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# 3D Biomechanical Analysis of Foot in diabetes with and without peripheral neuropathy-A pilot study.

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**ABSTRACT**

There has been a profound increase in the prevalence of diabetes mellitus especially among the Asian The biomechanical alteration in the foot structure and function are an important predictive risk factor for development of foot complications in type 2 diabetes mellitus. The routine biomechanical analysis using advanced motion analysis software in a clinical population like diabetes mellitus is still lacking in Indian settings. Therefore the aim of the study was to analyse and compare the biomechanical parameters of foot in diabetes mellitus with and without neuropathy and normal individuals of similar age group. The study was conducted in the biomechanical lab, diabetic foot clinic, Kasturba Hospital, Manipal, Karnataka, India. Sixty participants (n=20 DM type 2 with neuropathy, n=20 DM Type 2 without neuropathy and n=20 normal healthy) participated in the study. The kinematic analysis was done at knee and ankle joint using 3D SIMI REALITY MOTION SYSTEM GmbH, Germany, with two high speed Basler (1394a/b, GigE, 100fps@1Megapixel)cameras. For kinetics I step software, Aetrex, U.S.A and Wintrack dynamic Scan foot mat, Medicapteure software,France, U.S.A was used. Significant difference was seen in kinematic and kinetic variables like knee joint angle at toe off (p=0.002),knee velocity at static, heel strike, midstance and toe-off (p=0.000), knee acceleration at static, heel strike and midstance (p=0.001,0.002,0.006 respectively),ankle joint angle at midstance (p=0.006),ankle velocity at static, heel strike and midstance (p=0.022,0.001,0.002)ankle acceleration at static, heel strike, mistance and toe-off (p=0.013,0.002,0.000,0.000 respectively),gait cycle duration (p=0.000),max average plantar pressure (0.021) and max. great toe pressure (p=0.025). There biomechanical differences among the diabetes with and without neuropathy and normal healthy are high and therefore biomechanical analysis is an important tool and can be used for early screening and prediction of altered kinematic and kinetics in diabetes mellitus population in India.

**Keywords:** Type 2 diabetes mellitus, kinetics, kinematics, biomechanics, peripheral neuropathy

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## INTRODUCTION

Diabetes Mellitus (DM) has been an area of interest and intensive research over the years world-wide.There has been a profound increase in the prevalence of diabetes mellitus especially among the Asian Population. The World Health Organization has declared India as the Diabetic Capital of the world [1]. The prevalence of diabetes for all age-groups worldwide was 366 million (6.4%) in 2011 and it is estimated to increase to 552 million (7.7%) by 2030 [1].The International Diabetes Federation (IDF) estimated that the total number of people in India with diabetes to be approximately 50.8 million in 2010, rising to 87.0 million by 2030. The prevalence of peripheral neuropathy (DPN) among Diabetes has been reported tobe 33.33% in Indian population [2].Therefore; they are prone todevelop frequent and often severe foot problems with a relatively high risk of infections, plantar ulcer, gangrene and amputation [3].

Diabetic Foot Syndrome is a clinical trial of neuropathy, vascular and structural changes. Foot ulceration and amputation is a potential complication and manifestation of a diabetic foot. The exact mechanism behind diabetic foot is still unclear; however it is now very well known that diabetic foot ulcers are caused by mechanical trauma to an intensive foot with underlying vascular insufficiency and peripheral neuropathy [4]. The biomechanical alteration in the foot structure and function are an important predictive risk factor for development of foot complications in type 2 diabetes mellitus which is extensively studied and reported in previous literature [5]. The biomechanical factors include kinematic variables like joint angle, joint velocity, stiffness, joint acceleration, power etc whereas the most important kinetic variable of interest is ground reaction force and plantar pressure. Previous study has shown a positive relation between high plantar pressure and diabetic foot. High and abnormal plantar pressure distribution is the most common predictive risk factor for foot ulceration in diabetic population [3].The study done by Williams et al. 2007 also established a positive correlation between a kinematic variable like joint stiffness and diabetic neuropathy during late stance phase of the gait cycle [6].

