Musculoskeletal Complications of Diabetes Mellitus

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Diabetes may affect the musculoskeletal system in a variety of ways. The metabolic perturbations in diabetes (including glycosylation of proteins; microvascular abnormalities with damage to blood vessels and nerves; and collagen accumulation in skin and periarticular structures) result in changes in the connective tissue.

Musculoskeletal complications are most commonly seen in patients with a longstanding history of type 1 diabetes, but they are also seen in patients with type 2 diabetes. Some of the complications have a known direct association with diabetes, whereas others have a suggested but unproven association. This article will review the musculoskeletal and rheumatological manifestations commonly seen in patients with diabetes (Table 1).

**Hands**

Hands are a target for several diabetes-related complications. Diabetic cheiroarthropathy, also known as diabetic stiff hand syndrome or limited joint mobility syndrome, is found in 8–50% of all patients with type 1 diabetes and is also seen in type 2 diabetic patients. The prevalence increases with duration of diabetes. This condition is associated with and predictive of other diabetic complications.

This syndrome is characterized by thick, tight, waxy skin reminiscent of scleroderma. Limited joint range of motion (inability to fully flex or extend the fingers) and sclerosis of tendon sheaths are also seen. The underlying cause is thought to be multifactorial. Increased glycosylation of collagen in the skin and periarticular tissue, decreased collagen degradation, diabetic microangiopathy, and possibly diabetic neuropathy are thought to be some of the contributing factors. Flexion contractures of the fingers may develop at advanced stages. One indication of the presence of this condition is known as the “prayer sign” (Fig. 1). This is patients’ inability to press their palms together completely without a gap remaining between opposed palms and fingers.

The specific treatment of diabetic cheiroarthropathy (other than optimizing glycemic control) is unknown.

Flexor tenosynovitis (or trigger finger) is another frequent diabetic complication of the hands (Fig. 2). Patients complain of a catching sensation or locking phenomenon that may be associated with pain in the affected fingers. Examination shows a palpable nodule, usually in the area overlying the metacarpophalangeal joint, and thickening along the affected flexor tendon sheath on the palmar aspect of the finger and hand. Also, the locking phenomenon may be reproduced with either active or passive finger flexion.

This complication is thought to have the same pathogenesis as diabetic cheiroarthropathy, and its prevalence is similarly related to the duration of diabetes.

Initial treatment involves injecting local corticosteroids into the tendon sheath. If this is unsuccessful, patients will most likely need to see a hand surgeon for a minor operation that can provide permanent relief. (A small transverse incision just distal to the flexion crease over the metacarpal head exposes the flexor tendons and sheath. A complete longitudinal incision along the thickened fibrous tendon sheath relieves the constriction and allows the finger to move freely.)

Dupuytren’s contracture results from a thickening, shortening, and fibrosis of the palmar fascia. Nodule formation along the fascia is seen. Flexion contractures of the fingers may result, usually at the fourth finger, but sometimes involving any of the second through fifth digits. Dupuytren’s contracture has been reported in 16–42% of diabetic patients. Its pathogenesis is thought to be the same as that for cheiroarthropathy.

Once again, the prevalence of this condition increases with disease dura-
Dupuytren’s contracture may also be seen early in the course of disease. Physical therapy may be beneficial for early or mild cases. Varied success has been reported with local corticosteroid injections. Surgical intervention may be needed for severe cases.

Carpal tunnel syndrome (CTS) is seen in up to 20% of diabetic patients. Its specific relationship to diabetes is thought to be median nerve entrapment caused by the diabetes-induced connective tissue changes mentioned above. The prevalence of CTS in diabetic patients generally increases with duration of diabetes.

CTS is usually diagnosed based on patients’ history and clinical findings. Classically, patients complain of burning, paresthesias, or sensory loss in the median nerve distribution (the first three fingers as well as the radial half of the fourth finger). They may also complain of pain in the same area, often with radiation proximally into the forearm and arm. The pain may awaken patients from sleep and is aggravated by activities involving wrist flexion or extension, such as holding a newspaper or book, typing, driving, or using a knife and fork.

