Prevalence of Factors Predisposing to Foot Complication and Their Relation to Other Risks

Charnvate Sattaputh MD*,
Somkiat Potisat MD**, Amporn Jongsareejit MD***,
Udom Krairittichai MD****, Kunakorn Pooreesathian MD*

** Department of Surgery, Lerdsin Hospital, Ministry of Public Health, Bangkok, Thailand*
*** Institute of Medical Research and Technology Assessment, Department of Medical Services, MOPH, Nonthaburi, Thailand*
**** Prasat Neurological Institute, Department of Medical Services, MOPH, Bangkok, Thailand*
***** Department of Medicine, Rajavithi Hospital, College of Medicine, Rangsit University, Bangkok, Thailand*

Background: *Diabetic neuropathy and peripheral vascular disease (PVD) have been identified as major risk factors for diabetic foot ulceration and amputation in patients with type 2 diabetes mellitus (T2DM) but in Thailand have been a few of prospective studied about the prevalence and risk factors of diabetic neuropathy and PVD.*
Objective: *To evaluate the prevalence of diabetic neuropathy and PVD in Thai patients with T2DM and to determine other factors that related with them.*
Material and Method: *A cross-sectional study of 899 Thai T2DM patients from the out-patient department of seven public hospitals in Thailand between January 2007 and September 2008 was performed. Histories of these patients, complete physical examinations, feet examination, and blood with urine chemistry were obtained.*
Results: *Most of the patients were females with the average age of 59.64 years, the average of body mass index (BMI) was 27.32 kg/m^2, the average duration of diabetes was 8.12 years and 85.17% of patients were HbA1C ≥ 7%. Diabetic patients with PVD group compared with no PVD group, there were statistically significant differences for duration of having diabetes (OR 1.08; 95% CI [1.01-1.16]; p-value 0.047), creatinine level (OR 1.62; 95% CI [1.12-2.33]; p-value 0.01), present diabetic neuropathy (OR, 7.37; 95% CI [2.52-21.59]; p-value < 0.001). Patients with diabetic neuropathy group compared with no diabetic neuropathy group, there were statistically significant differences of age (OR, 1.04; 95% CI [1.01-1.06]; p-value 0.003), duration of having diabetes (OR, 1.04; 95% CI [1.01-1.07]; p-value 0.008), on ACEI or ARB drug (OR, 1.77; 95% CI [1.24-2.55]; p-value 0.002), HbA1C (OR, 1.14; 95% CI [1.03-1.27]; p-value 0.012), creatinine level (OR, 1.38; 95% CI [1.04-1.79]; p-value 0.014), present diabetic retinopathy (OR, 1.96; 95% CI [1.22-3.13]; p-value 0.005), present PVD (OR, 7.37; 95% CI [2.52-21.59]; p-value < 0.001), present diabetic nephropathy with microalbuminuria (OR, 1.74; 95% CI [1.12-2.69]; p-value 0.013).*
Conclusion: *Two percent of T2DM patients had PVD that associated with duration of diabetes, creatinine level, and diabetic neuropathy and 15% of T2DM patients had diabetic neuropathy that depended on age, duration of having diabetes, on ACEI or ARB drug, creatinine level, HbA1C, diabetic retinopathy, diabetic nephropathy, and PVD.*

Keywords: *Diabetic foot, Diabetes mellitus*

J Med Assoc Thai 2012; 95 (8): 1013-20
Full text. e-Journal: *http://jmat.mat.or.th*

Diabetic foot ulceration is a major cause of morbidity, disability, as well as emotional and physical costs for people with diabetes. Furthermore, it consumes a major portion of the resources allocated for the treatment of diabetes[1]. The number of people with diabetes worldwide was estimated at 131 million in 2000; it is projected to increase to 366 million by 2030[2]. Estimates show that the foot ulceration may occur in up to 15% of diabetic patients during their lifetime[3]. The diabetic foot ulceration is a significant cause of morbidity and can lead to prolonged hospital stays, which is evidenced by the fact that about 20% of hospitalizations related to diabetes involve diabetic foot ulceration[4]. Furthermore, about 60,000 major lower-extremity amputations are performed annually on American patients with diabetes[5]. In Thailand, Sirinate et al have determined the prevalence of lower extremity amputation in Thai diabetics patients was 1.5% and revealed a high risk of lower extremity

