Temporal redistribution of plantar pressure points in diabetic and control subjects: A time-series analysis of neuro-capillary chaos

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Abstract

Background
In diabetic individuals (DI), neuropathy hinders the redistribution of plantar pressure points thus leading to susceptible areas where there is constant capillary blanching which may develop into trophic ulcers. The redistribution of pressure points may precede evidence of clinical neuropathy. In this study we compare temporal redistribution of plantar pressure points (areas of capillary blanching) between normal subjects taken as controls and DI with no clinical signs of neuropathy.

Method
Four adults (45±4.55 years) diagnosed to have Type-2 Diabetes, without signs of clinical neuropathy and age -matched controls (43±3.74 years) were studied. The subjects were asked to stand on a glass slab and a 10 minute video recording of 10 selected plantar pressure points was made. Changes in the distance of these points with reference to a defined point on Mayer’s line were measured at every 10 seconds. Standard deviation of difference of redistributed consecutive pressure point (SDPP) in cms., and fractal dimension (FD) was used to compare the two groups.

Results
Combined mean SDPP (DI =0.013 ± 0.008 cms, controls= 0.196±0.233 cms, P <0.001) and FD (DI =1.000 ± 0.000, controls= 1.010±0.017, P <0.001) of diabetic patients were significantly lower than controls. Pressure point at base of the 4th toe and the lower limit of blanching to the left Mayer’s line at the heel did not differ significantly between DI and controls.

Conclusion
There is impaired redistribution of plantar pressure points in individuals with diabetes without signs of clinical neuropathy. This can be attributed to loss of chaos generating mechanisms in DI. Redistribution of pressure points may be essential in the prevention of trophic ulcers in susceptible individuals.

Key Words
Diabetes; neuropathy; trophic ulcers; fractal dimension; chaos
peripheral vascular disease, changes in foot architecture, peripheral sensory neuropathy and the plantar pressure are considered to be the prime etiological factors for the development of ulcers (2, 3). Studies in the past have demonstrated that, the capillary flow is increased rather than decreased in the diabetic neuropathic foot and the high pressure areas are presumed to be associated with increased basal skin blood flow as compared to low pressure areas (4-6). Currently it is believed that the development of plantar pressure ulcers is associated with high amount of pressure exerted on certain regions of the foot (7-10). Studies in the past have shown that the peak plantar pressure is a high risk for plantar ulcers yet it is a poor tool by itself to predict foot ulcers (11).

Considering the minimal amount of pressure (<30 mm Hg) required to occlude the capillary flow, any pressure greater than this is likely to cause an ulcer it would be more appropriate to suspect the prolonged sustenance of pressure at particular points causing unremitting tissue anoxia at these points rather than its magnitude as the prime cause for plantar ulcers. In our previous study we had demonstrated the role of redistribution of the pressure points or the weight bearing points in preventing the development of pressure ulcers in healthy individuals (12). The possible disruption of this bio-mechanism in diabetics owing to peripheral neuropathy may lead to the development of pressure ulcers in susceptible individuals. As sub-clinical neuropathy is may exist for long periods in diabetic individuals (DI) (13), we hypothesize that the loss of redistribution of pressure points may precede evidence of clinical neuropathy. In our current pilot study we aim to compare and quantify temporal redistribution of plantar pressure points (areas of capillary blanching) between normal subjects and DI with no clinical signs of neuropathy.

Method
This study was conducted at a premiere university teaching hospital in South India. The protocol of the study was approved by the Institute ethics committee. Written informed consent was obtained from each subject. Three adult male and 1 female diagnosed to have Type-2 Diabetes as per the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus criteria were selected (12). Patients underwent routine outpatient neurological examination. None of the patients were found to have any abnormalities in sensation or muscle weakness or any evidence of claudication pain. Age matched healthy male individuals were included as controls. The subjects were asked to stand on a transparent plexi glass slab of thickness 1 cm. A digital video camera (Sony DCR-SR87 with 690K pixel resolution) was placed 1 meter below the glass slab and a 10 minute video recordings of the plantar area was done for each of the study participants. Offline analysis of the images at every 10 second intervals was done. Ten pressure points (areas of capillary blanching) were selected from the right foot of all subjects. These pressure points were arbitrarily chosen from a previous study, analyzing pressure point changes in normal subjects (10). Figure 1 depicts the pressure points measured.

