

# A Cross Sectional Study to Determine the Distribution of the First Site of Infection in Diabetic Foot and the Etiologies that Caused the Infection

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## Abstract

**Aim:** To study the distribution of the initial site of infection in diabetic foot and associated initiating etiologies.

**Methods:** This cross sectional study conducted in the Department of Surgery, Vardhman Institute of Medical Sciences, Pawapuri, Nalanda, Bihar, India, for 2 years. 100 Patients who initially presented to the investigator and those who could be processed through well enough for identification of necessary data were included in the study.

**Results:** Of 100, 61% of the patients were above 60 years and 39% were below 60. 45% of the patients were male and 55% were females. 63% of them had duration of diabetes of more than 10 years and 37% had duration of less than 10 years. 41% had evidence of peripheral vascular occlusive disease and 59% had no clinical evidence of vascular occlusion. 29% had previous history of diabetic foot infection and 71% did not have. These patients are already predisposed and added with the scarring of previous infection if they were drained using plantar incisions or excisions. 41% of the patients were smokers and 59% non-smokers. Incidence of peripheral vascular disease and amputations is particularly high in smokers. 65% had a body mass index (BMI) above 30 and 35% had a BMI below 30. In addition to predisposing to diabetes, a greater body weight adds to the demand on the foot. 9% of these patients had chronic kidney disease and 91% did not have it. In our study, 29% affected the big toe, 18% the other 3 toes, 15% were in the interdigital spaces, 22% in metatarsal regions, 5% in mid foot, and 11% in heel. Trauma, callosities and foot deformities are some of the predominant causes in the big toe, other 3 toes interdigital spaces, metatarsal heads, mid foot and heel. Fungal infection is common in the interdigital space. Cracks and fissures are common in metatarsal heads, interdigital space and heel.

**Conclusion:** With this study, we are recommending that, during each visit, in addition to routine neural, and vascular clinical examination, the health care personnel should focus on the sites of foot, that has high chance of involvement and the associated risk factors that most commonly contribute to infection at the specific site to help prevent delay of detection of problems and thus save functional feet and limb.

**Keywords:** Diabetic foot, Charcot's foot, Initial site of infection, Associated aetiology of foot infections, Callosity

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## Introduction

Diabetes mellitus is a common disease affecting 1 out of 11 adults, world over. More than one third of the hospital admissions of these patients are consequent upon diabetic foot with infective complications. There is a high amount of expenditure incurred, labor investment and morbidity related to this condition. This condition is also responsible a lot of amputations, and invalidity in the population.

The site of initial infection is varied and its location is decided by multiple factors such as deformities, partial loss of parts of foot consequent upon previous infections requiring amputations, and change of physiological and architectural integrity.

In India, approximately 40,000 legs are being amputated every year, of which 75% are neuropathic with infection. Foot ulceration affects up to 25% of patients with diabetes. 85% of lower limb amputations are preceded by foot ulcers and diabetes remains the major cause of non-traumatic amputation across the world with rates being as much as 15 times higher than in the non-diabetic population. It is estimated that an ankle is lost to diabetes somewhere in the world every 30 seconds, a more important fact is that 85% Of all amputations in diabetes are preventable[1]

The international working group on the diabetic foot had recommended the international system of classification of risk which is given below[2]

Group 0 includes patients have diabetes, but no other risk factors, group 1 includes patients have both diabetes and neuropathy, group 2 includes patients have diabetes, neuropathy, vascular disease, and/or foot deformities, group 3 A includes patients that have a history of foot ulcers, and group 3 B includes patients with previous amputation[2]

Low risk diabetes is characterized by intact sensation, intact pulses and absence of foot deformities, moderate risk diabetes is

characterized by intact sensation and pulses with presence of foot deformities, and high risk diabetes is characterized by absent sensation, absent pulses, with present or absent foot deformities[3]

The common lower extremity neuropathic wound sites are interphalangeal joint sites of the toes, due to limited interphalangeal joint mobility, metatarsal head, due to high pressure and limited joint flexibility, interdigital, due to increased moisture, narrow foot wear, and deformity, bunion sites, due to narrow foot wear and foot deformity, dorsal toes, due to hammer and claw toe deformity and foot wear too shallow in toe box, distal toes, due to poor arterial perfusion, external force and short foot wear, midfoot, dorsal or plantar surface due to Charcot's foot and external trauma, heels, due to unrelieved pressure[2]

