# STRUCTURED EXERCISE PROGRAM ON FOOT BIOMECHANICS & INSULIN RESISTANCE AMONG PEOPLE LIVING WITH TYPE 2 DIABETES WITH AND WITHOUT PERIPHERAL NEUROPATHY

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**BACKGROUND**: The biomechanical changes in foot among people with type 2 diabetes mellitus often leads to various foot complications which could further add to diabetes related morbidity & mortality. These changes could be seen due to musculoskeletal factor like reduction in intrinsic foot muscle strength, tightness of lower limb muscles, postural changes, etc.

**AIM**: To design and determine the efficacy of a structured exercise program on foot kinetics and kinematics among type 2 participants

**MATERIALS AND METHODS**: A total of 35 participants with type 2 diabetes (n=15, type 2 diabetes mellitus without neuropathy and n=20, type 2 diabetes with peripheral neuropathy) were recruited. All participants were screened clinically & biochemically and given a set of structured exercise program, three times a week for 12weeks along with standard medical care.

**RESULTS**: The mean age of the participants was 56±10.5 (Non neuropathy) 62.3±7.35 (Neuropathy) years, mean duration of diabetes was 8.7±8.95 (Non neuropathy), 10.97±8 (Neuropathy) years, mean Body mass index was 26.54± 4.83(Non neuropathy), 24.39±3.58 (Neuropathy), Significant differences have been observed in kinetic and kinematic variables.

**CONCLUSION**: The present study shows that the structured exercise protocol is very effective in improving the biomechanics of foot in people with type 2 diabetes with and without neuropathy. It could help to correct the structure and function of the foot and eventually could reduce the risk of foot complications like diabetic foot ulcers.

*KEYWORDS: plantar pressure; HOMA-IR; exercise training; peripheral neuropathy, diabetic foot*

# СТРУКТУРИРОВАННАЯ ПРОГРАММА УПРАЖНЕНИЙ НА БИОМЕХАНИКУ НИЖНИХ КОНЕЧНОСТЕЙ СРЕДИ ПАЦИЕНТОВ С САХАРНЫМ ДИАБЕТОМ 2 ТИПА С ПОЛИНЕЙРОПАТИЕЙ И БЕЗ НЕЕ

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**ВВЕДЕНИЕ**. Биомеханические изменения нижних конечностей у пациентов с сахарным диабетом 2 типа (СД2) часто приводят к различным осложнениям стопы, которые могут еще больше усугубить заболеваемость и смертность, связанные с диабетом. Эти изменения возникают по причине костно-мышечных изменений, таких как снижение силы мышц стопы, упругости мышц нижних конечностей, постуральных изменений и т.д.

**ЦЕЛЬ**. Разработать и определить эффективность структурированной программы упражнений на кинетику и кинематику нижних конечностей среди пациентов с СД2.

**МЕТОДЫ**. Было привлечено в общей сложности 35 пациентов с СД2 (с СД2 без нейропатии – 15 и с СД2 с периферической полинейропатией – 20). Все участники были подвергнуты клинической и биохимической проверке, после чего три раза в неделю в течение 12 нед проделывали структурированную программу упражнений, получая при этом стандартную медицинскую помощь.

**РЕЗУЛЬТАТЫ**. Средний возраст участников составил 56±10,5 (без нейропатии) и 62,3±7,35 (с нейропатией) года, средняя продолжительность диабета составила 8,7±8,95 (без нейропатии) и 10,97±8 (с нейропатией) года, средний индекс массы тела – 26,54±4,83 (без невропатии), 24,39±3,58 (с нейропатией). Значительные различия наблюдались в кинетических и кинематических показателях.

**ЗАКЛЮЧЕНИЕ**. Настоящее исследование показывает, что протокол структурированных упражнений очень эффективен в улучшении биомеханики стопы у пациентов с СД2 как с нейропатией, так и без нее. Выполнение данных упраж-


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нений может корректировать структуру и функцию стопы и в конечном итоге может снизить риск развития диабетических язв.

