

# Clinical Assessment, Risk Factors, and Classification of Diabetic Foot: An Overview

<sup>1</sup>Zile S Kundu, <sup>2</sup>Milind Tanwar, <sup>3</sup>Kuldeep Singh, <sup>4</sup>Bikramjeet Singh

## ABSTRACT

Diabetes mellitus is one of the commonest medical conditions prevalent all over the globe. This is associated with many complications due to its progressive involvement of various organ systems. The basic mechanism includes gradual onset of neuropathies, vasculopathies, retinopathies, reduced immune systems, and thus increased incidence of infections. Foot involvement is quite common in diabetics. This should be prevented and treated early with a team approach. Every effort should be made to prevent ulcer formation and further complications like deep-seated infection and gangrene. For proper understanding, the basic concepts regarding the risk factors, pathogenesis, classification, and proper assessment/examination are of paramount importance. This study outlines these aspects, which will help in prevention of dreadful complications and early treatment of the diabetic foot.

**Keywords:** Classification, Clinical, Diabetic foot, Risk factors.

**How to cite this article:** Kundu ZS, Tanwar M, Singh K, Singh B. Clinical Assessment, Risk Factors, and Classification of Diabetic Foot: An Overview. *J Foot Ankle Surg (Asia-Pacific)* 2017;4(1):35-39.

**Source of support:** Nil

**Conflict of interest:** None

## INTRODUCTION

Diabetic foot is defined as the foot of a diabetic patient with ulceration, infection, and/or destruction of the deep tissues associated with neurological abnormalities and varying degrees of peripheral vascular disease of the lower limb.<sup>1</sup> The prevalence of foot ulcers in patients suffering from diabetes is about 4 to 10% in developed countries.<sup>2</sup> The incidence increases with age due to a long-standing combination of peripheral neuropathy, peripheral vascular deficiency, and associated autonomic

changes.<sup>3</sup> It is estimated that about 40 to 70% of the nontraumatic amputations of the lower limb are due to sequelae of diabetic foot.<sup>4</sup> About 5% of diabetic patients have a positive history of foot ulcers and 15% are proposed to experience such a complication in their lifetime. These patients need emergent management and help to assess the burden of complication.<sup>5</sup>

Diabetic foot is a much sought-after complication of diabetes but less cared for before of its occurrence. The focus of the present study is on early clinical assessment, identification of risk factors, and classification of the disease as identifying high-risk patients and preventing ulcers may decrease the comorbidity and burden of disease.

## PATHOGENESIS AND RISK FACTORS OF DIABETIC FOOT

Understanding the pathogenesis of the complication is the first step to early detection and prevention. The crucial factors involved in the development of a diabetic foot include the neuropathy and vasculopathy complicated by various systemic and local factors. The neuropathic component affects the sensory, autonomic, and motor nerves. This may lead to sensory loss in distal parts with associated sudomotor changes like drying and cracking of skin, leading to breach in the barrier<sup>6</sup> and changes in the foot anatomy and weight-bearing mechanics due to long-standing loss of proprioception. The abnormal planter pressures due to Charcot neuroarthropathy are also an important cause of development of these ulcers. It suggests that repetitive microtrauma lead to inflammatory cytokine (tumor necrosis factor alpha, interleukin 1 beta) release, causing uncontrolled osteolysis.<sup>7</sup>

This is further complicated by angiopathy of both macro- and microvascular origin. It is shown by various studies that diabetic patients suffer from a peripheral arterial disease, which is early onset and with a faster progression than in the general population. It has also been proven to be an independent risk factor for development and prognosis of the disease.<sup>8</sup>

The biochemical basis of pathogenesis is explained by accumulation of sorbitol and fructose in nerve cells leading to decreased myoinositol synthesis.<sup>9</sup> This leads to decreased nerve conduction velocities. Also associated vasculopathy is partially explainable by decreased nitric oxide production and endothelial cell damage due to hyperglycemia.<sup>10</sup>

