Key considerations for assessment and management of limited joint mobility in the diabetic foot

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Article points

1. Clinicians should be proactive in providing appropriate offloading devices and footwear modifications for people with diabetes who exhibit limited joint mobility in the foot.
2. It is recommend that people with diabetes receive an early assessment for limited joint mobility in the foot.
3. Assessing and treating the diabetic lower limb is an opportunity for clinicians to reinforce messages around optimising and maintaining good glycaemic control.

Key words

- Diabetic foot ulcer
- Mobility
- Offloading
- Pressure
- Risk

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Delbridge et al (1988) and Fernando et al (1991) recognised the potential risk of limited joint mobility (LJM) in the diabetic foot. These studies highlighted the influence of LJM in the diabetic foot during elevating plantar pressures, thus contributing to diabetic foot ulcers (DFUs). While there are no firm paradigms for the assessment and management of LJM in the diabetic foot, the concept of LJM as a risk factor in DFUs is a feature of the literature (Boyko et al 1999; Viswanathan et al, 2002; Zimny et al, 2004). Here, the author examines the impact of LJM and why it should feature in the assessment and management of the diabetic foot as recommended by Formosa et al (2013).

Limited joint mobility (LJM) occurs in people with diabetes and was first identified by Lundbaek in 1957 (Papanas and Maltezos, 2010), to describe a stiffening effect in the hand joints. At this time, LJM was also known as cheiroarthropathy ("cheiros" meaning "of the hand"). LJM was popularised in the literature by Rosenbloom from 1974 onwards (Papanas and Maltezos, 2010). LJM begins with a painless range of motion deficit at the fifth finger on each hand and spreads, affecting interphalangeal and metacarpophalangeal joints (Frost and Beischer, 2001).

The literature predominantly defines LJM in adolescents with type 1 diabetes as a hand-based phenomena. However, when Duffin et al (1999) measured the range of motion in the hands and feet of adolescents with type 1 diabetes compared to a control group, they determined the presence of LJM in both the hands and the feet of the diabetic group. While LJM in adolescents shows no significant reports of reduced mobility, over time, LJM may progress into the joints of the foot, having a greater impact on mobility.

The prevalence of LJM in the adult population with type 2 diabetes is variable. Lindsay et al (2005) reported a 23% prevalence of LJM in adults. Lazaro-Martinez et al (2011) reported in their population that 30–40% of patients with diabetes experienced LJM in most of the foot joints.

Aetiology of LJM

Amin et al (2005) and Umay et al (2011) reported a correlation between elevated HbA_{1c} levels and LJM. Craig et al (2008) describes the reaction between glucose and collagen leads to the formation of advanced glycated end products (AGEs). These AGEs, which are more pronounced with hyperglcaemia, increase cross-linking of collagen in connective tissue, causing collagen toughness and changes to the elastic modulus. The result is decreased muscle strength, poor joint function, and limitations in joint range of motion.

A high BMI may be a contributing factor to the aetiology of LJM. With increasing adiposity, tendons and joints are exposed to higher loads, which can lead to overuse and damage. Advancing age may contribute to LJM, however, Abate et al (2011) identified that people with diabetes compared to non-diabetic controls are at greater risk of experiencing changes to the foot joints, producing limitations of movement.

While the exact aetiology of LJM remains debatable, Umay et al (2011) maintain there are strong associations strong associations with hyperglycaemia.

What is the evidence for LJM in the diabetic foot?

Research suggests LJM in the diabetic foot can be assessed by undertaking the following:

- Measure range of motion in the joint.
- Measure association with high plantar pressure.
- Measure gait changes.
- Examine associated skin changes.
- Look for associated rheumatological or musculoskeletal manifestations.

Meanwhile, treatment for LJM in the diabetic foot should consider the following areas:

- Optimise glycaemic control.
- Reduce of high plantar pressure (orthotics).
- Improve gait and mobility (footwear provision).
- Improve movement in the joints.
- Prescribe anti-inflammatories for associated Achilles tendinopathy and plantar fasciitis.
- Consider surgery for intractable, painful complications.

Recommended assessment techniques for LJM in the foot

Lindsay et al (2005) explained that LJM in the hands is a marker for joint tissue changes. Assessment and diagnosis of LJM in the hands can be confirmed by two clinical tests. Firstly, the "prayer sign" is the inability to fully flatten the two palms when opposed and clasped together (Rosenbloom and Frias, 1974). Second, the "flattening sign" or "tabletop sign" is described as the inability to fully flatten the palm on a flat surface (Ikem et al, 2009).

Measure range of motion in the diabetic foot

Assessment of LJM in the diabetic foot can be undertaken by examining passive range of motion. This involves a manual examination of the available movement in the foot joints and appears to be the most frequent approach within the literature. Studies examining LJM in the foot, demonstrate that the ankle joint, subtalar joint, and first metatarsophalangeal joints are reliable locations for assessing LJM in the foot (Duffin et al, 1999; Chuter and Payne, 2001; Viswanathan et al, 2003; Zimny et al, 2004; Lazaro-Martinez et al, 2011). Instrumentation for measuring passive range of motion in the feet can be quantified using hand held or electronic goniometer techniques.