*КЛЮЧЕВЫЕ СЛОВА: подошвенное давление; HOMA-IR; тренировочное обучение; периферическая нейропатия; диабетическая стопа*

## BACKGROUND

Diabetes mellitus is a worldwide epidemic metabolic disorder causing significant morbidity and mortality [1]. The prevalence of type 2 diabetes mellitus has increased drastically with the highest impact on India [1]. Out of all 78.3 million diabetes population that is in South East Asia, India has become the global capital of diabetes. It is expected to rise to 131 million by the year 2040 [2]. Over the past 30 years, the status of diabetes has changed from being considered as a mild disorder of the elderly to one of the major cause of morbidity and mortality affecting youth and middle age people. In type 2 diabetes, there will be elevated glucose levels in circulating blood, caused by impairment in glucose tolerance which leads to the development of insulin resistance(IR). The kinetic and kinematics of the foot changes among people with type 2 diabetes mellitus and has been reported [3]. These changes often lead to various foot complications which could further add to morbidity, mortality, social and, economic burden associated with the disease [4].

Kinetic factors like increased plantar pressure have been regarded as the most important etiological factor for causing diabetic foot ulcers [5]. Studies have shown significantly higher plantar pressure among type 2 diabetes individuals with and without peripheral neuropathy compared to normal non-diabetes individuals [3,6]. Kinematic parameters like walking speed, joint velocity, joint acceleration, joint range of motion, joint power and spatial-temporal gait characteristics have also shown to be significantly affected [6]. These changes could be seen due to musculoskeletal factor like reduction in intrinsic foot muscle strength, tightness of lower limb muscles, postural changes, etc. A study concluded that there is a significant reduction in lower limb muscle strength (ankle and knee joint) in people with type 2 diabetes with and without neuropathy, and it is closely associated with altered kinetics and kinematics leading to impaired mobility and quality of life [7]. Reduction of proximal muscle strength is more evident in diabetes with neuropathy [7]. A study concluded that participants with diabetes peripheral neuropathy demonstrated a reduction in muscle strength , muscle quality and contractile slowing suggestive of loss in muscle power and functional impairments [8]. Currently, the most significant notable motor neuropathy of diabetes mellitus is ischemic infarction of thigh muscles including vastus lateralis, thigh adductors and biceps femoris which could be a major factor for reduced muscular forces [9]. As suggested above, the reduced joint range of motion/ limited joint mobility may lead to elevated plantar pressure and pressure time integrally predisposing to increased risk of foot complication [10]. Similarly, a close association between calf muscle tightness with increased plantar pressure among type 2 diabetes has been hypothesized under a mechanism called 'split site effect' [11]. A study also reported reduced passive range of dorsiflexion at ankle among diabetes participants compared to the non-diabetes individual at rest. This could lead to increased static plantar pressure in weight-bearing areas [12]. Postural changes with static and dynamic imbalance have also been reported in type 2 mellitus participants. It has been suggested to be more with diabetes with polyneuropathy due to sensory and motor deficits [13].

Changes in static and dynamic posture could be directly related to altered plantar pressure distribution [14]. From the previous study, it is evident that there are significant changes in foot kinetics and kinematic parameters which are biomechanically determinative. There could be a strong interaction between kinetics and kinematics predominated by musculoskeletal changes at knee and ankle joints. It is also very well known that exercises play a significant role in controlling the hyperglycemic state by utilizing the excess glucose with contraction of a larger group of muscles. The primary component of these exercises consists of physical activity with more significant importance to aerobic and resistance modes of exercises for upper and lower extremities. Physical activities have been shown to improve blood glucose control along with positive effects on lipid profile, blood pressure, cardiovascular events and weight management [15]. However, a comprehensive, structured exercise for correction of foot biomechanics in type 2 diabetes mellitus is lacking. Thus, there is a strong need to propose a set of specific exercises to control and prevent the musculoskeletal changes in foot among people with diabetes to prevent foot-related complications.

Therefore the study aimed to design and determine the efficacy of a structured exercise program on foot kinetics and kinematics among type 2 participants. The objective of the study is to find out the effect of a comprehensive, structured exercise program on foot biomechanics and Insulin resistance among people living with T2DM with and without peripheral neuropathy.

## METHODS

### Study design and setting

The pre-post experimental study conducted at the Diabetic Foot Clinic, Kasturba Hospital, Manipal, Karnataka, India.

