

Limited joint mobility and other musculoskeletal problems in diabetes

Lesley Hordon

While musculoskeletal conditions are common in the general population, people with diabetes are at greater risk than would be expected for their age. Identification and treatment of these problems are important to maintain quality of life. One specific problem of the diabetic hand, limited joint mobility, is difficult to treat, although better control of diabetes may reduce its prevalence in the future. Flexor tendon nodules, carpal tunnel syndrome and Dupuytren's contractures can also affect hand function and respond more readily to therapy. Painful shoulder problems are frequent in people with diabetes and require treatment. Obesity is a risk factor for type 2 diabetes, as well as for osteoarthritis and gout, but there is increasing interest in the effects of metabolic syndrome on the prevalence of gout and osteoarthritis.

The care of people with diabetes involves far more than the control of blood glucose levels. It is accepted that screening and management of retinopathy, microalbuminuria, neuropathy and cardiovascular risk factors, along with excellent foot care, are all necessary. However, joint problems, although common and often painful, are relatively neglected. This article covers some of the joint and soft tissue conditions commonly linked to diabetes, aiming to raise awareness of these in primary care and the potential for treatment that can improve the individual's quality of life.

Limited joint mobility

Limited joint mobility was first described in children with type 1 diabetes in 1974 by Rosenbloom and colleagues (Rosenbloom et al, 1974). It is characterised by a limitation of joint movement, most marked in the small joints of the hand, and is often associated with thickening and waxiness of the skin. Unlike arthritis, the condition is painless, although flexion contractures of the proximal interphalangeal (PIP) and metocarpophalangeal (MCP) joints can occur. Symptoms include stiffness, decreased

grip strength and difficulty with fine movements of the hands. The distal interphalangeal (DIP) joints and, less commonly, the wrists, elbows, shoulders, knees, neck and spine, may also be involved. Limited joint mobility in the feet may contribute to high foot pressures, which, along with other factors including neuropathy, peripheral vascular disease and trauma, can lead to foot ulceration (Turner et al, 2007).

Diagnosis is made by clinical examination. Two simple tests are the "prayer sign", where the hands are flattened together as in prayer, and the "table top sign", where the hand is pressed flat, palm downwards, on the table top (Figure 1). Contractures of the PIP, DIP and MCP joints can be readily detected by these quick and easy manoeuvres. Skin texture is also examined to detect changes of thickening and waxiness. It is important to exclude other diabetic hand conditions, which are described later in the article. These conditions can co-exist with limited joint mobility and respond more readily to treatment.

Limited joint mobility is caused by the deposition of abnormal collagen in the connective tissues around the joints. Enzymatic

Citation: Hordon L (2017) Limited joint mobility and other musculoskeletal problems in diabetes. *Diabetes & Primary Care* 19: 29–34

Article points

1. Limited joint mobility, particularly in the hand, can occur in people with diabetes. It is difficult to treat, but good glycaemic control may alleviate symptoms.
2. Other hand abnormalities that can occur in people with diabetes include Dupuytren's contracture, flexor tenosynovitis and diabetic sclerodactyly. Common shoulder problems include adhesive capsulitis and rotator cuff tendonitis.
3. There is increasing interest in the effects of metabolic syndrome on the prevalence of gout and osteoarthritis in type 2 diabetes.

Key words

- Gout
- Limited joint mobility
- Musculoskeletal problems
- Osteoarthritis

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1. The cause of limited joint mobility is the deposition of abnormal collagen in the connective tissues around the joints.
2. Estimates of the prevalence of limited joint mobility are difficult, as these depend on the populations of people with diabetes studied, ranging from those with mild, diet-controlled type 2 diabetes managed in primary care, to those with type 1 diabetes attending a specialist clinic. Furthermore, estimating prevalence is further complicated by the definition of limited joint mobility used.
3. The association between limited joint mobility and diabetic control has been equally difficult to determine due to differences in study populations.



Figure 1. Table top sign.

and non-enzymatic glycosylation of skin collagen and the production of advanced glycation end products, which lead to abnormal crosslinking and decreased turnover of collagen, are thought to be responsible (Brownlee et al, 1988; Kapoor et al, 1989; Rosenbloom, 2013).