In routine clinical practice, diabetes foot evaluation is focused on assessment of protective, vibratory sensation and ankle brachial index for screening neuropathy and vascular complications. However the routine biomechanical assessment and analysis using advanced motion analysis software of human movement in a clinical population like diabetes mellitus is still lacking in Indian settings. Even though the prevalence of type 2 diabetes mellitus with peripheral neuropathy is very high in Indian population, there is a dearth in studies on early assessment of biomechanics focusing on kinetic and kinematics movement analysis of diabetic foot to predict the plantar ulcer.Considering high burden of diabetes foot care in the Indian population, we have proposed thisstudy. Therefore, the aim of the present study is to find the biomechanical parameters for early prediction of plantar ulcer in T2DM with peripheral neuropathy and compare the kinetic and kinematics in T2DM without neuropathy, with neuropathy and age matched normal individuals.

## METHODOLOGY

Present cross sectional study was conducted at the Diabetic Foot clinic, Kasturba Hospital, Manipal University, Karnataka, India. After obtaining Institutional Ethical clearance (IEC) and an written informed consent from all the subjects, A total of sixty participants in three groups with n=20 in each group, volunteered into the study under the purposive sampling method. The mean and S.D for demographic data of all the participants in each group were as follows. For the neuropathy participantsage in years (52.83±11.78), height in cm (165.42 ±7.86), weight in kg (61.61±19.26), BMI (22.6±4.7) and duration of disease (9.16±5.13). For non-neuropathy participants age (56.5±11.57), height (163.92±11.97), weight (67.87±12.51), BMI (25.16±2.61) and disease duration (9.16±5.23) whereas for the normal participants age (57.33±12.33), height (164±6.26), weight (73.68±10.23), and BMI (25.2±3.52).

For the purpose of kinematic variables like joint angle, joint velocity and acceleration 3D SIMI REALITY MOTION SYSTEM GmbH, Germany was used with two high speed Basler (1394a/b, GigE, 100fps@1Megapixel)cameras. For plantar pressure analysis I step software, Aetrex, U.S.A and for walking speed the Wintrack dynamic Scan foot mat, Medicapteure software, France, U.S.A was used. Lower limb kinematics at knee and ankle joint was calculated using five retro-reflective markers (9mm) as shown in Fig 1.

As a standardized biomechanical procedure average of three trials of gait at normal walking speed was taken from each participant. The gait way consisted of 10 m walkway and a starting line was given for all

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participants. The retro-reflective markers were placed at 2nd MTP joint, lateral malleolus and shank for the ankle joint whereas for the knee joint markers were placed at lateral mid-thigh, lateral epicondyle, shank and lateral malleolus as shown in the Fig1. The raw data was filtered using second orderButterworth filter at a frequency of 10-400 Hz.