<sup>1</sup>Professor, <sup>2</sup>Senior Resident, <sup>3</sup>Associate Professor, <sup>4</sup>Resident

<sup>1,2</sup>Department of Orthopaedics, Pt. Bhagwat Dayal Sharma Post Graduate Institute of Medical Sciences, Rohtak, Haryana, India

<sup>3,4</sup>Department of Burns and Plastic Surgery, Pt. Bhagwat Dayal Sharma Post Graduate Institute of Medical Sciences, Rohtak Haryana, India

**Corresponding Author:** Zile S Kundu, Professor, Department of Orthopaedics, Pt. Bhagwat Dayal Sharma Post Graduate Institute of Medical Sciences, Rohtak, Haryana, India, e-mail: zskundu2003@rediffmail.com

The development of the ulcer usually follows a definitive pattern whereby callus formation is the first step. This chronic pressure and/or trauma leads to formation of an underlying hematoma, which eventually results in the breakdown of the overlying skin. This is further complicated by a superficial or deep infection secondary to low immunity and poor glycaemic control that lead to a nonhealing ulcer or even osteomyelitis, leading to amputation of the lower extremity in these cases (Figs 1 to 7).

Local factors that complicate the disease include infection, mechanical, and/or thermal insult. Smoking, visual impairment, alcohol consumption, uncontrolled hyperglycemia, and hyperlipidemias add to the nonhealing and further accentuation of the complication. The perpetual factor that leads to a recalcitrant form is a delayed presentation. Additionally, self-inflicted trauma like digging of nails, foreign body penetration, and use of traditional medication also adds to the problem. Nather et al<sup>11,12</sup> found that use of footwear is also important and noted that 70% of diabetics wear slippers or no footwear most of the time. Diabetic patients should be counseled to wear appropriate footwear at all times.

**Table 1:** Wagner’s classification

Grades	Description
0	No ulcer, foot at risk
I	Superficial ulcer involving full thickness but not underlying tissues
II	Deep ulcer, penetrating down to ligaments and muscles, but no bone involvement or abscess formation
III	Deep ulcer with cellulitis or abscess formation, often with osteomyelitis
IV	Localized gangrene
V	Extensive gangrene



**Fig. 1:** Normal foot with shining pink nails and normal skin texture

## CLASSIFICATIONS

Various classification systems have been proposed to evaluate the disease grade and progression. These are Wagner classification Table 1,<sup>13</sup> Kings College classification Table 2,<sup>14</sup> The University of Texas classification<sup>15</sup> Table 3, and Brodsky classification of diabetic ulcer<sup>16</sup> Table 4.

The higher the grade of ulcer, the poorer are its prognosis and longer is the duration of treatment.

This classification also includes the grade (depth) and stage (ischemia and infection) of diabetic foot in addition to the Wagner classification.

## CHARCOT (NEUROPATHIC ANTHROPATHY) JOINT

The involvement as Charcot’s joint or neuropathic arthropathy of the foot (Fig. 8) in diabetes mellitus can be further classified as detailed in Tables 5 and 6.<sup>17</sup>

## CLINICAL ASSESSMENT

Clinical assessment of a “foot at risk” involves a thorough clinical examination of the neurovascular component

**Table 2:** Kings College classification

Stages	Lesion
1	Normal foot
2	High risk
3	Ulcerated foot
4	Cellulitic foot
5	Necrotic foot
6	Major amputation



**Fig. 2:** Diabetic foot with brittle nails and a focus of redness just proximal to toe-bases which was well prevented with antibiotics and control of blood sugar levels



**Fig. 3:** Diabetic foot with cellulitis and swelling on the dorsum of foot which required treatment with prolonged antibiotics



**Fig. 4:** Diabetic foot with deep ulcer on the heel which required excision and partial calcaneotomy



**Fig. 5:** Diabetic foot with ulcer on the medial side of ankle and foot with induration of surrounding skin and necrotic slough in the base



