, the neurovascular and the neurotraumatic theories(4,5). The neurovascular theory postulates that joint denervation produces physiologic changes, such as increased blood flow from the loss of sympathetic regulation, these changes promote an imbalance between bone resorption and formation, resulting in osteopenia. The neurotraumatic theory proposes that repeated episodes of minor trauma to joints unprotected by the usual response to pain facilitate damage through further trauma and inadequate repair. Neuropathic arthropathy typically presents as an acute or subacute (monarthritis with swelling ,erythema and variable amount of pain) in the affected joint. The two most constant clinical features of neuropathic arthropathy are the presence of a significant sensory deficit and a degree of pain that is less than would be expected considering the amount of joint destruction evident on radiography(4). The most common sites of neuropathic joints are the tarsometatarsal joints followed by the metatarsophalangeal and tibiotalar joints(1,2,4,5). The diagnosis of neuropathic arthritis is based on the clinical features and characteristic radiographic finding(5). The radiographic picture in diabetes mellitus has been divided into a destructive type affecting tarsal bones and reabsorptive or mutilating type confined to the forefoot(1). Forgacs described the three roentgenographic stages of diabetic neuropathic joint diseases, with stage I(initial finding) demonstrating only osteoporosis and cortical defects leading to stage II (Progression) with osteolysis and fragmentation. In stage III (healing) there is deformity, ankylosis, refilling of cortical defects and restitution(7).

A resorptive osteoarthropathy (osteolysis of the forefoot; diabetic osteopathy) appears to be one distinct and unique complication of diabetes mellitus(8). It is usually first recognized as a "spotty" or generalized osteopenia of the distal metatarsal bones and proximal parts of the phalanges, often accompanied by variable degree of pain. The defect may progress to complete lysis of bone ends, resulting in partial or complete dissolution of distal metatarsals. proximal. phalanges ,and distal phalangeal tips, but usually sparing the central diaphysis. Eventually, the residual bone ends may become tapered and sharp, and secondary subluxation of the metatarsophalangeal joints commonly occurs.The etiology of diabetic osteolysis is unknown, it may develop in the absence of obvious infection and in the presence of an apparently intact peripheral nervous and vascular system. Indeed, the tissue microcirculation must be excellent in order to promote such active bone resorption(9). Conceivably. very subtle degrees of sepsis, peripheral nerve damage, tissue ischemia. and trauma acting in concert may all play a role in the development of this characteristic lesion(10). In diabetes of long duration. foot deformities are nearly universal. The most common abnormalities were angular., usually hallux valgus and hammer toe. Foot deformities in diabetic patients have several causes. Of course some are old, long tolerated abnormalities that only become problems when circulation and sensation diminish. Neuropathy of the motor nerves contributes to angular deformities of the toes and distal foot..At firs the most distal muscles. The intrinsic muscles of the foot, seems to be affected. The loss of balancing lumbrical function results in extension of the metatarsophalangeal joints of the toes and flexion of the proximal interphalangeal joints. The result of this imbalance is hammer toes. In addition, loss of abduction function of the dorsal interossei causes the toes to become crowded on the axis of the second digit, accentuating the angular prominences at the first and fifth metatarsophalangeal joints (bunion and bunionette)(11). The neuropathic arthropathy of diabetes, or charcot's foot, eventually results in a collapse of the mid foot(12,13). The subsequent rocker-bottom foot. medial tarsal subluxation, digital Subluxation and bone fragment 0ffer additional opportunity for ulceration, including the unique mid plantar ulcer(11).

