

Journal of Hand Surgery (British and European Volume)

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J Hand Surg [Br] 1992 17: 71

DOI: 10.1016/0266-7681(92)90015-T

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THE ASSOCIATION BETWEEN ALCOHOL, HEPATIC PATHOLOGY AND DUPUYTREN'S DISEASE

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We have looked at the incidence of Dupuytren's disease in alcoholics, those with non-alcoholic liver disease and a control population. Both alcoholic patients and those with non-alcohol related liver disease had a higher rate (28% and 22% respectively) than the controls (8%), but this did not quite reach statistical significance ($p > 0.05$). In addition we found no Dupuytren's disease in 50 Egyptian patients with bilharzia and no consistent biochemical abnormalities in 134 patients with significant Dupuytren's disease.

We conclude that alcoholics probably do have a higher rate of Dupuytren's disease and that this effect is largely due to the liver disease caused by alcohol abuse, but that the genetic factors are of greater aetiological importance.

Journal of Hand Surgery (British Volume, 1992) 17B: 71-74

Dupuytren (1834) was probably the first to imply an association between alcohol and the disease that now bears his name, whilst describing the hand affliction of his coachman. This was confirmed first by Wolfe et al. (1956) and later by Nazari (1966), but more recently Lamb (1981) has suggested that the associations between Dupuytren's disease and both alcoholism and liver disease were "probably just co-existence of this common condition and others equally frequent."

The exact role of alcohol in the pathogenesis of Dupuytren's disease is unclear. Some authors (Attali et al., 1987) have concluded that it is the alcohol that causes the disease, whilst others (Houghton et al., 1983) suspected that it is the hepatic damage caused by the alcohol that is important.

For this reason, we felt a controlled epidemiological study of alcoholics, those with non-alcoholic liver disease and a control group, should be performed to try and clarify the situation.

Patients and methods

The patients in each of the four groups (A to D) were examined and the distribution, severity and incidence of their disease recorded as our unit has previously described (Noble et al., 1984). This includes noting the presence of palmar or digital nodules, skin tethering, pretendinous bands or digital contracture as well as the site of the disease.

Group A

The hands of 100 members of Alcoholics Anonymous in Manchester were examined and recorded.

Group B

82 patients attending a hepatic clinic at Hope Hospital were similarly reviewed. None of these patients was

thought by the physician in charge of the clinic to have alcohol-related disease. This group consisted mainly of post-hepatic and primary biliary cirrhotics.

Group C

50 patients suffering from bilharzia were examined in Cairo in a similar manner.

Group D

A control group of 100 patients from a busy fracture clinic were age- and sex-matched to the populations of Groups A and B.

In addition, blood was taken from 134 patients attending a hand clinic in the same hospital with a proven diagnosis of Dupuytren's disease. This was analysed of serum enzymes, blood glucose, cholesterol, triglycerides and bilirubin and compared with the random analyses of 43 000 patients. The results of these were expressed as a percentage with values above the normal range.

The results were all analysed using the χ^2 test.

Results

Incidence (Table 1)

No evidence of Dupuytren's disease could be identified in any of the 50 patients with bilharzia (Group C).

The rate of Dupuytren's disease was 28% in alcoholic patients (Group A), 22% in those with non-alcoholic liver disease (Group B) and 8% in the control population (Group D). No statistical significance ($p > 0.05$) could be found between groups A and B, but was almost achieved between these two groups individually and the control population (Group D).

Distribution (Table 2)

All three groups (A, B and D) with Dupuytren's disease had a tendency for this to occur towards the ulnar border of the hand and, in particular, the ring finger. There was no significant difference in the site of the disease between any of these groups.

Severity (Table 3)

The mild forms of Dupuytren's, namely nodules, appeared to be more common in the alcoholic than the liver disease patients ($p < 0.01$). No other differences between groups A, B and D could be identified.

Biochemistry (Table 4)

All biochemical parameters showed little variation from a random sample, although this latter group obviously contained a mixed group of patients with multiple diagnoses.

Discussion

The aetiology of Dupuytren's disease remains unsolved and is undoubtedly multifactorial. Indeed some authors have found that Dupuytren's disease has an increased incidence in some chronic diseases such as diabetes mellitus (Noble et al., 1984) and hypertension (Larkin et

Table 1—Incidence of Dupuytren's disease (%)

Age	Alcoholics (100 patients)	Hepatic disease (82 patients)	Controls (100 patients)
<30	8	0	0
30-39	21	0	6
40-49	28	12	7
50-59	37	22	6
60-69	75	56	25
>70	60	33	20
TOTALS	28	22	7

Table 2—Distribution of Dupuytren's disease

Ray	Alcoholics (28 patients)	Hepatic disease (18 patients)	Controls (8 patients)
1st	15 (7)	11 (6)	13 (4)
2nd	0	4 (2)	6 (2)
3rd	4 (3)	8 (4)	16 (5)
4th	65 (32)	42 (22)	52 (17)
5th	16 (8)	35 (18)	13 (4)
Number of contractures	50	52	32