Correspondence to:
Sattaputh C, Department of Surgery, Lerdsin Hospital, Silom Rd, Silom, Bangrak, Bangkok 10500, Thailand.
Phone: 0-2353-9801, Fax: 0-2353-9801 ext. 5116
E-mail: scorpionpon2@hotmail.com

amputation in patients with a history of ulcer, peripheral vascular disease, diabetic retinopathy and insulin injection[6]. Sriussadaporn S et al was conducted to determine factors involved in foot ulceration in Thai non-insulin-dependent diabetes (Type 2). Diabetic patients were associated with three factors, which were peripheral neuropathy, visual impairment, and poor glycemic control[7]. The mortality rate in patients with diabetic foot ulceration is also high and is approximately twice that of patients without ulceration[8]. Early recognition and management of independent risk factors for ulcers and amputations can prevent or delay the onset of adverse outcomes.

Diabetic neuropathy and peripheral vascular disease (PVD) have been identified as major risk factors for diabetic foot ulceration and amputation[9,10]. Autonomic neuropathy can cause increased blood pooling and swelling in the foot. Motor neuropathy leads to atrophic changes in the foot musculature that cause foot deformity and decreased joint mobility. These problems subsequently lead to an area of increased plantar foot pressure. The lack of protective sensation from sensory neuropathy leads to repetitive trauma from an area of high pressure that results in ulceration. Peripheral vascular disease has been observed to affect vessels below the knee in patients with diabetes. Even in the face of non-obstructed vessels, impaired microvascular reactivity diminishes blood supply to the ulcerated areas[11].

The purpose of the present study was to evaluate the prevalence of diabetic neuropathy and peripheral vascular disease in diabetic patients and to evaluate other factors that related to diabetic neuropathy and PVD.

Material and Method

The present study was a cross-sectional study in Thai diabetes mellitus type 2 (T2DM) patients, performed between January 2007 and September 2008. The Ethics Review Committee for Research in Human Subjects, Ministry of Public Health approved the present study and all patients signed informed consent after reviewing a written summary of the study plan. The authors recruited T2DM patients diagnosed with the American Diabetes Association's criteria[12] from out-patient departments of seven public hospitals, including Rajvithi Hospital (Bangkok), Lerdsin Hospital (Bangkok), Nopparatrajathanee Hospital (Bangkok), Mettaphacharak Hospital (Nakhonpathom), Pathumthani Hospital (Pathumthani), Ladlumkeaw Hospital (Pathumthani) and Nongsau Hospital (Pathumthani). Exclusion criteria were pregnancy, breast feeding, acute systemic disease and other renal diseases. Histories of these patients and complete physical examinations were obtained. Height, weight, and blood pressure were measured using standard procedures. The mean of two separate blood pressure results was taken as the final blood pressure recording. Two fasting blood and three random spot urine samples from all patients were collected over three consecutive months.

A physical examination with emphasis on the lower extremities was performed by a group of physicians including the author as the team leader. All physicians were instructed and trained for specific examination and interpretation by the author. The examination consists of three main components, 1) Shapes of the foot and the toes, with special consideration on toe deformities, Bunions, Charcot deformities and prominent metatarsal head, 2) Characteristics of Dorsalis Pedis and Posterior Tibial pulses. 3) Detection of neuropathy, by testing for loss of protective sensation. The authors used 10-gramm Semmes-Weinstein 5.07 monofilament to determine the protective sensation[13,14] in seven areas of each foot and defined loss of protective sensation as the inability to feel the 10-gram Semmes-Weinstein 5.07 monofilament at one or more locations on the foot except the heel. Because the epidermis on the heel is thicker than the other area hence, only single area of sensation loss at the heel cannot represent sensory loss of the foot. PVD was defined by absent or diminished Dorsalis Pedis and/or Posterior Tibial artery pulses to palpation in the same limb.

Fasting blood sugar, HbA1c and serum creatinine were determined by hexokinase enzymatic, immunoturbidimertric (DCCT/NGSP) assay and the Jaffe method (rate-blanked and compensated) using a COBAS INTEGRA 400® analyzer (Roche Diagnostics, Indianapolis, IN, US). The quantity of urinary albumin concentration was determined by immunoturbidimetric assay and urine creatinine concentration by the Jaffe method (rate-blanked and compensated) using COBAS INTEGRA 400® analyzer.