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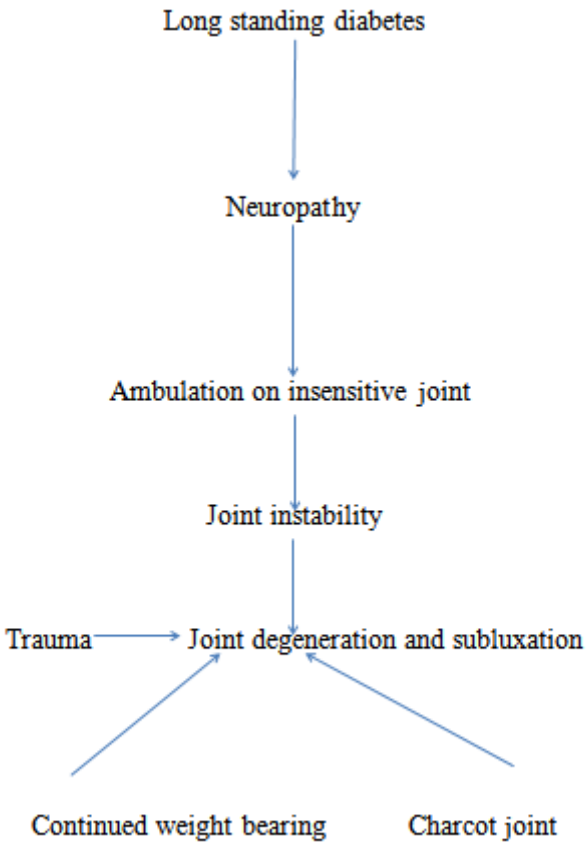

Pathogenesis of neuro-arthropathy in the diabetic foot (2)

Aims of the study: The aims of this study is to identify the feet bones and joints changes in diabetic patients with foot pain, their causes, relationship and risk factors.

Patients and methods:

Study design: The study was conducted as a case-control study at Al-Wafaa diabetes outpatient clinic in Mosul.

Period of study: The period of data collection and lab. work was six months started at the first of February, 2004 and completed the end of July, 2004.

Study population: The sample of the study includes 120 diabetic patients , 82 were females and 38 were males divided into two groups depending on the presence or absence of foot pain.

The first group (case group) consists of 60 diabetic patients, complaining from pain in both feet. their mean age was 55.56± 9.60 years and mean duration of diabetes was 9.60 ± 5.65 years. The second group (control group) consists of 6) diabetic patient, their mean age was 53.00±13.69 years and mean duration of diabetes was 9.02± 5.13 years and they have no foot pain.

Patient definition:

Inclusion criteria: Patients included in this study were those attending Al-Wafaa diabetes out-patient clinic who were known to be the diabetic for 3 years duration and more. This is because although symptoms of diabetic neuroarthropathies are usually related to the prolonged duration of diabetes, on occasion the neuropathic disorder can be the presenting feature of newly diagnosed diabetes (2,11).

Exclusion criteria: patients excluded from this study include:

I. Diabetes mellitus of less than 3 years duration
II. Patients who have a history of chronic rheumatic diseases causing foot problems e.g., R. A, Seronegative spondyloarthropathies and gout.
III. Trauma to the feet.
IV. Neuropathy not due to diabetes mellitus.

Data collection form and lab. Work

General information and history: A questionnaire form was filled for every patient through direct interviewing by the investigator himself (See Appendix 1 page 32). BMI was calculated as weight in kilograms divided by square of height in meter. The internationally accepted range of BMI, in adult are: Underweight (<18.5), normal (18.5-24.9) overweight (25.0-29.9) obese (30-39.9) and extremely obese (> 40) (14)**.**

Clinical examination

A. An Examination of both feet was conducted for all patients including the presence of joint swelling, erythema, deformity, ulcer, callus and muscle wasting.

The deformities were identified as follow:

Hammer toe: The MTP joint is hyper extended while PIP and DIP joints are in plantar flexion(15).
Claw toe: Fixed flexion deformity of the PIP joint with similar fixed deformity of the DIP joint(16).
Hallux valgus: Lateral deviation of the big toe causes prominence of the first MTP joint(17).
Mallet toe: Results from flexion deformities at the DIP joint(18).
Pes planus: Diminished longitudinal arch(18).
Pes cavus: Increase longitudinal arch(18).

Atrophy of the small muscles of the foot is recognized especially by a hollow in the distal sole (19)**.**

Also included in the examination is the assessment of peripheral pulses, tendon reflexes (ankle jerk), sensory examination including light touch, pinprick and position and the results were categorized as grade "1" present, grade"2" intermittently absent presentation and grade "3" absent. The latter two categories were used as a positive diagnosis of neuropathy. Intermittently absent was included as sensory neuropathy and often has a patchy distribution (20). Cutaneous perception was used to determine neuropathy rather than vibration perception because diminished vibration sensation can occur as a part of normal aging(21) whereas light touch is relatively preserved in old age, and is the most reliable indicator of clinical neuropathy(22). With advancing age, vibration is the sensation most commonly diminished, at the toes and ankles(23). Examination of the feet for the presence or absence of tenderness was also done.

