ALGODYSTROPHY AND ITS ASSOCIATION WITH DUPUYTREN'S DISEASE

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Seventy-two patients were examined 9 weeks after sustaining a Colles' fracture of the wrist for evidence of algodystrophy. They were examined 18 months later for evidence of Dupuytren's disease to determine the incidence of the association between the two conditions. Forty-one per cent of all patients had evidence of Dupuytren's disease at 18 months following Colles' fracture. Sixty-seven per cent of patients with algodystrophy had evidence of Dupuytren's disease compared with 19% of patients who showed no features of algodystrophy.

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The aetiology of Dupuytren's disease of the palmar fascia remains poorly understood. Although several associated factors are well recognized such as family history (Ling, 1963) diabetes (Noble et al., 1984), epilepsy (Critchley et al., 1976), alcoholism (Pojer et al., 1972), hyperlipidaemia (Sanderson et al., 1992) and more recently smoking (Burge et al., 1997), the mechanisms by which these conditions contribute to the disease process are ill-defined.

The role of trauma in the pathogenesis of Dupuytren's disease has been debated since the condition was first described yet there are no controlled studies linking acute trauma with the development of Dupuytren's disease (Liss and Stock, 1996). The reported incidence of Dupuytren's disease following fractures of the distal radius varies from 0.2% to 11% in prospective series (Bacorn and Kurtze, 1953; Stewart et al., 1985). Dupuytren's contracture has also been described following post-traumatic algodystrophy as part of the dystrophic soft tissue changes that are seen (Doury et al., 1981; Plewes, 1956). Algodystrophy has also been linked with hyperlipidaemia (Amor et al., 1980; Pinals and Jabbs, 1972).

The object of this study was to determine the frequency with which algodystrophy of the hand was associated with the development of Dupuytren's disease and to determine whether blood lipid status, blood sugar and liver function tests were related to the aetiology of algodystrophy.

PATIENTS AND METHODS

Diagnosis of algodystrophy

A consecutive series of 100 patients with fractures of the distal radius was reviewed at 9 weeks after fracture for features of algodystrophy. Four features of the disorder were specifically examined using previously described techniques: vasomotor instability using a questionnaire; finger tenderness using a dolorimeter (Atkins et al., 1990); finger stiffness using a goniometer (Field et al., 1994); and finger swelling using an arthrocircameter (Willkens et al., 1973). The patients were divided into three groups on the basis of this assessment. The algodystrophy group had abnormal values for all four tests. A borderline group had some but not all features of algodystrophy and a normal group had no features of algodystrophy.

Diagnosis of Dupuytren's disease

All patients were screened at 9 weeks after fracture and 72 of these patients were recalled at a mean of 18.3 months (range, 16–21 months). Patients were questioned about a history of hyperlipidaemia, diabetes mellitus, epilepsy and alcohol-related liver disease or a family history of Dupuytren's contracture and then examined for features of Dupuytren's disease. Any palpable thickening of the palmar fascia with skin tethering was regarded as diagnostic and the presence of palmar and finger contractures was noted.

Serum biochemistry

The blood lipid status was determined by measuring serum triglycerides and cholesterol; the presence of diabetes mellitus was determined by the fasting glucose and fructosamine level; and the degree of alcohol related liver damage was assessed by measurement of gamma glutyl transferase (gamma-GT).

Statistical analysis

For continuous data the variance of the three groups was determined using one way analysis of variance or the Kruskal–Wallis test. If the variance was significant ($P<0.05$) then paired groups were compared using Student's *t*-test or the Mann-Whitney U test. For categorical data 2x*k* tables with Yates' correction were used for comparison of proportions. If the results were significant ($P<0.05$) comparisons between individual groups were made using the x^2 or Fisher's exact tests.

RESULTS

Incidence of algodystrophy

The mean age of the study group of 72 patients was 70 years (range, 50–88 years). There were three men and 69

Table 1—Dupuytren's disease and algodystrophy following distal radius fracture

	No. of patients	*Age (years) Mean (95% CI)*	*Follow-up (months) Mean (95% CI)*	*No. of patients with Dupuytren's disease*
Algodystrophy	18	69.7 (64.6–74.8)	18.1 (17.4–18.9)	12 (67%)
Borderline	23	69.4 (65.4–73.4)	17.9 (17.6–18.4)	12 (52%)
Normal	31	70.7 (67.2–74.2)	18.6 (17.9–19.3)	6 (19%)

women. At 9 weeks following wrist fracture 18 (25%) patients had all four features of algodystrophy, 23 (32%) patients had some features and 31 (43%) patients had none of these features (Table 1). There was no significant age difference between the three groups.

Incidence of Dupuytren's disease

The mean age of patients with Dupuytren's disease was 70.2 years (range, 50–84 years) and was not significantly different from the age of 69.9 years (range, 51–88 years) in the unaffected group. Dupuytren's disease was present in 30 patients in this group of 72 Colles' fracture.

Dupuytren's and algodystrophy

There was a significant difference in the incidence of Dupuytren's disease between these groups. The algodystrophy group and had an increased incidence of Dupuytren's disease compared with the normal group ($P = 0.03$) as did the borderline group compared with the normal group ($P = 0.03$). There was no significant difference between algodystrophy and borderline groups. The distribution of disease affecting the palm and fingers in the different groups is shown in Table 2.

None of the patients with algodystrophy had any other risk factors for Dupuytren's disease. Two patients, one in the borderline group and one in the normal group, had evidence of pre-existing Dupuytren's disease. Both patients had other risk factors (family history and hypertriglyceridaemia). They did not report any progression of their disease.