Using the World Health Organization classification for adult Asians, BMI above 25 kg/m^2 was considered to constitute obesity. Subjects with systolic blood pressure over 130 mmHg and/or diastolic blood pressure over 80 mmHg were considered to have uncontrolled hypertension. Glomerular filtration rate (GFR) was estimated by using the abbreviated Modification of Diet in Renal Disease (MDRD)

formula[12]. Urine albumin to creatinine ratio (UACR) was classified into three groups, less than 30, 30 to 300 and more than 300 mg/gm. Normoalbuminuria, MA and macroalbuminuria were defined as the presence of at least two out of three spot urine tests for UACR showing less than 30, 30 to 300 and more than 300 mg/gm respectively.

The statistics for this research was the determination factor shown as mean ± standard deviation. Univariate comparisons of the independent variables of continuous and categorical dependent variables were made by using unpaired student's t-test and Pearson Chi-square test. Multivariate regression was used to assess the associated risk factors of PVD and loss of feet sensation with odds ratio and 95% confidence interval (95% CI). A p-value of less than 0.05 was considered as statistically significant. Analysis was made by SPSS, the software program for Windows version 17.0 (SPSS Inc., Chicago, Illinois, USA).

Results

Eight hundred ninety nine diabetes patients were studied and most of them were females. Six hundred forty two women (71.41%) with the average age was 59.64 ± 9.90 years were included in the present study. The average of body mass index (BMI) was 27.32 ± 4.71 and 751 cases (84.48%) were classified as overweight to obesity (BMI above 23.0 mg/m^2). Duration of diabetes was varied from less than 1 year to 34 years with average duration 8.12 ± 6.12 years. Seven hundred and fifty eight cases (84.31%) had HbA1C ≥ 7%. The average of low-density lipoproteins (LDL) was 139.12 ± 41.16 and 738 cases (82.74%) had LDL ≥ 100 mg%. Forty-three patients (4.78%) were ex-smokers and 75 patients (8.34%) were current smokers. The diabetic patients had systolic blood pressure (SBP) ≥ 130 mmHg or diastolic blood pressure (DBP) ≥ 80 mmHg, which were 435 (48.66%) and 177 (19.80%) patients, respectively. Foot deformities (toe deformities, Bunions, Charcot foot and prominent metatarsal head) were found in 5.89%, 3.44%, 0.22%, and 1.56% of patients. No patient had lower extremities amputations. One hundred forty two patients (15.97%) had loss of protective sensation. Nineteen patients (2.11%) had loss of pedal pulses. Retinopathy was detected in 234 patients (26.20%) and nephropathy, 326 patients (37.17%) as shown in Table 1.

Diabetes patients with pedal pulses deficit group compared with no pedal pulses deficit group, there were statistically significant differences for

Table 1. Characteristic (n = 899)

Clinical evaluation	n	%
Duration of diabetes (yr) (n = 888)		
≤ 10	617	69.48
> 10	271	30.52
Mean (SD)	8.12 (6.12)	
BMI (kg/m^2) (n = 889)		
< 18.5	15	1.69
18.5-22.9	123	13.84
23-24.9	160	18.00
25-29.9	355	39.93
≥ 30	236	26.55
mean(SD)	27.32 (4.71)	
HbA1C (%) (n = 899)		
< 7	141	15.68
≥ 7	758	84.31
Mean (SD)	8.77 (1.85)	
LDL (mg/dl) (n = 892)		
Optimal (< 100)	154	17.26
Abnormal	738	82.74
Mean (SD)	139.12 (41.16)	
Creatinine (mg/dl) (n = 892)		
< 1.4 in female / < 1.5 in male)	830	93.05
≥ 1.4 in female / ≥ 1.5 in male)	62	6.95
Mean (SD)	0.95 (0.56)	
Presence retinopathy (n = 893)		
No	659	73.80
Yes	234	26.20
PVD (n = 899)		
No	880	97.89
Yes	19	2.11
Smoking (n = 899)		
No	781	86.87
Ever	43	4.78
Yes	75	8.34
Systolic blood pressure (n = 894)		
< 130	459	51.34
≥ 130	435	48.66
Mean (SD)	130.88 (16.31)	
Diastolic blood pressure (n = 894)		
< 80	717	80.20
≥ 80	177	19.80
Mean (SD)	71.89 (9.41)	