### Conformity criteria

Participants with a known case of T2DM with neuropathy between the age group of 30 – 75 years were selected for the study. Participants with a known history of neurological disease, locomotor disability, and pregnancy were excluded from the study.

### Research facilities

The study was conducted at the Diabetic Foot Clinic, Kasturba Hospital, Manipal, Coastal Karnataka, Southern India.

**Research duration**

The study period was between June 2017 to May 2018.

**Participants**

A total of 35 participants with type 2 diabetes (n=15, type 2 diabetes mellitus without neuropathy and n=20, type 2 diabetes with peripheral neuropathy) were recruited under the purposive sampling method. An informed consent form was signed by each participant following which the testing protocol was demonstrated before data collection. Data were collected at baseline and 3rd month.

**Procedure description**

All participants were diagnosed with type 2 diabetes mellitus from the hospital biochemistry lab based on standard guidelines from American diabetic Association using fasting blood sugar level, 2-hr postprandial blood sugar level, 75 g oral glucose tolerance test, and $HbA_{1c}$. The test for presence the of peripheral neuropathy (sensory and motor) was confirmed using 10 g Monofilament test, Biothesiometer (Vibration Pressure Threshold) and Manual Muscle testing. Michigan Neuropathy Screening Instrument (MNSI) was also used for each participant. The kinetic and kinematic parameters that were considered in the given study have been described below along with the outcome measures used.

**Kinetic parameters**

Average plantar pressure, peak (Maximum) plantar pressure, forefoot, and hindfoot pressure were measured both statically and dynamically. The Wintrack software Medicapteur France, USA was used to measure this parameter. Muscular force for lower limb muscles like hip flexors, hip extensors, hip abductors, quadriceps, hamstrings, ankle dorsiflexors, ankle plantar flexors, ankle everters and intrinsic muscles of the foot (extensor hallicus, lumbricals, and interosseus) were measured using Manual Muscle Testing grading system.

**Kinematic parameters**

The ankle and knee joint range motion, angular velocity and angular acceleration in the sagittal and frontal plane were measured using SIMI 3D Motion Analysis software using 9mm retro reflective markers. The motion was captured using high-speed Basler Gig E cameras. The details of the motion capture could be seen in a study published by Hazari et al. 2016 [5]. Spatiotemporal parameters of gait like walking speed, step length, cadence, duration of double support, step time, etc. were measured using the foot scan Wintrack software. The postural evaluation was also performed using the Wintrack equipment.

**Other biomechanical parameters**

Some important clinical examination like postural deviations and deformities like clawing, hallux valgus and foot arch changes were recorded for each participant. Length testing for gastro-soleus, quadriceps, hamstring, Ilio-Tibial band was also performed at baseline and post-intervention exercises.

**Type of exercises prescribed**

Exercises were prescribed in two phases with different aims. At first, exercises were given to control blood sugar based levels as shown in table 1 below.

Following this, a specific set of exercises were designed and given to correct the kinetics and kinematics of lower limb joints and other biomechanical deviations. This exercise protocol was designed based on the findings from the previous literature and our clinical experience. The exercises consisted of major components of lower limb and feet with few components of the trunk and upper extremity. Each exercise was given twice daily ten repetitions with three sets and 10 seconds hold. A complete set of exercises were as follows:

*At ankle:*

**Strength Component**: ankle dorsiflexion and plantarflexion, toes curl, ankle eversion and inversion of intrinsic and extrinsic foot muscle exercises.

**Length Component**: active stretching of gastrocnemius, soleus, (Dorsiflexion with 15 sec hold) and plantar fascia stretch.

*At Knee:*

**Strength Component**: knee extension against manual resistance and weight cuffs (1 kg was kept as the standard for all participants), knee flexion against manual resistance and weight cuffs.