B. Examination for the range of movement of the feet joints(24).

Ankle joint: with the knee moderately flexed and gastrocnemius relaxed, support the lower leg with one hand, holding the foot firmly with the other hand, and passively move the ankle into dorsiflexion (about 20 degree) and plantar flexion (about 45 degree).

Subtalar joint: stabilize the distal leg with one hand and, grasping the heel with the other hand, move the foot into inversion (about 30 degree) and eversion (about 20 degree).

Midtarsal joints: stabilize the calcaneus in one hand and, holding the forefoot in the other hand, rotate the foot along its long axis into eversion (about 40 degree) and inversion (about 30 degree).

MTPJs: test each MTPJ by supporting the metatarsal head between the finger and thumb, and moving the proximal phalanx into extension and flexion. The first MTPJ has about 80-degree extension and 35 degree flexion; the other MTPJs have about 40 degree of extension and flexion.

IPJs: IPJ are tested by fixing the more proximal and moving the more distal phalanx. PIPJs flex to about 50 degree and DIPJs to about 40 degree, with varying extension up to 30 degree.

C. Because it had been mentioned that in patients with diabetic neuroarthropathy of the foot, the presence of lower limb sensory and autonomic neuropathy is invariable (2), and also because in patients with significant peripheral polyneuropathy, some evidence of autonomic neuropathy is almost always present(25), so assessment of the autonomic nervous system is done by measuring the blood pressure in supine and standing position and calculating the differences in the systolic blood pressure. A difference of more than 20 mmHg is regarded as significant i.e. (orthostatic hypotension)(26). Orthastatic hypotension occurs in patients with advanced autonomic neuropathy and is predominantly the result of sympathetic involvement(27).

Lab Investigations:

All patients were sent for ESR, FBG, renal function test (Blood urea, s. creatinine and GUE for proteinuria) and s. uric acid. To calculate the upper limit of ESR for a man, divide the age by two; for a women, add 10 to the age and divide by two(28). Glycated hemoglobin estimation to assess the degree of glycemic control was not done because it was not available at the time of study.

Radiology: X-ray of both feet (AP view) for all patients was done and X-ray of the ankle joint for some patients when indicated was conducted at the institution of Radiology in Mosul and the X-ray films was evaluated by one radiologist.

Data analysis:

The statistical analysis of data was carried out using:

1- Chi-Square test of independence to determine the significance of differences between categorical variables. P-Values of< 0.05 were considered to be statistically significant.
2- The relative risk estimate (Odds ratio) is taken as the ratio of the disease rates in the exposed (case group) and unexposed (control group) portion of the study population.

To determine whether the ods ratios (OR) were significantly different from one another, we calculated 95% confidence intervals (95%CI).

OR ＜ 1: The exposure is negatively related to the disease (protective).
OR = 1: The exposure is not related to the disease (no risk).
OR > 1: The exposure is positively related to the disease (high risk).

RESULTS

Table 1: Shows the relationship between some of the characteristics of the patient, and the feet bones and joints changes in diabetes

Parameters	Case (n=60) No.(%)	Control (n=60) No.(%)	P-value	OR	95% CI
Age (Years)			0.310		
<45	8(13)	14(23)	0.157	0.51	0.20-1.30
45-65	43(72)	40(66)	0.553	1.266	0.59-2.70
65+	9(15)	6(11)	0.408	1.59	0.53-4.76
Sex			0.050	2.19	1.00-4.79
Female	46(77)	36(60)			
Male	14(23)	24(40)			
Occupation			0.009		
Housewife	46(77)	34(57)	0.20	2.51	1.15-5.45

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Employer	13(21)	16(27)	0.522	0.76	0.33-1.76
Retired	1(2)	10(16)	0.004	0.08	0.01-0.46
Type of D.M			0.011		
Type I	17(28)	6(10)		3.55	1.28-9.77
Type II	43(72)	54(90)		0.28	0.10-0.77
Duration of D,M. (years)			0.087		
5<	15(25)	14(23)	0.231	1.10	0.46-2.65
5-10	18(30)	29(48)	0.040	0.46	0.22-0.96
10+	27(45)	17(29)	0.050	2.07	1.00-4.39