duration of having diabetes (adjusted odds ratio [OR], 1.08; adjusted 95% CI [1.01-1.16]; adjusted p-value = 0.047), creatinine level ([OR], 1.62; 95% CI [1.12-2.33]; p-value = 0.01), present diabetic neuropathy (loss of protective sensation) ([OR], 7.37; 95% CI [2.52-21.59]; p-value < 0.001) and estimated GFR (eGFR) but these were not statistically significant differences in adjusted data. The others are shown in Table 2.

Patients with diabetic neuropathy (loss of protective sensation) group compared with no diabetic neuropathy group, there were statistically significant differences of age (adjusted [OR], 1.03; adjusted 95% CI [1.01-1.06]; adjusted p-value 0.003), duration of having diabetes (adjusted [OR], 1.04; adjusted 95% CI [1.01-1.07]; adjusted p-value 0.008), on ACEI or ARB drug ([OR], 1.77; 95% CI [1.24-2.55]; p-value = 0.002), HbA1C (adjusted [OR], 1.14; adjusted 95% CI [1.03-1.27]; adjusted p-value = 0.012), creatinine level ([OR], 1.38; 95% CI [1.04-1.79]; p-value = 0.014), present diabetic retinopathy (adjusted [OR], 1.96; adjusted 95% CI [1.22-3.13]; adjusted p-value 0.005), present vascular complication (pedal pulses deficit) ([OR], 7.37; 95% CI [2.52-21.59]; p-value < 0.001), present diabetic nephropathy with microalbuminuria (adjusted [OR], 1.74; adjusted 95% CI [1.12-2.69]; adjusted p-value 0.013) and present diabetic nephropathy with macroalbuminuria but these were not statistically significant differences in adjusted data. The others are shown in Table 3.

Discussion

Risk identification is fundamental for effective preventive management of the foot in people with diabetes. The risk of ulcers or amputations is increased in people who have had diabetes ≥ 10 years, are male, who have poor glucose control, or have cardiovascular, retinal, or renal complications. The following foot-related risk conditions are associated with an increased risk of amputation[15]. The present study did not have patients with foot ulceration or amputation because most patients have had diabetes < 10 years.

Table 2. Diabetic patients with absent pedal pulses

Factors	Crude OR	Adjusted OR	Crude 95% CI of OR	Adjusted 95% CI of OR	Crude p-value	Adjusted p-value
Gender (M/F)	1.16	1.56	0.36-3.30	0.48-5.13	0.771	0.463
Age (yr)	1.05	1.03	0.99-1.11	0.96-1.11	0.115	0.421
Duration (yr)	1.12	1.08	1.05-1.19	1.01-1.16	0.001	0.047
SBP	1.01	0.70	0.97-1.04	0.20-2.55	0.747	0.593
DBP	0.95	1.62	0.89-1.01	0.28-9.23	0.077	0.588
BMI	0.90	0.89	0.79-1.02	0.76-1.04	0.102	0.143
HbA1C	0.85	0.85	0.61-1.17	0.59-1.22	0.318	0.376
LDL	0.99	-	0.99-1.01	-	0.780	-
Creatinine	1.62	-	1.12-2.33	-	0.010	-
eGFR	0.98	0.98	0.96-0.99	0.96-1.01	0.026	0.116
Diabetes retinopathy	1.89	2.75	0.67-5.33	0.82-9.20	0.230	0.100
Smoking						
Ever	1.01	-	0.13-7.74	-	0.993	-
Yes	-	-	-	-	-	-
On ACE or ARB*	0.84	-	0.33-2.16	-	0.722	-
Present neuropathy	7.37	-	2.52-21.59	-	<0.001	-
Present nephropathy						
Microalbuminuria	0.80	-	0.22-2.99	-	0.744	-
Macroalbuminuria	1.25	-	0.27-5.89	-	0.774	-

* This research study used a population with "Prevalence and Risk Factors of Diabetic Nephropathy among Thai Patients with Type 2 Diabetes Mellitus" is a study of risk factors for drug use ACEI or ARB{J Med Assoc Thai 2011;94 (Suppl.2): S1-S5}

