Original Article

Journal of Social Welfare and Management
Volume 6 Number 2, April – June 2014

Impact and Influence of Joint Mobility Impairment in Diabetic Peripheral Neuropathy: Implications for Social Welfare and Management

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Abstract

Background: One of the common peripheral nerve disorders affecting joint mobility is diabetic Charcot neuroarthropathy which is caused by the chronic disabling painfulcomplication of diabetes termed as Diabetic peripheral neuropathy (DPN).**Objective:** To evaluate the impact and influence of joint mobility due to DPN and vice versa, by exploring evidence from published studies on joint mobility and its evaluation and management of DPN. **Methods:** A systematic review using search terms of 'joint mobility' and 'diabetic neuropathy' was performed in three databases- PubMed, CINAHL and Google scholar to identify relevant citations which were later scrutinized based upon their title, abstract and full-text to include for data extraction and descriptive synthesis into evaluation and interventions for joint mobility in DPN. **Results:** Of the total list of 26 included studies, there were 21 studies on evaluation of joint mobility in DPN- 10 studies that measured joint mobility (hand function=1, large joints mobility=1, physical examination=1, gait-related joint mobility=7), and 11 studies that related it with other measures/factors (calluses/deformities=1, foot ulceration=7, neurodynamic mechanosensitivity=1, passive ankle stiffness=1, plantar flexor stiffness=1) and there were five studies on interventions and their effects on joint mobility in DPN (Achilles tendon lengthening=1, MABAL shoe=1, total contact cast=1, weight-bearing exercise=1, combined exercises and functional training=1). **Conclusion:** Limited evidence existed for presence of reduced ankle dorsiflexion and great toe extension in people with DPN, which was demonstrated in gait-related functional kinematic alterations and would predispose the diabetic patient to develop foot ulcers due to alterations in plantar pressures during loading phase of gait. Insufficient evidence existed for special shoe/contact casting for neuropathic ulcer healing, tendo-achilles lengthening surgery and supervised weight-bearing exercise training for improving joint mobility and preventing ulceration in people with DPN.

Keywords: Joint mobility; Mobilization; Diabetic neuropathy; Biomechanics; Kinesiology; Arthrology.

Introduction

Epidemiological triad for diseases in the field of public health includes the agent, host and environment that are collectively regarded as the social determinants of health and disease, which play a large part in the stimulus-response inter-relationship manifesting upon a structure-function continuum.[1] One such important social determinant is functional mobility which is dependent upon physiological[2] and pathophysiological[3] adaptations in spinal and peripheral joint mobility.

Joints form the basis for structural and functional mobility by providing link for moving bones, acting as fulcrum for contracting muscles, and acting as shock-absorbers in force distribution between rigid bony segments.[4,5,6] Synovial joints are the commonest type of joints which are recognized

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for their structural features of presence of synovial membrane and fluid, and functional features of stability and mobility.[7]

Upper extremity joints are well adapted to perform fine, precise skilled movements of picking up, carrying and transfering objects whilst lower extremity joints are well adapted to perform powerful quick weight-bearing movements.[8] Upper limb joints enable a person' activities of daily living such as eating, grooming and self-care whereas lower limb joints enable walking, functional ambulation, stair climbing and running.[9]

Joint mobility and its dysfunction is one of the leading impairments in body structure and function as recommended for International classification of functioning, disability and health (ICF) model of the World Health Organization (WHO).[10] Evaluation of joint mobility is an integral part of holistic disability evaluation in all medical conditions for people of all ages.[11]

Physical examination of joint mobility involve static and dynamic measures of alignment, active and passive physiological movement testing, joint play or accesory movement testing, and functional mobility testing during gait and other activities.[12] Disorders and dysfunction of joints lead to mobility impairments which present either as hypomobility (reduced mobility) or hypermobility (excessive mobility) of that particular joint tested on clinical examination.[13]

Inflammatory, dgenerative and auto-immune connective tissue disorders leading symptoms and signs of joint dysfunction such as joint pain, swelling, stiffness and deformity, with or without sounds (clicks, crepitus), locking and loss of function.[14] Although joint dysfunction occurs primarily due to musculoskeletal disorders, other physiological (ageing) and pathological conditions that cause secondary manifestation of joint dysfunction are neurological disorders that affect central and peripheral nervous systems.[15]