Tinel’s sign (tapping over the median nerve on the volar aspect of the wrist) may be helpful in diagnosis but is not universally positive. A positive Tinel’s sign produces paresthesias distally in the hand. Phalen’s test (the wrist flexion test) may also assist in diagnosis, but like Tinel’s sign, it is somewhat variable. Patients are asked to flex both wrists so that the dorsa of both hands are touching and to hold that position for 30–60 sec. A positive Phalen’s test consists of paresthesias being reproduced in the hand with this maneuver.

It is also important to examine patients for possible motor weakness caused by median nerve compression. This is done by assessing thenar muscle strength and examining the hand for the presence of thenar muscle atrophy.

Awareness on the part of the clinicians is necessary in order to intercede in

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**Figure 1.** The “prayer sign” indicates the presence of diabetic cheiroarthropathy. It is characterized by patients’ inability to completely close gaps between opposed palms and fingers when pressing their hands together.

**Figure 2.** This patient with flexor tenosynovitis (trigger finger) is trying to straighten out all of his fingers, but the middle finger is locked.
CTS before the development of thenar muscle atrophy. Diabetic patients may
have paresthesias caused by underlying peripheral neuropathy, and these two
entities must be differentiated.

Electromyogram/nerve conduction velocity (EMG/NCV) testing can con-
firm the diagnosis of CTS in uncertain cases and can also help to localize the
site of nerve entrapment. If more proximal entrapment is a concern, imaging
studies of the cervical region should also be completed.

Management of CTS is the same for diabetic patients as for nondiabetic
patients. Conservative treatment is tried initially for early or mild cases, using
volar wrist splints (particularly at night) with or without nonsteroidal anti-inflam-
matory drugs (NSAIDs). Ergonomic adjustment of computer workstations
should be made when appropriate. Local corticosteroid injection of the carpal tun-
nel may be tried as well. Patients with severe or refractory cases, as well as
those with thenar atrophy or progressive neurological changes on serial EMG/
NCV testing, should be sent for definit-
tive therapy with surgical release of the
transverse carpal ligament by a hand sur-
geon.

Shoulders

Diabetes can affect the shoulder in sev-
eral ways. First, adhesive capsulitis, or
frozen shoulder, has been reported in
19% of diabetic patients. This term
refers to a stiffened glenohumeral joint
usually caused by a reversible contrac-
tion of the joint capsule. Patients report
shoulder stiffness, along with decreased
range of motion. Therapy is largely con-
servative and involves minimizing over-
imobilization (gentle stretching/range
of motion exercises) and the use of anal-
gesics and/or intra-articular injections.

Calcific periarthritis of the shoul-
der is also seen in diabetes, where it is
roughly three times more common than
in people without diabetes. Shoulder
radiographs show calcium deposits out-
side of the joint, often in the area of the
rotator cuff tendons. However, in up to
two-thirds of the cases, this condition is
asymptomatic in patients with diabetes.

Reflex sympathetic dystrophy, also
known as “shoulder-hand syndrome,”
is seen in diabetic patients, although
whether it occurs with increased fre-
quency is controversial. It may be asso-
ciated with adhesive capsulitis (with
or without calcific periarthritis).

Patients may complain of pain from
shoulder to hand in the affected limb.

Classical examination findings include
swelling of the affected limb/area, skin
changes (changes in hair growth, shiny
skin, color and temperature changes),
increased sensitivity to temperature and
touch (hyperesthesia), and vasomotor
instability. Transient, patchy osteoporo-
sis is also often seen. Early intervention
is important. NSAIDs, other analgesics,
and corticosteroids have been used along
with physical therapy, and sympathetic
blocks may be helpful.

Feet

Diabetic osteoarthropathy (also known
as Charcot or neuropathic arthropathy)
is a condition involving destructive, lytic
joint changes. It is a severe, destructive
form of degenerative arthritis resulting
from a loss of sensation (brought on by
underlying diabetic neuropathy) in the
involved joints. It most commonly
affects the pedal bones. Loss of sensa-
tion leads to inadvertent (and unnoticed)
repeated microtrauma to the joints,
which leads to degenerative changes.