Table 3. Diabetic patients with loss of feet sensation

Factors	Crude OR	Adjusted OR	Crude 95% CI of OR	Adjusted 95% CI of OR	Crude p-value	Adjusted p-value
Gender (M/F)	0.87	0.75	0.76-1.72	0.45-1.24	0.487	0.259
Age (yr)	1.04	1.04	1.02-1.06	1.01-1.06	<0.001	0.003
Duration (yr)	1.07	1.04	1.04-1.09	1.01-1.07	<0.001	0.008
SBP	1.01	0.81	1.00-1.02	0.52-1.27	0.033	0.359
DBP	0.99	1.54	0.97-1.01	0.91-2.62	0.214	0.109
BMI	0.99	-	0.95-1.03	-	0.674	-
HbA1C	1.12	1.14	1.01-1.26	1.03-1.27	0.019	0.012
LDL	0.99	-	0.99-1.00	-	0.430	-
Creatinine	1.38	-	1.04-1.79	-	0.014	-
eGFR	0.99	0.99	0.98-0.99	0.99-1.00	0.001	0.210
Diabetes retinopathy	2.37	1.96	1.55-3.62	1.22-3.13	<0.001	0.005
Smoking						
Ever	1.54	2.58	0.72-3.31	1.08-6.19	0.266	0.033
Yes	1.66	1.70	0.93-2.95	0.83-3.47	0.085	0.145
On ACE or ARB	1.77	-	1.24-2.55	-	0.002	-
PVD	7.37	-	2.52-21.59	-	<0.001	-
Present nephropathy		-		-		-
Microalbuminuria	2.10	1.74	1.40 -3.14	1.12-2.69	<0.001	0.013
Macroalbuminuria	1.90	1.42	1.09 -3.30	0.76-2.66	0.024	0.269

Diabetic foot ulcers result from the simultaneous action of multiple contributing causes[16-18]. The major underlying causes are noted to be peripheral neuropathy and ischemia from peripheral vascular disease[19].

PVD is a contributing factor to the development of foot ulcers in up to 50% of cases[20,21]. It commonly affects the tibial and peroneal arteries of the calf. Endothelial cell dysfunction and smooth cell abnormalities develop in peripheral arteries as a consequence of the persistent hyperglycemic state. There is a resultant decrease in endothelium-derived vasodilators leading to constriction. Further, the hyperglycemia in diabetes is associated with an increase in thromboxane A2, a vasoconstrictor and platelet aggregation agonist, which leads to an increased risk for plasma hypercoagulability[22]. There is also the potential for alterations in the vascular extracellular matrix leading to stenosis of the arterial lumen[23]. Moreover, smoking, hypertension, and hyperlipidemia are other factors that are common in diabetic patients and contribute to the development of PVD. Cumulatively, this leads to occlusive arterial disease that results in ischemia in the lower extremity and an increased risk of ulceration in diabetic patients.

PVD is the risk for ulceration and interference with ulcer healing. Smoking is a risk factor for PVD. In this study, 13.13% of patients were smoking. Pedal pulse palpation is expedient and good screening. The present study showed 2.11% absence of pedal pulses. These patients had an indication for ankle brachial index (ABI) and further vascular evaluation. The present study showed that PVD are found to be higher in patients who have longer duration of diabetes and renal insufficiency because these problems are contributing factors of degenerative changes that are related with PVD[24,25].

More than 60% of diabetic foot ulcers are the result of underlying neuropathy[19]. The development of neuropathy in affected patients has been shown in animals and in vitro models to be a result of hyperglycemia-induced metabolic abnormalities[22]. Neuropathy in diabetic patients is manifested in the motor, autonomic, and sensory components of the nervous system[19]. Damage to the innervations of the intrinsic foot muscles leads to an imbalance between flexion and extension of the affected foot. This produces anatomic foot deformities that create

abnormal bony prominences and pressure points, which gradually cause skin breakdown and ulceration. Autonomic neuropathy leads to a diminution in sweat and oil gland functionality. As a result, the foot loses its natural ability to moisturize the overlying skin and becomes dry and increasingly susceptible to tears and the subsequent development of infection. The loss of sensation as a part of peripheral neuropathy exacerbates the development of ulcerations. As trauma occurs at the affected site, patients are often unable to detect the insult to their lower extremities. As a result, many wounds go unnoticed and progressively worse as the affected area is continuously subjected to repetitive pressure and shear forces from ambulation and weight bearing.

