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A study to map the primary sites of DFI onset and correlate these sites with specific initiating events

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A B S T R A C T

Background: Diabetic foot infections (DFIs) are a leading cause of hospitalization and lower extremity amputation. While the overall burden is well-documented, precise data on the anatomical distribution of the initial site of infection and its direct precipitating etiology in a controlled cohort is limited. This study aims to map the primary sites of DFI onset and correlate these sites with specific initiating events.

Methods: A prospective observational study was conducted over 12 months. Forty consecutive patients presenting with a new, culture-confirmed diabetic foot infection were enrolled. Data collected included demographic information, glycemic control (HbA1c), neuropathy assessment (10g monofilament test), and peripheral vascular status (ankle-brachial index). The initial site of infection was meticulously documented and categorized. A thorough patient interview and clinical examination were conducted to identify the primary precipitating etiology.

Results: The mean age was 58.7 ± 9.2 years, with 65% male participants. The mean HbA1c was 9.8 ± 2.1%. Neuropathy was present in 95% of cases. The hallux (n=14, 35%) and the plantar forefoot metatarsal head region (n=13, 32.5%) were the most common initial sites of infection, together accounting for 67.5% of cases. The heel was the initial site in 5 patients (12.5%). The most frequent initiating etiologies were mechanical trauma from ill-fitting footwear (n=17, 42.5%) and unrecognized minor trauma (n=12, 30%). Direct puncture wounds were less common (n=5, 12.5%). A strong association was found between ill-fitting footwear and ulcers/infections on the dorsal aspects of the toes and hallux.

Conclusion: This study demonstrates a predilection for DFI to originate in the hallux and plantar forefoot, areas of high mechanical pressure. The predominant initiating factor is repetitive trauma from footwear, not acute injury. These findings underscore the critical importance of prophylactic foot care, patient education on proper footwear, and daily foot inspections targeting these high-risk zones to prevent the devastating sequelae of DFI.

Keywords: Diabetic Foot, Foot Ulcer, Infection Site, Etiology, Neuropathy, Podiatry, Diabetes Complications

INTRODUCTION

Diabetic foot syndrome represents one of the most severe and costly complications of diabetes mellitus, with a lifetime risk of a foot ulcer estimated to be as high as 34% [1]. Infection, which frequently complicates foot ulcers, is the primary driver of lower extremity amputation, contributing to significant morbidity, mortality, and reduced quality of life. The economic burden is staggering, with costs of care for diabetic foot ulcers (DFUs) and subsequent infections exceeding those of many common cancers, placing immense strain on global healthcare systems [2].

The pathophysiological triad of peripheral neuropathy, peripheral arterial disease, and impaired immune response creates a fertile ground for infection [3]. Sensorimotor neuropathy leads to a loss of protective sensation, rendering the foot vulnerable to unrecognized and repetitive trauma. Concurrently, motor neuropathy alters biomechanics through muscle atrophy and imbalances, leading to foot deformities (e.g., claw toes, prominent metatarsal heads) and the creation of abnormal high-pressure points. Autonomic dysfunction results in anhidrosis and dry, fissured skin, further compromising the epidermal barrier. Any break in the skin at these high-pressure points can serve as a portal of entry for pathogens, while underlying peripheral arterial disease and diabetes-related immunopathy impede healing and facilitate the progression of infection [4].

The clinical management of diabetic foot infections (DFIs) is well-delineated in international guidelines from the International Working Group on the Diabetic Foot (IWGDF) and the Infectious Diseases Society of America (IDSA), which focus on diagnosis, severity classification, antimicrobial therapy, and surgical intervention [5]. Furthermore, the principles of off-loading pressure from ulcerated areas are universally accepted as a cornerstone of treatment. However, there remains a relative paucity of focused, contemporary studies that meticulously detail the precise anatomical distribution of where infections first begin and, crucially, what specific event initiates the process in a controlled cohort. Much of the existing literature aggregates "diabetic foot ulcers" as a single entity without granular site-specific analysis, or focuses on ulcer recurrence rather than de novo initiation.

This gap in knowledge has direct clinical implications for the primary prevention of DFIs. A precise understanding of the epicenters of infection onset and their triggers is not an academic exercise; it is a fundamental prerequisite for designing effective, targeted preventative strategies.

