Shoulder capsulitis in type I and II diabetic patients: association with diabetic complications and related diseases

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Abstract

***Objective*—To examine the association between shoulder capsulitis and chronic diabetic complications and diseases closely related to diabetes.**

***Methods*—A cross sectional study in 291 type I [mean (SD) age 33.2 (9.9) years] and 134 type II [61.1 (12.4) years] diabetic patients. The presence of shoulder capsulitis, Dupuytren disease, and limited joint mobility was sought. The patients were assessed for background and proliferative retinopathy, nephropathy, autonomic neuropathy, and peripheral symmetrical somatic polyneuropathy. Diseases closely related to diabetes (hypertension, history of myocardial infarction, coronary heart disease, and peripheral vascular disease) were also recorded.**

***Results*—Prevalence of shoulder capsulitis was 10.3% in type I and 22.4% in type II diabetic subjects. Shoulder capsulitis was associated with the age in types I ($P < 0.01$) and II ($P < 0.05$) diabetic patients, and with the duration of diabetes in type I patients ($P < 0.01$). Odds ratios for autonomic neuropathy in type I and type II diabetic subjects with shoulder capsulitis were 4.1 (95% confidence interval, 1.6 to 10.9) and 2.7 (95% CI, 1.1 to 7.0), respectively, after controlling for age and duration of diabetes. Odds ratio for history of myocardial infarction in type I diabetic subjects with shoulder capsulitis was 13.7 (95% CI, 1.3 to 139.5) after controlling for age, duration of diabetes, hypertension, and smoking habits. Other associations between shoulder capsulitis and diabetic complications, related diseases, and other hand abnormalities were fully explained by age and the duration of diabetes.**

***Conclusions*—Shoulder capsulitis is common in type I and type II diabetic patients. It is associated with age in type I and II diabetic patients and with the duration of diabetes in type I patients. It is associated with autonomic neuropathy in type I and II diabetic patients and with history of myocardial infarction in type I diabetic patients, independently of time related variables.**

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In 1872 Duplay described a condition, "péri-arthrite scapulo-humérale", which differed from arthritis in its symptoms and clinical course.[1] The true nature of this condition has been discussed ever since. Duplay himself believed that a subacromial bursitis was the basic cause of the pain and dysfunction. By 1916 Klapp and Riedel believed that the joint capsule was affected, and later, in 1931, Payr tried to reduce distension of the retracted capsular tissue by intra-articular injections (citation by Lundberg[2]). In 1934 Codman showed that stiffness and pain in the shoulder could occur without noticeable exogenous influences and he termed the condition "frozen shoulder", which has later been used as a synonym for humeroscapular periarthritis and adhesive capsulitis of shoulder (shoulder capsulitis).[3] Codman believed that shoulder capsulitis was caused by tendinitis in the short rotators, but more recently the thickening of the joint capsule and its adherence to the head of the humerus resulting in marked reduction in the volume of the glenohumeral joint have been considered as the basic pathological change in shoulder capsulitis.[4]

The aetiology of shoulder capsulitis is not clearly understood. Attempts have been made to relate it to various circumstances such as inactivity, strain, and pre-existing shoulder disorders, for example trauma.[5] Several conditions have been associated with shoulder capsulitis; these include cervical spondylosis,[6] coronary heart disease,[7 8] hemiplegia,[9] pulmonary tuberculosis,[10] bronchial carcinoma,[11] hyperthyroidism,[12] cerebral tumour, and epilepsy.[13] The relation between diabetes mellitus and shoulder capsulitis has been shown in few previous studies. Bridgman reviewed the medical records of 800 diabetic subjects and found evidence of periarthritis in 10.8%, compared with 2.3% in a control group of 600 non-diabetic subjects.[14] Pal *et al* found shoulder capsulitis in 20.4% of insulin-dependent and in 18.3% of non-insulin-dependent diabetes patients and in 5.3% of normal subjects.[15] In a study of 824 type II diabetic and 320 control subjects shoulder capsulitis was observed in 31.8% and 10.3% of subjects, respectively.[16]

Bilateral involvement of shoulder capsulitis was more common in diabetic (10%) than in the control subjects (3%).[16] Diabetic shoulder capsulitis seems to appear at a younger age, may be less painful, responds less well to treatment, and lasts longer than non-diabetic shoulder capsulitis.[17–19] A high frequency of other hand syndromes, such as limited joint

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mobility, has been found among diabetic patients with shoulder capsulitis.[18 19] The association between limited joint mobility and microvascular complications of diabetes has been well documented, but there are also two studies showing an association between shoulder capsulitis and retinopathy.[19-22] However, no association between shoulder capsulitis and diabetic neuropathy has been found.[14]

The purpose of this study was to investigate the prevalence of shoulder capsulitis and its association to the diabetic complications in type I and II diabetic subjects.