The 34 joints of the human foot and ankle form the base for stability and also enhance force distribution between body and the supporting surface, which is essential for almost all activities that involve standing, walking and/or running that are essential in regular social participation and contribution by an individual.[16,17] Joints are innervated by afferent sensory neurons[18] which when affected in peripheral nerve disorders lead to senseless or painless joints termed as neuropathic joints or neuroarthropathy or Charcot joints.[19]

Charcot's joints or Charcot neuroarthropathy is a leading complication of foot neuropathies which affect the individual's participation restriction is influenced by activity limitations and impairments in body structure and function due to its multidimensional manifestations.[20] Diabetic neuroarthropathy is a common form of Charcot arthropathy which is highly prevalent due to higher prevalence of its etiologically primary metabolic disorder globally.[21]

Whilst the cause for neuroarthropathy is neuropathy, diabetic neuroarthropathy is caused due to diabetic neuropathy and is often an overlooked complication in people with diabetes.[22] Early identification is thus essential in order to initiate appropriate management to prevent deformities and reduce the risk for amputations.[23] Management of Charcot neuroarthropathy is either conservative[24] or surgical,[25] depending upon the prognosis of the disorder and its impending biopsychosocial manifestations and their impact upon the person's social welfare and management.[26]

One of the common peripheral nerve disorders is peripheral neuropathy (PN) and a common cause for PN is diabetes mellitus (DM), which leads to chronic disabling neuropathic pain and is termed as Diabetic peripheral neuropathy (DPN). There is a need to know if articular manifestations occur in a non-articular disorder such as DPN so that early and appropriate evaluation and managment could be directed.

Mobility restrictions in diabetes-related foot

disease[27] impair the individual's social life thus affecting his/her social welfare[28] and directly influencing self-management of a person's state of health and disease.[29] The objective of this study was to evaluate the impact and influence of joint mobility due to DPN and vice versa, by exploring evidence from published studies on joint mobility and its evaluation and management of DPN.

Methodology

A systematic review using search terms of "('joint mobility'[Title/Abstract] OR mobilization[Title/Abstract] OR "range of motion"[Title/Abstract])AND (diabetic[Title] OR diabetes[Title]) AND (neuropathy[Title] OR neuropathic[Title]) NOT autonomic[Title]" was performed in three databases- PubMed, CINAHL and Google scholar to identify relevant citations which were later scrutinized based upon their title, abstract and full-text to include for data extraction and descriptive synthesis into evaluation and interventions for joint mobility in DPN. Two testers independently performed blinded search whose disagreements were resolved by consensus in presence of other testers.

Results

Of the total list of 26 included studies, there were 21 studies on evaluation of joint mobility in DPN- 10 studies that measured joint mobility (hand function=1, large joints mobility=1, physical examination=1, gait-related joint mobility=7), and 11 studies that related it with other measures/factors (calluses/deformities=1, foot ulceration=7, neurodynamic mechanosensitivity=1, passive ankle stiffness=1, plantar flexor stiffness=1) and there were five studies on interventions and their effects on joint mobility in DPN (Achilles tendon lengthening=1, MABAL shoe=1, total contact cast=1, weight-bearing exercise=1, combined exercises and functional training=1).

Evaluation of Joint Mobility

Hand Function

Travieso and Lederman[30] assessed subclinical impairments in tactual hand function by comparing 9 diabetic blind with 10 nondiabetic blind and 10 blindfolded sighted controls in terms of their performance on a battery of tests (cutaneous force and spatial resolution thresholds, haptic psychophysical functions for perceived roughness, weight, and size, and both accuracy and response times for haptic classification of 3-dimensional common objects, measures of joint mobility, muscular strength, and motor dexterity). The authors found poor performance of the diabetic blind in all the tests compared to the other two groups.

Large Joints Mobility

Andersen and Mogensen[31] studied movement performance (velocity, range of motion, reaction time, and strength of ankle dorsal and plantar flexion and knee extension) in 29 long-term patients with insulin-dependent diabetes mellitus (IDDM) and 29 matched control subjects. There was a 12% reduction in range of motion for ankle dorsal flexion and there was an inverse relationship between range of motion and neuropathy rank-sum score for ankle dorsal and plantar flexion.