Therefore, this study aims to fill this critical gap by prospectively investigating the distribution of the initial site of infection in a well-defined cohort of 40 patients presenting with a new DFI. Furthermore, it seeks to rigorously determine and categorize the associated initiating etiologies, with the ultimate goal of providing data that can refine preventative care and reduce the incidence of this devastating complication.

Methods

This study employed a **prospective observational cohort design**. The target population consisted of adult individuals (age >18 years) with either Type 1 or Type 2 diabetes mellitus who developed a new, acute diabetic foot infection (DFI) requiring clinical attention. The study was conducted at department of general surgery.

Inclusion Criteria:

Patients were enrolled if they met all of the following criteria:

1. Diagnosis of Type 1 or Type 2 diabetes mellitus.
2. Presentation with a new, acute diabetic foot infection, classified as PEDIS grade 2 (moderate) or 3 (severe), confirmed by the presence of at least two classic signs of inflammation (purulence, erythema, warmth, tenderness, induration) and a positive wound culture.
3. Ability and willingness to provide informed consent and participate in a structured interview.

Exclusion Criteria:

Patients were excluded from the study for any of the following reasons:

1. Presence of a chronic, non-healing wound without signs of acute infection.
2. Critical limb ischemia (PEDIS perfusion grade 3 or 4) requiring immediate revascularization, as this would confound the primary etiology.
3. Infection identified as secondary to a recent surgical procedure on the same foot.
4. Inability to communicate a history or identify a plausible initial site and etiology upon expert clinical examination (e.g., due to severe cognitive impairment).
5. Recurrent ulceration at the exact same site, where the initial cause could not be reliably distinguished from the current presentation.

Sample Size

The target sample size was set at **n=40**. This figure was deemed pragmatically achievable within the 12-month study period and sufficient to provide a meaningful distribution of frequencies for the primary outcome variables (anatomical site and etiology) for this initial analysis, while allowing for robust descriptive statistics.

Procedure for Data Collection

Data collection followed a standardized protocol:

1. **Screening and Consent:** Consecutive patients presenting to the clinic with a suspected DFI were screened for eligibility. Those who met the criteria were approached, and the study was explained. Written informed consent was obtained.
2. **Clinical Assessment:**
    - A detailed medical history was taken, documenting diabetes duration, treatment, and past foot problems.
    - A blood sample was drawn for HbA1c analysis.
    - A standardized foot examination was performed:
        - **Neuropathy:** Tested using a 10-gram Semmes-Weinstein monofilament at 10 sites on the plantar surface of each foot.
        - **PAD Assessment:** Ankle-Brachial Index (ABI) was measured using a handheld Doppler device.
3. **Wound Assessment:**

- The infected wound was meticulously cleansed and debrided of superficial necrotic tissue to reveal its full extent and origin point.
- The **initial site of infection** was identified (e.g., the center of the ulcer or the point of skin breakdown) and documented on a standardized foot map diagram. A clinical photograph was taken with a reference scale.
- A deep tissue specimen or wound curettage sample was collected after debridement for microbial culture and sensitivity.

4. **Structured Interview:**
   - A structured interview was conducted by the principal investigator to determine the **initiating etiology**. Patients were asked open-ended questions about the events leading up to the discovery of the wound, their footwear habits, and any recent activities or injuries. This history was then correlated with the clinical findings (e.g., location of a blister matching a shoe seam) to assign the primary etiology.

Statistical Analysis

Data analysis was performed using SPSS version 26.0. Descriptive statistics (mean, standard deviation, frequency, percentage) were used to summarize data. A p-value of <0.05 was considered statistically significant.