Estimates of the prevalence of limited joint mobility are difficult, as these depend on the populations of people with diabetes studied, ranging from those with mild, diet-controlled type 2 diabetes, to those with type 1 diabetes attending a specialist clinic. Furthermore, estimating prevalence is further complicated by the definition of limited joint mobility used. The condition can occur in both type 1 and type 2 diabetes and is linked to both duration of diabetes and diabetic control as measured by HbA_{1c}. The prevalence also increases with age and smoking (Eadington et al, 1991; Arkkila et al, 1994; Silverstein et al, 1998).

The correlation of limited joint mobility with the more serious microvascular complications of diabetes, such as retinopathy, nephropathy and neuropathy, has been controversial, with studies giving conflicting results. Again, differences in populations studied contribute to these conflicting results, as well as whether the study is a cross-sectional or a long-term prospective study. There are too many studies to list here, but two examples are given. In one prospective study, 44 individuals with and without limited joint mobility were followed for 10 years. These study participants did not have nephropathy and retinopathy at baseline. At 10 years, similar numbers in both groups developed retinopathy and microalbuminuria (McCance et al, 1993). A

larger prospective study examined 479 children for a median of 10.9 years following diagnosis of type 1 diabetes and found that 35% developed limited joint mobility at a median age of 13 years and a duration of diabetes of 5.2 years. The risk of developing limited joint mobility was related to higher HbA_{1c} levels, as well as puberty. Children with limited joint mobility had an increased risk of microalbuminuria (Amin et al, 2005).

The association between limited joint mobility and diabetic control has been equally controversial in the past, due to differences in study populations. Perhaps more importantly, the assessment of diabetic control has been very variable in different studies. Comparing people with diabetes with and without limited joint mobility using longitudinal measurements of HbA_{1c} gives a better idea of long-term control in these groups, rather than a cross-sectional study of the same groups comparing HbA_{1c} measured at a single time point. Supportive evidence that better long-term glycaemic control reduces the frequency and severity of limited joint mobility comes from two cross-sectional studies of children at diabetes camps in the US some 20 years apart. The 1976–1978 cohort had a prevalence of limited joint mobility of 30%, while the 1998 cohort had a prevalence of limited joint mobility of 7% (Infante et al, 2001). A similar cross-sectional British study looking at adults with type 1 diabetes (mean age 27 years) found the prevalence of limited joint mobility fell from 43% in 1981–1982 to 23% in 2002 (Lindsay et al, 2005).

Limited joint mobility does not respond well to treatment. Ensuring good glycaemic control and stopping smoking is sensible, and may reduce progression, although this is uncertain. Both actions, however, have other health benefits to the person with diabetes and should be encouraged. Function can be improved by physiotherapy with palmar stretching and by occupational therapy.

Other hand abnormalities in diabetes Carpal tunnel syndrome

Carpal tunnel syndrome is caused by the compression of the median nerve in the carpal tunnel at the wrist, and gives numbness and tingling, most commonly in the thumb, index,

middle and ring fingers, which often disturbs sleep. If severe, wasting of the thenar eminence (palm of the hand) can occur. Most studies cite a figure of around 5% for the prevalence of carpal tunnel syndrome in the general population (Bongers et al, 2007), while it has been reported to affect up to 20% of people with diabetes and up to 75% of those with limited joint mobility. Fortunately, the results of carpal tunnel release are similar in people with and without diabetes (Thomsen et al, 2009). Previous work has shown that carpal tunnel syndrome is associated with the duration of diabetes, but not metabolic control, nephropathy or retinopathy (Chaudhuri et al, 1989; Gamstedt et al, 1993). A recent secondary care study on people operated on for carpal tunnel syndrome has, however, suggested that type 2 diabetes may not be an independent risk factor for this condition but may reflect risk factors of age and BMI (Hendriks et al 2014). One electrophysiological study showed that carpal tunnel syndrome was more severe in people with metabolic syndrome than in people with diabetes alone (Yurdakul et al, 2015).

