Diabetes mellitus is a relatively common condition and is frequently encountered in patients seen by hand surgeons. This review examines the known problems associated with diabetes, as well as some of the clinical findings noted in these patients, who often have multiple visits for hand conditions over time. (J Hand Surg 2008;33A:771–775. Copyright © 2008 by the American Society for Surgery of the Hand. All rights reserved.)

**Key words** Carpal tunnel, diabetes mellitus, Dupuytren’s contracture, stiffness, trigger finger.

**LIMITED JOINT MOBILITY**

In 1957, Lundbaek published a description of hand stiffness as a complication of diabetes mellitus. Although the condition had been recognized earlier, this was the first widely published description of what was later dubbed limited joint mobility (LJM) by Rosenbloom et al., who recognized LJM as particularly associated with juvenile type 1 diabetes. Subsequent studies have corroborated similar findings. LJM is a condition of stiffness principally in the hands that occasionally extends to the proximal upper extremities and spine.

**Clinical findings**

Patients with LJM typically have limited extension of the metacarpophalangeal, proximal interphalangeal, and distal interphalangeal joints, generally beginning in the ulnar digits and spreading radially. Simple physical examination signs can be used to screen for LJM. The preacher’s sign involves the patient holding the hands opposed to one another vertically with elbows flexed and wrists extended. A positive sign is indicated by an inability of the patient to completely approximate the palmar surface of the digits (Fig. 1). The table top sign is a similar test in which the patient places the palms flat on a hard surface with the digits spread. Normally, the entire palmar surface of the digits should contact the table. If the test is positive, the digits and palm will not lie flat. In a similar test, Lawson et al. screened for LJM with the hands held in the preacher’s position and the digits spread. Positive screening tests warrant careful passive examination of each joint to assess limited extension. Both these tests can be positive with other clinical conditions, such as Dupuytren’s contracture or previous trauma, so a careful history and physical examination to rule out these conditions are warranted.
Relation to diabetic disease

In an effort to elucidate the pathophysiology of LJM and to assess its usefulness as a sign of the severity of generalized disease, multiple studies have tried to correlate LJM with both complications of diabetes mellitus, and demographic characteristics of patients. Almost universally, studies have documented a positive relationship of LJM with age and duration of diabetic disease. The prevalence of LJM has ranged from 8% to 76%, with most studies identifying the rate at approximately 30%. Notably, a study that looked for LJM in 78 patients with a relatively short duration of type 2 diabetes (<10 years) found no evidence of the condition.

Several of these studies involved type 1 diabetes and found a strong relationship between LJM and retinopathy. Lawson et al. examined the relationship of LJM with retinopathy and found an overall higher prevalence of LJM among patients with severe retinopathy, whereas Rosenbloom et al. showed a higher prevalence with any degree of retinopathy. Subgroup analysis in the study by Lawson et al. indicated that in insulin-dependent diabetes mellitus, LJM was related to retinopathy independent of age and duration of disease; in non–insulin-dependent diabetes mellitus, however, LJM was linked to age and duration but not independently to retinopathy. This study is a good example of the difficulty inherent in studying diabetes mellitus, particularly in the older population with type 2 diabetes. It is difficult to recruit enough patients to independently assess the various complications (LJM, retinopathy, nephropathy, neuropathy) and sufficiently investigate what mechanism may be responsible for the LJM. Additionally, establishing a true duration of disease, or extent of blood sugar control, is near impossible. Just as many insulin-resistant patients escape clinical study, subclinical joint stiffness has been found in diabetic patients who would not clinically qualify as having LJM, indicating that pathologic mechanisms are at work long before patients are identified.

DUPUYTREN’S DISEASE

A disease with clear similarities to LJM, Dupuytren’s disease (DD) is more common among those with diabetes than in the general population. Unlike LJM, however, DD has an increasingly well-understood etiology in the absence of diabetes, one that is clearly independent of the glycosylation involved in diabetic complications. This difference, along with a few distinct differences in clinical findings, suggests that the pathophysiology of DD differs in people with and without diabetes. Furthermore, although LJM and DD may coexist in people with diabetes, the two are distinct clinical entities.