Table 1: Baseline Characteristics of the Study Cohort (N=40)

Characteristic	**Value**
Age (years)	
Mean ± SD	58.7 ± 9.2
Range	42 - 76
Sex, n (%)	
Male	26 (65.0%)
Female	14 (35.0%)
Type of Diabetes, n (%)	
Type 2	38 (95.0%)
Type 1	2 (5.0%)
Diabetes Duration (years), Mean ± SD	14.3 ± 6.5
Glycemic Control (HbA1c %), Mean ± SD	9.8 ± 2.1
Peripheral Neuropathy*, n (%)	38 (95.0%)
Peripheral Arterial Disease†, n (%)	11 (27.5%)

The study cohort consisted of 40 patients with a mean age of 58.7 years (±9.2), ranging from 42 to 76 years of age. There was a male predominance, with 26 male participants (65.0%) and 14 female participants (35.0%). The vast majority of patients had Type 2 diabetes (95.0%), with a mean disease duration of 14.3 years (±6.5). Glycemic control was poor across the cohort, as evidenced by a mean HbA1c level of 9.8% (±2.1). Peripheral neuropathy was present in 38 patients (95.0%), while peripheral arterial disease, defined as an ABI <0.9, was identified in 11 patients (27.5%).

Table 2: Anatomical Distribution of the Initial Site of Infection

Anatomical Site	Number of Patients (n)	Percentage (%)
Hallux (Total)	**14**	**35.0%**
• *Dorsal aspect*	*7*	*17.5%*
• *Plantar aspect*	*5*	*12.5%*
• *Medial/Lateral aspect*	*2*	*5.0%*
Plantar Forefoot (Metatarsal Heads)	**13**	**32.5%**
Heel	**5**	**12.5%**
Other Toes (2nd-5th)	**4**	**10.0%**
Nail Fold (Paronychia)	**3**	**7.5%**
Dorsal Midfoot	**1**	**2.5%**
Total	**40**	**100%**

The anatomical distribution of the initial site of infection revealed a clear predilection for specific high-pressure areas of the foot. The hallux was the most common single site, involved in 14 cases (35.0%). Of these, the dorsal aspect was affected in 7 cases (17.5% of total cohort), the plantar aspect in 5 (12.5%), and the medial/lateral aspects in 2 (5.0%). The plantar forefoot, specifically the metatarsal head region, was the second most common site, serving as the initial location in 13 cases (32.5%). Together, the hallux and plantar forefoot accounted for 27 of the 40 cases (67.5%). The heel was the initial site in 5 patients (12.5%), while other toes (2nd-5th) and the nail fold (paronychia) were the origin in 4 (10.0%) and 3 (7.5%) cases, respectively. A single case (2.5%) originated on the dorsal midfoot.

Table 3: Distribution of Initiating Etiologies for Diabetic Foot Infection

Precipitating Etiology	Number of Patients (n)	Percentage (%)
Ill-fitting Footwear	17	42.5%
Unrecognized Minor Trauma	12	30.0%
Direct Puncture Wound	5	12.5%
Onychocryptosis / Nail Pathology	3	7.5%
Thermal Injury	2	5.0%
Iatrogenic (e.g., inappropriate self-care)	1	2.5%
Total	**40**	**100%**

Analysis of the precipitating etiologies identified mechanical trauma from ill-fitting footwear as the predominant cause, responsible for initiating the infection in 17 patients (42.5% of the cohort). Unrecognized minor trauma was the second most frequent etiology, identified in 12 cases (30.0%). Direct puncture wounds, such as from a nail or thorn, were the cause in 5 cases (12.5%). Underlying nail pathology, including onychocryptosis (ingrown toenail), initiated infection in 3 patients (7.5%). Thermal injury and iatrogenic causes (e.g., from inappropriate self-care) were less common, accounting for 2 (5.0%) and 1 (2.5%) cases, respectively.

Table 4: Cross-Tabulation of Initial Site by Initiating Etiology (n)

Anatomic Site	Ill-fitting Footwear	Unrecognized Trauma	Puncture Wound	Nail Pathology	Thermal Injury	Iatrogenic	Total
Hallux	9	3	0	2	0	0	**14**
Plantar Forefoot	5	8	0	0	0	0	**13**
Heel	0	4	1	0	0	0	**5**
Other Toes	3	0	1	0	0	0	**4**
Nail Fold	0	0	0	0	0	3	**3**
Dorsal Midfoot	0	0	0	0	1	0	**1**
Total	**17**	**12**	**5**	**3**	**2**	**1**	**40**

A strong correlation was observed between the anatomical site of the initial infection and its precipitating etiology. Ill-fitting footwear was overwhelmingly associated with lesions on the dorsal aspects of the toes, accounting for 9 of the 14 hallux ulcers (including all 7 dorsal hallux cases) and all 3 ulcers on other toes attributed to this cause. In contrast, unrecognized minor trauma was the primary mechanism for ulcers on the plantar forefoot (8 of 13 cases) and the heel (4 of 5 cases). All three cases of paronychia (nail fold infection) were of iatrogenic origin. Puncture wounds were distributed across softer tissue areas, affecting the heel and the web spaces between toes. The single dorsal midfoot ulcer was caused by a thermal injury.