**Length Components**: active stretching of quadriceps (3) and self-patellar mobilization

**Table 1.** Regular Exercise protocol to control the diabetes

| Variable | Type 2 diabetes mellitus |
|---|---|
| Frequency | 3 days / week aerobic exercise<br>2 days / week of resistance exercise |
| Intensity | 40-85% HRR [Aerobic Exercise] , 60-80% 1 RM [Resistance Exercise]<br>RPE = 11-16 |
| Time | Aerobic: 30-60 mins per session<br>Resistance :8-12 reps/ exercise, 1-3 sets/exercise |
| Type of exercise | Aerobic : Brisk Walking , running , cycling<br>Resistance : Muscle groups- Plantar & Dorsiflexor, Quadriceps, Hip abductors , Hamstring, Biceps, Triceps & Deltoid<br>Flexibility: stretching exercises – Intrinsic foot muscle, Tendon Achilles, Hamstrings, Calf , wrist flexors and extensors |

*At Hip:*

**Strength Component**: active straight leg raise for hip flexors in supine lying, leg rise with knee extended in side lying for hip abductors, straight leg raise in the prone position for hip extensors. The hip exercises were gradually progressed. For the first month, all participants were instructed to perform the exercise against gravity itself. For the next two months up to the third month against half kg weight cuff and after three months to 6 months against 1 kg weight cuff was given.

**Length Component**: Piriformis Stretch with side rotation.

*Trunk and Back:*

Back and trunk extensor strengthening exercises were given to all participants. Pelvic bridging was taught to control pelvic tilt and rotation. Transverse abdominus control and strengthening of the diaphragm against self-resistance was taught to strengthen abdominal component. Postural exercises were also taught and demonstrated to participants with any posture deviation at neck, shoulder, and trunk.

**Resistance Training**: As mentioned above resistance training exercises prescribed for upper and lower extremity using Theraband. Biceps curls; shoulder flexion, abduction and external rotation were explicitly focused on avoiding chances of shoulder capsulitis and restricted range of motion.

**Exercise Intervention adherence and monitoring**: The exercises were routinely monitored for adherence. All exercises were demonstrated and supervised by the physical therapist for the initial ten days as an institutional based intervention program. Following this, all participants were instructed to perform the exercises at home based up to 3 months. These were monitoring by log book and progressed over the telephonic conversation.

### Ethical review

The study protocol was approved by Institutional ethics committee, Kasturba Hospital, Manipal (IEC 339/2016)

### Statistical analysis

All the analyses were performed using the Statistical Package for the Social Sciences (SPSS) version 16. The descriptive statistics were performed for mean and standard deviation along with the test of normality. The paired t-test was performed to analyze the statistically significant difference at baseline and three months. A p-value of 0.05 was set for statistical significance.

## RESULTS

The demographic details and Biochemical parameters of all participants have been presented in Table 2 and Table 3. Tables 4 and 5 represent the results of the structured exercises program for kinetic parameters. Tables 6 and 7 represent the results of the structured exercise program for kinematic parameters.

## DISCUSSION

The present study has focused on the benefits of structured exercises on specific kinetic and kinematic parameters in a type 2 diabetes mellitus with and without peripheral neuropathy. The kinetic parameter like plantar pressure distribution has significant clinical relevance in determining the load and prediction of foot ulcers in diabetes population [16]. In the present study, we found that the average and the maximum plantar pressure was higher among participants with type 2 neuropathy compared to non-neuropathy participants (216.41±74.05, 421.31±77.32 kPa & 125.53±20.83, 260.43±54.29 kPa respectively. It is well known that a higher plantar pressure could lead to the breakdown of soft tissue and cause a diabetic foot ulcer within 2.5 years [17]. In the present study, we found from Table 4 and Table 5 that the exercise protocol was very effective and statistically significant in lowering the average and peak plantar pressure at three months both in participants with and without neuropathy (p-value 0.00). Also, the mean changes in participants with neuropathy were higher compared to participants without neuropathy. The reduction in the plantar pressure could be seen due to beneficial effects of exercises on the strength of intrinsic foot muscles and stretching of plantar fascia and calf muscles. Increased strength and length of foot muscles could lead to even plantar pressure distribution while maintaining normal arch [18]. It has been proposed that there are structural changes in the medial longitudinal arch of the foot among diabetes mellitus often leading

**Table 2.** Demographics characteristics and Biochemical parameters of all participants

| Parameters | Type 2 DM without neuropathy (n=15) | Type 2 DM with neuropathy (n=20) |
|---|---|---|
| Age in years | 56±10.5 | 62.3±7.35 |
| Body Mass Index | 26.54±4.83 | 24.39±3.58 |
| Fasting Sugar in mg/dL | 128.6±31.65 | 168.7±63.71 |
| Post-Prandial Sugar in mg/dL | 238.7±85.66 | 246.9±75.05 |
| Vibration Pressure Threshold in Volts | 13.8±3.19 | 39.65±10.57 |
| Ankle Brachial Index | 1±0.15 | 1.16±0.23 |
| Duration of Diabetes Mellitus in years | 8.7±8.95 | 10.97±8 |