Clinical findings

Dupuytren’s disease consists of palmar and digital nodules and cords, palmar skin tethering, and digital contractures. Studies have noted that in the setting of diabetes mellitus, involvement is predominantly of the ring and middle digits, as opposed to DD, which more commonly involves the small and ring digits. Additionally, several studies have noted that diabetic patients often experience a milder form of disease, with few patients complaining of symptoms, a finding that was recently confirmed in a retrospective study of 2,919 patients with DD. This study also found that the prevalence of diabetes mellitus in their population of DD patients was only slightly higher than in the general population (11 vs. 7%). The lack of a large difference in prevalence in this study may be due to an inability to capture the mild forms of the disease that would not present to physicians. If true, this further supports the idea that the pathophysiologic processes of DD differ in people with and without diabetes.

Relation to diabetic disease

As with LJM, DD has been found to have an increased incidence among diabetic patients, and age and duration are likewise associated with an increased inci-
dence of DD. Studies of association with diabetic complications vary. Some studies have found an association between DD and retinopathy independent of age and duration of disease. Others have identified age and duration as the principal factors. Dupuytren’s disease has also been associated with neuropathy and LJM, independent of age and duration. Notably, DD does not appear to be related to glucose control, although this may be a function of the difficulty in assessing long-term glucose control.

CARPAL TUNNEL SYNDROME

Carpal tunnel syndrome has a well-documented correlation with diabetes. Unlike DD, which tends to present in a milder form in people with diabetes, carpal tunnel syndrome tends to be more problematic in this group. Additionally, carpal tunnel release seems to provide less reliable symptom relief in the diabetic population.

Clinical findings

Carpal tunnel syndrome presents with similar clinical findings in diabetic and nondiabetic patients alike. Anecdotally, diabetic patients are thought to have worse outcomes from carpal tunnel release than nondiabetic patients. Studies investigating such a difference have met with mixed results, although evidence of poor nerve regeneration in diabetes lends support to the theory. Among clinical studies, some find no difference between diabetic and normal control subjects, whereas others find that improvement in symptoms, though still important, is not as great among diabetics.

Relation to diabetic disease

The incidence of carpal tunnel syndrome in the diabetic population has consistently been reported as between 11% and 21%, in numerous studies. The mechanism of carpal tunnel syndrome in the setting of diabetes is not known, but two general theories prevail. One is that glycosylation of connective tissues increases collagen cross-linking, leading to increased stiffness and thickening of the transverse carpal ligament or peritendinous tissue. A second possibility, not exclusive of the first, is that the polyneuropathy caused by diabetic microvascular leads to increased susceptibility of the median nerve to a compressive injury. Evidence supports both theories. One histologic study suggests that both tissue changes, such as thickening of collagen bundles in tendon sheaths, and small vessel arteriosclerosis are contributors to carpal tunnel syndrome. In the diabetic population, carpal tunnel syndrome has been connected independently to retinopathy, general peripheral neuropathy, stenosing tenosynovitis, and Dupuytren’s disease. These relationships suggest both a microvascular and a gross compressive mechanism of injury to the median nerve.

STENOSING TENOSYNOVITIS (TRIGGER FINGER)

Although stenosing tenosynovitis, commonly referred to as trigger finger, is less well-documented among diabetics than are LJM, Dupuytren’s disease, and carpal tunnel syndrome, the condition has been clearly shown to be more common in the diabetic population.

Clinical findings

Trigger finger in the diabetic population does not differ considerably from that in the general population. Clinically, patients present with complaints of stiffness, pain, or locking in the digit, with tenderness and often a palpable nodule at the A1 pulley. Studies have shown that, compared with a nondiabetic population, trigger finger in diabetic patients is more common in female patients, more often bilateral, more often multidigit, and relatively sparing of the index and small fingers. Trigger finger in diabetic patients responds less well to corticosteroid injection, a common initial treatment, and more often requires surgery.