According to Seattle diabetic foot study, the following factors were related independently to diabetic foot risk - neuropathic foot: relative risk 2.2 (1.5-3.1), past history of amputation: 2.8 (1.8-4.3), foot ulcer: 1.6 (1.2-2.3), insulin use 1.6 (1.1-2.2), Charcot deformity 3.5 (1.2-9.9), 15 mm higher dorsal foot transcutaneous PO2: 0.8 (0.7- 0.9), and high body mass index: 1.2 (1.1- 1.4). Higher ulcer risk was associated with hammer/claw toe deformity[4] The risk of ulcers and amputations increases with age and duration of diabetes[5] Average duration of diabetes in patients presenting with foot infections tend to be 10 years or more[6] Males have a 1.6- fold increased risk of diabetic ulcers[7]

Charcot's foot, Charcot's arthropathy, Charcot's joint or neuropathic joint is a progressive condition of disruption of musculoskeletal architecture characterized by pathological fractures, joint dislocations, and debilitating deformities of the foot. Once seen mostly associated with syphilis, now it is most commonly associated with diabetes. There is continuous destruction of

bone and soft tissues at weight bearing joints of foot and ankle[8,9]

Based on location of arthropathy 5 patterns have been described by Saunders: pattern 1 involves forefoot, which includes interphalangeal joints, the phalanges, and the metatarsophalangeal joint; pattern 2 involves the tarsometatarsal joint; pattern 3 involves Cuneo navicular, talonavicular, and the calcaneocuboid articulations; pattern 4 involves the talocrural or ankle joint which is the articulation of tibia and fibula with the talus; and pattern 5 involves the posterior calcaneus[10]

Studies show that 45% involves pattern 2 and 35% involves pattern 3.

Brodsky and Rouse system describes 3 types: type 1 involves midfoot, type 2 involves hind foot, type 3A involves the ankle, and type 3B is a pathological fracture of the os calcis tubercle[10]

The multilevel Schon classification system has 4 types. Each of the 4 types has A, B, and C subsets depending on severity of involvement: type 1-lisfranc pattern, type 2- cuneonavicular pattern, type 3-peri navicular pattern, and type 4-transverse tarsal pattern.[11]

Hyperglycemia causes increased levels of intracellular glucose in nerves, leading to saturation of normal glycolytic pathway with shunting of extra glucose into the polyol pathway and converted to sorbitol and fructose by the enzymes, aldose reductase and sorbitol dehydrogenase. Accumulation of these leads to decreased myoinositol, decreased membrane  $\text{Na}^+/\text{K}^+$  ATPase activity, impaired axonal transport and structural breakdown of nerves, causing abnormal potential propagation[12]

The nonenzymatic reaction of excess glucose with proteins, nucleotides, and lipids results in advanced glycation end products that may have a role in disrupting neuronal integrity and repair mechanisms through interference with nerve cell metabolism and axonal transport[13] The increased production of free radicals in

diabetes causes direct damage to blood vessels leading to nerve ischemia and facilitation of advanced glycation end products (AGE) reactions[14]

Diabetic vasculopathy is associated with microvascular retinopathy, nephropathy, and neuropathy and macrovascular complications associated with structural and functional changes in large arteries that lead to increased stiffness, abnormal pulse wave travel, and systolic hypertension. Structural changes mainly result from glycation of wall components and functional changes originating in endothelial dysfunction, smooth muscle cell dysfunction impaired platelet function and coagulation abnormalities, along with protein kinase C

(PKC) activation, foam cell formation due to monocyte activation from oxidative stress and insulin resistance giving rise to plaque formation, all leading to increased arterial stiffness[15,16]

### Material and methods

This cross-sectional study conducted in the Department of Surgery, Vardhman Institute of Medical Sciences, Pawapuri, Nalanda, Bihar, India, for 2 years, after taking the approval of the protocol review committee and institutional ethics committee.

### Inclusion criteria

Patients who initially presented to the investigator and those who could be processed through well enough for identification of necessary data were included in the study.

### Exclusion criteria

Patients who are not willing to participate in the study and already treated cases, where initial site of infection or cause cannot be ascertained with any surety were excluded

### Methodology

This study was conducted on 100 patients who presented with diabetic foot, which

includes all the age groups, who presented with diabetes.

### Statistical analysis

Categorical and continuous variables expressed as frequency. Group comparisons with respect to categorical variables performed using Chi-square test. P less than 0.05 was considered threshold for statistical significance. Statistical

package for the social sciences (SPSS) version 25.0 used for statistical analysis.

