Type 2 diabetic foot complications: an overview

Amit Kumar C. Jain, Mounia Sabasse

Key words:

- Charcot foot
- Foot ulcer
- Necrosis
- Peripheral arterial disease

Authors

Amit Kumar C. Jain is Diabetic Lower Limb & Podiatric Surgeon, at the Department of Surgery, St Johns Medical College, Bangalore, India; Mounia Sabasse is Diabetic Foot and Wound Care Specialist, Dubai Health Authority, United Arab Emirates. Amit Jain's classification of diabetic foot complications is a popular tool that encompasses the common complications seen in the diabetic foot around the world. This classification makes us look at various lesions commonly affecting diabetic foot apart from the foot ulcers that are seen in diabetic patients. Studies have shown that type 1 diabetic foot complications are the most common complications that are encountered in hospitalised patients in developing countries. This article discusses the more common type 2 diabetic foot complications seen in the clinical practice. Type 2 diabetic foot complications are non-infective complications that occur in patients with long-standing diabetes.

iabetes mellitus is a serious, noncommunicable disease that affects almost all the vital organs of the human body^[1]. It is estimated that there were about 387 million people with diabetes worldwide in 2014 and it is predicted to increase to 592 million by 2035^[2].

Diabetic foot ulcers is one of the most distressing complications of diabetes and constitutes a major public health problem^[1,3]. Diabetic foot ulcers are known to affect around 15% of people with diabetes in their lifetime^[1]. For many years, diabetic foot ulcers were examined using Wagner's classification^[4,5]. In the last two to three decades, there has been a rise in the incidence of a number of other diabetic foot complications, such as Charcot foot and necrotising fasciitis, which were not covered by Wagner's classification. To address such emerging complications, which are now frequently encountered, a new classification known as Amit Jain's classification for diabetic foot complications was proposed in 2012^[4,5,6].

Amit Jain's classification of diabetic foot complications included for the first time all the common complications seen in the diabetic foot^[4,7,8]. The Amit Jain three-tier classification system, which divides diabetic foot complications into three simple types^[5,6,7], is proving to be a popular tool and effective in teaching^[7] *Table 1*.

The aim of this article is to review some of the common Amit Jain's type 2 diabetic foot complications encountered in day-to-day clinical practice.

Callus ulcers/trophic ulcers

Around 50-70% of patients with diabetes have some form of neuropathy^[1,10,11], and 15% of people with diabetes will develop some form of foot ulcer during their lifetime^[11,12]. Neuropathic ulceration often results from a neglected callus^[13]. Changes in the biomechanics of foot function due to sensorymotor neuropathy leads to increased pressure in certain areas, especially on the ball of the great toe and in the first metatarsal head region [Figures 1 and 2]. Increased pressure leads to the formation of a callus at these pressure points^[10,13]. A callus is a hypertrophy of the stratum corneum with excessive keratinisation, which indicates abnormal foot pressure^[14]. High pressures are known to cause neuropathic ulcers in prominent pressure areas. In fact, the relative risk of ulceration beneath a callus is 11 times greater than that in other regions of the foot^[10].

Foot ulcers in those people with diabetes who lack protective sensation and have adequate blood flow to their foot are termed diabetic neuropathic foot ulcers^[11]. The management of these ulcers consists in the removal of necrotic tissue (slough), the use of a

Table 1. Amit Jain's classification of diabetic foot complications [4]	
Type of diabetic foot complication	Lesions
Type 1: infective	Wet gangrene, abscesses, cellulitis, necrotising fasciitis, etc.
Type 2: non-infective	Callus ulcers, Charcot foot, peripheral arterial disease, diabetic bullae, etc.
Type 3: mixed	Ex-non-healing ulcer with osteomyelitis, ischaemic ulcer with infection, etc.

moist wound dressing, and offloading^[11]. Infection of diabetic neuropathic foot ulcers should be prevented as far as possible.

Diabetic bulla

A diabetic bulla, also known as bullosis diabeticorum, is an uncommon, spontaneous, non-inflammatory blistering condition which is characteristic of diabetes^[15,16,17]. Diabetic bulla was first described by Kramer in 1930^[1] and was named bullosis diabeticorum by Cantwell and Martz in 1967^[17].

A diabetic bulla occurs in around 0.5% of people with diabetes in the USA^[17]. It is associated with long-standing diabetes mellitus and more frequently occurs in men, with a male-to-female ratio of 2:1^[15,17]. It typically affects the legs and foot of diabetic patients, particularly over the dorsum and sides of the feet^[16] [*Figure 3*].

