Dupuytren's Disease, Carpal Tunnel Syndrome, Trigger Finger, and Diabetes Mellitus

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A comparative prospective study of 120 adult diabetics (60 insulin dependent, 60 non-insulin dependent) and 120 non-diabetic adults as controls showed significantly higher incidence of Dupuytren's disease, limited joint motion, carpal tunnel syndrome, and flexor tenosynovitis in the diabetic population. Of the diabetic patients one third had a mild non-progressive form of Dupuytren's disease, which commonly involved the long and ring rays. Limited joint motion was noted in a third of diabetics, and carpal tunnel syndrome was observed in 15-25%, and flexor tenosynovitis in about a fifth. Limited joint motion co-existed with Dupuytren's disease in 57% of insulin-dependent diabetics. Diabetic polyneuropathy was found in two thirds of insulin-dependent diabetics and in one third of non-insulin dependent diabetics. All these hand changes were more marked in insulin-dependent diabetics and they showed a positive correlation with increasing age of the patient, duration of the diabetes, and the presence of a microangiopathy. (J Hand Surg 1995;20A:109-114.)

Diabetes mellitus has been cited as an etiologic factor of several conditions affecting the hand, such as Dupuytren's disease, carpal tunnel syndrome, trigger finger, and reflex sympathetic dystrophy. In 1974 Rosenbloom reported a syndrome of painless limitation of digital motion with involvement of small joints and thick, tight, waxy skin occurring in young patients with insulin-dependent diabetes (type I diabetes). This condition, named limited joint motion by Rosenbloom and cheiroarthropathy by Benedetti, also occurs in non-insulin dependent diabetics (type II diabetes) and in the non-diabetic older population. This condition can also affect large joints such as the wrist and the elbow.

This study aims to analyze the prevalence and the unique clinical behavior of Dupuytren's disease, limited joint motion, carpal tunnel syndrome, and flexor tenosynovitis in patients with diabetes mellitus, and to clarify surgical management of Dupuytren's disease and carpal tunnel syndrome in these patients.

Materials and Methods

The diabetic population studied consisted of 120 adults who had a diagnosis of diabetes for at least 1 year. Of these, 60 were insulin dependent (type I diabetes; 19 men and 41 women) and 60 were non-insulin dependent (type II diabetes; 20 males and 40 females). The control series consisted of 120 non-diabetic patients, matched for age and sex.

A family history of Dupuytren's disease or any other disease process that could be implicated in the occurrence of Dupuytren's disease (i.e., epilepsy, alcoholism), carpal tunnel syndrome (generalized neuropathy, pregnancy, hypothyroidism) or flexor
Figure 1. The prayer sign in a diabetic patient with evidence of limited joint motion.

The diagnosis of LJM included skin thickening and the inability to mobilize the skin over the dorsum of the involved finger.

The diagnosis of carpal tunnel syndrome (CTS) required the presence of intermittent symptoms of numbness or tingling, a positive provocative test (Tinel’s sign, Phalen’s test, or Paley and MacMurtry’s test) and an abnormal electrodiagnostic study of the median nerve at the wrist.

The diagnosis of flexor tenosynovitis included the observation of palpable crepitus at the palm or locking of a digit in flexion or in extension, or both.

Diabetic patients were investigated for glucose control using the glycosylated hemoglobin (HbA1c) measurement; for microvascular complications by retinal angiography; and for diabetic peripheral neuropathy by an electrodiagnostic study of the median and ulnar nerves in the upper limbs and of the peroneal and tibial nerves in the lower limbs. Dorsal skin biopsies of digits with LJM were performed in eight digits and were analyzed by means of light microscopy. The significance of the results was evaluated using the chi-square test and the Student t-test.

Results

In diabetic patients, we found a higher incidence of Dupuytren’s disease (p < .01 in type I and type II diabetes), LJM (p < .01 in both types of diabetes), CTS (p < .01 in type I and p < .05 in type II diabetes) and flexor tenosynovitis (p < .01 in both types of diabetes) than in the non-diabetic population (Fig. 2).

In types I and II diabetics, Dupuytren’s disease had an almost similar incidence and was observed four times more frequently than in the non-diabetic population (Fig. 1). In both types of diabetes men were equally affected (10 of 19 in type I, and 10 of 20 in type II), whereas women with type I diabetes were more frequently affected than those with type II (11 of 41 vs. 7 of 40). The prevalence of Dupuytren’s disease was also significantly related to the age of the patient (Table 1) and the duration of diabetes (p < .01 in type I and p < .05 in type II) (Table 2). The incidence of Dupuytren’s disease in patients who had been diabetic for over 15 years was significantly more frequent (p < .001 in type I and p < .01 in type II diabetes) (Table 2). Both hands were involved in one-third of the diabetic patients who had been diabetic for over 15 years was significantly more frequent (p < .001 in type I and p < .01 in type II diabetes) (Table 2). Both hands were involved in one-third of the diabetic patients. Features of Dupuytren’s fasciitis in diabetics that differ from those observed in non-diabetics include the predominant involvement of the fourth and the third rays (Fig. 3). The fifth digit was involved in only 7 of the 38 diabetics showing evidence of Dupuytren’s disease, but was more severely affected than the
other rays (Fig. 3). Contractures of the digits were observed only in men. No knuckle pads were seen in this study but one diabetic had Ledderhose's disease. In the 21 patients who had both Dupuytren's disease and type I diabetes, LJM was observed in 12 patients (p < .01), CTS in 9 patients (p < .01), and a microangiopathy in 13 patients (p < .01).