Measure plantar pressure in the diabetic foot

Delbridge et al (1988), Fernando et al (1991), and Viswanathan et al (2003) all identified the relationship between LJM, neuropathy, and elevated plantar pressures and an increased risk of developing diabetic foot ulcers (DFUs).

The evidence for LJM as a risk factor for development of DFUs is not conclusive. Armstrong et al (2001) indicated DFUs are the result of an interplay between vascular, neurological, and musculoskeletal alterations causing high pressures on the foot. Despite this, there is research to support the hypothesis that high pressures and limited range of motion, can increase the risk of ulceration in the diabetic foot (Viswanathan et al, 2003; Turner et al, 2007).

Gait assessment

Turner et al (2007) highlighted that LJM in the first metatarsophalangeal joint (MPJ), coupled with peripheral neuropathy, generated increased plantar pressure associated with diabetic foot ulceration. However, Turner et al (2007) recommended assessing both the passive and gait range of motion at the ankle subtalar joint and first metatarsophalangeal joint. The study concluded that passive range of motion at the first MPJ provides a gauge for identifying patients with higher foot pressure and ulceration. Such studies reinforce the recommendation of an early assessment of LJM in the diabetic foot. Turner (2007) et al's research indicated that LJM in the diabetic foot may amplify the damage caused by underlying biomechanical dysfunction.

Assess skin changes

The tissue properties of LJM in the foot were investigated by Abouaesha et al (2001) and Craig et al (2008). These studies hypothesised a relationship between LJM with hardening and thickening of the plantar skin on the foot, as a predictor of high plantar pressure. These high pressures are, in turn, highly predictive of DFU. Hardening and thickening of the tissues associated with LJM is also strongly associated with persistent hyperglycaemia.

History of musculoskeletal manifestations

Arkilla (2003) and Cagliero (2003) associated LJM with a broad group of manifestations unique to people with diabetes. These include; capsulitis of the shoulder, Dupuytren's contraction, flexor tenosynovitis, carpal tunnel syndrome, and neuroarthropathy.

Page points

1. Assessment of limited joint mobility (LJM) in the diabetic foot can be undertaken by examining passive range of motion.
2. Relationships between LJM, neuropathy, and elevated plantar pressures and the increased risk for developing diabetic foot ulcers have been shown.
3. LJM has been associated with a range of manifestations unique to people with diabetes.

Page points

1. There are no firm paradigms for the treatment of limited joint mobility (LJM) in the diabetic foot.
2. Optimisation of glycaemic control should be reinforced as the first stage of LJM management.
3. Reduction of high plantar pressures caused by LJM is a key management strategy for clinicians responsible for preventing and treating diabetic foot ulcers.

Abate et al (2013) considered LJM as a rheumatological manifestation, which is more pronounced in people with diabetes. Abate et al (2013) suggested that frozen shoulder, rotator cuff tears, Dupuytrens contracture, trigger finger, Achilles tendinopathy, and plantar fasciitis are all associated with LJM.

Management of LJM

While there are no firm paradigms for the treatment of LJM in the diabetic foot, the concept of managing it has remained a significant feature within the literature. It is from this evidence base the following suggestions are derived.

Optimising glycaemic control

Several authors (Amin et al, 2005; Lindsay, 2005; Umay et al, 2011) have identified a relationship between LJM and hyperglycaemia in people with diabetes. For people with diabetes, this means LJM can act as an important marker of their glycaemic control. Lindsay's (2005) longitudinal study suggested a decreasing incidence of LJM due to improved metabolic control of diabetes. Therefore, optimisation of glycaemic control by careful monitoring of HbA_{1c} levels should be reinforced as the first stage of LJM management (Papanas, 2010; Somai and Vogelgesang, 2011; Umay et al, 2011; Abate et al, 2013).

Mechanical reduction of high plantar pressures

Delbridge et al (1988), Fernando et al (1991), and Viswanathan et al (2003) identified a relationship between the presence of LJM, neuropathy, and high plantar pressure, which increased the risk of developing DFUs. NICE (2004; 2011) recommended that the intensity of diabetic foot care provision should reflect the risk status of the individual. Therefore, any risk associated with the formation of DFUs, such as high plantar pressures associated with LJM, should result in a prophylactic treatment plan being put in place for that individual.

Reduction of high foot pressures is a key management strategy for clinicians responsible for preventing and treating DFUs. Prophylactic pressure reduction in the foot can be achieved by a variety of offloading techniques via the provision of foot orthotics/insoles.

For patients with DFUs, foot immobilisation may be desirable to achieve ulcer healing. This may include the provision of slipper casting and total contact casting to offload the ulcerated foot.

However, Lazaro-Martinez et al (2011) explained joint mobility can be reduced as a consequence of DFU treatment, particularly if it involves foot immobilisation. Therefore, DFU treatment involving immobilisation should include consideration of restoring joint mobility after the immobilisation stage.