Physical Examination

Sacco Ide *et al*[32] described a clinical assessment protocol which included: (1) preliminary investigation with identification of relevant clinical diabetes and neuropathy characteristics; (2) thermal, tactile and proprioceptive sensitivity tests on the feet; (3) evaluations of muscle function, range of motion, lower limb function, foot anthropometry. The authors found a generalized reduction in range of motion due to DPN.

Gait-related joint mobility:

Mueller *et al*[33] compared (1) the gait characteristics, (2) the plantar-flexor peak torques, and (3) the ankle range of motion of 10 subjects with diabetes mellitus (DM) and peripheral neuropathy with those of 10 age-matched controls and found that the DM group subjects showed less ankle mobility, ankle moment, ankle power, velocity, and stride length during walking than the NODM group subjects. The decrease in ankle strength and mobility appeared to be the primary factor contributing to the altered walking patterns of the DM group.

Yavuzer *et al*[34] defined the gait deviations by comparing 20 patients with diabetic peripheral neuropathy (DPN), 26 patients without diabetic peripheral neuropathy (NDPN) and 20 healthy control subjects (C) and found that NDPN, but not DPN, group revealed slower gait, shorter steps, limited knee and ankle mobility, lower ankle plantar flexor moment and power than C group.

Rao *et al*[35] examined the relationship between ankle dorsiflexion (DF) range of motion (ROM) and stiffness measured at rest (passively) and plantar loading during gait in 10 individuals with and 10 without diabetes mellitus (DM) and sensory neuropathy and found that subjects with DM have reduced passive ankle DF ROM and increased stiffness compared to non-diabetic control subjects.

Rao et al[36] examined segmental foot mobility during gait in 15 subjects with and 15 without diabetes and neuropathy. The study had important findings; "Calcaneal pronation, noted in early stance in both groups, was reduced in subjects with diabetes and may have important consequences on joints proximal as well as distal to it. Subjects with diabetes showed reduced foot 'splay' in early stance, indicated by first metatarsal and forefoot eversion. At terminal stance, decreases in calcaneal plantarflexion, first metatarsal and forefoot supination were noted in subjects with diabetes, suggesting that less supination is required in subjects with diabetes to create a rigid lever."

Sacco *et al*[37] investigated the ankle range of motion during neuropathic gait and its influence on plantar pressure distribution in two phases during stance: at heel-strike and at push-off in 31 adults (control group, n=16; diabetic neuropathic group, n=15). Diabetic neuropathy patients were found to walk with a lesser ankle range of motion in stance phase and smaller ankle flexion at heel-strike.

Rao *et al*[38] examined dynamic foot mobility during gait as it relates to plantar loading in 15 individuals with DM (diabetes mellitus and neuropathy) compared to 15 matched control subjects. The findings of this study indicated that reductions in segmental foot mobility were accompanied by increases in local loading in subjects with DM. Reduction in frontal plane calcaneal mobility during walking also served as an important functional marker of loss of foot flexibility in subjects with DM.

Gomes *et al*[39] studied kinematic and electromyographic data in 46 subjects (healthy and DN) who walked at two cadences (self-selected and 25% higher). However, during the imposed cadence, DN individuals showed reduced ankle range of motion along the entire cycle compared with the self-selected condition and healthy individuals.

Relationship with other measures/Factors:

Calluses and Deformities

Lázaro-Martínez *et al*[40] studied the interrelationship of diabetic neuropathy with the range of joint mobility and the presence and locations of calluses and foot deformities in 281 patients amd found that neuropathy was a risk factor for reduced passive range of motion of the first metatarsophalangeal joint.

Foot Ulceration

Crausaz *et al*[41] studied the physical capacity to perform the preventive footcare measures previously taught, among three groups of diabetic outpatients: (1) 38 patients with neuropathic ulcers; (2) 21 patients with neuropathy, but no ulcers; (3) 30 patients

without neuropathy. Joint mobility measured as heel-buttock distances, did not differ between uncomplicated neuropathic and non-neuropathic patients, and was in the normal range in both groups. However, 71% of complicated neuropathic patients had reduced joint mobility compared with uncomplicated neuropathic and non-neuropathic patients.