Notes: DM - diabetes mellitus

**Table 3.** Pre and Post changes of Biochemical parameters of the participants

| Parameter | PRE | POST | P value ( <0.05) | PRE | POST | P value ( <0.05) |
|---|---|---|---|---|---|---|
| HOMA IR | 3.34±2.24 | 2.95±2.2 | 0.02 | 2.7±2.21 | 2.45±1.38 | 0.04 |
| Fasting Insulin | 18.56±4.59 | 16.23±4.07 | 0.01 | 20.68±5.83 | 19.63±5.26 | 0.05 |
| Hb1Ac | 7.02±1.52 | 6.78 ± 1.11 | 0.001 | 7.38±1.29 | 6.99 ± 1.04 | 0.002 |

Notes: HOMA-IR - Homeostatic Model Assessment for Insulin Resistance

**Table 4.** Pre-Post changes for kinetic parameters test at baseline and three months among type 2 diabetes mellitus with neuropathy

| Kinetic Variables | Baseline (Mean ± s.d) | At three months (Mean ± s.d) | p-value |
|---|---|---|---|
| Average pressure in kilopascal (kPa) | 216.41±74.05 | 188.66 ± 57.32 | 0.00 |
| Maximum pressure in kilopascal (kPa) | 421.31±77.32 | 305.13 ± 70.30 | 0.00 |
| Max. Forefoot Pressure in (kPa) | 1.80±.51 | 1.60 ± .45 | 0.00 |
| Max. Hindfoot Pressure in (kPa) | 1.40±.17 | 1.31± .05 | 0.00 |
| Forefoot/ Hindfoot Pressure ratio | 1.00±.34 | 0.96 ± .41 | 0.08 |
| Max. Pressure surface area in (kPa) | 104.05±18.81 | 107.25 ± 17.82 | 0.02 |
| Max. Pressure at Great Toe in kilopascal (kPa) | 224.32±30.95 | 190.77 ± 31.26 | 0.00 |

**Table 5.** Pre-Post changes for kinetic parameters at baseline and three months among type 2 diabetes mellitus without neuropathy

| Kinetic Variables | Baseline (Mean ± s.d) | At three months (Mean ± s.d) | p-value |
|---|---|---|---|
| Average pressure in kilopascal (kPa) | 125.53±20.83 | 119.75 ± 19.78 | 0.00 |
| Maximum pressure in kilopascal (kPa) | 260.43±54.29 | 243.05 ± 49.83 | 0.00 |
| Max. Forefoot Pressure in (kPa) | .35±.23 | .43 ± .27 | 0.01 |
| Max. Hindfoot Pressure in (kPa) | .48±.28 | .48 ± .26 | 0.82 |
| Forefoot/ Hindfoot Pressure ratio | .73±.12 | .67 ± .13 | 0.00 |
| Max. Pressure surface area in (kPa) | 123.2±22.19 | 118.87 ± 22.33 | 0.00 |
| Max. Pressure at Great Toe in kilopascal (kPa) | 197.74±11.04 | 194.18 ± 9.28 | 0.00 |

to high arch foot [19]. From the given study it can be suggested that the exercise protocol design for intrinsic foot muscles and ankle joint was effective in maintaining and reversing the arch changes thereby producing an even plantar pressure distribution with significantly lower mean values of average and maximum/peak plantar pressure. The result could also be supported by the significant reduction in the great toe pressure as seen in Table 4 and Table 5. The results of the study also suggest that the reduction of great toe pressure and peak pressure could reduce the risk of future foot complication and ulcer development [20]. From the given study and Table 4 it can also be seen that there is a significant reduction the mean forefoot and hindfoot pressure following 3 months exercise regimen. These results further support the effectiveness of exercises on producing even pressure distribution. It should be noted that aerobic and resistance training could have also influenced the results with its beneficial effects on weight reduction. The mean changes the BMI for participants with neuropathy was 0.56 and 0.24 for participants with non-neuropathy. The reduction in average and peak plantar pressure could also be supported by the significant increase in the contact surface area among type 2 diabetes mellitus with neuropathy (Table 4). However, the changes were small among participants without neuropathy.