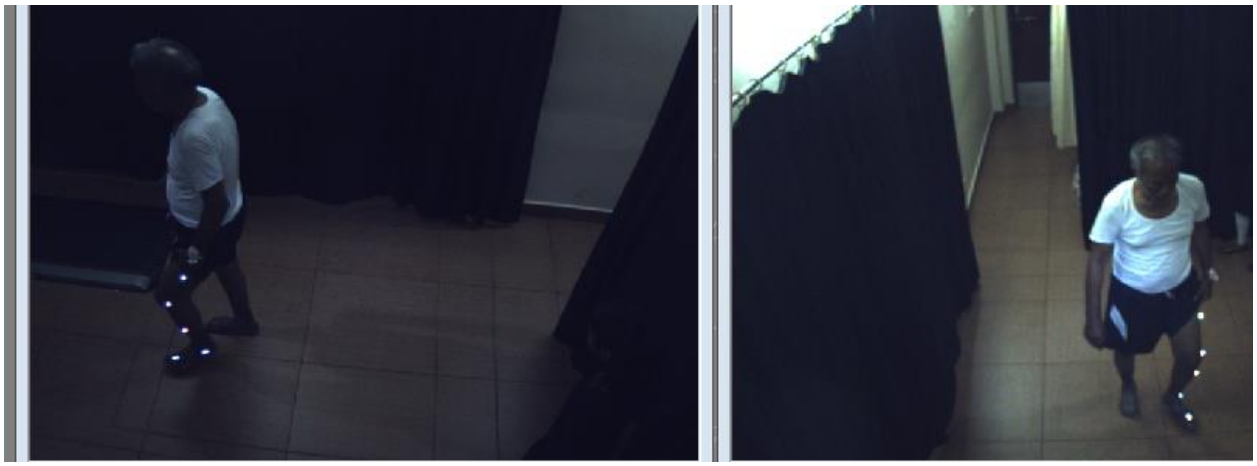

**Fig.1. Showing marker placement for knee and ankle joint kinematics.**

Statistical analysis was carried out using SPSS package version 16 for Microsoft windows. Descriptive statistics was done to calculate mean and standard deviation for demographic variables. The test of normality was done using Shapiro Wilko test after which one way ANOVA was used to compare all the outcome variables of interest. In all cases SPSS version 16 was used and significant statistical difference was set as p≤0.05.

## RESULTS

The descriptive analysis of kinematic and kinetic variables along with comparison of means (p value) using one way Anova between three groups have been presented in table 1 whereas table 2 represents the point of higher difference using post-hoc test.

**Table1. Kinematic and Kinetic characteristics of the subjects in the groups**

| **KNEE JOINT ANGLE (°)** | **MEAN ± S.D** | **p value** |
|---|---|---|
| Static | 170.94±5.33 | 0.253 |
| At heel strike | 167.57±4.97 | 0.774 |
| At mid-stance | 167.68±6.19 | 0.119 |
| At toe-off | 151.56±8.52 | 0.002** |
| KNEE JOINT VELOCITY (°/S) | MEAN± S.D | p value |
| Static | 1.89±2.6 | <0.001 |
| At heel strike | 72.85±47.85 | <0.001 |
| At mid-stance | 38.28±17.55 | <0.001 |
| At toe-off | 226.41±75.40 | <0.001 |
| KNEE JOINT ACCELERATION (°/s²) | MEAN± S.D | p value |
| Static | 9.79±7.76 | 0.002** |
| At heel strike | 1229.1±817 | 0.001** |
| At mid-stance | 431.7±382.16 | 0.006** |
| At toe-off | 1543±78.3 | 0.338 |
| ANKLE JOINT ANGLE (°) | MEAN± S.D | p value |
| Static | 142.48±6.35 | 0.067 |
| At heel strike | 134.72±6.58 | 0.268 |
| At mid-stance | 131.77±7.6 | 0.006** |
| At toe-off | 116.54±12.45 | 0.176 |
| ANKLE JOINT VELOCITY (°/S) | MEAN± S.D | p value |
| Static | 0.59±0.68 | 0.022* |

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| | | |
|---|---|---|
| At heel strike | 43.48±24.2 | <0.001 |
| At mid-stance | 42.34±31.38 | 0.002** |
| At toe-off | 156±93.05 | 0.358 |
| ANKLE JOINT ACCELERATION ($^o/s^2$) | MEAN± S.D | p value |
| Static | 6.06±5.63 | 0.013* |
| At heel strike | 1098.6±966.2 | 0.002** |
| At mid-stance | 129.5±105.77 | <0.001 |
| At toe-off | 1238±627.22 | <0.001 |
| GAIT CYCLE DURATION (ms) | 1227.5±320.81 | <0.001 |
| MAX. AVG.PLANTAR PRESSURE | 1905.5±444.53 | 0.021* |
| MAX.GREAT TOE PRESSURE | 0.38±0.28 | 0.025* |