**Fig. 6:** Diabetic foot with gangrenous patches with slough extending in to the leg and foot required above knee amputation



**Fig. 7:** Foot of a diabetic patient with gangrene of toes and foot required below knee amputation

and the systemic factors that lead to its emergence. It is needless to emphasize on detailed history and general physical examination to look for diabetes-associated insults like acanthosis nigricans, acne, hirsutism, and vitiligo. Signs to detect retinopathy and associated nephropathy should not be missed. Associated sudomotor dysfunction (raised local temperature, anhidrosis) may also be evident on local examination and this may be examined using the starch test or the ninhydrin test.<sup>18</sup>

Documentation of peripheral pulses and measurement of ankle brachial index (ABI) is a key to measure vascular insufficiency. An ABI  $<0.9$  is suggestive of a subclinically existing peripheral vascular disease.<sup>19</sup>

In addition to the sudomotor abnormalities, assessment of neuropathy involves examination of both motor and sensory modalities. This includes examination of

**Table 3:** The University of Texas classification

Stage	Grade	1	2	3
A	Pre- or postulcerative completely epithelized lesion	Superficial wound	Deep wound penetration up to tendon or capsule	Wound penetration up to bone or joint
B	Infection	Infection	Infection	Infection
C	Ischemia	Ischemia	Ischemia	Ischemia
D	Infection and ischemia	Infection and ischemia	Infection and ischemia	Infection and ischemia

**Table 4:** Brodsky classification of diabetic ulcer

Grades	Description
0	Intact skin, a preulcerative lesion with erythema, callus or sheer hemorrhage over bony prominence
I	Superficial full thickness ulcer – down but not through subcutaneous tissue
II	Ulceration down tendon and joint capsule, but neither the joint nor the bone is visible
III	Ulceration with exposed bone or joints and osteomyelitis or pyarthrosis

power, tone, muscle bulk, and the reflexes (superficial/deep). Comparative sensory examination of bilateral lower limbs should be done to assess integrity of spinothalamic (pain, temperature, fine touch, pressure) and posterior column (vibration, crude touch, and proprioception) sensations. The Semmes-Weinstein 5.07 gauge monofilament test using 10 gm force until it bends is conducted and 10 sites are examined (Fig. 9). Sensations present in seven or fewer sites equal loss of protective sensation.<sup>20</sup> For touch sensation and tuning, fork test (128 Hz) is of particular importance. The sense of proprioception can be assessed by joint position matching.<sup>21</sup> Local examination of ulcer and its characteristics should be done in detail as they define the prognosis at presentation. This will also help to assess response to treatment at subsequent visits of the patient. Biochemical analysis in the form of routine complete hemogram with differential and total counts, kidney function tests, precise monitoring of blood glucose and HbA1c help to regulate treatment of local and systemic diseases.

**Table 5:** Eichenholz classification: It is based more on radiographic rather than clinical findings

Stage	Description
Stage 0	Unilateral edema, erythema, and associated warmth; no break in skin integrity; radiographs negative or shows local osteoporosis
Stage 1	Unilateral edema, erythema, and unilateral warmth; radiographs show osseous destruction, joint dislocation/subluxation
Stage 2	Decreased local edema, erythema, and associated warmth; radiographs show coalition of small fracture fragments and absorption of fine bone debris
Stage 3	No or minimal edema, erythema, or increased warmth; radiographs show consolidation and remodeling of fracture fragments