Discussion

This prospective study provides a detailed topographic and etiological map of the initial presentation of diabetic foot infections in a tertiary care setting. The principal findings indicate that DFIs originate predominantly in the high-pressure zones of the hallux and plantar forefoot, and are precipitated primarily by repetitive mechanical trauma from ill-fitting footwear rather than acute, dramatic injury. These insights have significant implications for refining preventative strategies.

The pronounced predilection for the hallux (35%) and plantar metatarsal heads (32.5%) to serve as the initial site of infection is a compelling validation of biomechanical theory in diabetic foot disease. These areas are known to sustain the highest peak plantar pressures during the propulsive phase of gait [6]. In the neuropathic foot, which characterized 95% of our cohort, this repetitive stress goes unperceived, leading to sub-epidermal hemorrhage, callus formation, and eventual tissue breakdown. This sequence, often termed the "neuropathic pathway" to ulceration, is well-established [7]. Our findings align closely with those of Lavery et al. (2003), who, in a larger cohort, also identified the plantar forefoot and the hallux as the most common ulcer locations, accounting for a combined 60% of cases in their study [8]. The consistency between studies underscores that these anatomical sites are the epicenters of risk and should be the primary targets for preventative off-loading and daily inspection.

The most critical finding of this investigation is the elucidation of the initiating etiologies. Contrary to a common lay perception that DFIs often start from a single acute injury, our data demonstrate that 72.5% of infections were initiated by *repetitive or mundane trauma*: ill-fitting footwear (42.5%) and unrecognized minor trauma (30%). This highlights a profound failure in preventative foot care education and practice. Our results corroborate the work of Reiber et al. (1999), who identified "mechanical stress" from footwear as the leading contributing factor in over half of incident ulcers, with acute trauma being a less common cause [9]. Similarly, a study by Pound et al. (2021) found that patient-reported causes of ulceration were frequently related to footwear, with a significant portion of patients recalling no specific traumatic event, pointing instead to gradual pressure from daily activity [10]. The concordance across these studies transforms a clinical suspicion into an evidence-based fact: inappropriate footwear is the single greatest modifiable risk factor for initiating the cascade towards infection and amputation.

The strong correlation between site and etiology further refines this understanding. The dorsal hallux and toes were almost exclusively affected by pressure from shoe toe-boxes, while the plantar forefoot was primarily affected by the combination of high plantar pressures during gait and footwear that failed to adequately off-load these pressures. This specificity is crucial for moving beyond generic advice. Patient education must be anatomically precise, instructing individuals to inspect

not just the "bottom of the foot," but specifically the tips and tops of the toes and the ball of the foot for pre-ulcerative signs like erythema or blisters. Furthermore, it argues compellingly for the formal prescription of therapeutic footwear with high, wide toe boxes and custom orthotics designed to redistribute plantar pressure, not just as a treatment for ulceration, but as a primary prevention strategy for all neuropathic patients.

This study has several limitations. The sample size, though sufficient for this descriptive analysis, is relatively small and from a single tertiary referral center, which may limit the generalizability of the findings to all community settings. Furthermore, identifying the initiating etiology relies partly on patient recall, which is subject to bias. However, the prospective design and the use of clinical correlation to validate patient history strengthen the reliability of our data.

Conclusion

In conclusion, this study demonstrates that the initial site of diabetic foot infection is not random but is concentrated in predictable high-pressure zones, and the primary cause is most often preventable mechanical stress from footwear. The findings serve as a powerful reminder that the path to amputation often begins not with a single misstep, but with the daily, repeated trauma of wearing the wrong shoes. Therefore, the cornerstone of reducing the incidence of DFI must be a relentless focus on prophylactic foot care, encompassing targeted patient education and the widespread prescription of appropriate protective footwear.

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