As with Dupuytren's disease, LJM was also more common in the diabetic patients than in the control population (Fig. 1). Women were equally affected regardless of their type of diabetes (10 of 41 in type I diabetes, and 11 of 40 in type II diabetes), whereas men with type I diabetes were more likely to have LJM than those with type II (10 of 19 vs. 6 of 20). The occurrence of LJM increased significantly with the increasing age of the patient, only in type I diabetics (p > .01) (Table 1). No correlation was found between LJM and the duration of diabetes. In both types of diabetics showing evidence of LJM stage I was observed in one third of cases, stage III in one fifth of cases. The remaining affected patients which represented the most important group (16 of 38 patients), had an intermediate deformity. Note that Dupuytren's disease occurred in the majority of type I diabetics with LJM (12 out of 21 patients). A similar correlation was not observed in type II diabetics. In both types of diabetes when Dupuytren's disease and LJM co-existed (38 patients), a pretendinous cord was responsible for a contracture of the digit in only 4 patients. Eleven of the 21 patients with LJM and type I diabetes also had a microangiopathy (p < .01) and 15 demonstrated a diabetic peripheral neuropathy (p < .01). Biopsy studies of the skin and periarticular tissues in eight hands of diabetics with LJM showed an abnormal thickening of the dermis with accumulation of collagen and fibroblasts on light microscopic examination.

Table 1. Relation Between the Age and Hand Abnormalities in Diabetic Patients

<table>
<thead>
<tr>
<th>Age (Years)</th>
<th>Diabetes Type I</th>
<th>Diabetes Type II</th>
</tr>
</thead>
<tbody>
<tr>
<td>DD +</td>
<td>52 ± 11†</td>
<td>66 ± 7†</td>
</tr>
<tr>
<td>(n = 21)</td>
<td>(n = 18)</td>
<td></td>
</tr>
<tr>
<td>DD –</td>
<td>33 ± 13</td>
<td>59 ± 9</td>
</tr>
<tr>
<td>(n = 39)</td>
<td>(n = 42)</td>
<td></td>
</tr>
<tr>
<td>LJM +</td>
<td>48 ± 13†</td>
<td>61 ± 7</td>
</tr>
<tr>
<td>(n = 21)</td>
<td>(n = 17)</td>
<td></td>
</tr>
<tr>
<td>LJM –</td>
<td>34 ± 14</td>
<td>60 ± 16</td>
</tr>
<tr>
<td>(n = 39)</td>
<td>(n = 43)</td>
<td></td>
</tr>
<tr>
<td>CTS +</td>
<td>46 ± 11‡</td>
<td>54 ± 12</td>
</tr>
<tr>
<td>(n = 16)</td>
<td>(n = 9)</td>
<td></td>
</tr>
<tr>
<td>CTS –</td>
<td>37 ± 16</td>
<td>62 ± 9</td>
</tr>
<tr>
<td>(n = 44)</td>
<td>(n = 51)</td>
<td></td>
</tr>
<tr>
<td>FTS +</td>
<td>44 ± 9‡</td>
<td>37 ± 18</td>
</tr>
<tr>
<td>(n = 14)</td>
<td>(n = 10)</td>
<td></td>
</tr>
<tr>
<td>FTS –</td>
<td>37 ± 18</td>
<td>60 ± 2</td>
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<tr>
<td>(n = 46)</td>
<td>(n = 50)</td>
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</table>

* Mean and standard deviations. Significant differences at p < 0.01(†), at p < 0.05(‡) compared to diabetics without hand abnormalities.

N = 120.