One patient in the borderline group had a positive family history of Dupuytren's disease and developed Dupuytren's nodules following fracture.

Table 2—The distribution of Dupuytrens' disease in algodystrophy

	Palmar	*Finger*	*Both*	*Total/Number in group*
Algodystrophy	9	1	2	12/18
Borderline	12	0	0	12/23
Normal	5	1	0	6/31

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There was no association between triglyceride, cholesterol, glucose, fructosamine or gamma GT levels and the presence of algodystrophy or the presence of Dupuytren's disease (Tables 3 and 4).

There was similarly no significant difference in the biochemical indices between patients with and without Dupuytren's disease in either the algodystrophy, borderline or normal groups.

DISCUSSION

Although the aetiology of both Dupuytren's disease and algodystrophy remain uncertain an association between the two conditions has been described. Plewes (1956) reported 37 cases of Sudek's atrophy of the hand and noted palpable thickening of the palmar fascia in all but one case. The frequency with which Dupuytren's disease occurs following algodystrophy has not been previously determined. The reported incidence of Dupuytren's disease following fractures of the distal radius varies from 0.2% to 11% (Bacorn and Kurtze, 1953; Stewart et al., 1985). In our group of 72 patients at a mean of 18.3 months following Colles' fracture, examination revealed

Table 3—Serum fasting biochemistry and algodystrophy

	Triglycerides Mmol/l Mean (95% CI)	*Glucose Mmol/l Mean (95% CI)*	*Fructosamine Mmol/l Mean (95% CI)*	*Gamma GT Iu/l Mean (95% CI)*	*Cholesterol Mmol/l Mean (95% CI)*
Algodystrophy	1.3 (1.07–1.53)	4.9 (4.31–5.59)	1.9 (1.81–2.08)	30.8 (22.3–39.2)	6.1 (5.41–6.85)
Borderline	1.6 (1.37–1.87)	5.5 (5.10–5.92)	2.0 (1.86–2.16)	21.3 (17.8–24.7)	6.3 (5.54–7.01)
Normal	1.6 (1.39–1.90)	5.1 (4.85–5.28)	2.1 (1.88–2.29)	30.8 (20.7–41.0)	6.4 (5.72–7.05)
P-value	NS	NS	NS	NS	NS

Table 4—Serum fasting biochemistry and Dupuytren's disease

	Triglycerides Mmol/l Mean (95% CI)	*Glucose Mmol/l Mean (95% CI)*	*Fructosamine Mmol/l Mean (95% CI)*	*Gamma GT Iu/l Mean (95% CI)*	*Cholesterol Mmol/l Mean (95% CI)*
Dupuytren's	1.5 (1.33–1.72)	5.1 (4.71–5.51)	2.1 (1.85–2.27)	25.5 (20.4–30.5)	6.2 (5.56–6.86)
No Dupuytren's	1.6 (1.36–1.78)	5.2 (4.97–5.48)	2.0 (1.9–2.11)	29.4 (21.7–37.1)	6.3 (5.83–6.85)
P-value	NS	NS	NS	NS	NS

that 40% had evidence of thickening of the palmar fascia. This is a much larger incidence than has previously been reported. Patients in the algodystrophy group and the borderline group had an increased incidence of Dupuytren's disease compared to patients with no features of algodystrophy. The incidence of Dupuytren's disease in patients with all four features of algodystrophy was 67%.

We found no association between Dupuytren's disease and diabetes mellitus, epilepsy or excess alcohol consumption. Previous authors have found evidence that hyperlipidaemia is associated with algodystrophy (Amor et al., 1980). This was not the case in this study and our findings are similar to those reported by Eulry et al. (1992). Previous studies that have described Dupuytren's disease following Colles' fractures (Cooney et al., 1980; Smaill, 1965) have not specifically sought to identify changes in the palmar fascia or to identify algodystrophy, which occurs in up to 25% of Colles' fractures when identified using our techniques (Atkins et al., 1990; Field et al., 1994). One prospective series reported the presence of early Dupuytren's disease in 11% of patients at 6 months after Colles' fracture in the form of nodules or palmar bands which were non progressive (Stewart et al., 1985). These reports have not distinguished palmar and finger involvement. Our figure of 40% at 18 months is high and is explained partly by our inclusion of patients with palpable palmar thickening.

It is difficult to propose a mechanism that might link algodystrophy with Dupuytren's disease as the pathogenesis of both conditions is essentially unknown. We have looked at recognized risk factors but found that these do not explain the frequency or association between algodystrophy and Dupuytren's disease in this series of patients. There is little evidence in the literature to support a link between trauma and Dupuytren's disease (Kelly et al., 1992). In a recent review Liss and Stock (1996) found no controlled studies of acute trauma and Dupuytren's contracture but did find some support for an association between vibration exposure and Dupuytren's disease. Trauma is also the commonest precipitator of algodystrophy and both conditions result in varying degrees of soft tissue contracture.

Much of our understanding of Dupuytren's disease is based on histological and immunohistochemical studies of end stage disease and the mechanisms by which this process is initiated are not fully understood. Doury et al. (1981) found only a few incomplete reports of the pathological examination of periarticular tissues in algodystrophy. Biopsy of fascial tissue from a palmar contracture in a patient with shoulder-hand syndrome was indistinguishable microscopically from the histology of classical Dupuytren's disease (Steinbrocker and Argyros, 1958). However, it may be that until further studies are performed any link between algodystrophy and Dupuytren's disease will be difficult to clarify.

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