Methods

PATIENTS

We evaluated 425 Finnish diabetic patients (200 men and 225 women) attending the departments of medicine in Turku University Central Hospital (n = 111) and Päijät-Häme Central Hospital (n = 57) in Lahti, in a private diabetes outpatient clinic in Turku (n = 217), and in the municipal health centre of Turku (n = 40). Of these, 291 had type I and 134 type II diabetes according to the classification of the National Diabetes Data Group.[23] Patients were considered to have type II diabetes if there was no previous history of ketoacidosis and they had not suffered from severe weight loss. All the insulin treated patients in the type II diabetes group were over 50 years at the time of the diagnosis of diabetes, or their fasting plasma C peptide value was over 0.3 nmol litre^{-1}, or the stimulated plasma C peptide value was over 0.7 nmol litre^{-1}.

CLINICAL EXAMINATION

The criteria for shoulder capsulitis, previously described by Pal *et al*, were shoulder pain for at least one month, an inability to lie on the affected shoulder, and restricted active and passive shoulder joint movements in at least three planes.[15] A reliably documented record of the patient having had these symptoms and signs was accepted as evidence of a shoulder capsulitis in the past. The diagnosis of Dupuytren disease was made by the method earlier used by Noble *et al.*[24] Limited joint mobility was assessed by the method of Rosenbloom *et al.*[20]

All subjects gave a detailed history. Special emphasis was placed on age and the duration of diabetes. Weight and height were measured and the body mass index (BMI) was calculated from the formula BMI = weight (kg)/[height (m)]^2. The occupation of subjects (manual or intellectual work) was recorded, as was the history of injuries, operations, or infections in the upper extremities.

Retinal examination was performed by experienced ophthalmologists, with fundoscopy after dilatation of the pupils with a mydriatic agent. Retinopathy was classified as background or proliferative. Background retinopathy was characterised by microaneurysms, hard exudates, cotton wool or soft exudates, and retinal haemorrhages. Proliferative retinopathy was characterised by neovascularisation, fibrous proliferations, vitreous haemorrhage obscuring the retina, phthisis bulbi, or enucleation secondary to a complication of diabetic retinopathy.

The symptoms of somatic peripheral symmetrical polyneuropathy included pain, paraesthesiae, and muscle weakness. Knee and ankle jerks and vibration sense over the lateral malleoli and over the styloid processes of radii (using a 256 Hz tuning fork) were tested. The patients were considered to have peripheral symmetrical somatic polyneuropathy if they had typical symptoms and impaired vibration sense, or if both ankle jerks were absent. The diagnosis of autonomic neuropathy was based on structured questions regarding anhidrosis or hyperhidrosis of the extremities or body, or a gustatory sweating disorder. Heart rate variation was assessed in 121 subjects by measuring the standard deviation of R-R intervals in the supine position[25] and by calculating the difference between the maximum and minimum heart rates during deep breathing, as described elsewhere.[26] The diagnosis of postural hypotension was based on a structured question regarding symptoms of hypotension on stand up.

Blood pressure was measured sitting after a period of rest of at least 10 minutes using a standard mercury sphygmomanometer. The reading when the fifth Korotkoff sound disappeared was taken as the diastolic blood pressure. Subjects were defined as having hypertension if they used antihypertensive drugs for hypertension or had a systolic blood pressure of $\geq$ 160 mm Hg or a diastolic blood pressure of $\geq$ 95 mm Hg.

The subjects' smoking history was recorded. An ex-smoker was defined as a person having smoked regularly at any time in the past.

A history of myocardial infarction was based on symptoms of chest pain, electrocardiogram (ECG) abnormalities, and enzyme determinations. The criteria for coronary heart disease were a history of definite myocardial infarction or ischaemic ECG abnormalities during angina pectoris type chest pain at rest or during an exercise test. Peripheral vascular disease was present if one or two tibialis posterior pulses were absent.

BIOCHEMICAL METHODS

The metabolic control of diabetes was evaluated by the annual average glycated haemoglobin A_{1c} ($GHbA_{1c}$) over the previous five years. $GHbA_{1c}$ was determined by fast protein liquid chromatography (Pharmacia, Uppsala, Sweden). The endogenous insulin secretion capacity was assessed by plasma C peptide measurement (fasting or stimulated). Plasma C peptide was assessed either by radioimmunoassay [antisera M No 299-029P, Cambridge Medical Diagnostics, Billerica, MA, USA), the tracer was human Tyr-C-peptide labelled with ^{125}I (Novo, Bagsvaerd, Denmark), with C peptide standard (Novo, catalogue No 820)] or by the Byk-Sangtec radioimmunoassay-coat C peptide method (standardised to WHO 84/150). Twelve hour or six hour overnight urine samples were collected

