Effects of Limb Length Discrepancy on the Development of Diabetic Ulcers Using Computer Assisted Gait Analysis

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Introduction

Diabetes mellitus is the most rapidly growing cause of the global disease burden.[1] Diabetic foot ulcerations (DFUs) are a manifestation of diabetes associated with difficult/delayed wound healing and a five-year mortality rate of 13-42%.[2] Prevention and effective management of DFUs is crucial to reduce the risk of lower limb amputations and improve the quality of life for patients with diabetes.

Increased plantar pressure is thought to be the major cause of DFUs. The impact of other variables such as limb length discrepancy (LLD) are less understood. Computer assisted gait analysis (CAGA) is an emerging technology that allows physicians to “see the unseen”. Qualitative and quantitative data derived from CAGA can be used to interpret disturbances of gait caused by variables such as LLD.

Objectives

A strategy to reduce costs associated with DFUs is through early identification and treatment before the ulcer reaches a stage that requires inpatient admission. LLD as an independent variable may play a larger role in the development of diabetic ulceration than previously understood. By using empiric data obtained through CAGA, we seek to validate the effects of LLD on the development of diabetic ulceration. We hypothesized that LLD can be used to predict ulcer occurrence.

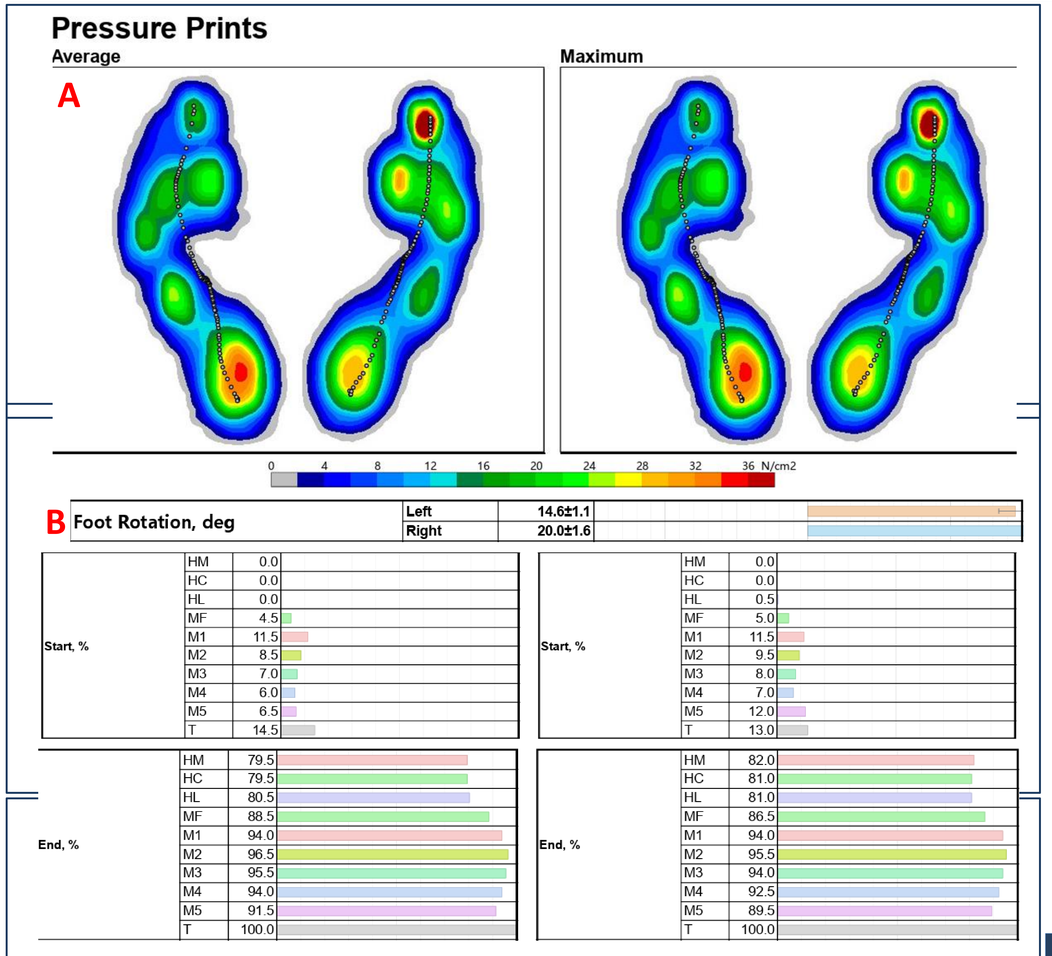
Materials and Methods

108 patients were enrolled in the study. Inclusion criteria were patients with a history of healed diabetic ulceration, participation in CAGA to identify temporospatial gait disturbances, and a LLD of 0.6 mm or greater. Patients were excluded with a LLD < 0.6 mm, history of Charcot neuroarthropathy, or macro traumatic inciting event that could be attributed to the development of their ulceration.