**Fig. 8:** Charcot neuroarthropathy of the ankle in a diabetic patient

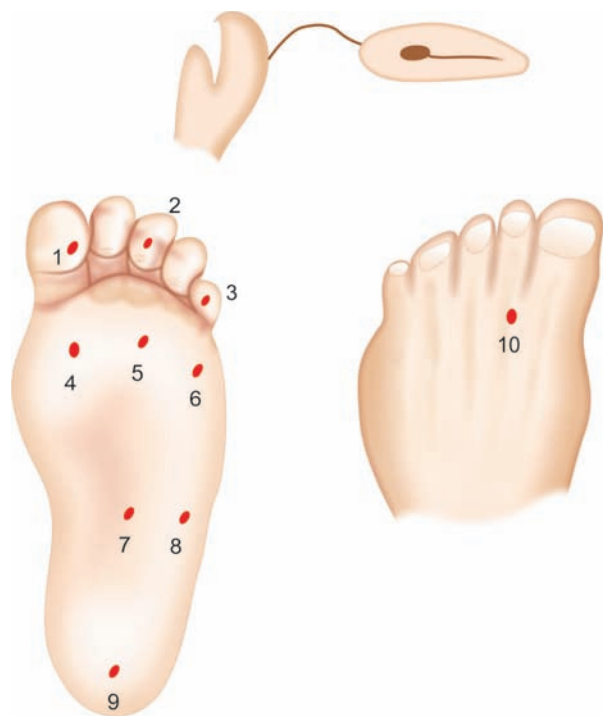
Before the start of an empirical therapy, it is essential to diagnose the colonizing organism by biopsy/pus culture sensitivity testing.<sup>11,12</sup>

The use of radiological examination may help to assess the extent of involvement of bone and soft tissues. Skiagrams are the first radiological investigation of choice in ruling out fractures, dislocations, and Charcot joints.<sup>22</sup> The hallmark of physical clue to the underlying neuropathic skeletal abnormality, regardless of the innocent radiographic appearance, is localized warmth and swelling, usually out of proportion to the injury (if the patient remembers an injury) that persists (Tables 5 and 6).<sup>17</sup>

Magnetic resonance imaging scan has evolved as an important modality to assess the depth and area of involvement. Angiography and color Doppler have emerged as new diagnostic modalities as confirmatory evidence of peripheral vascular disease. Role of pedobarography has also been suggested in understanding the development of foot ulcers and further decide the type of orthosis to be used for offloading.<sup>23</sup>

**Table 6:** Anatomical classification: Charcot neuropathic arthropathy classification based on anatomical location of the involved joint

Types	Description
I	Tarsometatarsal joints involvement
II	Triple joint complex involvement
IIIA	Tibiotalar joint involvement
IIIB	Pathological fracture of calcaneus



**Fig. 9:** The Semmes-Weinstein 5.07 gauge monofilament test and various sites to be tested

## CONCLUSION

Proper and systematic clinical assessment, identification of risk factors, and classification of the diabetic foot are of paramount importance as this will help in early detection of the disease before the development of the frank ulcer and infection, which are dreadful complications. Diabetic foot is a much sought after complication of diabetes but less cared for before its occurrence. These feet need treatment with a team approach, including endocrinologist, chiropodist, surgeon, and physiotherapist. Additionally, the education of the patients for taking care of their feet is very important, and preventing ulcers may decrease the comorbidity and burden of disease. Thus, the role of patient and attendant education and detection by health care providers at all levels remains indispensable. The incidence of amputation can be certainly reduced by early detection and holistic care.

## REFERENCES

1. International Working Group on the Diabetic Foot. In: International consensus on the diabetic foot. The Netherlands: International Working Group on the Diabetic Foot; 1999. p. 20-96.
2. Boulton AJ. The diabetic foot: from art to science. The 18th Camillo Golgi lecture. *Diabetologia* 2004 Aug;47(8): 1343-1353.
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 Jan;158(2):157-162.
4. Moxey PW, Gogalniceanu P, Hinchliffe RJ, Loftus IM, Jones KJ, Thompson MM, Holt PJ. Lower extremity amputations – a

review of global variability in incidence. *Diabet Med* 2011 Oct;28(10):1144-1153.

