

The Musculoskeletal Complications of Diabetes

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The musculoskeletal system can be affected by diabetes in a number of ways. Some of the musculoskeletal conditions seen in diabetics are the same as those found in the general (nondiabetic) population, whereas others are unique to diabetes. The hands, shoulders, feet, muscles, and skeleton are some of the frequently affected sites. Although there is often no “cure” for these problems, there are treatments available that can significantly improve function and quality of life for diabetics with rheumatologic problems.

Introduction

Diabetes can cause a number of changes in the connective tissue. Microvascular abnormalities, with subsequent damage to blood vessels and nerves, along with protein glycosylation, and excess collagen accumulation in the skin and connective tissue are some of the metabolic perturbations thought to play a role in the development of musculoskeletal problems in diabetics. In general, musculoskeletal complications are most often seen in patients with a longstanding history of type 1 diabetes, but are also encountered (not infrequently) in patients with type 2 diabetes. This article reviews the common rheumatologic problems seen in diabetics and their appropriate management.

The Hand

The hand may be affected in several ways by diabetes. First, diabetic cheiroarthropathy (also known as syndrome of limited joint mobility or diabetic stiff hand syndrome) may occur in 8% to 50% of type 1 diabetics, and is also seen in patients with type 2 diabetes [1••]. The prevalence increases with the duration of diabetes. This syndrome is characterized by thick, tight, waxy skin over the hands, reminiscent of scleroderma. Patients may complain of pain and/or paresthesias early on. Limited range of motion in the joints of the hands ensues, and sclerosis of the tendon

sheaths is seen. Flexion contractures of the fingers may develop at advanced stages. The “prayer sign” characterized by an inability of the patient to press the palms of both hands completely together may be observed in diabetic cheiroarthropathy.

A multifactorial etiology is thought to underlie the syndrome. Increased glycosylation of the skin and peri-articular tissue, diabetic microangiopathy with a thickened basement membrane, decreased collagen degradation, and possibly diabetic neuropathy have been implicated as possible contributing factors [1••,2•]. Diabetic cheiroarthropathy is associated with and predictive of other diabetic complications, such as retinopathy and nephropathy. The specific treatment of diabetic cheiroarthropathy is unknown. Optimizing glycemic control is postulated to be helpful in arresting its progression.

Dupuytren’s contracture is a thickening, shortening, and fibrosis of the palmar fascia. Nodules may be observed on the palms of affected patients, and flexion contractures of the fingers may result. The fourth finger is most commonly affected, but any of the second through fifth digits can be involved. Dupuytren’s contracture, although not unique to diabetes, has been reported in 16% to 42% of diabetic patients [1••]. In diabetics, the pathogenesis is thought to be the same as in cheiroarthropathy. The prevalence of this condition also increases with disease duration, but Dupuytren’s contracture may be seen early in the course of diabetes. Treatment for Dupuytren’s contracture is rather empiric. Physical therapy may be of some benefit for early or mild cases, and varied success has been reported with local corticosteroid injections. Surgical intervention may be necessary for severe or refractory cases.

Flexor tenosynovitis, or trigger finger, is another frequent musculoskeletal complication seen in diabetes. This condition, however, is also found in nondiabetics. Patients complain of a catching sensation or locking phenomenon of the finger(s) on flexion, which may or may not be painful. On examination, there is usually a palpable nodule at the base of the affected finger, along the flexor tendon sheath. The thickened tendon sheath causes the tendon to get “stuck” during finger flexion. This locking may often be reproduced on examination with active or passive flexion. In diabetics, this complication is thought to share the same pathogenesis as diabetic cheiroarthropathy. Its prevalence is similarly related to the duration of diabetes also.

Initial treatment of flexor tenosynovitis consists of a local corticosteroid injection into the tendon sheath. If, after two or three injections, this approach is unsuccessful the next step is referral to a hand surgeon who performs a minor operation that can provide permanent relief. A complete longitudinal incision along the thickened fibrous tendon sheath relieves the constriction.

Carpal tunnel syndrome (CTS) is observed in up to 20% of diabetic patients [1••,2•]. Although CTS is commonly observed in nondiabetics as well, its specific relationship to diabetes is thought to be median nerve entrapment resulting from the diabetes-induced changes in connective tissue as mentioned above. The prevalence of CTS in diabetics generally increases with diabetes duration.

The diagnosis of CTS is usually a clinical one, based on the patient's history along with physical examination findings. A classic presentation is the complaint of burning, pain, paresthesias, or sensory loss in the median nerve distribution. The pain may radiate proximally into the forearm, or even the arm. It is often worse at night, and with activities involving wrist flexion and extension, such as holding a newspaper or book, typing, driving, or with the use of eating utensils.

A positive Tinel's sign (the production of paresthesias distally in the hand on tapping over the median nerve on the volar aspect of the wrist) may be helpful in diagnosis, but is not universally positive. Similarly, a positive Phalen's test may assist in diagnosis. The patient fully flexes both wrists, with the dorsa of the hands opposed to one another, and maintains that position for 30 seconds. A positive test consists of the reproduction of paresthesias in the hand.