Table 1 Clinical characteristics of type I diabetic patients with and without shoulder capsulitis

Study group	Shoulder capsulitis		
	Present (n=30; 10.3%)	*Absent (n=261; 89.7%)*	*Total (n=291; 100%)*
Age (years)			
Mean (SD)	46.7 (8.2)*	31.7 (8.9)	33.2 (9.9)
Range	33-61	16-67	16-67
Sex			
Men (% of total)	40.0	48.7	47.8
Occupation			
Manual work (% of total)	51.9	45.2	45.9
BMI (kg m^{-2})			
Mean (SD)	24.7 (3.2)	24.0 (3.0)	24.1 (3.1)
Range	20.0-31.7	17.3-35.3	17.3-35.3
Duration of diabetes (years)			
Mean (SD)	28.7 (8.2)*	17.1 (9.1)	18.3 (9.7)
Range	11-48	1-47	1-48
$GHbA_{1c}$ (%) (5 years)			
Mean (SD)	7.9 (1.5)	8.4 (1.5)	8.4 (1.5)
Range	3.1-10.2	5.0-12.9	3.1-12.9
Non-smokers			
(% of total)	63.3	66.5	66.2
Ex-smokers			
(% of total)	20.0*	5.4	6.9
Smokers			
(% of total)	16.7	28.1	26.9

BMI, body mass index; $GHbA_{1c}$, glycated haemoglobin. * $P < 0.01$ compared to the patients without shoulder capsulitis (ANOVA).

Table 2 Clinical characteristics of type II diabetic patients with and without shoulder capsulitis

Study group	Shoulder capsulitis		
	Present (n=30; 22.4%)	*Absent (n=104; 77.6%)*	*Total (n=134; 100%)*
Age (years)			
Mean (SD)	65.3 (10.0)*	59.9 (12.8)	61.1 (12.4)
Range	44-81	38-84	38-84
Sex			
Men (% of total)	50.0	44.2	45.5
Occupation			
Manual work (% of total)	69.2	58.0	60.5
BMI (kg m^{-2})			
Mean (SD)	27.3 (4.7)	27.3 (4.6)	27.3 (4.6)
Range	19.1-39.8	15.6-43.3	15.6-43.3
Duration of diabetes (years)			
Mean (SD)	12.6 (6.1)	10.7 (7.2)	11.2 (7.0)
Range	2-26	1-32	1-32
$GHbA_{1c}$ (%) (5 years)			
Mean (SD)	9.1 (2.0)	8.5 (1.7)	8.6 (1.8)
Range	5.7-13.9	5.1-13.9	5.1-13.9
Non-smokers			
(% of total)	53.3	73.8	69.2
Ex-smokers			
(% of total)	10.0	13.6	12.8
Smokers			
(% of total)	36.7†	12.6	18.0

BMI, body mass index; $GHbA_{1c}$, glycated haemoglobin. * $P < 0.05$ and †$P < 0.01$ compared to the patients without shoulder capsulitis (ANOVA).

from 256 subjects, each of whom was instructed in writing to avoid exercise during the collection—only light indoor walking was allowed. The urine volumes were measured and the albumin concentrations were analysed after centrifugation from dipstick-negative samples by nephelometry (Behring nephelometer analyser; Behring Marburg, Germany).[28] Microalbuminuria and macroalbuminuria were considered to be present when the urinary albumin excretion rate (UAE) exceeded 20 but was less than 201 $\mu g\ min^{-1}$ in at least two of three consecutive samples and when the samples were dipstick-positive, respectively. Before establishing the presence of nephropathy associated micro- or macroalbuminuria, other causes of albuminuria such as physical exercise and concurrent urinary tract infection were carefully excluded.

STATISTICAL METHODS

For statistics, Pearson's χ^2 test, one way analysis of variance (ANOVA), multinominal polychotomous, and stepwise logistic regression analyses were used.[29]