Dupuytren's contracture

Dupuytren's contracture (shown in *Figure 2*) is the result of fibrosis in and around the palmar fascia, with nodule formation and contracture of the palmar fascia, leading to flexion contractures of the fingers, commonly the ring and little fingers. It is often painless. Dupuytren's contractures have been reported in 16–42% of outpatients with diabetes, the prevalence increasing with age and duration of diabetes (Gamstedt et al, 1984). Other associations of Dupuytren's contracture include racial and genetic factors, and chronic liver disease. Dupuytren's contracture is often asymptomatic, but severe cases may require surgery.

Flexor tenosynovitis

Flexor tenosynovitis, sometimes known as trigger finger, is characterised by a palpable nodule formation and thickening localised to the flexor tendon sheath. When the patient flexes the finger, it may lock in a fixed position and straightening the finger can be painful. The ring and middle fingers and thumb are most often affected, and the condition may be bilateral (Yosipovitch et al,



Figure 2. Dupuytren's contracture.

1990). The prevalence ranges from 1.5–20% in people with diabetes, depending on the population studied and can occur in 0.7% of people without diabetes, although those with diabetes are more likely to have multiple digit involvement (Vance et al, 2012). Collagen abnormalities induced by diabetes are thought to be responsible.

Local corticosteroid injection can be successful (Kapoor et al, 1989) but some people require repeated surgery (Yosipovitch et al, 1990). Flexor tenosynovitis is more common in those with limited joint mobility (Kameyama et al, 2009), and it is important to distinguish between the two conditions as flexor tenosynovitis responds to treatment.

Diabetic sclerodactyly

Diabetic sclerodactyly is characterised by thickening and waxiness of the skin, most marked on the dorsum of the fingers. It is associated with limited joint mobility, but can occur separately from this condition. In most cases, the frequency of diabetic sclerodactyly increases with the duration of diabetes. It can be distinguished from scleroderma, which it resembles, by the absence of Raynaud's phenomenon, digital ulceration and calcinosis, and autoantibodies are negative. Improving diabetic control is the only option for treatment and has limited benefit.

Shoulder problems

Adhesive capsulitis

Adhesive capsulitis, commonly known as frozen shoulder, is characterised by painful restriction of glenohumeral movement in all planes of motion,

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Figure 3. Both women and men with gout have a high risk of diabetes.

in the absence of joint degeneration sufficient to explain the condition. Both active and passive movements are restricted. The aetiology is unknown. While the prevalence is approximately 5% in those without diabetes, it increases to 19–29% in people with diabetes, depending on the diagnostic criteria used and the population studied. In people with diabetes, the condition is more likely to be bilateral (Pal et al, 1986; Balci et al, 1999).

Adhesive capsulitis in those with diabetes is associated with increased age, longer duration of diabetes, the presence of limited joint mobility and Dupuytren's contractures. Some studies have shown associations with retinopathy and neuropathy, as well as poorer glycaemic control. For both those with and without diabetes, treatment is with analgesia, intra-articular steroids and physiotherapy. Arthroscopic release may help in resistant cases, but people with diabetes are slightly less likely to respond than those without diabetes (71% versus 90%; Mehta et al, 2014). Adhesive capsulitis commonly resolves slowly over 1–3 years, but a significant

minority of patients in both groups (7–15%) are left with residual functional disability.

Rotator cuff tendonitis

Rotator cuff tendonitis gives an aching discomfort in the shoulder, with pain both at night and on movement. Pain is felt in the deltoid area of the upper arm and is aggravated by abduction, with a painful arc of movement between 70–120 degrees. Calcific tendonitis is three times more common in those with diabetes compared to those without, although only one-third have symptoms (Mavrikakis et al, 1991). Of 60 people with diabetes with shoulder pain, 58% had adhesive capsulitis and 28% tendonitis (Moren-Hybbinette et al, 1987). Treatment is by steroid injection and physiotherapy, although surgery may be required in persistent cases.