A bulla may range from 0.5–10 cm in diameter^[15]. A diabetic bulla is sub epidermal^[16] and there is no history of preceding trauma^[16]. There is no surrounding erythema and the bulla contains clear fluid^[15,17]. The exact mechanism of occurrence of diabetic bulla remains unclear^[15,17,18]. An earlier

3)

Figure 3. Bullosis diabeticorum. Note the absence of surrounding erythema.

opinion proposed that a bulla results from an occlusion of the arteriole supplying the local area of the skin, resulting in necrosis^[18], although this hypothesis remains to be confirmed. Other theories propose that diabetic bulla is the result of a combination of increased venous pressure and microangiopathy^[15], and of poor regulation of blood glucose with instances of hypoglycaemia^[15].

No specific treatment is required, and a bulla usually heals within 2 weeks with much scarring, although there are instances of ulcerations and secondary infection^[15, 16, 17].

Toe abnormalities

The common toe deformities seen in diabetic foot are hammer toe, mallet toe, claw toe, hallux valgus and hallux rigidus^[19]. Most of these toe deformities occur due to motor neuropathy that leads to muscle atrophy^[20].

In hammer toe deformity, there is hyperextension of the metatarsophalangeal joint and flexion deformity of the proximal interphalangeal joint *[Figure 4]*. There is no deformity at the distal interphalangeal joint^[21]. In mallet toe, there is distal interphalangeal joint flexion deformity of the toe. In claw toe,



Figure 4. Hammer toe deformities.



Figure 1. Callosity over the left great toe.



Figure 2. Trophic ulcer after removal of the callus (same patient as in Figure 1).

"Charcot foot accounted for 7.69% of major amputations in people with diabetes in a 2015 study from the Indian subcontinent."



Figure 5. Claw toe deformity.



Figure 6. Ischaemic ulcer over the right foot (S/P great toe amputation).



Figure 7. Chronic Charcot foot of the left foot.

there is hyperextension of the MTP joint along with flexion deformity at both proximal and distal interphalangeal joint^[21, 22] [*Figure 5*].

Hallux valgus is a complex deformity where there is abduction and external rotation of the great toe. The deformity is said to exist when abduction of the hallux is greater than $12^{\circ[19]}$. In hallux rigidus, the dorsiflexion of the hallux at the first metatarsophalangeal joint is restricted^[19].

The prevalence of these deformities differs in different regions. In a study by Mansour et $al^{[20]}$, hammer toes were observed in 10.9% of those with diabetes studied, and claw toes were observed in 3.8% of patients^[20]. In a series by Ogbera et $al^{[23]}$, claw toe was present in 7.4%, hallux valgus in 6.3% and hammer toe in 5.5% of patients studied.

The presence of toe deformities is associated with an increased risk of ulcer development^[24]. These ulcers are managed with wound care, debridement and offloading^[24]. Depending on the type of toe deformity, treatment includes tenotomies, arthroplasties, osteotomies and arthrodesis^[19, 24].

Peripheral arterial disease

Peripheral arterial disease (PAD) a major macro vascular complication of diabetes mellitus^[25]. Patients with diabetes are four times more likely to develop PAD^[26]. It is estimated that 8% of the patients already have underlying PAD at the time of diagnosis^[26].

The onset of PAD is earlier in people with diabetes than in non-diabetic individuals^[27]. There is a geographical difference in the prevalence of PAD in people with diabetes: it has been estimated at 3.2% in a south Indian study and 15.9% in a western population^[28]. In Germany, the incidence of PAD was found to be 48% in patients with foot ulcers^[29]. The low prevalence of PAD in southern India is in marked contrast to the higher rate of coronary artery disease^[28]. PAD is likely to occur more frequently in older individuals and is present in more than 70% of people with diabetes aged over 70 years who have a foot ulcer^[30].

PAD in people with diabetes differs from that in non-diabetics in terms of epidemiology (increased prevalence), clinical presentation (involves lower leg arteries) and in therapeutic interventions (more limitations in surgical reconstruction and endovascular recanalization)^[29,31]. The medium sized arteries (tibial vessels) of the leg are predominantly involved in diabetes with relative sparing of the proximal larger and pedal vessels^[32]. Calcifications of the pedal vessel are also common^[30]. Most often patients with diabetic foot problems with underlying PAD present either with an ischaemic ulcer (Figure 6) or with gangrene. Absent foot pulses, monophasic Doppler signals or ankle brachial index of less than 0.9 usually suggest underlying PAD^[30]. Often, in the case of non-healing ulcers, further investigations such as an angiogram are needed, and there may be a requirement for bypass surgery or endovascular intervention^[29]. Morbidity, mortality and amputations are higher in patients with diabetes and PAD^[30].