Parameters	Case (n=60) No.(%)	Control (n=60) No.(%)	P-value	OR	95% CI
Compliance with treatment			0.000	5.44	2.45-12.07
NO	33(55)	11(18)			
Yes	27(45)	49(82)			
Family history of D.M			0.001	3.47	1.66-7.28
ve+	37(62)	19(32)			
Ve-	23(38)	41(68)			
Associated diseases			0.153		
Ve-	32(54)	41(68)	0. 092	0.53	0.25-1.11
HT	25(41)	15(25)	0.053	2.14	1.00-4.62
HD+	3(5)	4(7)	0.697	0.74	0.16-3.36
BMI			0.639		
<18.5	0(00)	1(2)	0.315	0.00	
18.5-24.9	10(16)	7(11)	0.432	1.51	0.54-4.22
25-29.9	24(40)	24(40)	1.000	1.00	
30-39.9	22(37)	26(44)	0.456	0.76	0.37-1.56
>40	4(7)	2(3)	0.402	2.07	0.38-1.35

1) Patients older t than 65 are more liable feet bones and joints changes in diabetes
2) Female are more liable to have feet bones and joints changes in diabetes.
3) Housewives are at increased risk of feet joint changes in diabetes.
4) Those patients who were type I D.M. are more liable feet bones and joints changes (both).
5) Patients with more than 10 years duration of diabetes are at increased risk of having feet bones and joints changes.
6) Non-compliance with treatment is a presdisposing factor for feet bones and joints changes in diabetes.
7) Those patients with family history of diabetes are more liable feet bones and joints changes.
8) Hypertension associated with diabetes increased the risk of feet bones and joints changes.
9) Extremely obese patients are more liable feet bones and joints Changes.

Table 2: Shows the main feet symptoms at the time of assessment

Parameters	**Case (n=60) No.(%)**	**Control (n=60) No.(%)**	**P-value**
Paraesthesia	57(95)	1(2)	0.000
Swelling (not odema)	17(28)	1(2)	0.000
ulcer	6(10)	0.(0.0)	0.012

Of course the main presenting symptom of the case group is feet pain which might be either burning soles, aching pain or shooting pain, While the control group have no feet pain at all, so the pain symptom is not included in this table. It is obvious from the table that paraesthesia is the main symptom associated with

feet pain. Other symptoms are swelling of the ankle joint or the dorsum of the feet. Ten percent of the case group has foot ulcer.

Table 3: The relationship between the signs at clinical examination and the feet joint changes in diabetes

Parameters	Case (n=60) No.(%)	Control (n=60) No.(%)	P-value
Deformity			0.006
ve+	21(35)	8(13)	
Ve-	39(65)	52(87)	
Ulcer			0.012
ve+	6(10)	0(0.0)	
Ve-	54(90)	60(100)	
Callus			0.037)
ve+	16(27)	7(12)	
Ve-	44(73)	53(88)	
Muscle wasting			0.023
ve+	11(18)	3(5)	
Ve-	49(82)	57(95)	
Peripheral pulse			0.094
Normal	55(92)	59(98)	
Absent	5(8)	1(2)	
Tendon reflex (Ankle jerk)			0.000
Normal	11(18)	46(77)	0.000
Sluggish	10(17)	5(8)	0.168
Present with reinforcement	6(10)	4(7)	0.509
Absent	33(55)	5(8)	0.000
Light touch			0.000
Present	20(34)	56(93)	0.000
Intermittently absent	26(43)	4(7)	0.000
Absent	14(23)	0(0.0)	0.000
Pin prick			0.000
Present	12(20)	55(92)	0.000
Intermittently absent	31(52)	3(5)	0.000
Absent	17(28)	2(3)	0.000
Position			0.000
Present	39(65)	59(98)	0.000
Intermittently absent	9(15)	0(0.0)	0.002
Absent	12(20)	1(2)	0.001
Tenderness			0.000
+ve	24(40)	4(6)	
-ve	36(60)	56(94)	

There is a significant association between deformities, callus, ulcer muscle wasting with the feet bones and joints changes in diabetes. The main types of deformities were hammer toe, claw toe and hallux valgus (Fig 1). Peripheral pulse is not associated with feet bone and joint changes in diabetes (P-value 0.094). Loss of the ankle jerk is associated with the feet joint changes (P-value 0.000). Sensory neuropathy (light touch, pin prick and position) is of statistically significant difference on comparison with the control group. Regarding tenderness a significant difference was demonstrated between the cases and the control group.