**Table2. Comparision of kinetic and kinematic characteristics between the groups**

| Dependent variables | Comparable groups | 95% Confidence Interval | | p |
|---|---|---|---|---|
| | | Lower bound | Upper bound | |
| Knee joint angle at toe -off | Neuropathy to Normal | 2.8272 | 15.2318 | 0.002 |
| Static knee joint velocity | Neuropathy to non-neuropathy | 4.1091 | 0.6029 | 0.006 |
| | Neuropathy to Normal | 1.1184 | 4.6246 | 0.001 |
| Knee joint velocity at heel strike | Neuropathy to Normal | 77.2511 | 103.9389 | 0.000 |
| | Non-neuropathy to Normal | 83.5276 | 110.2154 | 0.000 |
| Knee joint velocity at midstance | Neuropathy to non-neuropathy | 5.7748 | 30.1362 | 0.002 |
| | Non-neuropathy to Normal | 7.6768 | 32.0382 | 0.001 |
| Knee joint velocity at toe off | Neuropathy to Normal | 28.1361 | 123.3249 | 0.001 |
| | Non-neuropathy to Normal | 64.6521 | 159.8409 | 0.000 |
| Static Knee acceleration | Neuropathy to non-neuropathy | 1.4056 | 12.6614 | 0.011 |
| | Neuropathy to Normal | 1.8146 | 13.0704 | 0.006 |
| Knee acceleration at heel strike | Neuropathy to Normal | 1263.7659 | 1843.5791 | 0.000 |
| | Non-neuropathy to Normal | 1244.9349 | 1524.7481 | 0.000 |
| Knee acceleration at midstance | Non-neuropathy to Normal | 77.6889 | 645.1481 | 0.009 |
| Static Ankle joint angle | Neuropathy to Normal | 0.9172 | 8.8882 | 0.033 |
| Ankle joint angle at midstance | Neuropathy to Normal | 1.5329 | 12.7701 | 0.009 |
| | Non-neuropathy to Normal | 0.1161 | 11.1211 | 0.056 |
| Static ankle joint velocity | Neuropathy to Normal | 0.0668 | 1.0682 | 0.023 |
| Ankle joint velocity at heel strike | Neuropathy to non-neuropathy | 17.4694 | 43.2906 | <0.001 |
| | Neuropathy to Normal | 0.2961 | 25.5251 | 0.057 |
| | Non-neuropathy to Normal | 30.0839 | 55.9051 | <0.001 |
| Ankle joint velocity at midstance | Neuropathy to Normal | 50.9457 | 82.8573 | <0.001 |
| | Non-neuropathy to Normal | 42.0192 | 73.9308 | <0.001 |
| Static ankle joint acceleration | Non-neuropathy to Normal | 0.8434 | 9.2826 | 0.015 |
| Ankle acceleration at heel strike | Neuropathy to Normal | 190.0383 | 1590.7257 | 0.009 |
| | Non-neuropathy to Normal | 211.1550 | 1611.8473 | 0.007 |
| Ankle acceleration at midstance | Neuropathy to Normal | 106.3037 | 293.1722 | <0.001 |
| | Neuropathy to non-neuropathy | 114.1233 | 300.9917 | <0.001 |
| Ankle acceleration at Toe-off | Neuropathy to non-neuropathy | 402.2798 | 1286.3441 | <0.001 |
| | Neuropathy to Normal | 151.3999 | 995.4561 | 0.005 |
| Gait cycle duration | Neuropathy to non-neuropathy | 194.3417 | 554.7873 | <0.001 |
| | Neuropathy to Normal | 390.6127 | 741.0873 | <0.001 |
| | Non-neuropathy to Normal | 21.0627 | 371.5373 | 0.024 |
| Max. avg. Plantar pressure | Neuropathy to Normal | 17.4246 | 689.7463 | 0.037 |
| | Non-neuropathy to Normal | 26.9954 | 645.3514 | 0.059 |
| Max. great toe pressure | Neuropathy to Normal | 0.0246 | 0.4528 | 0.025 |