Delbridge *et al*[42] studied 62 subjects in three groups (controls, diabetic patients without foot problems, and diabetic patients with neuropathic ulceration) to assess joint-related changes and their relationship to foot ulceration. "there was a significant impairment of mobility in the range of motion of the sub-talar joint in diabetic patients with ulcers when compared with controls or with the other diabetic patients. There was a significant correlation between sub-talar range of motion and mobility in other joints of the foot such as at the hallux, or with mobility of the 5th finger. There was also a significant association between the clinical presence of limited joint mobility in the hand, Dupuytren's contracture, and mobility of the sub-talar joint. Furthermore, impairment of mobility of the sub-talar joint was greatest on the affected side in those diabetic patients with neuropathic ulceration. We conclude that the syndrome of limited joint mobility also affects the joints of the feet of diabetic patients and may predispose to ulceration in susceptible neuropathic feet."

Katoulis *et al*[43] listed factors such as previous foot ulceration, diabetic neuropathy, limited joint mobility, high plantar pressures, microangiopathy, macroangiopathy and diabetic nephropathy as risk factors for diabetic foot ulceration.

Bennett *et al*[44] investigated the role of nonenzymatic glycosylation and pressure beneath the sole of the foot in the pathogenesis of neuropathic foot ulcers in twenty-seven subjects with diabetes with a recent history of neuropathic foot ulceration who were matched by age and sex with a group of 50 control subjects without neuropathy or history of foot ulceration. Ankle joint flexibility was found to be reduced in cases with neuropathic foot ulceration compared with the control group.

Brink[45] assessed joint mobility (passive dorsal flexion of the metatarsophalangeal joints) in ten diabetic feet with polyneuropathy and a history of recurrent plantar ulcers in comparison with non-diabetic control feet and found slightly lesser joint mobility and thinner plantar pads in the diabetics as compared with control subjects. The author thus proposed that induration of the periarticular soft tissue may predispose to recurrent foot ulcers in diabetics by decreasing the shock-absorption capacity of the plantar pad.

Nubé et al[46] reported presence of pronated foot type among patients with history of foot ulceration, which also had reduced range of active first metatarsophalangeal joint dorsiflexion, ankle dorsiflexion, subtalar joint range of motion during gait.

Frykberg *et al*[47] ascertained the risk of ulceration associated with high foot pressures and peripheral neuropathy in a large and diverse diabetic population of 251 diabetic patients of Caucasian (group C) (n=121), black (group B) (n=36), and Hispanic (group H) (n=94) racial origins and found that joint mobility was significantly greater in the Hispanic cohort compared with the other groups at the first metatarsal-phalangeal joint, while the subtalar joint mobility was reduced in the Caucasian group. The study concluded that both high foot pressures (> or =6 kg/cm2) and neuropathy are independently associated with ulceration in a diverse diabetic population, and in black and Hispanic diabetic patients, joint mobility and plantar pressures are less predictive of ulceration than in Caucasians.

Mechanosensitivity during Lower Extremity Neurodynamic Testing

Boyd *et al*[48] determined the unique effects T2DM and DSP on nerve mechanosensitivity in the lower extremity on 43 people with T2DM. Straight leg raise neurodynamic tests were performed with ankle plantar flexion (PF/SLR) and dorsiflexion (DF/SLR). The addition of ankle dorsiflexion during SLR

testing was found to reduce the hip flexion ROM by 4.3° at P1 and by 5.4° at P2.

Passive Aankle Stiffness

Salsich *et al*[49] quantified and compared passive ankle stiffness and dorsiflexion (DF) range of motion in 17 subjects with DM and PN versus 17 age-matched healthy subjects. The subjects with DM and PN had smaller maximal dorsiflexion angles and less plantar flexor muscle excursion than the healthy group, without differences in stiffness.

Plantar Flexor Stiffness

Salsich et al[50]determined the relationships between plantar flexor (PF) muscle stiffness, strength (concentric peak torque), and dorsiflexion (DF) range of motion (ROM) in 17 subjects with diabetes who have peripheral neuropathy and 17 age-matched controls. The subjects with diabetes and peripheral neuropathy (DM-PN) had peak concentric PF torque which was positively correlated with passive torque at 5 degrees DF, and stiffness. The percentage of passive PF torque at 5 degrees DF was greater in subjects with DM-PN, compared to control subjects.