### Results

61% of the patients were above 60 years and 39% were below 60. 45% of the patients were male and 55% were females. 63% of them had a duration of diabetes of more than 10 years and 37% had a duration of less than 10 years.

**Table 1: Age and gender distribution of patients**

| Gender       | No. | %  |
|--------------|-----|----|
| Male         | 45  | 45 |
| Female       | 55  | 55 |
| Age in years |     |    |
| Below 60     | 61  | 61 |
| Above 60     | 39  | 39 |

41% had evidence of peripheral vascular occlusive disease and 59% had no clinical evidence of vascular occlusion. In the assessment of neuropathy, both abnormal 10 G monofilament test and biothesiometry measure of more than 25 is accounted. 11% of patients had previous history of amputation, and 89% did not have such a history. Any amputation alters the normal weight transmission and predisposes patients to repeated infections. 29% had previous history of diabetic foot infection and 71% did not have. These patients are already predisposed and added with the scarring of previous infection if they were drained using plantar incisions or excisions. 41% of the patients were smokers and 59% non-smokers. Incidence of peripheral vascular disease and amputations is

particularly high in smokers. 65% had a body mass index (BMI) above 30 and 35% had a BMI below 30. In addition to predisposing to diabetes, a greater body weight adds to the demand on the foot. 9% of these patients had chronic kidney disease and 91% did not have it. In our study, 29% affected the big toe, 18% the other 3 toes, 15% were in the interdigital spaces, 22% in metatarsal regions, 5% in mid foot, and 11% in heel.

Trauma, callosities and foot deformities are some of the predominant causes in the big toe, other 3 toes interdigital spaces, metatarsal heads, mid foot and heel. Fungal infection is common in the interdigital space. Cracks and fissures are common in metatarsal heads, interdigital space and heel.

**Table 2: Site specific etiologies**

| Etiologies              | Big toe | Other 3 toes | Metatarsal heads | Interdigital spaces | Midfoot | Heel |
|-------------------------|---------|--------------|------------------|---------------------|---------|------|
| Trauma                  | 70      | 67           | 54               | 27                  | 75      | 31   |
| Callosity               | 59      | 32           | 76               | 11                  | 12      |      |
| Foot deformity          | 59      | 74           | 42               | 9                   | 88      | 12   |
| Cracks or fissures      |         |              | 30               | 18                  |         | 64   |
| Fungal infection        |         |              |                  | 50                  |         |      |
| Nail infection          | 7       | 22           |                  |                     |         |      |
| Etiology not identified | 2       | 7            | 23               | 18                  | 25      |      |

## Discussion

A review of the literature did not reveal any study of similar nature. Big toe is the commonest site of initial infection, then the ball of foot or region over metatarsal heads, then the four toes, then the interdigital spaces, due to fungal infections, then the heel and finally the midfoot, either due to nails or foreign body.

The initiating factors are often a combination of factors like callosities, deformities and trauma as the introducer of infection. Diabetic neuropathy affects the sensory, motor and autonomic nerves.

In sensory neuropathy, the patient's foot is insensate, yet he has paresthesia, an uncomfortably numb feeling, some complaining of pain, all of which forces them to seek remedy. The usual means they choose locally here is to apply heat and often they present with burnt foot with blebs if dry heat like hot bricks or burning coconut husk are stepped on, or scalds if they put their feet in hot water, which is rarer in incidence. They also find it easy to catch hold of hot vessels by their rim out of stoves presenting 3 or 4 days later in the outpatient department (OPD) alarmed by the blebs on the fingers, not knowing how they got it! They can also wander around their house or in the roads stepping on hot tar or into the ashes of burned leaves, with embers in it[6,8]

So, loss of pain and temperature fibers occur first and are more significant in cause and perpetuation of the malady. These fibers are more prone to damage because they are non-myelinated with unprotected cell membranes which offer a large surface area for leak of  $K^+$  and  $Na^+$  across membrane, each time an action potential is conducted. So, they require many times more energy for restoration of membrane potential and recovery.

When the resources are scarce due to lack of supply, ineffectiveness of what is available due to excessive glycation giving rise to loss of three-dimensional confirmation of proteins, rendering them

dysfunctional, apoptotic changes and damages are a natural result and occur many times faster in these nerves.