Peripheral neuropathy is common in diabetic patients. The callus and ulcer sometimes are found among diabetic patients who denied foot numbness. The reason is that diabetic patients often do not recognize plantar pressure while walking or abnormal pressure from inappropriate footwear. The authors preferred to increase sensitivity for protective sensation testing by defining the absence of protective sensation as the inability to feel monofilament at least one or more locations on the foot[24,25]. The present study showed that peripheral neuropathy are found often in elderly patients and a long duration of having diabetes is giving eye problems included diabetic retinopathy, sensory deficits or physical limitation. If patients had poor blood sugar control (HbA1C $\geq$ 7%), renal insufficiency, or nephropathy (presence microalbuminuria or macroalbuminuria), the peripheral neuropathy was found more than these condition. Most common sensory deficit was loss of protective sensation, which was considered as one of the risk factors to develop foot ulceration and future limb amputation.

Conclusion

About 2% of diabetes patients had PVD that related with duration of having diabetes, creatinine level, and diabetic neuropathy. Additionally, about 15% of diabetes patients had diabetic neuropathy that depend on age, duration of having diabetes, on ACEI or ARB drug, creatinine level, HbA1C, diabetic retinopathy, diabetic nephropathy, and vascular complication.

These risks may be detected and reduced to some degree by appropriate screening and intervention measures. Early detection, effective management of foot problems, and scheduled follow-up must be emphasized to prevent diabetes related lower extremities amputations and decreased mortality rate.

Acknowledgements

The present study was supported by a grant from the Department of Medical Services, Ministry of Public Health. The authors wish to thank coordinators and subjects at Rajvithi Hospital (Bangkok), Lerdsin Hospital (Bangkok), Nopparatrajathanee Hospital (Bangkok), Mettaphacharak Hospital (Nakhonpathom), Pathumthani Hospital (Pathumthani), Ladlumkeaw Hospital (Pathumthani) and Nongsau Hospital (Pathumthani). The authors wish to thank the staff of Institute of Medical Research and Technology Assessment, Department of Medical Services.

Potential conflicts of interest

None.

References

1. Cowie CC. Diabetes 1996 vital statistics. Alexandria, VA: American Diabetes Association; 1996.
2. Wild S, Roglic G, Green A, Sicree R, King H. Global prevalence of diabetes: estimates for the year 2000 and projections for 2030. Diabetes Care 2004; 27: 1047-53.
3. Palumbo PJ, Melton LJ 3rd. Peripheral vascular disease and diabetes. In: Aubert R, editor. Diabetes in America. 2nd ed. Washington, D.C.: US Government Printing Office; 1985: 401-9.
4. Gibbons GM, Eliopoulos GM. Infection of the diabetic foot. In: Kozak GP, Campbell DR, Frykberg RG, Habershaw GM, editors. Management of diabetic foot problems. Philadelphia: WB Saunders; 1995: 121-9.
5. Reiber GE, Boyko EJ, Smith DG. Lower extremity foot ulcers and amputation in diabetes. In: Harris MI, Cowie CC, Stern MP, Boyko EJ, Reiber GE, Bennett PH, editors. Diabetes in America. Washington, D.C.: US Government Printing Office; 1995: 409-28.
6. Krittiyawong S, Ngarmukos C, Benjasuratwong Y, Rawdaree P, Leelawatana R, Kosachunhanun N, et al. Thailand diabetes registry project: prevalence and risk factors associated with lower extremity amputation in Thai diabetics. J Med Assoc Thai 2006; 89 (Suppl 1): S43-8.
7. Sriussadaporn S, Mekanandha P, Vannasaeng S, Nitiyanant W, Komoltri C, Ploybutr S, et al.