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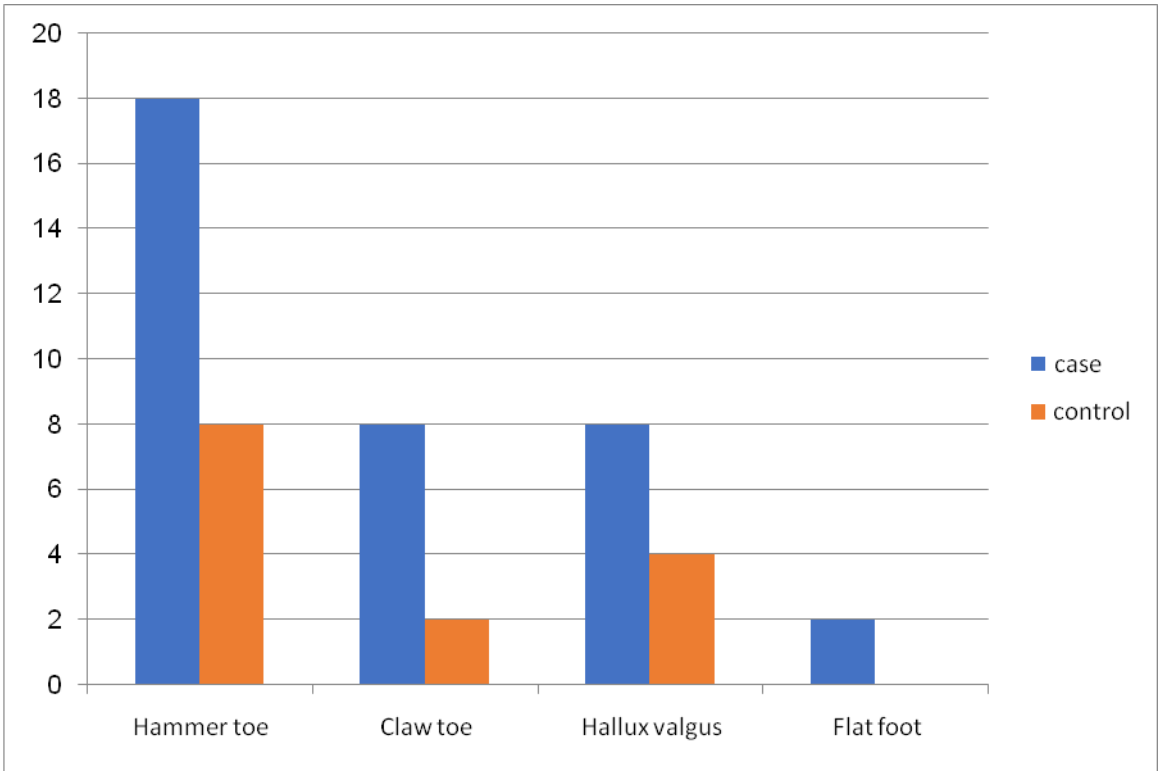

Fig 1: Types of foot deformities in cases and controls

Table 4: Shows the relationship between the restriction of movement of the feet joints and diabetes with pain

Restriction of movement	Case (n=60) No.(%)	Control (n=60) No.(%)	P-value
Ankle: dorsi flexion	16(26)	0(0.0)	0.000
Plantar flexion	9(15)	1(2)	0.008
Subtalar			
Inversion of the calcanium	18(30)	1(2)	0.000
Eversion of the calcanium	18(30)	1(2)	0.000
Mid tarsal:			
Inversion of the mid foot	17(28)	1(2)	0.000
Eversion of the mid foot	17(28)	1(2)	0.000
MTPJs:			
Flexion	9(15)	1(2)	0.008
Extension	9(15)	1(2)	0.008
IPJs Flexion	6(10)	0(0.0)	0.012
Extension	6(10)	0(0.0)	0.012

It is obvious from this table that there is a significant association between the restriction of mobilization of the feet joints and diabetes, also it is obvious from this table that the main affected joints are the subtalar and the midtarsal joints.