Motor neuropathy leads to atrophy and loss of function of the small muscles of the foot faster, and these are responsible as the dynamic elements of weight transmission as well as preservers of the arches of feet. The interossei and lumbricals attached by a common tendon to the extensor expansion seizes to pull, resulting in extension of metatarsophalangeal and flexion of interphalangeal joints giving rise to clawing of the toes, and hammer toe, this makes the toes dysfunctional making the metatarsal heads take the brunt of weight[9,10]

Continuous irritation of any site of skin will initially give rise to a bleb. Removal of this bleb is painful if on the hand, but on the insensate foot of a diabetic, nobody notices it. If the bleb is not thus removed, it gives rise to a callosity, which is an advantage on the hand of a manual laborer, but on the foot, it is like a stone which has been tied under the foot. Walking on such feet, carrying the crushing weight of the entire body will immediately give rise to further accumulation of fluid, which will also add to the callus. Each time the patient walks, this accumulation of fluid occurs, begging to get infected. This will get infected, in due course and it bursts out without deeper destruction if the person is not advanced in his diabetes. Thus, originates a trophic ulcer over a weight bearing area. Any scar of a healed wound in the plantar feet also gives rise to the same problem that is why draining from plantar aspect should be avoided.

Autonomic neuropathy renders the natural shunting of blood away from the skin to deeper structures ineffective, and all the arterioles open up, shunting whatever little blood is available to the skin, leaving deeper tissues more prone to ischemia if vascularity is compromised. So, these people who get a preferential blood supply to dermis has a nice and supple skin, which does not reveal an underlying infection, in

deeper tissues till it is far advanced. Finally, it is revealed by leaking of foul- smelling fluid from the original site of injury, though skin looks perfect. It is on opening up this site for drainage that the destruction underneath is revealed. Unless the patient and bystander are warned beforehand about what is going to be underneath, the surgeon can get blamed for it[11,12]

This also gives rise to another phenomenon, bleb forming burns with live skin below the burn will heal very fast in diabetics, even faster than in non-diabetics! Also, the rate of healing of skin is faster in a deeper wound, than the deeper parts of the wound which is disadvantageous, because these wounds particularly on plantar aspect, if long and deep can heal with a depression of the surface, which opposes the opposite walls, giving rise to cuticular accumulation, acting like callosities described above.

There is a difference between a Charcot's foot getting infected as compared to a usual diabetic foot. In a usual infected diabetic foot, the soft tissues are infected and necrosed and if debrided, leaving the bone surfaces, will heal in time, the blood supply to the bone is usually preserved better, and a bit of decortication once the infection gets under control, or scraping of periosteum will aid in granulation of the wound.

In an infected Charcot's foot, the soft tissues will be intact, the bones also appear well underneath, but pus will keep continuing to pour out from the wound copiously. An x-ray reveals multiple dislocations, loss of shape of bone and crumbling, loss of arches and sometimes rocker bottom feet. Charcot's foot is a situation with multiple underlying fractures and loss of integrity of joints; this will be associated with severe inflammatory reaction, resembling cellulitis, with strong committing to a diagnosis of infection, among the ill-informed and non-suspecting. This is often accompanied by investigation using Doppler and ultrasound, often generating report of fluid accumulation. They will mark it out for

you, and you may have an enthusiastic resident offering incision and drainage in the ward! Instead of pus, some sero-sanguineous fluid or clot will be removed and the wound dressed. An open fracture is an orthopedic emergency, when multiple fractures and joints are converted into an open wound under insufficiently sterile atmosphere, and dressed the same way for multiple days, till one day, when pus starts flowing out of the wound.

During the study period 8 Charcot's feet were encountered, 6 diagnosed on OPD basis and two coming from outside after 6 months of dressing, ending up in below knee amputations. Those diagnosed beforehand were treated with strict rest to the feet. These were excluded from the study as 6 were not infected, 2 presented with infection due to misguided incision.

The 6 cases of burns which came during the study period never got infected and recovered fully by dressing with soframycin and non-adherent gauze once in 5 days.

Also, during the study period three cases of diabetes were diagnosed because they presented with mid foot claudication. Diabetic vasculopathy particularly affects the smaller vessels of the leg like posterior tibial, anterior tibial and dorsalis pedis giving rise to midfoot claudication. Midfoot claudication alone, is pathognomonic of diabetes.

### Conclusion

With this study, we are recommending that, during each visit, in addition to routine neural, and vascular clinical examination, the health care personnel should focus on the sites of foot, that has high chance of involvement and the associated risk factors that most commonly contribute to infection at the specific site to help prevent delay of detection of problems and thus save functional feet and limb.

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