A very high statistically significant difference can be seen in both kinematic and kinetic variables among the three groups. Table1. clearly shows that the difference in kinematic variables like knee joint angle at toe-off, knee joint velocity at static, heel strike, midstance and toe-off, joint acceleration at static, heel strike and midstance, joint angle at midstance, ankle joint velocity at static, heel strike and midstance, ankle acceleration at static, heel strike, midstance and toe-off and gait cycle duration was significant. The kinetic

variables like maximum average plantar pressure and maximum great toe pressure also showed significant difference. Table2 represents the post-hoc analysis results and the point of higher difference between the two groups can be clearly understood. The results from post hoc also show that the difference was higher between neuropathy and normal individuals compared to non-neuropathy and normal. Some variables also showed a significant difference between non-neuropathy and neuropathy participants.

## DISCUSSION

The biomechanical analysis of foot in diabetic population can be a very useful tool for early prediction of diabetic foot complications including ulcers as well as their prevention by altering and managing the altered mechanics[7]. As stated earlier the detailed biomechanical investigation has been lacking in Indian setting. However it is very evident from the present study that there was a lot of difference in the biomechanical parameters of foot between normal individuals and diabetic participants with and without neuropathy in India. Also the presence of neuropathy in diabetic population has worsened the underlying risks to this group. Table 1 suggests that there was a significant clinical and statistical difference among diabetic participants with neuropathy, without neuropathy and age matched normal while reporting kinematic parameters like knee joint angle at toe-off (p=0.002),knee joint velocity at static, heel strike, midstance and toe-off with a highly significant p value of 0.000 each. Apart from these there was also a significant difference for knee joint acceleration at static, heel stike and midstance position (p=0.002, 0.001 and 0.006 respectively). Similar findings were seen at the ankle joint and the significant difference in kinematic variables included ankle joint velocity and acceleration at heel strike, midstance and toe off as depicted in table 1. It is important to note that unlike knee joint the ankle joint showed a significant p value of 0.000 for acceleration at toe-off which suggests that the ankle joint requires more power and velocity to propel the body forward at toe-off. The results from post –hoc analysis in Table 2 suggested the point of difference between the two groups for the significant variables as discussed in detail below:

### Kinematics of the foot

Joint angle: The significant difference for knee joint angle at toe-off on post-hoc analysis with p value of 0.002 was obtained between neuropathy and normal group. The descriptive analysis showed that the neuropathy participants walked with more knee flexed angle (147.15±7.4) compared to normal group (155.84±8.92). The higher knee flexion at toe-off in neuropathy could be associated with musculoskeletal changes as a consequence of motor neuropathy. It could lead to proximal weakness as well as tightness of hamstrings and calf muscles [8].

Joint velocity: As reported earlier from Table.1 that there was a highly significant difference in knee and ankle joint velocity at multiple phases of gait cycle, the results from the post-hoc test suggested that there joint velocity in neuropathy and non-neuropathy was lower compared to normal individuals. However between the neuropathy and non-neuropathy lower joint velocity was seen in neuropathy group (Table2). A Study has shown that large diameter neuropathy can lead to slower nerve conduction [9].The reason for slower joint velocity in non-neuropathy could be related to psychological status, undergoing neuropathy that might be latent and not symptomatic. The other reason could be attributed to the musculoskeletal changes like tightness and weakness in lower extremity along with painful neuropathy so that the limb has lesser arc or range of motion to generate higher joint velocity. Apart from this painful neuropathy like burning and tingling sensation could be a major cause for generating lesser power and thus reduced joint velocity could be found. It should be noted that mid-stance joint velocity was minimal as the limb segment moved by smaller arc, therefore the larger the arc of movement the higher could be the joint velocity and thus the difference in joint velocity at different phases of gait cycle could be a dependent variable on available range of motion.