Table 5: Difference in systolic blood pressure measurement in supine and standing position (assessment of autonomic nervous system)

Parameters	Case (n=60) No.(%)	Control (n=60) No.(%)	P-value	OR	95% CI

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Total			0.000		
No difference	33(55)	53(88)	0.000	0.16	0.07-0.39
10-20 mmHg	4(7)	2(3)	0.402	2.07	0.38-11.35
20-30	23(38)	5(9)	0.000	6.84	2.59-18.04

Results of this table shows that there is a significant association between autonomic neuropathy and the feet bones and joints changes diabetes, also those with autonomic neuropathy are more liable feet bones and joints changes (P-value 0.000, OR - 6.84 and 95% CI=2.59-18.04) while normal autonomic nervous system is a protective factor (OR =0.16 and 95% CI= 0.07-0.39).

Table 6: Results of basic laboratory investigations

Parameters	Case (n=60) No.(%)	Control (n=60) No.(%)	P-value	OR	95% CI
FB.G (mmol/L)			0.002		
10<	14(23)	32(54)	0.001	0.27	0.13-0.58
10-15	34(57)	23(38)	0.044	2.10	1.02-4.33
> 15	12(20)	5(8)	0.067	2.75	0.93-8.11
Blood area (mmol/L)			0.008	0.15	0.04-0.61
7.1≤	49(82)	58(97)			
7.1>	11(18)	2(3)			
S-creatinine (umol/L)			0.658	0.82	0.32-2.11
<133	46(77)	48(80)			
≥ 133	14(23)	12(20)			
GUE			0.019	0.35	0.15-0.84
Nil	40(66)	51(85)			
Proteinuria-	20(34)	9(15)			
S-uric acid (pumol/L)					
Male			0.154		
480≤	14(23)	33(37)			
480>	0(00)	2(3)			
Female			0.697	0.74	0.16-3.36
360≤	42(70)	33(55)			
360>	4(7)	3(5)			
ESR (mm/hr)			0.695	1.17	0.53-2.56
Increase	20(33)	18(30)			
Normal	40(67)	42(70)			

Patient with high F. B. G are more liable to develop feet bones and joints changes. Normal renal function is a protective factor against feet bones and joints changes. There was no association between ESR and S. uric acid with the feet bones and joints changes.

Table 7: Shows the result of X-ray findings.

Parameters	Case (n=60) No.(%)	Control (n=60) No.(%)	P-value
Osteopenia	13(20)	5(8)	0.041
Osteoarthritis of the first MLTPJ	4(7)	1(2)	0.171
Osteoarthritis of the ankle Joint	4(7)	1(2)	0.171
Calcanial spur	6(10)	0(0.0)	0.012
Vascular calcification	2(3)	0(0.0)	0.154
Flattening of the metatarsal head and the	3(5)	3(5)	1.00

base of the proximal phalanges (early changes of neuropathy).			
Fragmentation and irregularity at the insertion of the Achilles tendon.	3(5)	0(0.0)	0.079
Hyperostosis of the shaft of the metatarsal (early changes of neuropathy)	5(8)	0(0.0)	0.022
Changes of osteomyelitis.	1(2)	0(0.0)	
Resorption of the tuft of distal phalanx.			0.043
A. Of the fifth toe.	2(3)	0(0.0)	
B. Of the first and second toes.	4(7)	0(0.0)	

There is a significant association between osteopenia, calcanial spur, hyperostosis of the shaft of the metatarsals and resorption of the tuft of the distal phalanges of the toes with the feet bones and joints changes in diabetes, while there is no association with vascular calcifications and osteoarthritis of the ankle and first MTPJ.