The points were:

a) The lowest point of the pale area on the 2nd toe.
b) The lowest point of the pale area on the 3rd toe.
c) The lowest point of the pale area on the 4th toe.
d) The lowest point of the pale area on the 5th toe.
e) A point to the left of the mid-point of the Meyer’s lines (an anatomical line passing through the middle of the great toe and the heel).
f) A point to the right of the mid-point of the Meyer’s line.
g) A point 2 cm above the base of heel, to the left of Meyer’s line.
h) A point 2 cm above the base of heel, to the right of Meyer’s line.
i) A point on the Meyer’s line corresponding to the lower limit of the pale area on the foot.
j) A point on the Meyer’s line corresponding to the upper limit of the pale area on the foot.

Measurement of perpendicular distances of the first eight points with reference to Mayer’s line and of the last 2 points with reference to the lowest point on the heel (also the lowest point on the Meyer’s line).
was done at every 10 seconds. A total of 60 measurements were obtained per point per subject.

For each point the following parameters were calculated:

a) Standard deviation of difference between consecutive redistribution of centre of the pressure point (SDPP) in cm. SDPP was used rather than raw distances since it is invariant to foot size.

b) Fractal dimension (FD) as described by Katz, 1988 (13)

\[ FD = \log (N-1) \div \left[ \log (N-1) + \log (D/L) \right] \]

N = Number of samples (i.e. 60), d = Maximum & L = Total ‘distance’ on waveform. FD (range 1-2) itself has no units.

**Statistical Analysis**

All statistical analysis were done using SPSS version 11. Continuous variables were expressed as mean ± standard deviation. Comparison of parameters between patients and controls was performed using Mann-Whitney U test for exact significance values.

**Results**

Demographics of the subjects are shown in Table 1. The age range for DI was 41-51 years and 38-47 years for controls. Duration of illness for DI at diagnosis ranged from 132-192 months.

(25.1±0.98 kg/m²) (23.6±0.49 kg/m²) Figure 2 depicts the mean distances of the centre of these points with reference to Mayer’s line DI and control subjects Overall variability is higher in normal individuals when compared to diabetics.

Comparison of combined points show SDPP (DI =0.013 ± 0.008 cm, controls= 0.196±0.233 cm, P <0.001) and FD (DI =1.000 ± 0.000, controls= 1.010±0.017, P <0.001) of diabetic patients to be significantly lower than controls. On examination of individual pressure points, (Table 2) two pressure points on the plantar aspect of the foot in diabetics (point 3,8) (pressure point at base of 4th toe, right of the midpoint of Meyer’s line) appear to have similar SDPP and FD in both patients and controls and do not reach significance levels. The FD of point 1 (base of second toe) is comparable between diabetics and controls.

**Discussion**

The present study is a pilot study with measurements done manually. This severely limited the number of patients, recordings and sampling frequency possible. The small numbers would have made any correlation analysis impossible with nerve conduction studies to determine relation with degree of neuropathy. The present study shows that even in a single individual there is variability in the magnitude of redistribution of weight bearing points (Figure 2). Redistribution appears to form a complex time series in normal individuals. In DI this is lost giving rise to a monotonous time series. The resultant waveforms of DI has less fluctuations and lower magnitude that that of controls. The SDPP is a useful measure as it minimizes the affect of foot size and shape. The results suggest that point 3 and 8 (pressure point at base of 4th toe, right of the midpoint of Meyer’s line, have the least variability of redistribution in both controls and diabetics. These points thus can be presumed to be the points through which maximum weight channelized and thus balance is obtained. These points indecently lie along the lateral osseous ridge and give rise to the observation that pressure ulcers occur under osseous pressure points (7-9). Pressure changes appear chaotic in nature and do not follow any set pattern. The overall frequency and magnitude of the redistribution of these points is significantly less in diabetics as compared to healthy individuals, demonstrated by both fractal dimension and statistical measures. As fractal dimension is related to other nonlinear measures of chaos like correlation dimension, maximal Liapunov exponent, we expect that these measures too would be lower in diabetic individuals (DI). It would a much larger time series to calculate these measures and may require image processing tools to extract higher frequency changes. This is in line with the continuous wavelet transformation (CWT) plots that show the time dependent frequency changes of different pressure points in both healthy and diabetics. The CWT plot for healthy individual shows pronounced chaotic redistribution of pressure points
displayed by high frequency color changes depicted by yellow and white (Figure 3a) while those with diabetic sub clinical neuropathy with minimal to nil redistribution of weight bearing points produce a darker images depicted by maroon to completely black image on the CWT (Figure 3b).

