

Musculoskeletal manifestations of diabetes mellitus

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Rheumatic complaints are common in patients with diabetes. Maintaining good glycaemic control by exercise, diet, and medication improves or prevents the development of rheumatic conditions.

This review will discuss some of the musculoskeletal manifestations of diabetes mellitus. Table 1 shows the prevalence of these conditions in patients with and without diabetes. Sports physicians and patients with diabetes should be aware of these manifestations, so that optimal physiotherapy programmes can be devised that do not exacerbate existing complaints and encourage continuing physical activity in this group.

Diabetes mellitus is a chronic metabolic condition characterised by persistent hyperglycaemia with resultant morbidity and mortality related primarily to its associated microvascular and macrovascular complications. It is common, with recent estimates showing that 7.5% of Australians over 25 have diabetes, of which 50% of cases are undiagnosed.¹ Most of these patients (85%) have type 2 diabetes mellitus. Type 1 generally starts in the young as an autoimmune mediated disease of pancreatic dysfunction, and requires lifelong parenteral insulin supplementation. Type 2 generally occurs in people over 40 and is characterised by insulin resistance.

Physical activity is an attractive treatment for patients with diabetes given its low cost, non-pharmacological nature and additional aerobic and cardiovascular benefits.

Exercise is considered to be one of the three cornerstones of optimal diabetes treatment, along with diet and pharmacotherapy.² It is important to obtain and maintain optimal glycaemic control, as poor glycaemic control is associated with increased prevalence of diabetic complications.³ A recent meta-analysis has shown that exercise training in patients with type 2 diabetes mellitus reduces HbA_{1c} by an amount that should decrease the risk of diabetic complications. This was not associated with an appreciably greater change in body mass in the intervention group compared with the control group,⁴ suggesting that exercise training in patients with type 2 diabetes is valuable in its own right rather than being just an avenue to weight loss. Physical activity is therefore an attractive treatment for patients with diabetes given its low cost, non-pharmacological nature, and additional aerobic and cardiovascular benefits.

FROZEN SHOULDER

The most disabling of the common musculoskeletal problems is adhesive capsulitis, which is also known as frozen shoulder, shoulder periarthritis, or obliterative bursitis. It is characterised by progressive, painful restriction of shoulder movement, especially external rotation and abduction.⁵ The thickened joint capsule is closely applied and adherent to the humeral head, resulting in considerable reduction in the volume of the glenohumeral joint (fig 1). The exact origins of adhesive capsulitis are not known, although it has been associated with several other conditions, including shoulder trauma, cerebral conditions, cardiac conditions, and respiratory conditions. The natural history of the disease is characterised by three distinct phases: painful, adhesive, and resolution phases.⁵ Adhesive capsulitis appears at a younger age in patients with diabetes and is usually less painful,⁶ although it responds less well to treatment and lasts longer.⁷ The estimated prevalence is 11–30% in diabetic patients and 2–10% in non-diabetics.^{8–11} Adhesive capsulitis is associated with the duration of diabetes and age.^{10, 11}

Diabetic patients with frozen shoulder are more likely to have other diabetic complications such as limited joint mobility than diabetics without a frozen shoulder, although this may be explained by age.^{10, 11}

Most cases of adhesive capsulitis will resolve over time, but, in the interim, management consists of adequate analgesia and intra-articular corticosteroid injections in the painful early stages if required. Corticosteroid injections may increase blood sugar levels in diabetics over the 24–48 hour period after the injection, and

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Table 1 Prevalence of musculoskeletal disorders in patients with or without diabetes

Musculoskeletal disorder	With diabetes	Without diabetes
Adhesive capsulitis (frozen shoulder) ^{5–8}	11–30%	2–10%
Limited joint mobility ^{9–11}	8–50%	0–26%
Dupuytren's contracture ^{12–15}	20–63%	13%
Carpal tunnel syndrome ^{16, 17}	11–16%	125/100000*
Flexor tenosynovitis ¹⁸	11%	<1%
Diffuse idiopathic skeletal hyperostosis ^{10, 15}	13–49%	1.6–13%

*Incidence estimate over 5 years.



Figure 1 Shoulder arthrogram showing a contracted and adherent joint capsule in adhesive capsulitis.



Figure 2 Limited joint mobility shown by the inability to press the two palms completely together, with the wrists maximally flexed, forming the prayer sign.



Figure 4 Hands of patient with reflex sympathetic dystrophy.

therefore blood sugar monitoring and contingency plans for elevated blood sugar levels should be considered. Distension or manipulation under anaesthesia are occasionally considered. An appropriately graded, regular physiotherapy programme should be maintained, after the painful phase, throughout the course of the condition.

HAND SYNDROMES

There is increased incidence of hand abnormalities in patients with type 1 and type 2 diabetes compared with the general population. The association between the hand abnormality and the duration of diabetes but not age or sex is a consistent finding.¹²



Figure 3 Dupuytren's contracture of the hand.



Figure 5 Hands of patient with reflex sympathetic dystrophy.