DISCUSSION

Diabetes mellitus is the most common cause of neuroarthropathy, although less than 1% of patients with this disease develop such changes . The articulation of the foot are affected in the majority of cases(29). In the present study, feet bones and joints change in diabetes in diabetes mellitus were demonstrated that female, housewives, non-compliance with treatment, family history of diabetes and prolonged duration ,autonomic neuropathy, high FBG and impaired renal function are the major risk factors, while there is no association between feet joint changes in diabetes and BMI,s.uric acid and ESR. The e main associated features for those with foot pain are paraesthesia and swelling of the feet and the main findings are sensory neuropathy, deformity, callus, ulcer, in addition to muscle wasting and limitation of movements of the feet joints .Our study shows that old age (> 65 Years) were more susceptible to feet bones and joints changes, however, other workers had shown that usual age of onset is ≥ 50 years(5,11). Esses et al., described neuropathic arthritis in a 21 years-old diabetic(30). Also female sex appears to be a risk factor which is not in agreement with other studies, which concluded that both sexes are equally affected, (1,31), while other studies had shown that male sex is a risk factor(2,32). However, most of the patients in our study were female which might explain our finding. Regarding the occupation, the present study shows that being housewife is a risk factor while retirement is a protective factor. This might be explained by the fact that feet bones and joints changes are induced by stress of weight bearing that leads to gradual damage of the foot(1). In addition to the repeated minor trauma to the joints unprotected by the usual response of pain facilitate damage through further trauma and inadequate repair(4). For the type of diabetes, in the present study those patients who were type I D.M. were more liable to develop feet bones and joints changes. This is in accordance with other studies(2,3,9)**.** For the duration of diabetes,the present study shows that duration of more than 10 year is a risk factor for feet bones and joints changes, this is in line other studies (5,11,33). Non-compliance with treatment in the present study appears to be a strong risk factor for feet bones and joints changes in diabetes that leads to unproper control of the disease, this is in agreement with other studies (1,3,5). Regarding family history of diabetes, the present study shows that family history of diabetes is a risk factor for feet bones and joints changes. Although I couldn't find such a study, but genetic factors are known to run in families with diabetes. Of the associated diseases, hypertension appears to increase the risk of feet joint changes in diabetes. This is in line with other studies(5). BMI: The present study shows that extreme obesity is associated with increased risk of feet bones and joints changes in diabetes. (OR= 2.07 and 95% , CI= 0.38-1.35) I could not find a study correlating the obesity with diabetic neuroarthropathy, but it had been mentioned that the majority of type2 diabetes are obese(2,19,34). The other possible explanation is that overweight leads to more trauma to the non sensitive feet joints which in turn causes more joints destruction. Regarding the symptoms: The case group of course are complaining from pain which may be burning sole. aching pain and shooting pain, this is in line with other studies(21,35). Other symptoms like paresthesia, swelling, and ulcer are associated with feet bones and joints changes in diabetes. These are in agreement with other studies (4,5,9,36). In the present study there was a significant association between deformities and the feet joint changes in diabetes. The main deformities were hammer toe. claw toes and hallux valgus, this is in agreement with other studies(1,2,11,37), however, another study show no association between diabetes and claw toes(38).

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Also in the present study there was a significant association between ulcer, callus, muscle wasting, and the feet bones and joints changes. This is in agreement with other studies, which had mentioned atrophy of the interossei muscles of the foot, leading to foot deformities with dorsal prominence or plantar protrusions. Callus formation occur over weight bearing areas in these anaesthetized areas leading to ulcer formation, this is in agreement with other studies(1,5,9). Peripheral pulses are not associated with the feet bones and joints changes in diabetes. This is not agreement with other studies which had mentioned that diabetic neuroarthropathy is associated with normal or bounding pulse(I,2,21). Neurological examination shows that loss of ankle jerk, and sensory neuropathy are associated with feet bones and joints changes. These are in agreement with other studies which had shown that feet joint changes in diabetes are mainly due to the impact of neuropathy(1,2,4 9,19,22). Regarding autonomic neuropathy, in our study there is a very highly significant association between autonomic neuropathy and the feet bones and joints changes, and autonomic neuropathy is a risk factor. This is in concordance with other studies(2,4,21). Regarding the results of basic laboratory investigations, the present study shows that there is a very highly significant association between FBG and the feet bones and joints changes and that high FBG is a risk factor. This is in agreement with other studies(5,13,19), Some studies show no significant relationship between the development of neuropathy and the severity of the disease or the degree of blood glucose control(11). Normal renal function appears to be a protective factor. This is in agreement with other studies, which had shown that peripheral neuropathy is associated with chronic renal insufficiency(23), also diabetic nephropathy is associated with prolonged duration and poor glycemic control which are important predisposing factors feet bones joints changes in diabetes. The present study shows no significant association between the serum uric acid and the feet bones and joints changes in both groups. This is in line with other study(1). Other studies show that hyperuricemia had been detected in a significant number of diabetic patients(26,35). Other studies show a correlation between serum uric acid level and body weight in diabetes(2,9). Other causes of hyperuricemia in diabetic patients are the chronic renal diseases and the use of thiazide diuretics(2). Regarding ESR, the present study shows no significant association between the level of ESR and the feet joint changes. This is in line with other studies(4,24,27). The main radiographic finding in the present study is osteopenia, this is agreement with other studies (27). This might be related to the fact that sympathetic autonomic neuropathy lead to increased blood flow and Osteopenia. The other significant finding is the calcanial spur, this is in agreement with other study (24,25,38). Also there is a significant association between hyperostiesis of the shafts of the metatarsals, periosteal reaction, resorption of the tufts of the distal phalanges and the feet joint changes. These are in consistence with other studies(2,5,22,31). Of the non significant finding is the osteoarthritis of the ankle and the first MTPJ. This is not in agreement with other studies (1,24,25,28). The other non-significant finding is vascular calcification this is line with other studies(2). Others had mentioned although vascular calcification is a characteristic finding, but it is not associated with severity of the disease(30,39). There was only one case of osteomyelitis. Flattening of the metatarsal head and the base of the proximal phalanges which are early changes of neuropathy is present in equal number of the case and control group.