It may be concluded that the causation of diabetic ulcers or pressure ulcers may be the result of loss of mechanisms leading to generation of chaos in the pressure distribution in feet. This in contrast to the popular belief that the development of foot ulcers is related to the amount of pressure at a given point (8, 9). We suggest a more intuitively plausible causality for foot ulceration. Ulceration may be linked to the sustenance of pressure rather than the degree as evidenced that very little pressure is required to cause capillary blanching. Tissue anoxia thus should occur if the pressure continues to cause blanching rather than a large pressure that disappears occasionally and does not cause sustained tissue anoxia.

The variation in the frequency of redistribution in different areas of the foot may pose a higher risk of ulcer formation in some specific parts of the foot. These pressure points in the diabetics which display a low frequency of redistribution may be at a higher risk for development of plantar ulcers as compared to other parts of the foot. Points over the heel and third toe seem especially vulnerable with lower frequency of redistribution.

Earlier theories, proposing high pressure point areas on the foot to be at a higher risk for developing plantar ulcers have led to several expensive diagnostic instruments for the recognition of such point (6, 7). However, all such futuristic diagnostic technology may not be accessible to low income group patients in the in remote and economically challenged areas especially in the developing countries. Our method of analysis of such high risk points on the foot of diabetics is simple and inexpensive.

**Conclusion**

Our analysis of 10 randomly selected points shows that the sites of low to minimal frequency of chaotic redistribution of these points is specific for an individual and may vary from person to person. There is impaired redistribution of plantar pressure points in individuals with diabetes without signs of clinical neuropathy. This can be attributed to loss of chaos generating mechanisms in DI. Redistribution of pressure points may be essential in the prevention of trophic ulcers in susceptible individuals. The recognition of such points through such simple techniques may even help low-end shoe manufacturers to design comfortable footwear for sensory neuropathy patients specific to their need which shall provide them protection against the development of pressure ulcers.

**References**

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PEER REVIEW

Not commissioned. Externally peer reviewed.

CONFLICTS OF INTEREST

The authors declare that they have no competing interests.

CONTRIBUTIONS

DVO, CJK: Idea development, data collection and manuscript writing

CP: Data analysis and interpretation and manuscript writing

SCD: Mentored the project
Figures and Tables

Figure 1. Image of plantar aspect of foot with points measured with reference to Meyer’s line

1: The lowest point of the pale area on the 2nd toe. 2: The lowest point of the pale area on the 3rd toe. 3: The lowest point of the pale area on the 4th toe. 4: The lowest point of the pale area on the 5th toe. 5: A pressure point to the left of the mid-point of the Meyer’s lines (an anatomical line passing through the middle of the great toe and the heel). 6: A pressure point to the right of the mid-point of the Meyer’s line. 7: A point 2 cms above the base of heel, to the left of Meyer’s line. 8: A point 2 cms above the base of heel, to the right of Meyer’s line. 9: A point on the Meyer’s line corresponding to the lower limit of the pale area on the foot. 10: A point on the Meyer’s line corresponding to the upper limit of the pale area on the foot.
Figure 2. Mean distances of the centre of these points with reference to Mayer’s line in controls (2a) and diabetic individuals (2b.). The DI time series has fewer fluctuations and lower magnitude than that of controls.
Figure 3. Continuous wavelet transformation plots

a: CWT plot for the healthy individual

b: CWT plot for the chronic diabetic individual with subclinical neuropathy
Table 1: Demographic data of controls and diabetic patients.