Table 3—Severity of Dupuytren's disease (%)

Disease type	Alcoholics (28 patients)	Hepatic disease (18 patients)	Controls (8 patients)
Nodule	18 (17)	12 (6)	16 (5)
Tethering	35 (10)	42 (22)	31 (10)
Pretendinous band	20 (12)	34 (18)	22 (7)
Contracture	27 (11)	12 (6)	31 (10)
Number of contractures	50	52	32

Table 4—Biochemical profiles

Test	% Abnormality	
	Dupuytren's disease (134)	Random samples (43,000)
Bilirubin	4	9
Alk. Phos.	16	28
A.L.T.	23.5	33
A.S.T.	8	13
L.D.H.	28	31
γ -G.T.	32	44
Random glucose	14	11
Triglycerides*	40	48
Cholesterol*	27	42

* 95 Dupuytren's versus 946 random samples.

al., 1986), whilst a reduced incidence has been found in others, for example rheumatoid arthritis (Arafa et al., 1984).

The incidence of Dupuytren's disease has been noted to be low in non-Europeans (Pojer 1972), and our finding of none in a middle-Eastern country, even in patients with a chronic liver disease, underlines the importance of the genetic factors in the aetiology of this condition (Ling, 1963).

Dupuytren's disease has long been associated with alcohol abuse. The incidence in this study of 28% compares well with others who have looked at this phenomenon (Attali et al., 1987; Houghton et al., 1983; Pojer et al., 1972).

Nazari (1966) found a higher incidence of Dupuytren's disease in alcoholics (55%) than in patients with non-alcohol related liver disease (35%). However, Houghton et al. (1983), found a similar pattern to ours: 25% in alcoholics and 28% in non-alcoholic liver disease.

Wolfe et al. (1956), looking at alcoholics with and without liver disease, found a higher incidence of Dupuytren's disease in those with alcoholic cirrhosis (47%) than in alcoholics without clinical or biochemical evidence of hepatic disease (29%). However Attali et al. (1987) could not confirm these findings and found no

significant differences between alcoholics with and without liver disease (32.5% and 28% respectively), though they did notice a significantly lower incidence (6%) in those with non-alcoholic hepatic disease.

The severity of the disease appears to be unrelated to alcohol consumption, as confirmed by Nazari (1966), although we have found significantly more mild disease in alcoholics than in those with liver disease.

Attention has also been focussed on the biochemical factors that may be responsible for Dupuytren's disease. Pojer et al. (1972) found various abnormalities, including raised γ -GT and alkaline phosphatase, which they felt may be of importance. The percentage with abnormalities was of a similar order to those found in our series, so we doubt if they are of any aetiological or pathological significance as they closely mirror the random samples we analysed.

What, then, can we make of this apparently rather confusing picture from the literature (Table 5)? All the authors appeared to have used a similar method of analysis of their Dupuytren's disease, noting its severity on a three or four-point scale. This would not appear to account for the different incidences reported.

The high incidences reported by Wolfe (1956) are probably related to the fact that all of his alcoholics were

Table 5—Review of previous studies

Author	Year	Controls (%)	Alcoholics (%)	Alcoholics with liver disease (%)	Non-alcoholic liver disease (%)
Wolfe	1956	8	27	47	22*
Nazari	1966	—	—	55	35
Pojer	1972	—	25	—	—
Houghton	1983	—	—	25	28
Attali	1987	12	28	32.5	6
Noble	1991	8	28	—	22

*2 out of 9 patients.

hospital in-patients, often with complications. Nazari (1966), who also found a very high incidence in those with alcoholic liver disease, included six cases with only occasional alcohol consumption, and indeed his figures, although apparently different, fail to reach statistical significance. The very low incidence of Dupuytren's found by Attali et al. (1987) in those with non-alcoholic liver disease does not appear to be in keeping with several other authors' findings, including our own.

We believe it is reasonable to conclude from our findings (although they just fail to reach statistical significance) and those of other authors that Dupuytren's disease is more common in alcoholics. The causal factor appears to be mainly the effect of the alcohol on the liver rather than a direct effect of the alcohol, as the incidence in alcoholics and those with non-alcoholic liver disease appears to be similar.

However, in implicating these factors in the aetiology, it is important to remember the very important genetic predisposition that we have highlighted by finding no cases in Egypt.

Finally, the biochemical changes seen in Dupuytren's disease appear to be of little, if any, aetiological significance.

Acknowledgements

The authors would like to thank Dr. D. Marsh, Consultant Physician at Hope Hospital, for permission to examine his patients and Mrs. J. R. McMurray, Top Grade Biochemist at Hope Hospital for her help.

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Accepted: 15 February 1991

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