CONCLUSION

From the results of the present study.it may be concluded that. Feet bones and joints changes in diabetes are mainly due to peripheral neuropathy and that poor control of diabetes and prolonged duration are the main leading causes of these changes. In addition, it appears that female, housewives, and those with hypertension. family history of diabetes, autonomic neuropathy and impaired renal function are more liable to have feet joint changes in diabetes while there was no association with BMI, s. uric acid and ESR. The main feet were bones and joint changes were swelling, tenderness, deformities and limitation of movements. The main radiographic findings were osteopenia. calcanial spurs hyperostosis of the shafts of the metatarsals and resorption of the tufts of the distal phalanges.

Appendix 1

Serial no.		Date:		
Name:		Age		Sex
Occupation		Address		
Type of diabetes	Type I	Type II	Duration:	
Compliance with treatment		Yes	No	

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Family history of diabetes	+ve	-ve
Associated diseases:		
Weight (kg):	Height (m):	B.M.I:

For cases only	Duration of feet pain
	Types of pain:
	Burning soles:
	Aching pain:
	Shooting pain:

Symptoms at the time of assessment

	Site	Duration
Paraesthesia:		
Swelling (not odema):		
Ulcer:		

Appendix 2 (Examination of feet)

Parameters		RT	LT
Deformity:	+ve		
	-ve		
Ulcer	+ve		
	-ve		
Muscle wasting:	+ve		
	-ve		
Peripheral pulse	Normal		
	Reduced		
	Absent		
Tendon reflexes: Ankle jerk:	Normal		
	Sluggish		
	Present with reinforcement		
	Absent		
Light touch:	Present		
	Intermittently absent		
	Absent		
Pin Prick:	Present		
	Intermittently absent		
	Absent		
Position:	Present		
	Intermittently absent		
	Absent		
Tenderness	+ve		
	-ve		

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Parameters		Right	Left
Ankle	Dorsiflexion		
	Plantar flexion		
Subtalar:	Inversion of calcanium		
	Eversion of calcanium		
Midtarsal	Inversion of mid foot		
	Eversion of mid foot		
MTPJ	Flexion		
	Extension		
IPJ	Flexion		
	Extension		

Blood Pressure (mmHg):	Supine:	Standing
Investigations:		
FBG (mmol/L)		
Bd. Urea (mmol/L)		
S. creatinine (u mol/L)		
GUE (Protcinuria)		
S. unic A. (u mol/L)		
E.S.R (mm/hr.)		

Appendix 3: Rheumatologic manifestations of diabetes mellitus)

Neuropathy	
	Monoeuropathy multiplex
	Diabetic amyotrophy
	Radiculopathy
	Autonomic neuropathy
Neuroarthropathy:	Osteolysis
	Osteoporosis
	Charcot joint
	Coexistent osteomyelitis
	Periarthritis of the shoulder (adhesive capsulitis)
Other Associations:	Hyperuricemia and gout?
	Ankylosing hyperostosis.
	Flexion contractures (Duputren's conrasturcs, limited joint mobility).
	Tenosnyovitis

Recommendations:

1- Good control of diabetes.
2- Any patient with peripheral neuropatly should be regarded as potential victim of neuroarthropathy, so regular examination and follow up is mandatory.
3- Regular attendance of the patients at the diabetes outpatient clinic to identify, advise and educate those patients at risk.
4- Every diabetic should have examination of the feet at routine consultation for the diabetic state.

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