When CTS is suspected, it is very important to look for any associated motor weakness caused by median nerve entrapment, as this finding warrants prompt and aggressive (surgical) therapy. Evaluation should consist of assessing thenar muscle strength, and looking for the presence of any thenar muscle atrophy. In the absence of these findings, conservative measures may generally be tried first.

An important point to remember when evaluating a diabetic patient for possible CTS is that these patients may have paresthesias due to an underlying peripheral neuropathy, and the two entities must be differentiated. Electromyogram/nerve conduction velocity (EMG/NCV) testing can confirm the diagnosis of CTS in uncertain cases.

Management of CTS in diabetics does not differ from the approach in nondiabetics. For early or mild cases, with no evidence of motor compromise, conservative measures are tried first. Volar wrist splints are worn (especially at night), usually with nonsteroidal anti-inflammatory drug (NSAID) therapy. When appropriate, ergonomic adjustments of workstations should be done. Another option is local corticosteroid injection of the carpal tunnel. For patients with severe or refractory cases, evidence of thenar atrophy or weakness, or progression on serial EMG/NCV testing,

referral to a hand surgeon for definitive therapy is appropriate (surgical release of the transverse carpal ligament).

The Shoulder

Diabetes can affect the shoulder in a number of ways. Adhesive capsulitis, or frozen shoulder, has been reported in approximately one fifth of diabetic patients, and is also observed in nondiabetics [2•]. In this self-limited disorder, the glenohumeral joint capsule undergoes (reversible) contraction. Patients most often report shoulder stiffness and decreased range of motion. Pain is a less frequent complaint. The etiology is not well understood. Therapy consists of minimizing over-immobilization with gentle stretching and range-of-motion exercises daily. Analgesics and/or intra-articular injections (both corticosteroid and saline have been used) may also be employed.

Calcific periartthritis of the shoulder is also seen in diabetics, who are affected roughly three times more often than their nondiabetic counterparts. The diagnosis is based on shoulder radiographs, which demonstrate calcium deposits outside of the joint (often in the area of the rotator cuff tendons). This may be an incidental finding, as up to two thirds of affected diabetics are asymptomatic.

Reflex sympathetic dystrophy (RSD), also known as "shoulder-hand syndrome," is another condition seen in diabetics. Whether RSD occurs with increased frequency in diabetics is contentious. RSD is a pain syndrome that often results in intractable pain and severe disability. Patients may report pain from the shoulder to the hand in the affected limb (lower extremities may also be affected). Occasionally, there is a report of antecedent trauma or illness as an inciting event, but often there is no such history. Although the pathophysiology of RSD is unclear, autonomic nervous system dysregulation is believed to be of central importance.

Physical examination reveals vasomotor changes, including swelling, hyperesthesia, allodynia, and skin changes (changes in hair growth, shiny skin, color, and temperature changes). The affected area is often tender, and demonstrates impaired mobility. RSD may be associated with adhesive capsulitis (with or without calcific periartthritis).

Triple-phase radionuclide scans typically show changes of significantly increased, very early uptake in blood flow and pool studies, with a delayed increase in uptake in the periarticular tissues of the affected limb/area (compared with the contralateral side) [1••]. Radiographs show patchy osteopenia in the affected area, but these changes are usually not evident until later (after several weeks) and are not specific for RSD.

Early intervention is believed to be important. Rigorous physical therapy is central to treatment. Pain control is generally instituted with NSAIDs and analgesics. Oral corticosteroids may be useful early on. Sympathetic blockade may be used in refractory or severe cases.

Recently, some success with pain control has been reported using spinal cord stimulation in chronic RSD [3].

The Foot

Diabetic osteoarthropathy (also known as Charcot or neuropathic osteoarthropathy) most commonly affects the pedal bones. This condition, which in diabetics is always accompanied by an underlying diabetic peripheral neuropathy, is a severe and destructive form of degenerative arthritis. Loss of sensation (due to neuropathy) is thought to lead to inadvertent and repeated microtrauma to the joints, which leads to the characteristic degenerative changes. Diabetic peripheral neuropathy is thought to play the greatest pathogenic role in the development of diabetic osteoarthropathy.

Diabetic osteoarthropathy is rare, affecting 0.1% to 0.4% of diabetics [1••]. It is observed in both type 1 and type 2 diabetes. Affected patients typically have a long-standing history of diabetes, with an average disease duration of 15 years. Physical examination invariably demonstrates the presence of a peripheral neuropathy. There may be skin changes, such as erythema, swelling, hyperpigmentation or purpura, or soft tissue ulcers overlying the affected area. Joint instability and gross joint deformities may also be evident.