Osteoarthritis

Obesity is a risk factor for gout and also for osteoarthritis, particularly of the knee, as well as for type 2 diabetes. There is increasing interest in whether diabetes itself is a risk factor for these two conditions, independent of the mechanical and metabolic effects of obesity. Two studies, both of which adjusted for age and BMI, have linked osteoarthritis to diabetes. A cross-sectional study of people with diabetes in Puerto Rico showed a two-fold increased risk of hand and knee osteoarthritis, with the risk lower in those who only used insulin for their diabetes, compared to people without diabetes (Nieves-Plaza et al 2013). A longitudinal cohort study showed that type 2 diabetes was an independent risk factor for severe osteoarthritis leading to hip and knee replacement (Schett et al, 2013).

Gout, metabolic syndrome and type 2 diabetes

Gout is a painful, episodic and inflammatory arthritis caused by deposition of uric acid crystals in tissues and joints. Tophi, uric acid crystal deposits surrounded by granulomatous inflammation, may form in and around joints and under the skin, in some cases leading to bony erosions. Metabolic syndrome is described as a combination of clinical risk factors for cardiovascular disease, which are also associated

Table 1. NCEP ATP III (2001) criteria for the clinical diagnosis of the metabolic syndrome (adapted from Alberti et al, 2009).

The presence of three of any of the five criteria constitutes a diagnosis of metabolic syndrome	Categorical cut points
1. Elevated waist circumference	Men ≥ 102 cm; women ≥ 88 cm*
2. Elevated triglycerides OR drug treatment for elevated triglyceride	≥ 1.7 mmol/L
3. Reduced HDL-cholesterol OR drug treatment for reduced HDL-cholesterol	Men ≥ 1.0 mmol/L; women ≥ 1.3 mmol/L
4. Elevated blood pressure OR treatment for elevated blood pressure	Systolic ≥ 130 mmHg and/or diastolic ≥ 85 mmHg
5. Elevated fasting glucose OR drug treatment for elevated fasting glucose	≥ 5.6 mmol/L

*Note this value is for Caucasians and there are different thresholds for different ethnic groups.

NCEP ATP III=National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III).

with insulin resistance. There are a number of slightly different definitions of metabolic syndrome, but a commonly used criterion is the NCEP ATP III* (2001) guideline (Table 1). Given that metabolic syndrome is associated with chronic kidney disease (Chen et al, 2004) and obesity, it is not surprising there is also an association of this condition with hyperuricaemia. In a large population study of adults in the United States, the prevalence of metabolic syndrome increased substantially with increasing levels of serum uric acid (Choi and Ford, 2007).

What is harder to determine is whether hyperuricaemia is involved in the pathogenesis of metabolic syndrome. A study following people aged 18–30 for 15 years, who were initially free of diabetes, found that the incidence rate of diabetes and prediabetes were higher among those with hyperuricaemia. When adjusted for multiple confounding factors, including age, sex and BMI, the relative risk of diabetes was 1.87 in those with hyperuricaemia compared to those without (Krishnan et al, 2012). In their discussion, the authors considered whether hyperuricaemia could be a marker for the renal effect of hyperinsulinaemia, as reduced excretion of urate is inversely related to the degree of insulin resistance. However, as there was a negligible association between serum uric acid and plasma insulin at baseline, they concluded that hyperuricaemia precedes rather than follows

insulin resistance.

Between the sexes, men with gout (Figure 3) appear to have a higher risk of type 2 diabetes independent of other risk factors such as age, BMI and family history of diabetes (Choi et al, 2008). In a large insurance database study, women with gout appeared to be at higher risk of diabetes than men with gout (Kim et al, 2015). In the same study, the risk of diabetes was found to be higher in people with gout, compared to people with osteoarthritis, even when adjusted for comorbidities, such as obesity.

The association of serum uric acid with hyperinsulinaemia, typical of type 2 diabetes, is complex (Choi and Ford, 2008). Initially, serum uric acid levels increase with increasing levels of HbA_{1c}, probably due to decreased renal uric acid excretion linked to hyperinsulinaemia. As HbA_{1c} levels rise, serum uric acid decreases. This is thought to be due to the uricosuric effects of high blood glucose. When treatment with insulin is instituted, uric acid levels increase (Lindsay et al, 2005), perhaps due to a combination of the effects of insulin and the drop in blood glucose reducing renal uric acid clearance. In theory, starting insulin may put the person with type 2 diabetes at an increased risk of gout or gout flares, although this remains to be confirmed clinically.