The effect of exercises on kinematics was similar to kinetics (Table 6 and Table 7). It was found that there were significant changes in the ankle range of dorsiflexion at different phases of the gait cycle (static, heel strike, midstance, and toe-off). There was a significant change in the static range of motion at ankle for neuropathy participant (p-value 0.01), suggesting that calf stretching and strengthening of ankle dorsiflexors was very effective. The significantly increased range of dorsiflexion at the ankle during midstance and toe phase of the gait cycle could be are more clinically important. Reduced dorsiflexion range at toe-off hinders with metatarsal break and could lead to excessive pressure at first toe predisposing to the risk of callus, and ulceration [21] Similar findings were seen at the knee joint with a significant increase in knee extension range of motion at the static phase of the gait cycle. Reduced knee extension at static phase could be suggestive of hamstring tightness and weakness of quadriceps. In the presents study, strengthening of knee extensors and stretching of hamstring was a useful exercise regimen to improve the knee range of motion. It can be seen from Table 6

**Table 6.** Pre-Post changes for kinematic parameters at baseline and 3 months among type 2 diabetes mellitus with neuropathy

| Kinematics | Baseline (Mean ± s.d) | At three months (Mean ± s.d) | p-value |
|---|---|---|---|
| Ankle static joint angle in degrees | 103.46±2.97 | 101.55 ± 1.87 | 0.06 |
| Ankle heelstrike joint angle in degrees | 106.69±3.91 | 104.6 ± 2.31 | 0.08 |
| Ankle midstance joint angle in degrees | 102.88±3.88 | 99.75 ± 3.95 | 0.04 |
| Ankle toe off joint angle in degrees | 98.61±3.89 | 101.48 ± 2.49 | 0.00 |
| Knee joint static angle in degrees | 168.45±4.25 | 171.14 ± 3.45 | 0.09 |
| Knee heelstrike joint angle in degrees | 165.27±2.89 | 167.99 ± 2.59 | 0.59 |
| Knee midstance joint angle in degrees | 156.25±3.17 | 155.14 ± 3.08 | 0.26 |
| Knee joint toe-off angle in degrees | 149.14±5.21 | 147.96 ± 4.87 | 0.42 |
| Peak Ankle joint velocity | 258.77±45.51 | 265.94 ± 34.74 | 0.13 |
| Peak Ankle joint acceleration (°/sec$^2$) | 1689.21±112.47 | 1789.14 ± 124.4 | 0.05 |
| Peak Knee joint angular velocity (°/sec) | 198.33±23.47 | 213.42 ± 22.74 | 0.09 |
| Peak Knee joint angular acceleration(°/sec$^2$) | 1995.41±214.33 | 2014.15 ±241.32 | 0.23 |

**Table 7.** Pre-Post changes for kinematic parameters at baseline and three months among type 2 diabetes mellitus without neuropathy

| Kinematics | Baseline (Mean ± s.d) | At three months (Mean ± s.d) | p-value |
|---|---|---|---|
| Ankle static joint angle in degrees | 103.36±1.24 | 102.21 ± 2.19 | 1.02 |
| Ankle heelstrike joint angle in degrees | 101.27±3.12 | 101.05 ± 1.24 | 0.91 |
| Ankle midstance joint angle in degrees | 98.21±3.14 | 96.18 ± 3.47 | 0.13 |
| Ankle toe off joint angle in degrees | 92.44±1.08 | 90.23 ± 2.04 | 0.08 |
| Knee joint static angle in degrees | 172.14±3.99 | 173.53 ± 2.17 | 0.26 |
| Knee heelstrike joint angle in degrees | 169.42±4.18 | 168.15 ± 3.56 | 0.87 |
| Knee midstance joint angle in degrees | 175.29±2.37 | 175.33 ± 2.04 | 0.25 |
| Knee joint toe-off angle in degrees | 167.41±4.07 | 163.78 ± 3.85 | 0.06 |
| Peak Ankle joint velocity | 168.17±4.78 | 169.44 ± 5.02 | 0.47 |
| Peak Ankle joint acceleration (°/sec$^2$) | 1551.11±124.78 | 1599.47± 134.08 | 0.09 |
| Peak Knee joint angular velocity (°/sec) | 70.03±9.87 | 74.29 ± 8.45 | 0.14 |
| Peak Knee joint angular acceleration(°/sec$^2$) | 1451.24±213.47 | 1524.48 ± 219.44 | 0.05 |