DD, Dupuytren's disease. LJM, Limited joint mobility. CTS, Carpal tunnel syndrome. FTS, Flexor tenosynovitis. +, presence. –, absence.
CTS was six times more frequent in the patients with type I diabetes (16 of 60) and four times more frequent in the patients with type II diabetes (9 of 60 patients), than in the non diabetics (5 of 120) (Fig. 1). Eleven of 19 men and 5 of 41 women were affected in type I diabetes, whereas in type II diabetes 5 of 50 men and 4 of 40 women showed evidence of CTS. In the non-diabetic patients, CTS (5 cases) was found in women. In diabetics with CTS, both hands were affected in 16 of 25 patients. The prevalence of CTS increased with the increasing age of the patient only in type I diabetes (p < .05) (Table 1) and did not correlate with the duration of the diabetes. A diabetic peripheral neuropathy was found in 11 of 16 type I diabetic patients who had CTS. Conversely, 11 of 44 type I diabetics without evidence of CTS had a diabetic peripheral neuropathy (p < .01). Also, in type I diabetes, a microangiopathy was observed in 14 of 16 patients who had median nerve entrapment at the carpal tunnel (p < .01). The occurrence of the combination of CTS and diabetic neuropathy was noted in only one third of type II diabetics with evidence of median nerve compression syndrome. Of the patients with type II diabetes without carpal tunnel syndrome, one sixth (8 of 51) showed evidence of diabetic polyneuropathy.

Flexor tenosynovitis was observed in 14 of 60 patients with type I diabetes and in 10 of 60 patients with type II diabetes (Fig. 1). This condition was only diagnosed in 3 of 120 controls (Fig. 1). Women were almost equally affected in the two types of diabetes (10 out of 41 in type I and 9 of 40 in type II diabetics). Men were more frequently affected in case of type I diabetes (4 of 19) than in case of type II diabetes (1 of 20). Concerning diabetic patients with flexor tenosynovitis, palpable crepitus in the palm was observed in 14 of 24, true trigger finger in 10 of 24. More than one digit was affected in the same hand in two-thirds of the diabetic patients (16 of 24). In the 14 patients who had both flexor tenosynovitis and type I diabetes, CTS was observed in 8 patients (p < .01), microangiopathy in 11 patients (p < .01) and a diabetic peripheral neuropathy in 10 patients (p < .01).

**Discussion**

Our study clearly demonstrates a higher incidence of hand abnormalities, such as Dupuytren’s disease, LJM, CTS, and flexor tenosynovitis in the diabetic than in the non-diabetic population (Fig. 1). The prevalence of Dupuytren’s disease in the diabetic patients studied here (32%) does not differ signifi-
cantly from that reported recently by Noble (42%). The surprising range (2–63%) found previously in the literature may be explained by the fact that the most common manifestation of Dupuytren’s disease in diabetics is mild disease without contracture. Thus, mild expressions of Dupuytren’s disease are missed by surgical case studies and by studies performed by physicians not accustomed to seeing patients with Dupuytren’s disease. Our findings confirm the atypical pattern of Dupuytren’s disease in diabetics: predominant involvement of the ring and long finger rays that rarely leads to severe contractures. We have also noted that the small finger, while less frequently involved, seems to be more severely affected. We did not find any knuckle pads in our diabetic population. We confirm the previously observed fact that contractures are more common in diabetic men than in women. In our series, the incidence of Dupuytren’s disease increased with increasing duration of diabetes, confirming observations in the literature. The most significant increase occurred after 15 years of having had a history of diabetes, both type I and type II. This study confirms the frequent association between Dupuytren’s disease and LJM in diabetes but only for type I diabetes. The combination of diabetes, Dupuytren’s disease, and LJM is important, considering the poor results of the surgical treatment of the digital contractures in patients diagnosed as having LJM.

In our study of diabetic patients, CTS occurred more frequently than previously noted (12% by Fraser). In two large studies of carpal tunnel syndrome, the prevalence of diabetic patients varied between 77% and 16%. In the diabetic carpal tunnel disease analyzed here, as opposed to the idiopathic form, men were more frequently affected than women, especially in type I diabetes. A significant association between CTS and diabetic peripheral neuropathy in type I diabetes was found in this study. Our results can be explained by both the age-related incidence of carpal tunnel syndrome found here and also the increasing prevalence of diabetic neuropathy with time seen in diabetics. When CTS is suspected in the diabetic an electrodiagnostic study is recommended, not only at the wrist but in all four limbs. If a diabetic peripheral neuropathy is found surgery at the carpal tunnel site will be less effective because of the polyneuropathy.

In the diabetic patients studied here, flexor tenosynovitis typically affected more than one digit in the same hand. The high incidence of flexor tenosynovitis seen here has been suggested previously.

Despite several differences between types I and II diabetes and their resultant hand changes, the coexistence of these problems suggests a common etiology. The connective tissue proliferation inherent in Dupuytren’s disease, LJM, and flexor tenosynovitis also seem to be a consequence of the abnormal accumulation of stable end products of non-enzymatic collagen glycosylation, which are thought responsible for increased cross-linking, packing, and stiffening of collagen. However, this phenomenon may also require a constitutional predilection and a microangiopathy.

Diabetic neuropathy is probably a significant factor in the development of carpal tunnel syndrome in the diabetic population. While the median nerve is sensitive to entrapment within the canal tunnel, a significant correlation between the occurrence of CTS and the presence of flexor tenosynovitis and microangiopathy suggests that proliferation of connective tissue and microvascular insufficiency may play a role in the pathogenesis of nerve entrapment.

References