Conclusion

While treatment of some of the musculoskeletal problems associated with diabetes is outside the scope of primary care, their prompt recognition and management in primary care where possible can dramatically improve quality of life. Good

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*National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, And Treatment of High Blood Cholesterol In Adults (Adult Treatment Panel III).

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blood glucose control and weight management may play a part in the reducing the prevalence of some of these musculoskeletal conditions, as well as other diabetic complications. ■

Acknowledgment

The original version of this article was published in *Journal of Diabetes Nursing* (2016) **20**: 166–70.

Alberti KG, Ekel RH, Grundy SM et al (2009) Harmonising the metabolic syndrome. *Circulation* **120**: 1640

Amin R, Bahu TK, Widmer B et al (2005) Longitudinal relationship between limited joint mobility, height, insulin like growth factor 1 levels and risk of developing microalbuminuria: The Oxford Regional Prospective Study. *Arch Dis Child* **90**: 1039–44

Arkkila PE, Kantola IM, Viikari JS (1994) Limited joint mobility in type 1 diabetic patients: Correlation to other diabetic complications. *J Intern Med* **236**: 215–23

Balci N, Balci MK, Tuzuner S (1999) Shoulder adhesive capsulitis and shoulder range of motion in type II diabetes mellitus: Association with diabetic complications. *J Diabetes Complications* **13**: 135–40

Bongers FJ, Schellevis FG, van den Bosch WJ, van der Zee J (2007) Carpal tunnel syndrome in general practice (1981 and 2001): incidence and role of occupational and non occupational factors. *Br J Gen Pract* **57**: 36–9

Brownlee M, Cerami A, Viassara H (1988) Advanced glycosylation end products in tissue and the biochemical basis of diabetic complications. *N Eng J Med* **318**: 1315–21

Chaudhuri KR, Davidson AR, Morris IM (1989) Limited joint mobility and carpal tunnel syndrome in insulin dependent diabetes. *Br J Rheumatol* **28**: 191–4

Chen J, Muntner P, Hamm L et al (2004) The metabolic syndrome and chronic kidney disease in US adults. *Ann Intern Med* **140**: 167–74

Choi HK, Ford ES (2007) Prevalence of the metabolic syndrome in individuals with hyperuricaemia. *Am J Med* **120**: 442

Choi HK, Ford ES (2008) Haemoglobin A1C, fasting glucose, serum C-peptide and insulin resistance in relation to serum uric acid levels – the Third National Health and Nutrition Examination Survey. *Rheumatology* **47**: 713–7

Choi HK, De Vera MA, Krishnan E (2008) Gout and the risk of type 2 diabetes among men with a high cardiovascular profile. *Rheumatology (Oxford)* **47**: 1567–70

Eadington DW, Patrick AW, Frier BM (1991) Association between connective tissue changes and smoking habit in Type 2 diabetes and non diabetic humans. *Diabetes Res Clin Pract* **11**: 121–5

Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (2001) Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *JAMA* **285**: 2486–97

Gamstedt A, Noble J, Heathcote JG, Cohen H (1984) Diabetes mellitus in the aetiology of Dupuytren's disease. *J Bone Joint Surg Br* **66**: 322–5

Gamstedt A, Holm-Glad J, Ohlson CG, Sundstrom M (1993) Hand abnormalities are strongly associated with the duration of diabetes mellitus. *J Intern Med* **234**: 189–93

Hendriks SH, van Dijk PR, Groenier KH et al (2014) Type 2 diabetes seems not to be a risk factor for the carpal tunnel syndrome: a case control study. *BMC Musculoskelet Disord* **15**: 346

Infante JR, Rosenbloom AL, Silverstein JH et al (2001) Changes in frequency and severity of limited joint mobility in children with type 1 diabetes mellitus between 1976-78 and 1998. *J Pediatr* **138**: 33–7