Factors associated with diabetic foot ulceration in Thailand: a case-control study. Diabet Med 1997; 14: 50-6.
8. Boyko EJ, Ahroni JH, Smith DG, Davignon D. Increased mortality associated with diabetic foot ulcer. Diabet Med 1996; 13: 967-72.
9. Pecoraro RE, Reiber GE, Burgess EM. Pathways to diabetic limb amputation. Basis for prevention. Diabetes Care 1990; 13: 513-21.
10. 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: 157-62.
11. Pham HT, Economides PA, Veves A. The role of endothelial function on the foot. Microcirculation and wound healing in patients with diabetes. Clin Podiatr Med Surg 1998; 15: 85-93.
12. American Diabetic Association. Diabetic nephropathy. Diabetes Care 2002; 25 (Suppl 1); S85-9.
13. Patout CA Jr, Birke JA, Wilbright WA, Coleman WC, Mathews RE. A decision pathway for the staged management of foot problems in diabetes mellitus. Arch Phys Med Rehabil 2001; 82: 1724-8.
14. Armstrong DG, Lavery LA, Vela SA, Quebedeaux TL, Fleischli JG. Choosing a practical screening instrument to identify patients at risk for diabetic foot ulceration. Arch Intern Med 1998; 158: 289-92.
15. Mayfield JA, Reiber GE, Sanders LJ, Janisse D, Pogach LM. Preventive foot care in diabetes. Diabetes Care 2004; 27 (Suppl 1): S63-4.
16. Wheeler S, Singh N, Boyko EJ. The epidemiology of diabetic neuropathy. In: Veves A, Malik RA, editors. Diabetic neuropathy: clinical management. 2nd ed. Totowa, NJ: Humana Press; 2007: 7-30.
17. Clayton W Jr, Elasy TA. A review of the pathophysiology, classification, and treatment of foot ulcers in diabetic patients. Clin Diabetes 2009; 27: 52-8.
18. Kelkar P. Diabetic neuropathy. Sem Neurol 2006; 25: 168-73.
19. Bowering CK. Diabetic foot ulcers. Pathophysiology, assessment, and therapy. Can Fam Physician 2001; 47: 1007-16.
20. Huijberts MS, Schaper NC, Schalkwijk CG. Advanced glycation end products and diabetic foot disease. Diabetes Metab Res Rev 2008; 24 (Suppl 1): S19-24.
21. Boulton AJ, Armstrong DG, Albert SF, Frykberg RG, Hellman R, Kirkman MS, et al. Comprehensive foot examination and risk assessment: a report of the task force of the foot care interest group of the American Diabetes Association, with endorsement by the American Association of Clinical Endocrinologists. Diabetes Care 2008; 31: 1679-85.
22. Zochodne DW. Diabetic polyneuropathy: an update. Curr Opin Neurol 2008; 21: 527-33.
23. Paraskevas KI, Baker DM, Pompella A, Mikhailidis DP. Does diabetes mellitus play a role in restenosis and patency rates following lower extremity peripheral arterial revascularization? A critical overview. Ann Vasc Surg 2008; 22: 481-91.
24. Khan NA, Rahim SA, Anand SS, Simel DL, Panju A. Does the clinical examination predict lower extremity peripheral arterial disease? JAMA 2006; 295: 536-46.
25. American Diabetic Association. Peripheral arterial disease in people with diabetes. Diabetes Care 2003; 26: 3333-41.

ความชุกและปัจจัยเสี่ยงที่มีความสัมพันธ์กับผลแทรกซ้อนบริเวณเท้าในผู้ป่วยเบาหวานชนิดที่ 2

ชาญเวช ศรัทธาพุทธ, สมเกียรติ โพธิสัตย์, อัมพร จงเสรีจิตต์, อุดม ไกรฤทธิชัย, คุณากร ภูรีเสถียร

ภูมิหลัง: *โรคเส้นประสาทส่วนปลายจากเบาหวาน (diabetic neuropathy) และโรคหลอดเลือดส่วนปลายตีบตัน (peripheral vascular disease) จากเบาหวานเป็นปัจจัยเสี่ยงหลักของการเกิดแผลที่เท้าและการสูญเสียนิ้วเท้า เท้า หรือ ขาในผู้ป่วยเบาหวานชนิดที่สอง แต่อย่างไรก็ตามยังมีการศึกษาถึงเรื่องความชุกและปัจจัยเสี่ยงต่าง ๆ ของโรคแทรกซ้อนดังกล่าวข้างต้นไม่มากนัก*