Charcot foot

Charcot foot is a progressive, non-infectious destructive disease of the bones and joints in a person with neuropathy^[33]. Although it used to be considered an uncommon complication, it is now more frequently encountered in patients with diabetes, with the foot and ankle being the most commonly affected sites^[33]. Charcot foot was first described in 1868 by Jean Martin Charcot^[33,34].

The incidence of Charcot neuroarthropathy is around 0.1–0.5% in patients with diabetes^[35,36]. The duration of diabetes is usually greater than 12 years in patients with Charcot foot^[37]. Although it is often unilateral, Charcot foot occurs bilaterally in up to 30% of the cases^[35]. Charcot foot affects individuals most frequently in the fifth decade of life and has a similar prevalence in men and women^[33,35].

Charcot foot may be acute or chronic *[Figure 7]*. Acute Charcot foot manifests as a red hot swollen foot or ankle with clinically bounding pulses^[33]. Ulcer manifestations are more common in chronic Charcot foot, and the most frequent locations are plantar medial followed by plantar lateral and plantar central^[38]. Conservative management includes wound care if ulcer is present and strict offloading^[35,38]. Surgical treatment ranges from exostectomy to arthrodesis^[38].

Charcot foot requires early diagnosis and treatment to prevent complications^[34]. It is considered a limbthreatening condition and has been associated with increased mortality^[34]. Charcot foot accounted for 7.69% of major amputations in people with diabetes in a 2015 study from the Indian subcontinent^[39].

Type 2 diabetic foot complications: an overview

Conclusions

Amit Jain's classification of diabetic foot complications is the simplest of all such classifications till date in diabetic foot and includes all the more common complications observed in the diabetic foot. Amit Jain's classification serves as an easy tool for teaching and disseminating the knowledge of the diabetic foot across countries.

- Singh S, Pai DR, Yuhhul C. Diabetic foot ulcer: Diagnosis and management. Clinical Research on Foot & Ankle 2013; 1:120
- International Diabetes Federation (2014) *IDF Diabetes Atlas.* 6th edn. http://www.idf.org/sites/default/files/Atlasposter-2014_EN.pdf (accessed 23 September 2015)]
- Abbas Z, Morbach S. Diabetic Foot damage in developing countries: The urgent need for education. *Diabetic Voice* 2005; 50: 15–17
- Jain AKC. A new classification of diabetic foot complications: A simple and effective teaching tool. *The Journal of Diabetic Foot Complications* 2012; 4(1): 1–5
- 5. Jain AKC. Joshi S. Diabetic foot classifications: review of literature. *Medicine Science* 2013; 2(3): 715–21
- 6. Kalaivani V, Vijaya Kumar HM. Diabetic foot in India. Reviewing epidemiology and the Amit Jain's Classifications. *Scholars Acad J Biosci* 2013; 1(6): 305–8
- Dhubaib HA. Understanding diabetic foot complications: In praise of Amit Jain's classification. *The Diabetic Foot Journal Middle East* 2015; 1(1): 10–11
- Kalaivani V. Evaluation of diabetic foot complications according to Amit Jain's classification. J Clin Diagn Resv 2014; 8(12): 7–9
- 9. Jain AKC, Viswanath S. Distribution and analysis of diabetic foot. OA Case Reports 2013; 2(12): 117
- 10. Boada A. Skin lesions in the diabetic foot. Actas Dermosifiliogr 2012; 103: 348–56.
- Margolis DJ, Taylor LA, Hoffstad O, Berlin JA. Diabetic neuropathic foot ulcers. *Diabetes Care* 2002; 25(10): 1835–9
- 12. Boulton AJM, Krisner RS, Vileikyte L. Neuropathic diabetic foot ulcers. *N Eng J Med* 2004; 351: 48–55
- Habershaw GM, Chzran J. Biomechanical consideration of diabetic foot. In: Kozak GP, Campbell DR, Frykberg RG, Habershaw GM, eds, Management of Diabetic Foot Problems. Pennysylvania: WB Saunders. 1995;53-65
- Colagiuri S, Marsden LL, Naidu V, Taylor L. The use of orthothic devices to correct plantar callus in people with diabetes. *Diabetes Res Clin Pract* 1995; 28(1): 29–34
- Wilson TC, Snyder RJ, Southerland CC. Bullosis diabeticorum: Is there a correlation between hyperglycemia and this symptomatology? *Wounds* 2012; 24(12): 350–5
- Sreedevi C, Car N, Pavlic-Renar I. Dermatologic lesions in diabetes mellitus. *Diabetologia Croatica* 2002; 31(3): 147–51
- Ghosh SK, Bandyopadhyoy D, Chatterjee G. Bullosis diabeticorum: A distinctive blistering eruptions in diabetes mellitus. *Int J Diabetes Dev Ctries* 2009; 29(1): 41–2
- Cope RL. Spontaneous bulla formation in the skin in diabetes mellitus. Ann Intern Med 1950; 32(5): 964–7
- Thomson CE, Campbell RH, Wood AR, Rendall CC. Adult foot disorder. In: Lorimer DL, French G, Donnell M, Burrow JG, eds, Neale's Disorders of the Foot. London: Churchill Livingstone. 2002;113-179

