ULTRASONOGRAPHIC FEATURES OF DIABETIC CHEIROARTHRPATHY

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SUMMARY

Ultrasonography was used to measure flexor tendon sheath thickness in 14 insulin-dependent (IDDM) diabetics with diabetic cheiroarthropathy (DCA) and compared to 17 IDDM patients without DCA along with 10 healthy volunteers. Assessment was also made of the presence of systemic diabetic microvascular disease complications. A blinded visual 'eyeball' report on the ultrasound scans by a radiologist found hypoechoic thickening of the flexor tendon sheaths in 12 of the 14 patients with DCA, three of the 17 unaffected diabetics and two of the healthy volunteers (Fisher's exact, \( P < 0.001 \)). However, further quantification of tendon sheath thickness separated patients with DCA from others. In all patients with DCA, tendon sheath thickness was \( \geq 1 \text{ mm} \) (median 1.8 mm, range 1.0–2.3 mm) and \( < 1 \text{ mm} \) in the other two groups (medians 0.6 and 0.5 mm, range 0.3–1.0 mm) (Kruskal-Wallis, \( P < 0.001 \)). All patients with DCA had evidence of systemic microvascular disease complications, particularly proliferative retinopathy (82%). It appears that flexor tendon sheath thickening in the hand is an integral part of the pathology in DCA and is easily demonstrated by ultrasound. It is closely associated with overt diabetic microvascular disease complications.

KEY WORDS: Diabetic cheiroarthropathy, Tendon sheath, Insulin-dependent diabetes, Diabetic microvascular complications.

A LARGE proportion of morbidity and mortality due to diabetes mellitus is due to chronic microvascular complications such as retinopathy, nephropathy and neuropathy. An under-recognized complication is diabetic cheiroarthropathy (DCA). DCA is a syndrome characterized by painless limitation of the finger joints and thick, tight, waxy skin. The cardinal feature is the 'prayer sign'. Studies largely agree on a prevalence figure of 30–35% and it may be related to other microvascular disease complications [1].

The aims of this study were to assess (1) the flexor tendon and tendon sheath using ultrasound and (2) the presence of systemic diabetic microvascular disease complications in patients with DCA.

SUBJECTS AND METHODS

The diagnosis of DCA was made independently by two clinicians using the 'prayer sign', characterized by incomplete approximation of one or more of the digits when the patient attempts apposition of the palmar surfaces of the proximal and distal interphalangeal joints with palms pressed together and the fingers fanned. Particular attention was also given to the skin of the hands.

Fourteen insulin-dependent diabetes mellitus (IDDM) patients with DCA were identified from the diabetic clinic at Southend Hospital (male = 8, female = 6, mean age 43.4 yr, age range 27–54 yr). A group of 17 insulin-dependent diabetics (male = 9, female = 8, mean age 41.2 yr, age range 27–59 yr) with no evidence of DCA were included in the study, along with 10 healthy volunteers with no evidence of diabetes mellitus. The diabetics without DCA were matched as closely as possible for age (± 5 yr), sex and duration of diabetes (DCA group: mean duration = 26 yr and range 10–40 yr; non-DCA group: mean duration = 25.4 yr and range 10–43 yr). Details of the patients' insulin dose and diabetic control, as measured by fructosamine levels in the preceding 2 yr, were obtained from the diabetic clinic records.

High-frequency ultrasonography using a 7.5 MHz, and subsequently a 10 MHz, probe was used to examine the volar aspects of both hands. The examination was performed in the transverse plane with special attention paid to the evaluation of all flexor tendons and tendon sheaths in the hand, but only the tendon sheaths with the greatest thickness were used for measurements used in the analysis. First, all the ultrasound scans underwent a simple blinded visual 'eyeball' report by a consultant radiologist (ABT) and the measurements of the tendon sheaths performed using the facilities on the ultrasound scanner.

Retinopathy was assessed by fundoscopy and screening retinal photography performed in the diabetic clinic. Peripheral neuropathy was assessed by clinical examination of the ankle jerks and vibration sense, and nephropathy by the presence of persistent proteinuria and/or impaired renal function (creatinine > 125 \( \mu \text{mol/l} \)). Clinical examination was performed for Dupuytren's contracture and any underlying joint disease, and these subjects were not included in the study if present. In addition, flexor tenosynovitis (palpable crepitus) and symptoms and signs of carpal tunnel syndrome (Tinel's sign) were sought.

RESULTS

Ultrasound scans from all patients with DCA showed a hypoechoic thickening of the flexor tendon sheaths. Quantitative analysis of the tendon sheath thickness from DCA patients revealed measurements \( \geq 1 \text{ mm} \) (median 1.8 mm, range 1.0–2.3 mm).
(Fig. 1), compared to diabetic patients without DCA and healthy controls (≤1 mm, medians 0.6 and 0.5 mm, respectively, range 0.3–1 mm) (Fig. 2). Figure 3 shows the medians and 95% confidence intervals of tendon sheath measurements in the three groups (Kruskal–Wallis test, $P < 0.001$).

The ultrasound scans also underwent blinded visual report by the radiologist and tendon sheath thickening was found in 12 of the 14 patients with DCA, and in only three of the unaffected diabetics and two of the healthy volunteers ($P < 0.001$, Fisher’s exact test). No difference in the tendon itself was observed between the groups. Specifically, there was no evidence of tendon rupture or any other soft-tissue abnormalities.

All the 14 patients with DCA had systemic microvascular disease, particularly proliferative
Figure 3.—Graphical representation of measurements of tendon sheath thickness in the DCA group, healthy volunteers (non-DM) and diabetic controls (non-DCA). The median and 95% CI are shown.

Retinopathy in 13 out of 14 patients (82%), neuropathy in five patients and nephropathy in five patients (Table I). On the other hand, in the IDDM group without DCA, four had evidence of microvascular disease and only one patient had proliferative retinopathy (Fisher's exact test, \( P < 0.001 \)) (Table I). No significant differences were observed between groups for carpal tunnel syndrome, flexor tenosynovitis, and diabetic control or insulin requirements over the preceding 2 yr (Table I).

### Discussion

Using ultrasound, the thickness of the flexor tendon sheath appears to be significantly more profound in diabetics with DCA when compared with IDDM patients without DCA and non-diabetic controls. This suggests that flexor tendon sheath thickening is an integral part of DCA. The pathogenesis of DCA is still not known, but increased skin thickness has been demonstrated both histologically [2] and using ultrasound [3]. Glycosylation of collagen is thought to be important in the pathogenesis of increased skin thickness in DCA [4].

Real-time high-frequency ultrasound has not been used to evaluate the tendons and tendon sheath in DCA. It proved to be an easy and quick investigation to perform on the diabetic hand, and effectively demonstrated tendon sheath thickening, and the resolution was significantly improved using the 10 MHz probe.

A significant association was found between DCA and systemic microvascular complications, particularly proliferative retinopathy. The difference in the prevalence of microvascular disease complications in the two groups may be attributed to genetic differences.
It is also important to point out that diabetic control was documented in our patients only in the 2 yr prior to the study. The degree of diabetic control before this period could not be established and may in fact have influenced the disease progress in the DCA group. However, it appears abundantly clear that DCA shares a common pathogenetic link with systemic microvascular complications. Further work is needed to determine whether hand ultrasound could provide an easy, early test for microvascular disease which may complement conventional screening methods, e.g. retinal screening.

In conclusion, flexor tendon sheath thickening appears to be a major component of DCA, and is easily demonstratable on ultrasound. DCA is closely associated with microvascular disease.

REFERENCES