วัตถุประสงค์: *เพื่อหาความชุกและปัจจัยเสี่ยงที่มีความสัมพันธ์กับโรคเส้นประสาทส่วนปลายจากเบาหวาน และโรคหลอดเลือดส่วนปลายตีบตันจากในผู้ป่วยที่เป็นเบาหวานชนิดที่ 2*

วัสดุและวิธีการ: *ศึกษาผู้ป่วยเบาหวานชนิดที่ 2 จำนวน 899 ราย จากคลินิกผู้ป่วยนอกของโรงพยาบาลรัฐบาล 7 แห่ง ระหว่างมกราคม พ.ศ. 2550 จนถึงกันยายน พ.ศ. 2551 โดยการซักประวัติ ตรวจร่างกาย ตรวจเท้า และตรวจทางห้องปฏิบัติการจากเลือดและปัสสาวะ*

ผลการศึกษา: *ผู้ป่วยส่วนใหญ่เป็นเพศหญิง, อายุเฉลี่ย 59.64 ปี, ดัชนีมวลกายเฉลี่ย 27.32 kg/m^2, ระยะของการเป็นเบาหวานเฉลี่ย 8.12 ปี และผู้ป่วยร้อยละ 85.17 มี HbA1C มากกว่าหรือเท่ากับร้อยละ 7 ในผู้ป่วยเบาหวานที่มีโรคหลอดเลือดส่วนปลายตีบตันมีความแตกต่างอย่างมีนัยสำคัญทางสถิติเมื่อเปรียบเทียบกับกลุ่มที่ไม่มีโรคหลอดเลือดส่วนปลายตีบตันในเรื่องระยะของการเป็นเบาหวาน (OR, 1.08; 95% CI [1.01-1.16]; p-value 0.047), ระดับ creatinine (OR, 1.62; 95% CI [1.12-2.33]; p-value 0.01), มีโรคเส้นประสาทส่วนปลายจากเบาหวาน (OR, 7.37; 95% CI [2.52-21.59]; p-value < 0.001) ในผู้ป่วยเบาหวานที่มีโรคเส้นประสาทส่วนปลายชาจากเบาหวานมีความแตกต่างอย่างมีนัยสำคัญทางสถิติเมื่อเปรียบเทียบกับกลุ่มที่ไม่มีโรคเส้นประสาทส่วนปลายชาจากเบาหวานในเรื่องของอายุ (OR, 1.04; 95% CI [1.01-1.06]; p-value 0.003), ระยะของการเป็นเบาหวาน (OR, 1.04; 95% CI [1.01-1.07]; p-value 0.008), การรับประทานยากลุ่ม ACEI หรือ ARB (OR, 1.77; 95% CI [1.24-2.55]; p-value 0.002), HbA1C (OR, 1.14; 95% CI [1.03-1.27]; p-value 0.012), ระดับ creatinine (OR, 1.38; 95% CI [1.04-1.79]; p-value 0.014), มีโรคจอประสาทตาเสื่อมจากเบาหวาน (diabetic retinopathy) (OR, 1.96; 95% CI [1.22-3.13]; p-value 0.005), มีโรคหลอดเลือดส่วนปลายตีบตัน (OR, 7.37; 95% CI [2.52-21.59]; p-value < 0.001), มีโรคไตจากเบาหวานร่วมกับมี microalbuminuria (OR, 1.74; 95% CI [1.12-2.69]; p-value 0.013)*

สรุป: *มีผู้ป่วยเบาหวานประมาณร้อยละ 2 ที่มีโรคหลอดเลือดส่วนปลายตีบตัน โดยมีความสัมพันธ์กับระยะของการเป็นเบาหวาน, ระดับ creatinine และมีโรคเส้นประสาทส่วนปลายจากเบาหวาน ในส่วนของผู้ป่วยเบาหวานที่มีโรคเส้นประสาทส่วนปลายจากเบาหวานมีประมาณร้อยละ 15 โดยมีความสัมพันธ์กับอายุ, ระยะของการเป็นเบาหวาน, การรับประทานยากลุ่ม ACEI หรือ ARB, HbA1C, ระดับ creatinine, มีโรคจอประสาทตาเสื่อมจากเบาหวาน, มีโรคหลอดเลือดส่วนปลายตีบตัน และมีโรคไตจากเบาหวาน*