Limited joint mobility

Limited joint mobility is also known as diabetic cheiroarthopathy (after the Greek word "cheiros" for hand). It is characterised by thick, tight, waxy skin mainly on the dorsal aspect of the hands, with flexion deformities of the metacarpophalangeal and interphalangeal joints (increased resistance to passive extension of the joints). Limited joint mobility can be shown clinically by the inability of the two palms to come completely together, with the wrists maximally flexed, forming the prayer sign (fig 2). In the early stages, paraesthesiae and slight pain develop. The symptoms increase very slowly, and greater pain, aggravated by movement of the hands, may supervene. Biopsy specimens of involved skin



Figure 6 Radiograph (lateral view) of the thoracic spine showing flowing osteophytes in diffuse idiopathic skeletal hyperostosis.

show pronounced thickening of periarticular rather than articular collagen, which may be due to non-enzymatic glycosylation of collagen.¹³

This condition is most commonly seen in type 1 diabetics, with a prevalence of 8–50%,^{6, 12} compared with 0–26% in controls,¹⁴ with differences in prevalence estimates possibly related to differences in the definitions used and perhaps differences in glycaemic control.¹⁵

Limited joint mobility is more prevalent in patients with diabetic neuropathy than in those without.^{16, 17} Limited joint mobility and Dupuytren's contracture are commonly found in the same patient.^{12, 17, 18} Treatment consists of optimising glycaemic control and an individualised hand therapy programme if a patient's symptoms warrant it.

Dupuytren's contracture

Dupuytren's contracture is the palmar or digital thickening, tethering, or contracture of the hands (fig 3). In patients with diabetes, the ring and middle finger are more commonly affected, compared with the fifth finger in patients without diabetes.¹⁹ The prevalence of Dupuytren's contracture in diabetic patients ranges from 20 to 63%,^{16, 19, 20} compared with 13% in the general population.²¹ Among patients with Dupuytren's contracture, 13–39% have diabetes.^{19, 22} The contractures are generally milder in diabetics than in patients with Dupuytren's contracture who do not have diabetes, and the prevalence increases with advancing age.²³ Treatment consists of optimising glycaemic control, physiotherapy, and hand exercises if required, and surgery only if function is severely affected. The contractures are usually mild, however, and rarely require surgery.⁶



Figure 7 Radiograph (anterior view) of the lumbar spine in diffuse idiopathic skeletal hyperostosis.

Carpal tunnel syndrome

Carpal tunnel syndrome (CTS) is a disorder characterised by paraesthesia over the median nerve cutaneous distribution of the thumb, index, middle, and lateral half of the ring fingers, which is often worse at night. The symptoms may be caused by compression of the median nerve within the carpal tunnel, diabetic neuropathy, or a combination of both.²⁴ CTS is common in patients with diabetes, with an estimated prevalence of 11–16%,^{25, 26} compared with an incidence of about 125 per 100 000 population over a five year period.²⁷ About 5–8% of patients with CTS have diabetes.²⁸ CTS is more common in women than in men. Associations between carpal tunnel syndrome and age and the duration of diabetes have been suggested.²⁴ Treatment of CTS consists of the use of simple analgesics, splints, and possibly local steroid injections for the milder cases of compressive CTS. Surgery is indicated in those patients who fail the above conservative measures.

Flexor tenosynovitis

Flexor tenosynovitis (trigger finger or stenosing tenovaginitis) is caused by fibrous tissue proliferation in the tendon sheath leading to limitation of the normal movement of the tendon. The prevalence of flexor tenosynovitis is estimated at 11% in diabetic patients, compared with <1% in non-diabetics.²⁹ There is also an increased incidence in people with impaired glucose tolerance.³⁰ Flexor tenosynovitis is associated with the duration of diabetes but not age.²⁹ A corticosteroid injection into the symptomatic flexor tendon sheath is often curative.

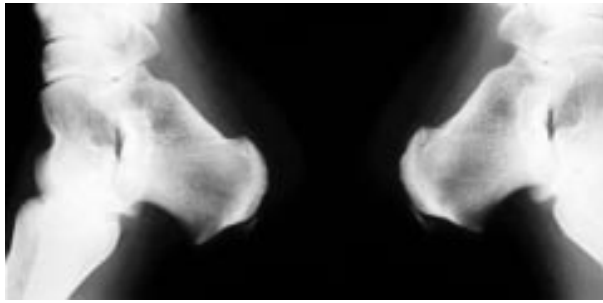


Figure 8 Bilateral calcified Achilles tendons in a patient with diabetes.



Figure 9 Joint destruction such as is seen in Charcot's disease.



Figure 10 Joint destruction such as is seen in Charcot's disease.