that was a significant difference in knee flexion range at midstance and toe-off phase among participants with diabetes neuropathy (p=0.04 & p=0.01 respectively). These findings are suggestive of better eccentric control of quadriceps due to the strengthening of muscles with exercise training compared to baseline producing a better gait pattern. Strengthening and stretching of hamstring muscle could produce better H/Q ratio and there could be better co-contraction of agonist and antagonist knee muscles producing a stable dynamic joint [22].

The hip joint kinematic was not assessed quantitatively. However, the effect of exercises was clinically evident. Strengthening exercises were prescribed for hip flexors, extensors, and abductors. These exercises could be helpful in maintaining the joint integrity and delaying the adverse effects of diabetes on joint capsules commonly seen as osteoarthritis of hip [23]. The exercises prescribed for back and trunk were effective in controlling the balance and postural deviation among both participants with neuropathy and without neuropathy. This exercise could also be helpful to manage back pain commonly seen among diabetes mellitus individual [24]. The exercise for upper limb and shoulder could also be beneficial to maintain the shoulder range of motion and to avoid periarthritis [25]. Regarding joint velocity and acceleration, the results of the study suggest that there were significant changes in ankle and knee joint. However, there are very few data on the given parameters, and therefore the results of the study should be compared with future findings. The clinical implication of these results suggests that the increased velocity and acceleration could be seen due to stronger joint muscles. Since joint power is a function of muscular force and joint velocity it can be suggested that the power may have increased at ankle and knee joint with strengthening exercises. Studies have shown a reduction in joint power among diabetes with and without peripheral neuropathy [26].

The effect of the given structured exercises was also seen on the blood profile of all participants. The

mean value for $HbA_{1c}$ dropped from 7.02 to 6.78 at three months which was similar to the results from the previous findings. A study showed a significant reduction in $HbA_{1c}$ over a period of 10 weeks of training. The study concluded that resistance training could produce a more significant reduction in $HbA_{1c}$ than aerobic training [27]. There was also a significant clinical reduction in fasting insulin values. A previous study conducted from our center reported that even distribution of plantar pressure and correction of altered foot biomechanics could be an important factor for increasing foot mobility [3]. The present study found that there was a significant clinical mean reduction of 2.33 among participants without neuropathy and 1.05 among participants with neuropathy at three months post exercise intervention. The reduction in the given parameter for neuropathy participants was lesser compared to non-neuropathy suggestive that neuropathy mechanism could lead to higher insulin resistance. These findings were also supported by a significant clinical reduction in HOMA IR blood values (Table 3). The reason for these results could be attributed to increased insulin sensitivity with regular exercise. For instance strengthening of intrinsic foot muscles lead to better load pattern and kinematics like joint angle, velocity and acceleration at ankle joint. It has been also seen that appropriate loading of plantar pressure through strengthening exercises could have an beneficial effect in controlling hyperglycemia by increasing insulin sensitivity .The study conducted by Kimberley et al. 2016 suggested that there was a significant improvement in insulin sensitivity between 48 and 72 hours after exercise (ES,-0.702; CI,-1.392 TO -0.012; p= 0.046). The present study used a combination of aerobic and resistance training along with a targeted group of muscle exercises. Therefore, it would be difficult to suggest the predominance of the type for lower $HbA_{1c}$ value and HOMA IR. However, the given results are suggestive of better utilization of glucose.

## CONCLUSION

The present study showed the structured exercise protocol was very effective in improving the kinetics and kinematics of foot in type 2 diabetes mellitus with and without neuropathy. These exercises could be used in clinical practice. It could help to correct the structure and function of the foot and eventually could reduce the risk of foot complications like diabetic foot ulcers.

## ADDITIONAL INFORMATION

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