Interventions for Joint Mobility

Achilles Tendon Lengthening

Salsich *et al*[51] studied the effects of a tendo-Achilles lengthening (TAL) procedure on ankle muscle performance by comparing the effects of TAL and total-contact casting (TCC) with TCC alone in 29 subjects with diabetes mellitus (DM) and a neuropathic plantar ulcer. Maximal dorsiflexion angle was found to increase 11 degrees after TAL and remained increased at 8 months follow-up.

MABAL shoe

Hissink *et al*[52] used the MABAL shoe (removable fiberglass combicast shoe) to treat 23 plantar ulcers and found 21 ulcers had healed, with a mean healing time of 34 days.

Total Contact Cast

Caputo *et al*[53] explained the use of total contact casts as a treatment option for recalcitrant foot ulcers as it allowed mobilization and resulted in diminished edema and decreased pressure over the ulcerated area, complete healing occurring within 8 weeks.

Weight-Bearing Versus Nonweight-Bearing Exercise

Mueller *et al*[54] determined the effects of weight-bearing (WB) (exercise performed in standing and walking) versus nonweight-bearing (NWB) (Group-specific progressive balance, flexibility, strengthening, and aerobic exercise conducted sitting or lying) exercise for 29 persons with diabetes mellitus (DM) and peripheral neuropathy (PN) who were randomly assigned to WB (n=15) and NWB (n=14) exercise groups. WB group fared better than the NWB group.

Combined Strengthening, Stretching and Functional Training Program Versus Usual-Care

Sartor *et al*[55] reported an ongoing randomised controlled trial, to study the effect of a physiotherapy intervention on foot rollover during gait, range of motion, muscle strength and function of the foot and ankle, and balance confidence.

Discussion

The present study aimed to explore the impact and influence of joint mobility in DPN from a social welfare perspective and found that limited evidence existed for presence of reduced ankle dorsiflexion and first MTP joint extension in people with DPN, which was demonstrated in gait-related functional kinematic alterations and would predispose the diabetic patient to develop foot ulcers due to alterations in plantar pressures during loading phase of gait. Insufficient evidence existed for special shoe/contact casting for

neuropathic ulcer healing, tendoachiles lengthening surgery and supervised weight-bearing exercise training for improving joint mobility and preventing ulceration in people with DPN.

There were not many studies found on evaluation of hip and knee in people with DPN and this had thrown light on scope for future studies in this area. Intervention studies on medical,[56] surgical,[57] physiotherapeutic, [58] neurodynamic[59] and acupuncture[60] treatments were available but many did not evaluate joint mobility as as outcome measure per se.

Diabetes modulates collagen remodeling thereby affecting ligaments, cartilage and synovia which promote degradation of the extracellular matrix thus compromising joint structure and function.[61] Whilst previous reports had found reduced joint mobility in wrist and hands of diabetics compared to non-diabetics,[62,63] and also the presence of diabetic neuroarthropathy in the shoulder joint.[64]

Joint dysfunction leads to neuromuscular re-organization initiating muscle imbalances of strength and length impairments both in people with and without pain.[65] The resulting proprioceptive impairment secondary to joint dysfunction[66] is worthwhile considering the prevalence of balance issues in people with DPN.

Clinicians need to emphasize physical examination of joint mobility in their patient assessments so as to identify joint dysfunctions in upper and lower extremities.[67] It is also essential to follow an impairment-based approach as described by Murphy[68] in his case report where he described the use of intertarsal and ankle joint manipulative treatment with calf muscle myofascial therapy in a male patient with diabetic polyneuropathy.

This study had few limitations: lack of meta-analysis and use of non-validated search strategy. However, these were presumably shadowed by independent blinded search method which enhanced internal validity of our findings.

Future assessment studies should identify intra-articular, peri-articular and extra-articular causes of joint mobility impairments in DPN which were operated through central and peripheral mechanisms. Intervention studies should focus on application of orthopedic manual physical therapy examination[69] and interventions in people with DPN to address the articular, myofascial and neural components of joint mobility dysfunctions.

Conclusion

Limited evidence existed for presence of reduced ankle dorsiflexion and first MTP joint extension in people with DPN, which was demonstrated in gait-related functional kinematic alterations and would predispose the diabetic patient to develop foot ulcers due to alterations in plantar pressures during loading phase of gait. Insufficient evidence existed for special shoe/contact casting for neuropathic ulcer healing, tendoachiles lengthening surgery and supervised weight-bearing exercise training for improving joint mobility and preventing ulceration in people with DPN.

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