Footwear and gait rehabilitation

Studies have identified there are noticeable gait range of motion changes associated with LJM in the foot (Turner et al, 2007). Due to the generalised stiffness accompanying LJM there is reduced shock absorption at initial contact of the gait cycle. In addition, there is reduced dorsiflexion in the forefoot at push off phase of the gait cycle. For these reasons, when LJM advances in the diabetic foot, Dahmen et al (2001) recommend a shoe with shock absorption through the heel, a toughened outsole, a rocker bottom sole, and an insole that distributes pressure evenly (*Figure 1*).

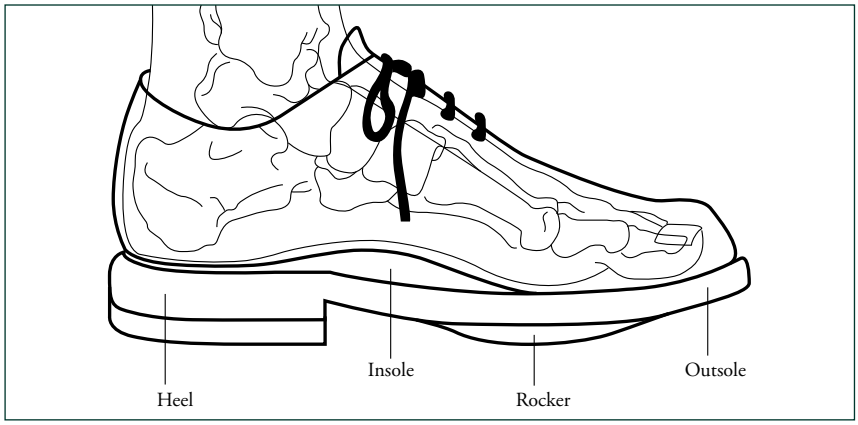

Figure 1. A recommended shoe with shock absorption through the heel, a toughened outsole, a rocker bottom sole, and an insole that distributes pressure evenly.

Improving movement

Restoring joint mobilisation for generalised LJM in the foot may be achieved by physical therapy.

Dijs et al (2000) suggested preventative measures focusing on a reduction of plantar foot pressures by insoles, hosiery and footwear alone is insufficient treatment for LJM in the diabetic foot. They also proposed supplementary intervention should be explored and undertook a pilot study involving 11 patients providing physical therapy by passive

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joint mobilisation. The joint mobilisation resulted in an improvement in mobility after 10 sessions. Unfortunately, this improvement was temporary, and the author recommended further research to maintain the joint mobility effect over the long term.

Documenting skin changes

LJM in the diabetic foot may be detected by assessment of skin changes. It is important that clinicians assess and recognise any early clinical changes that might indicate LJM in the foot.

Anti-inflammatory agents

Low-grade persistent inflammation is often a factor in joint and musculoskeletal (MSK) conditions, such as Achilles tendinopathy and plantar fasciitis.

Anti-inflammatory drugs and corticosteroids (including local injections) have been used to achieve good short-term outcomes for patients with painful hand symptoms (Somai and Vogelgesang, 2011). There are however, some controversies, with some clinicians preferring to explore physical therapies such as stretching and strengthening exercises.

Surgery

Minor foot surgery may be a consideration to restore function at the first metatarsophalangeal joint affected by LJM, as Turner et al (2007) suggested reduced gait range of motion at this location is strongly associated with high plantar pressures leading to DFUs

The future: therapy to counter the effect of AGEs

Pharmacological compounds to counter the negative effects of AGEs may be a part of future clinical trials. However, this research is in its relative infancy and research to date has only been tested in experimental models.

Conclusion

Although there is some evidence of the clinical link between LJM and diabetic foot ulceration, the scientific support for this relationship is still relatively weak. LJM is not apparent in some ethnic groups with no apparent reason. Hence, it is recommended that clinicians and researchers to continue their work into the complexities of LJM and diabetic foot ulceration, as it remains an important area of investigation.

Treatment of LJM in the foot warrants further evaluation, as Ardic et al (2003) suggested the musculoskeletal manifestations of diabetes are poorly treated compared to other pathologies, such as nephropathy, neuropathy, and retinopathy. Somai and Vogelgesang (2011) concurred with this and said, in general, that the treatment of LJM remains largely unsatisfactory and controversial.

While several treatment pathways have been explored for the wider manifestations of LJM in the body, it is not fully understood how the mechanisms operate for LJM in the diabetic foot.

At present complete functional recovery has not been reported in the diabetic foot as a consequence of treatments for LJM. Therefore, there is a requirement to explore the potential benefits of physical therapy for LJM in future clinical trials.

The current recommendation to clinicians is to assess and recognise any early clinical changes that might indicate LJM in the foot. It is crucial to continue researching the management of LJM in the diabetic foot if we are to appraise the efficacy of our care. Management of LJM in the diabetic foot requires wider appraisal by the clinical and research communities to generate meaningful treatment models. ■

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