Diagnosis of diabetic osteoarthropathy is based on radiographic findings. There is usually no history of overt trauma, and symptoms are often milder than would be expected based on the radiographic appearance. Radiologic signs range from osteoporosis, cortical defects, periosteal reactions, and subluxation to osteolysis, fragmentation, dislocation, ankylosis, and fracture, depending on the stage when radiographs are taken. Computed tomography (CT) scans are insensitive when used to evaluate disease activity, whereas magnetic resonance imaging (MRI) and bone scintigraphy studies can be valuable adjuncts to plain films in this regard. The differential diagnosis of diabetic osteoarthropathy includes neuropathic arthropathy secondary to other conditions, infection (osteomyelitis), inflammatory processes, degenerative processes, tumor, and deep venous thrombosis or thrombophlebitis.

Treatment is generally conservative and unsatisfactory. Splinting or bracing to protect the area from weight bearing, and maintaining good glycemic control are mainstays of therapy. Broad-spectrum antibiotics are frequently employed when soft tissue ulceration accompanies the arthropathy. Podiatrists will sometimes use a total-contact cast for the acute Charcot joint. This cast must be applied by an experienced technician, and frequent monitoring and changing are required. Unfortunately, because of its tight fit and patients' underlying neuropathy, the total-contact cast carries a fairly high risk of causing new injuries and ulcers. Surgical treatment and amputation are employed cautiously only as appropriate.

Muscle

Diabetic muscle infarction is a rare complication. Patients with a long-standing history of poorly controlled diabetes are most often affected. This spontaneous infarction, with no history of trauma, is seen more commonly in insulin-requiring diabetes, and those affected tend to have multiple microvascular complications (retinopathy, nephropathy, and neuropathy).

Patients present with the acute onset of pain and swelling over days to weeks in the affected muscle groups (usually the thigh or calf), with varying degrees of tenderness. Muscle enzymes, such as creatinine phosphokinase, may be normal or elevated. Laboratory studies are done primarily to exclude other conditions, including tumor, muscle infection or abscess, thrombosis or thrombophlebitis, localized myositis, and osteomyelitis.

CT scans are nonspecific. MRI may show a high signal of the affected muscle on T2-weighted images. Incisional muscle biopsy may be required for diagnosis. Histologic findings consist of muscle edema and necrosis. Excisional biopsy may actually worsen this condition, and should only be done to rule out malignancy or infection.

Therapy for diabetic muscle infarction comprises rest and analgesia. Routine daily activities are not thought to be deleterious, but physical therapy may worsen the condition. Most cases resolve over a period of weeks to months.

The Skeleton

Diffuse idiopathic skeletal hyperostosis (DISH) is a term for metaplastic calcification of the spinal ligaments, which can be diagnosed on lateral spine radiographs, along with prominent osteophyte formation. In DISH, the apophyseal joints, sacroiliac joints, and disc spaces are spared, thus helping to differentiate it from other conditions. The thoracic spine is most commonly affected. DISH may be accompanied by a more widespread calcification of other (extra-axial) ligaments and tendons.

The pathophysiology is not understood. DISH has a higher prevalence among diabetics than among the non-diabetic population, and is commonly seen among obese type 2 diabetics. Patients tend to complain of neck and back stiffness, with loss of some range of motion. Pain is generally not a prominent symptom.

Treatment consists of physical therapy and NSAIDs or other analgesics. As of yet, there is no evidence that good glycemic control delays the onset of or improves this condition.

Osteoarthritis

Diabetes itself is not clearly a risk factor for osteoarthritis (OA). Obesity is a risk factor for both conditions. Several studies reported an association between early OA and diabetes, and OA has been reported to be increased in type

2 diabetes. However, OA of the weight-bearing joints in affected type 2 diabetic patients may be related to their obesity rather than to the diabetes itself. At this time, it is not yet known whether diabetes is a risk factor for OA independent of obesity.

Conclusions

Diabetes quite commonly affects the musculoskeletal system in a number of ways. These conditions, although resulting in significant morbidity, are often unrecognized or overlooked. However, many of the rheumatologic complications of diabetes, while not necessarily curable, are treatable to varying degrees. Such treatments can afford improvement in quality of life and allow more independence in activities of daily living. Thus, having an awareness of the potential musculoskeletal complications of diabetes can be an invaluable part of diabetes care.

References and Recommended Reading

Papers of particular interest, published recently, have been highlighted as:

- Of importance
- Of major importance

1. •• Forgas SS: **Endocrine and hemoglobin-related arthropathies and storage diseases.** In *Rheumatology*. Edited by Klippel JH, Dieppe PA. London: Mosby; 1998:8.23.1–8.23.6.

This is a two-volume comprehensive and authoritative textbook on rheumatology.

2. • **UpToDate Clinical Reference Library [database online]. Release 9.2. Various topics in diabetes and rheumatology.** Wellesley, MA: UpToDate; 2001.

UpToDate is an educational program available on CD-ROM as well as online, that covers many topics in internal medicine. On a monthly basis, physician editors review more than 270 journals for up-to-the-minute medical findings. The rather comprehensive literature review is updated (several times each year) with the publication of new important information.

3. Kemler MA, Barendse GA, van Kleef M, *et al.*: **Spinal cord stimulation in patients with chronic reflex sympathetic dystrophy.** *N Engl J Med* 2000, 343:618–624.