Joint Acceleration: Similar to joint velocity, the knee and ankle joint acceleration was significantly reduced in neuropathy and non-neuropathy compared to normal participants. Among the neuropathy and non-neuropathy higher values of acceleration was seen in non-neuropathy which was expected. Since the knee and ankle joint segment had lower values of joint velocity in neuropathy group, therefore the change in velocity over time could also be less and thus lower knee and ankle joint acceleration was seen compared to normal and non-neuropathy participants. It can also be suggested that the knee and ankle joint in neuropathy participants has lesser power to generate the higher velocity and indeed joint acceleration due to muscular weakness and other possible causes as explained above. It should be noted that the midstance phase and toe

off phase requires greater strength and velocity to support and shift body weight over the other extremity but since diabetic neuropathy could affect with joint strength, joint sensation and proprioception the lesser values of joint velocity and acceleration were accounted.

Gait Cycle Duration: Walking speed or gait cycle duration has been a very important kinematic variable reported by many previous studies. A marked difference was seen in the walking speed among the comparable groups in different studies. Gomes et al. 2011; Savelberg et al. 2010, Sawacha et al. 2009 and Mansoo et al. 2011 found that DPN( diabetes with neuropathy) subjects had slower walking speed than DMC (without neuropathy) and HC( healthy control) subjects [3,9,10,11]. These findings could be seen as a compensatory and adaptive mechanism to reduce plantar pressure as slower walking speed has been reported to reduce the peak plantar pressure at forefoot during barefoot walking [11]. But these findings were controversial to the result obtained by Yavuzer et al. 2006 who reported that the walking speed of the DPN was faster than both DMC and HC [12]. On the contrary the meta-analysis report by Fernando et al. 2013 demonstrated no significant difference in walking speed and stride length. However the recently published study by Fortleza et al. 2014 reported a significant slower walking speed in DPN compared to control participants [13]. Our study reported that there was significant difference in gait cycle duration hence walking speed among the three comparable groups with a mean of 1227.5 ±320.81 Ms and p value of 0.000. The post hoc analysis suggested that the difference lied between each group and neuropathy group had the least walking speed.

**Kinetics of the foot**

Maximum Average Plantar Pressure: Previous literature has reported that high plantar pressure is the most common predisposing factor for developing foot ulcers in diabetes population [7]. It can be acknowledged that diabetes lead to characteristics changes to the structure of foot which affects the biomechanics of the patients in several ways. Such structural changes often lead to high and abnormal plantar pressure distribution [14]. Boulton and associates reported that a peak pressure of 1100 kPa is a higher threshold for developing a foot ulcer in diabetes with neuropathy. Armstrong et al. 1998 also suggested that a peak pressure of 65N/cm² in diabetes patients can leak to high risk of ulceration by six times [15]. In our study we found that was a significant difference in peak average plantar pressure among diabetic and normal group. In our study, we found the mean average plantar pressure of 1905.5 ±444.53 kPa with a p value of 0.021 suggestive of high statistical significant difference and higher risk of plantar pressure. On post hoc analysis greater difference was found between neuropathy to normal (p=0.037) compared to non-neuropathy to normal (p=0.059).

Maximum Great Toe pressure: The great toe or the metatarsal head is the most common area for developing diabetic foot ulcers as reported in previous studies. Our study has shown that there was a significant difference in great toe pressure between the neuropathy and normal group with a p value of 0.025. The neuropathy group showed the highest pressure at the great toe which suggest that they are more prone for developing plantar ulcer in that area.

## CONCLUSION

The findings of the study are novel in the Indian context and thus hold a high significance for clinical assessment and analysis of biomechanical aspects of diabetic foot. The results can be used as a screening method for kinetic and kinematic analysis of foot in clinical population and the altered biomechanics can be corrected through appropriate intervention exercises. Correction of altered biomechanics could reduce the abnormal pressure on foot which in turn could prevent ulcers and future amputation and salvage.

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