Reflex sympathetic dystrophy

Reflex sympathetic dystrophy is also known as algodystrophy, Sudeck's atrophy, and chronic regional pain syndrome type 1. It is characterised by localised or diffuse pain, usually with associated swelling, trophic changes, and vasomotor disturbances,³¹ with impaired mobility of the affected region (figs 4 and 5). There have been difficulties with the definitions of reflex sympathetic dystrophy, and the cause, pathogenesis, and natural history are unclear. The condition may occur spontaneously, or after minimal trauma—following surgery or a fracture. Concurrent medical conditions may predispose to reflex sympathetic dystrophy, including diabetes mellitus, hyperthyroidism, hyperparathyroidism, and type IV hyperlipidaemia.³² A variety of treatments have been used with anecdotal success, including analgesics, physiotherapy, intravenous bisphosphonates, calcitonin, oral corticosteroids, and sympathetic ganglion blocks.³² The outcome is usually good, although some patients develop chronic pain and contractures.



Figure 11 Bilateral painless ulcers in a patient with diabetic peripheral neuropathy.



Figure 12 The nail through the sole of this slipper (see arrow) worn by a patient with diabetic peripheral neuropathy, was only noticed at the end of the day when the slipper was removed.

DIFFUSE IDIOPATHIC SKELETAL HYPEROSTOSIS

Diffuse idiopathic skeletal hyperostosis, also known as ankylosing hyperostosis or Forestier's disease, is characterised by new bone formation, particularly in the thoracolumbar spine (figs 6 and 7). New bone appears to "flow" from one vertebra to the next, and is more prominent on the right side of the thoracic vertebra.³³ Ossification of ligaments and tendons elsewhere may occur, such as the skull, pelvis, heels, or elbows^{15 34} (fig 8). A proposed mechanism of causation is the prolonged and high levels of insulin or insulin-like growth factors occurring in diabetic patients, stimulating new bone



Figure 13 Diabetic amyotrophy of the shoulder girdle.

growth, and may explain the higher prevalence in type 1 compared with type 2 diabetes (ratio 3:1).²⁰ There may be associated pain in one third of patients who have hyperostosis of the heels or elbows. Patients with hyperostosis of the spine may have associated mild stiffness on arising in the morning, and 16% of affected persons may develop dysphagia.¹⁴ In most cases affected persons have normal mobility of the spine and may be asymptomatic, with the diagnosis of the condition an incidental radiographic finding.¹⁵ Estimated prevalence is 13–49% in diabetic patients⁶ and 1.6–13% in non-diabetics.²⁰ Among patients with diffuse idiopathic skeletal hyperostosis, 12–80% have diabetes or impaired glucose tolerance. The high prevalence of abnormal glucose tolerance tests in patients with diffuse idiopathic skeletal hyperostosis is partly a result of an association with obesity, with 83% of patients being male and 30% obese.¹⁴ Obesity and diabetes seem to have independent contributions to the development of the condition.¹⁴

Rates of hyperostosis increase with age in both the normal and diabetic populations, although the age related increase in incidence begins earlier in diabetics.⁶ Management consists of education, diabetic control, and physiotherapy.

NEUROPATHIC (CHARCOT'S) JOINTS

Charcot's disease, or joints, is a result of diabetic peripheral neuropathy. A reduction in the normal afferent protective neural impulses, and therefore loss of protection from trauma to the joint leads to progressive, painless joint destruction (figs 9 and 10). Charcot's joints are typically seen in patients over the age of 50 who have had diabetes for many years and have existing neuropathic complications (figs 11 and 12). The joints most commonly affected are weight-bearing joints such as the foot, ankles, and knees; joints such as the hand and wrist are

rarely affected.³⁵ Initial warmth and erythema mimic osteomyelitis or septic arthritis, but the absence of fever, elevated white cell count, and elevated erythrocyte sedimentation rate helps to differentiate the latter two conditions. Management consists of optimising glycaemic control and regular foot care and review, particularly in those with grossly impaired sensation. The use of orthotics and crutches can relieve pressure on the affected joints during ambulation.³⁵ Occasionally surgery may be required if complicated fractures develop.

DIABETIC AMYOTROPHY

Diabetic amyotrophy is a disabling illness that is distinct from other forms of diabetic neuropathy. It is characterised by muscle weakness and wasting, and by diffuse, proximal lower limb muscle pain, and asymmetrical loss of tendon jerks. The shoulder girdle may be affected, but less commonly (fig 13). It typically occurs in older men with type 2 diabetes, and is often associated with weight loss, sometimes as much as 40% of pre-morbid body mass. The exact cause and incidence of diabetic amyotrophy is uncertain. It is a diagnosis of exclusion: sinister causes must be sought and excluded because of the clinical picture of weight loss and new neurological signs. Management consists of stabilising glycaemic control and use of physiotherapy. Most cases improve, but the improvement is gradual and often incomplete.³⁶

CONCLUSION

The complications of diabetes mellitus are numerous and include involvement of the musculoskeletal system. Several rheumatic conditions are more prevalent or caused by the long term metabolic consequences of diabetes mellitus. When the control of diabetes is poor, higher levels of diabetic complications result.³ Poor glycaemic control can lead to worsening of certain rheumatic conditions. Pharmacotherapy, diet, and a regular, sensible physiotherapy programme should be the cornerstone of diabetes management. It is our recommendation that all patients with diabetes have an appropriate exercise programme, overseen by their medical practitioner, as an integral part of their diabetes management in order to reduce the frequency and severity of complications.

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