5. Reiber, GE.; Boyko, E.; Smith, DG. Lower extremity ulcers and amputations in individuals with diabetes. In: Harris, MI., editor. *Diabetes in America*. 2nd ed. Bethesda: National Institutes of Health; 1995. p. 409-427.
6. Brem H, Sheehan P, Boulton AJ. Protocol for treatment of diabetic foot ulcers. *Am J Surg* 2004 May;187(5A):1S-10S.
7. Madan SS, Pai DR. Charcot neuroarthropathy of the foot and ankle. *Orthop Surg* 2013 May;5(2):86-93.
8. Norgren L, Hiatt WR, Dormandy JA, Nehler MR, Harris KA, Fowkes FG. Trans Atlantic Inter-Society Consensus II Working Group: inter-society consensus for the management of peripheral arterial disease (TASC II). *Eur J Vasc Endovasc Surg* 2007 Dec;33(1):S1-S75.
9. Boulton AJ, Kirsner RS, Vileikyte L. Clinical practice. Neuropathic diabetic foot ulcers. *N Engl J Med* 2004 Jul;351(1): 48-55.
10. Lüscher TF, Creager MA, Beckman JA, Cosentino F. Diabetes and vascular disease: pathophysiology, clinical consequences, and medical therapy: part II. *Circulation* 2003 Sep;108(13):1655-1661.
11. Nather A, Bee CS, Huak CY, Chew JL, Lin CB, Neo S, Sim EY. Epidemiology of diabetic foot problems and predictive factors for limb loss. *J Diabetes Complications* 2008 Mar-Apr;22(2):77-82.
12. Nather, A.; Singh, E. Diabetic foot wear current status and future directions. In: Nather, A., editor. *Diabetic foot problems*. Singapore: World Scientific; 2008. p. 527-539.
13. Wagner FW Jr. The dysvascular foot: a system for diagnosis and treatment. *Foot Ankle* 1981 Sep;2(2):64-122.
14. Edmonds, M.; Foster, AVM. *Managing the diabetic foot*. 3rd ed. Chichester: Wiley-Blackwell; 2014.
15. Oyibo SO, Jude EB, Tarawneh I, Nguyen HC, Harkless LB, Boulton AJ. A comparison of two diabetic foot ulcer classification systems: the Wagner and the University of Texas wound classification systems. *Diabetes Care* 2001 Jan;24(1):84-88.
16. Brodsky JW. Outpatient diagnosis and care of the diabetic foot. *Instr Course Lect* 1993;42:121-139.
17. Ishikawa, SN. Diabetic foot. In: Canale, ST.; Beaty, JH., editors. *Campbell's operative orthopaedics*. 11th ed. Philadelphia: Mosby Elsevier; 2008. p. 4697-4716.
18. Schlereth T, Brosda N, Birklein F. Spreading of sudomotor axon reflexes in human skin. *Neurology* 2005 Apr;64(8): 1417-1421.
19. Doobay AV, Anand SS. Sensitivity and specificity of the ankle-brachial index to predict future cardiovascular outcomes: a systematic review. *Arterioscler Thromb Vasc Biol* 2005 Jul;25(7):1463-1469.
20. Bell-Krotoski JA, Fess EE, Figarola JH, Hiltz D. Threshold detection and Semmes-Weinstein monofilaments. *J Hand Ther* 1995 Apr-Jun;8(2):155-162.
21. Goble DJ. Proprioceptive acuity assessment via joint position matching: from basic science to general practice. *Phys Ther* 2010 Aug;90(8):1176-1184.
22. Rajbhandari SM, Jenkins RC, Davies C, Tesfaye S. Charcot neuroarthropathy in diabetes mellitus. *Diabetologia* 2002 Aug;45(8):1085-1096.
23. Lobmann R, Kayser R, Kasten G, Kasten U, Kluge K, Neumann W, Lehnert H. Effects of preventative footwear on foot pressure as determined by pedobarography in diabetic patients: a prospective study. *Diabet Med* 2001 Apr;18(4):314-319.