<table>
<thead>
<tr>
<th></th>
<th>Control (n=4)</th>
<th>DI (n=4)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)</td>
<td>43±3.74</td>
<td>45±4.55</td>
</tr>
<tr>
<td>Gender (M:F ratio)</td>
<td>4:0</td>
<td>3:1</td>
</tr>
<tr>
<td>Body mass index (kg/m²)</td>
<td>23.6±0.49</td>
<td>25.1±0.98</td>
</tr>
<tr>
<td>Duration of illness (months)</td>
<td></td>
<td>156±25.92</td>
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</table>

DI: Diabetic individuals

Table 2: Statistical and Fractal Dimensional comparison between Diabetic individuals and controls

<table>
<thead>
<tr>
<th>Pressure points</th>
<th>SDPP (Cms.)</th>
<th>Control (n=4)</th>
<th>P*</th>
<th>DI (n=4)</th>
<th>Control (n=4)</th>
<th>P*</th>
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</thead>
<tbody>
<tr>
<td>DI (n=4)</td>
<td></td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>0.017±0.013</td>
<td>0.184±0.129</td>
<td>0.029</td>
<td>1.000±0.000</td>
<td>1.009±0.009</td>
<td>0.057</td>
</tr>
<tr>
<td>2</td>
<td>0.016±0.006</td>
<td>0.291±0.294</td>
<td>0.029</td>
<td>1.000±0.000</td>
<td>1.021±0.024</td>
<td>0.029</td>
</tr>
<tr>
<td>3</td>
<td>0.010±0.007</td>
<td>0.302±0.323</td>
<td>0.114</td>
<td>1.000±0.000</td>
<td>1.023±0.029</td>
<td>0.114</td>
</tr>
<tr>
<td>4</td>
<td>0.019±0.000</td>
<td>0.316±0.258</td>
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<td>1.000±0.000</td>
<td>1.018±0.018</td>
<td>0.029</td>
</tr>
<tr>
<td>5</td>
<td>0.020±0.006</td>
<td>0.081±0.022</td>
<td>0.029</td>
<td>1.000±0.000</td>
<td>1.002±0.001</td>
<td>0.029</td>
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<tr>
<td>6</td>
<td>0.014±0.012</td>
<td>0.115±0.082</td>
<td>0.029</td>
<td>1.000±0.000</td>
<td>1.004±0.004</td>
<td>0.029</td>
</tr>
<tr>
<td>7</td>
<td>0.011±0.008</td>
<td>0.079±0.058</td>
<td>0.029</td>
<td>1.000±0.000</td>
<td>1.002±0.002</td>
<td>0.029</td>
</tr>
<tr>
<td>8</td>
<td>0.008±0.009</td>
<td>0.051±0.025</td>
<td>0.057</td>
<td>1.000±0.000</td>
<td>1.001±0.001</td>
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<tr>
<td>9</td>
<td>0.008±0.009</td>
<td>0.113±0.123</td>
<td>0.029</td>
<td>1.000±0.000</td>
<td>1.006±0.010</td>
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<tr>
<td>10</td>
<td>0.007±0.008</td>
<td>0.429±0.456</td>
<td>0.029</td>
<td>1.000±0.000</td>
<td>1.024±0.027</td>
<td>0.029</td>
</tr>
</tbody>
</table>

SDPP: Standard deviation of difference between consecutive pressure points in Cms. FD: Fractal dimension; DI: Diabetic individuals. Pressure points (1: The lowest point of the pale area on the 2nd toe. 2: The lowest point of the pale area on the 3rd toe. 3: The lowest point of the pale area on the 4th toe. 4: The lowest point of the pale area on the 5th toe. 5: A pressure point to the left of the mid-point of the Meyer’s line (an anatomical line passing through the middle of the great toe and the heel). 6: A pressure point to the right of the mid-point of the Meyer’s line. 7: A point 2 cms above the base of heel, to the left of Meyer’s line. 8: A point 2 cms above the base of heel, to the right of Meyer’s line. 9: A point on the Meyer’s line corresponding to the lower limit of the pale area on the foot. 10: A point on the Meyer’s line corresponding to the upper limit of the pale area on the foot.)