Relation to diabetic disease

Trigger finger has been shown to have a prevalence of approximately 20% in multiple studies of diabetic populations, compared with roughly 2% in the general population. As with other hand complications, age and duration of disease are often cited as significant contributing factors. In one study, trigger finger was found to be independently associated with carpal tunnel syndrome in type 1 diabetic patients. This finding is consistent with a study of flexor tendon sheath histology in patients having carpal tunnel release that revealed thickening of the sheaths, suggesting a similar etiology for both conditions.

INFECTION

Hyperglycemia has a negative effect on cell-mediated immunity and phagocyte function, increasing the risk of infection in the diabetic population. Accordingly, people with diabetes are at increased risk for hand infection. Several series have reported on the treatment of multiple hand infections in the diabetic population. The apparent significant incidence of diabetic hand infections in tropical regions, and particularly in sub-Saharan Africa, has led to the coining of the term “tropical diabetic hand syndrome.”

None of these studies has shown statistically an increased risk of incidence or virulence of hand infec-
tions in the diabetic population, but some findings from an example case series of 25 patients are worthy of mention. First, a history of trauma played a role in only 16% of patients, and other studies reflect either a relatively low incidence of trauma or minimal severity of the antecedent trauma. Second, intraoperative cultures grew out multiple organisms in 55% of specimens, gram-negative organisms in 73%, and Staphylococcus aureus in only 36%, a pattern of flora also mirrored in other series. This representative case series suggests that all diabetic hand infections should be treated with caution, and the possibility of polymicrobial infection should be considered when determining an initial approach to antibiotic coverage.

HAND WEAKNESS

Hand weakness has been demonstrated in the diabetic population, compared with normal control subjects, and this observation mirrors similar findings in the lower extremities. In the setting of the numerous hand complications described so far, functional disability may not seem surprising. As noted, however, both LJM and DD in the diabetic population tend not to cause significant functional disability, and many such patients likely do not present to physicians for treatment. Reduced grip and pinch strength, however, have been found in at least one study to be independent of LJM, DD, and trigger finger. In addition, no correlation has been established with age, duration of disease, or control of diabetes. Although not proven clearly, neuropathy has been suggested as a possible etiology for diabetic hand weakness.

DERMATOLOGIC LESIONS

In the diabetic population, numerous cutaneous lesions may occur in the hands, as well as in other locations.

Bullous diabeticorum

Bullous diabeticorum, or diabetic blisters, occur in patients with severe diabetes and often in the setting of neuropathy. The blisters (0.5–3.0 cm) are typically painless, irregular in shape, have no surrounding inflammation, and arise acutely. They are self-limiting and heal without significant scarring in 2 to 4 weeks.

Granuloma annulare

Granuloma annulare lesions, which have an anecdotal relationship to diabetes, may range from a few to many hundreds of small (1–2 mm) papules that may combine to form annular plaques. These may appear on the dorsum of the hand and fingers, as well as on extensor surfaces, and do not resolve spontaneously. Generally, no specific treatment is required, although several topical dermatologic medications may be effective.

Huntley’s papules

Huntley’s papules, or “skin pebbles,” typically occur on the dorsum of the hands or interphalangeal joints of the fingers and are a self-limiting marker of diabetic disease.

Necrobiosis lipoidica diabeticorum

Necrobiosis lipoidica diabeticorum lesions are painless, scaly papules that develop into sclerotic plaques that may eventually ulcerate. These uncommon lesions are found most often on the legs but may also affect the hands. Necrobiosis lipoidica is considered a marker for the development of diabetes, in that they are often found in nondiabetic patients who are found, on further history and testing, to have impaired glucose tolerance or a family history of diabetes. Topical steroids may be attempted on enlarging lesions, but in the absence of ulceration no specific treatment is necessary.
REFERENCES