Kameyama M, Meguro S, Funae O et al (2009) The presence of limited joint mobility is significantly associated with multiple digit involvement by stenosing flexor tenosynovitis in diabetics. *J Rheumatol* **36**: 1686–90

Kapoor A, Sibbitt WL (1989) Contractures in diabetes mellitus: the syndrome of limited joint mobility. *Semin Arthritis Rheum* **18**: 168–80

Kim SC, Liu J, Solomon DH (2015) Risk of incident diabetes in patients with gout: a cohort study. *Arthritis Rheumatol* **67**: 273–80

Krishnan E, Pandya BJ, Chung L et al (2012) Hyperuricaemia in young adults and risk of insulin resistance, prediabetes, and diabetes: A 15-year follow-up study. *Am J Epidemiol* **176**: 108–16

Lindsay JR, Kennedy L, Atkinson AB et al (2005) Reduced prevalence of limited joint mobility in type 1 diabetes in a UK clinic population over a 20 year period. *Diabetes Care* **28**: 658–61

Mavrikakis ME, Sfrikakis PP, Kontoyannis SA et al (1991) Clinical and laboratory parameters in adult diabetics with and without calcific shoulder peri-arthritis. *Calcif Tissue Int* **49**: 288–91

McCance DR, Crowe G, Quinn MJ et al (1993) Incidence of microvascular complications in Type 1 diabetic subjects with limited joint mobility: A 10 year prospective study. *Diabet Med* **10**: 807–10

Mehta SS, Singh HP, Pandey R (2014) Comparative outcome of arthroscopic release for frozen shoulder in patients with and without diabetes. *Bone Joint J* **96-B**: 1355–8

Moren-Hybbinette I, Moritz U, Scherstein B (1987) The clinical picture of the diabetic shoulder–natural history, social consequences and analysis of concomitant hand syndrome. *Acta Med Scand* **221**: 73–82

Nieves-Plaza M, Castro-Santana LE, Font YM et al (2013) Association of hand or knee osteoarthritis with diabetes mellitus in a population of Hispanics from Puerto Rico. *J Clin Rheumatol* **19**: 1–6

Pal B, Anderson J, Dick WC, Griffiths ID (1986) Limitation of joint mobility and shoulder capsulitis in insulin- and non-insulin-dependent diabetes mellitus. *Br J Rheumatol* **25**: 147–51

Rosenbloom AL (2013) Limited joint mobility in Childhood diabetes: Discovery, description and decline. *J Clin Endocrinol Metab* **98**: 466–73

Rosenbloom AL, Grigic A, Frias JL (1974) Diabetes mellitus, short stature and joint stiffness – a new syndrome. *Pediatr Res* **8**: 441

Schett G, Kleyer A, Perricone C et al (2013) Diabetes is an independent predictor for severe osteoarthritis: Results from a longitudinal cohort study. *Diabetes Care* **36**: 403–9

Silverstein JH, Gordon G, Pollock BH, Rosenbloom AL (1998) Long term glycaemic control influences the onset of limited joint mobility in type 1 diabetes. *J Pediatr* **132**: 944–7

Thomsen NO, Cederlund R, Rosen I et al (2009) Clinical outcomes of surgical release among diabetic patients with carpal tunnel syndrome: prospective follow up with matched controls. *J Hand Surg Am* **34**: 1177–87

Turner DE, Helliwell PS, Burton AK, Woodburn J (2007) The relationship between passive range of motion and range of motion during gait and plantar pressure measurements. *Diabet Med* **24**: 1240–6

Vance MC, Tucker JJ, Harness NG (2012) The association of haemoglobin A1C with the prevalence of stenosing tenosynovitis. *J Hand Surg Am* **37**: 1765–9

Yosipovitch G, Yosipovitch Z, Karp M, Mukamel M (1990) Trigger finger in young patients with insulin dependent diabetes. *J Rheumatol* **17**: 951–2

Yurdakul FG, Bodur H, Çakmak OO et al (2015) On the severity of carpal tunnel syndrome: diabetes or metabolic syndrome. *J Clin Neurol* **11**: 234–40