The condition is quite rare, affecting
only 0.1–0.4% of diabetic patients, and
is seen in both type 1 and type 2 dia-
betes. The average duration of disease
in affected patients is 15 years.

Physical examination will invariably
demonstrate peripheral neuropathy.
There may be skin such as erythema,
swelling, hyperpigmentation or purpura,
and soft-tissue ulcers over the affected
area, as well as joint loosening or insta-
bility and joint deformities.

The diagnosis is made based on radi-
ographic findings, with symptoms often
milder than would be expected based on
the radiographs. There is usually no his-
tory of overt trauma.

Depending on the stage and severity
of the arthropathy, radiographs can show
degenerative changes with subluxation,
bone fragments, osteolysis, perioseal
reaction, deformity, and/or ankylosis.
Computed tomography (CT) scans are
insensitive when evaluating for disease
activity, whereas magnetic resonance
imaging (MRI) and bone scintigraphy
studies are valuable adjuncts to plain
films in this regard. The differential
diagnosis includes infection, inflamma-
tory processes, degenerative processes,
tumor, deep venous thrombosis or
thromboembolitis, and neuropathic
arthropathies secondary to other condi-
tions. Diabetic peripheral neuropathy is
thought to play the greatest pathogenic
role in diabetic osteoarthropathy.

Treatment is generally conservative
and unsatisfactory, involving both splint-
ning/bracing to protect the area from
weight bearing and good glycemic con-
trol. Podiatrists sometimes use a total-
contact cast for acute Charcot joints.
This must be applied by an experienced
cast technician and monitored and
changed frequently. Unfortunately, it
carries a fairly high risk of causing new
injuries and ulcers because of the tight fit
and patients’ underlying neuropathy.
Broad-spectrum empiric antibiotics are
also frequently used when skin ulcers
accompany the arthropathy.

Muscles

Diabetic muscle infarction is a rare con-
dition. This spontaneous infarction, with
no history of trauma, tends to affect
patients with a long history of poorly
controlled diabetes. It is seen more com-
monly in patients with insulin-requiring
diabetes, and most affected patients
have multiple microvascular complica-
tions (neuropathy, nephropathy, and
retinopathy).

The clinical presentation is an acute
onset of pain and swelling over days to
weeks in the affected muscle groups
(usually the thigh or calf) along with
varying degrees of tenderness. Creati-
ine phosphokinase levels may be nor-
ligaments and tendons, as well.

The underlying pathophysiology is not understood. DISH has a higher prevalence among diabetic patients than among people without diabetes. Specifically, it is commonly seen in association with type 2 diabetes, particularly in obese patients.

Patients complain of stiffness in the neck and back with decreased range of motion. Pain is generally not a prominent symptom. Treatment consists of physical therapy and NSAIDs or other analgesics. There is no evidence yet that good glycemic control delays the onset of or improves this condition.

Osteoarthritis
Diabetes is not clearly a risk factor for osteoarthritis (OA). However, obesity is a risk factor for both conditions. Several studies have reported an association of early OA and diabetes. Both large and small joint OA have been reported to be increased in type 2 diabetes. However, OA of the weight-bearing joints in the affected type 2 diabetic patients may be related to their obesity and not to the diabetes itself. It is not yet known whether diabetes is a risk factor for OA independent of obesity.

Conclusion
Diabetes quite commonly affects the musculoskeletal system, resulting in significant morbidity. These manifestations may go unrecognized or simply be overlooked in daily clinical practice. However, many of these rheumatological complications are treatable (to varying degrees), with resultant improvements in quality of life and more independence in activities of daily living. Thus, clinicians should be aware of the possible musculoskeletal complications of diabetes in order to intervene and provide the best care for affected patients. Asking patients about their symptoms and monitoring for signs of musculoskeletal complications can be an invaluable part of overall diabetes care.

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