A trained physical therapist (PT) conducted the initial CAGA evaluation whereby patients ambulate on a specialized treadmill which collects temporospatial gait parameters for analysis. Actual and functional LLD measurements were also recorded with the patient laying supine and recording the distance from the anterior superior iliac spine and umbilicus to the medial malleolus.

CAGA data were then interpreted by a team of specialists from podiatry, PT, biomedical engineering, biomechanist, and pedorthist. This data was used to identify abnormal temporospatial gait parameters related to LLD that leads to ulceration.

Results



CAGA stance phase temporospatial gait adaptations of LLD

CAGA data is shown in the figures above. **A.** Center of pressure (COP) mapping demonstrates average and maximum values. The right foot displays a hotspot at the plantar medial forefoot. **B.** Measurement of rotation is shown in which the right foot displays an additional 5.4 degrees of external compared to the contralateral foot. **C.** Start and end time percentages of the foot during stance phase. Average start times of the metatarsals demonstrate that the right foot has an earlier average metatarsal start time compared to the contralateral side during stance, 9.6 versus 7.9%.

32 of 108 patients enrolled had clinically significant LLD as determined by the inclusion criteria. Of these 32 individuals, 17 were found to have macro traumatic inciting events. These macro traumatic events were multifactorial in nature, including improper shoe gear, falls/mechanical injury, or vascular insult. Of the remaining 18 patients, 16 met the hypothesis of the study whereby LLD gait parameters were shown to correlate with at risk areas for ulceration. 2 patients went against the hypothesis. Average LLD 1.36 cm.

LLD can be described one of three ways: anatomic/structural resulting from osseous inequality, functional resulting from unilateral asymmetry without an osseous shortening component, and functioning which is a dynamic measurement of differences of the feet during weight bearing.[3] CAGA provides data that can measure the effects of all forms of LLD. Adaptations to LLD are seen through CAGA with alterations of the center of pressure (COP), internal/external rotation of the foot, impulse recording (force over time) and early heel off/early metatarsals start times.

Although Limb length discrepancies affect 60-95% of the general population, no consensus has been made regarding its clinical significance. White et al. found that discrepancies as high as 3 cm can result in load asymmetry due to compensation.[4] El-nahas artificially induced a LLD of 2 cm and demonstrated that the short limb compensates with significantly increased peak pressure to the lesser metatarsal heads.[5] Even less is understood regarding the biomechanical relevance of more subtle LLD measurements. Pereira et al. found that runners with LLD of 0.5 to 2 cm adopt a compensatory mechanism for their lower limb asymmetry.[6] LLD accommodations are seen in both feet. Walsh et al. found that the short limb adapts by supination and greater degrees of plantar flexion during stance and early heel rise. The long limb accommodates by pronation and increased dorsiflexion at terminal stance and a delay in the timing of this peak dorsiflexion.[7] Perttunen et al. suggested that loading of the long limb is greater, with the foot loading patterns shifted more to the forefoot.[8] There remains paucity in the literature regarding effects of LLD on the diabetic gait.

Our study validates the assumptions of Pereira, with CAGA data demonstrating that the long limb is prone to plantar medial ulceration and the short limb prone to plantar lateral ulceration. The data shows that subtle LLD of even ~1.36 cm may play a crucial role in the development of ulceration. In essence, gait adaptations to LLD can develop in one of several ways, including alterations of the COP line, increased external rotation of the long limb, early heel rise or early start time of the metatarsals during stance phase. These abnormalities are shown through CAGA and typically lead to a plantar medial forefoot ulcer in the long limb or plantar lateral forefoot in an adapted short limb.

Conclusion

We demonstrate the temporospatial gait abnormalities in LLD which may correlate to diabetic ulceration. The practical application of this knowledge is that treatment devices can be made for prevention. We therefore advocate for increased awareness and routine screening of LLD for all diabetic patients in the initial risk assessment for diabetic ulceration.

Citations