- 20. Mansour AA, Imran HJ. Foot abnormalities in diabetes: Prevalence and predictors in Basrah. *Middle East Journal of Family Medicine* 2006; 14(3): 1–6
- 21. Johnson S, Branthwaite H, Naemi R, Chockalingam N. The effect of three different toe props on plantar pressure and patient comfort. *J Foot Ankle Res* 2012; 5: 22
- 22. Ledoux WR, Schoen J, Lovell M, Huff E. Clawed toes in the diabetic foot: Neuropathy, intrinsic muscle volume and plantar aponeurosis thickness. *J Foot Ankle Res* 2008; 1(Suppl): 2
- 23. Ogbera AO, Aedokun A, Fasanmade OA, Ohwouoriole AE, Ajani M. The foot at risk in Nigerians with diabetes mellitus: The Nigerian Scenario. *Int J Endocrinol Metab* 2005; 4: 165–73
- 24. Netten JJV, Bril A, Baal JGV. The effect of flexor tenotomy on healing and prevention of neuropathic diabetic foot ulcers on the distal end of the toe. *J Foot Ankle Res* 2013; 6: 3
- 25. Khurana A, Dhoat P, Marwaha TS. Peripheral vascular disease: A silent assassin: Its rising trend in Punjab. *Journal, Indian Academy of Clinical Medicine* 2013; 14(2): 111–4
- 26. Albayati MA, Shearman CP. Peripheral arterial disease and bypass surgery in the diabetic lower limb. *Med Clin North Am* 2013; 97: 821–34
- 27. Huysman F, Mathieu C. Diabetes and peripheral vascular disease. Acta Chir Belg 2009; 109: 587–94
- 28. Agrawal AK, Singh M, Arya V, Garga U, Singh Vp, Jain V. Prevalence of peripheral arterial disease in type 2 diabetes mellitus and its correlation with coronary artery disease and its risk factors. J Assoc Physicians India 2012; 60: 28–32
- 29. Jain AKC, Varma Ak, Mangalanandan, Kumar H. Revascularization in the diabetic lower limb. *The Journal of Diabetic Foot Complications* 2013; 5(1): 18–23
- Brownrigg JRW, Schaper NC, Hinchliffe RJ. Diagnosis and assessment of peripheral arterial disease in diabetic foot. *Diabet Med* 2015; 32: 738–47
- Coce F, Metelko Z, Jaksic B, Car N, Pavkovic P. Peripheral arterial disease and diabetes mellitus. *Diabetologia Croatica* 2008; 37(2): 47–53
- Yii MK, Liew NC. Revsculariztion for foot salvage in diabetic critical foot ischaemia. *Med J Malaysia* 1999; 54(3): 325–8
- van der Ven A, Chapman CB, Bowker JH. Charcot neuroarthropathy of the foot and ankle. J Am Acad Orthop Surg 2009; 17(9): 562–71
- 34. Gouveri E, Papanas N. Charcot osteoarthropathy in diabetes: A brief review with an emphasis on clinical practice. World J Diabetes 2011; 2(5): 59–65
- 35. Vella S, Cachia MJ. Charcot neuroarthropathy: Pathogenesis, diagnosis and medical management. *Malta Medical Journal* 2008; 20(3): 13–19
- 36. Pakarinen TK, Laine HJ, Maenpaa H, Mattila P, Lahtela J. The effect of zoledronic acid on the clinical resolution of Charcot neuroarthropathy: A pilot randomized controlled trial. *Diabetes Care* 2011; 34(7): 1514–16
- 37. Gupta PPK, Mohan V. Charcot foot: An update. J Assoc Physicians India 2003; 551: 367–72
- Ku□era T, Šponer P, Šrot J. Surgical reconstruction of Charcot foot neuroarthropathy: A case based review. Acta Medica (Hradec Kralove) 2014; 57(3): 127–32
- 39. Jain AKC, Viswanath S. Studying major amputations in a developing country using Amit Jain's typing and scoring system for diabetic foot complications: Time for standardization of diabetic foot practice. *International Surgery Journal* 2015; 2(1): 26–30