Results

PREVALENCE OF SHOULDER CAPSULITIS

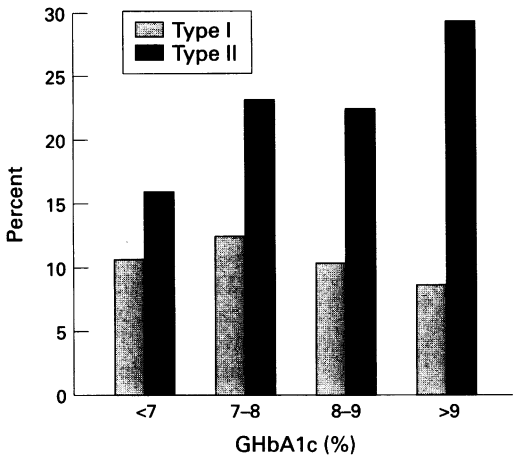
The overall cumulative prevalence of shoulder capsulitis was 14% (13% in men and 15% in women). The prevalence was 10% in type I and 22% in type II diabetic patients ($P < 0.01$). The mean age was 46.7 (SD 8.2) years in type I diabetic subjects with shoulder capsulitis and 31.7 (8.9) years in subjects without shoulder capsulitis ($P < 0.01$) (table 1). A significant association between shoulder capsulitis and the age was also found in type II diabetic patients [65.3 (10.0) *v* 59.9 (12.8) years] ($P < 0.05$) (table 2). Type I diabetic subjects with and without shoulder capsulitis had had diabetes for 28.7 (8.2) and 17.1 (9.1) years, respectively ($P < 0.01$) (table 1). The duration of diabetes was not significantly different in type II diabetic subjects with and without shoulder capsulitis, at 12.6 (6.1) *v* 10.7 (7.2) years (table 2). Shoulder capsulitis was not associated with subjects' height, weight, BMI, or occupation (tables 1 and 2). A surprising association between ex-smoking and shoulder capsulitis was found in type I diabetic subjects ($P < 0.01$), whereas shoulder capsulitis was associated with present smoking in type II diabetic subjects ($P < 0.01$) (tables 1 and 2). The association between shoulder capsulitis and ex-smoking in type I diabetic subjects was fully explained by the fact that ex-smokers were older [40.3 (11.6) years] than non-smokers [32.5 (9.7) years] or present smokers [33.4 (9.7) years]. Present smoking was not associated with patients' age, duration of diabetes, or control of diabetes in type II diabetic subjects.

GLYCAEMIA CONTROL

There was no significant difference in mean levels of $GHbA_{1c}$ over the previous five years in type I or II diabetic patients with or without shoulder capsulitis (tables 1 and 2). Type II diabetic patients with poor control of diabetes ($GHbA_{1c} > 9\%$) seemed to have more shoulder capsulitis than patients with better control of diabetes (figure), but this association was not significant.

MICROVASCULAR COMPLICATIONS

The proportions of shoulder capsulitis were 4%, 10%, and 21% in type I patients without retinopathy, with background and with proliferative retinopathy, respectively (table 3). When a χ^2 test was used, shoulder capsulitis was associated with proliferative retinopathy ($P < 0.01$) in type I diabetic subjects (table 3), whereas no significant association was found in type II diabetic patients ($P = 0.55$) (table 4). No association was found between shoulder



Proportion of diabetic patients (%) (type I and II) with shoulder capsulitis according to the control of diabetes (mean $GHbA_{1c}$ during the last five years) (%).

capsulitis and micro- or macroalbuminuria in type I or II diabetic subjects (tables 3 and 4).

Because shoulder capsulitis is strongly associated with the duration of diabetes and with age in type I and with age in type II diabetic patients (tables 1 and 2), it is to be expected that shoulder capsulitis is correlated with the duration and age dependent complication of diabetes such as retinopathy. Because retinopathy was divided into three categories, the association with shoulder capsulitis was studied using a multinominal logistic model. No significant correlation between shoulder capsulitis and background or proliferative retinopathy in type I patients was found when the confounding effects of the duration of diabetes and age were controlled for.

PERIPHERAL SYMMETRICAL SOMATIC POLYNEUROPATHY AND AUTONOMIC NEUROPATHY

The prevalence of peripheral symmetrical somatic polyneuropathy (neuropathy) was 44% in type I and 56% in type II diabetic subjects. Three per cent of type I diabetic subjects without neuropathy and 20% with neuropathy had shoulder capsulitis ($P < 0.01$) (table 3). No significant association remained when the confounding effect of the duration of diabetes and age were controlled for by logistic regression analysis. No association between shoulder capsulitis and neuropathy was found in type II diabetic subjects (table 4).

Nineteen per cent and 25% of type I and II diabetic patients, respectively, had autonomic neuropathy. Twenty five per cent of type I and 35% of type II diabetic subjects with autonomic neuropathy had shoulder capsulitis compared to 7% and 18% without autonomic neuropathy, respectively. The odds ratios for autonomic neuropathy were 4.1 (95% confidence interval, 1.6 to 10.9) and 2.7 (95% CI, 1.1 to 7.0) in type I and type II diabetic subjects with shoulder capsulitis, respectively, compared to subjects without shoulder capsulitis. If the diagnosis of autonomic neuropathy was based on hyperhidrosis, anhidrosis, or postural hypotension, the odds ratio for autonomic neuropathy in type I diabetic subjects with shoulder capsulitis was 3.6 (95% CI, 1.6 to

Table 3 Diabetic complications and related diseases according to age, the duration of diabetes, the control of diabetes (mean $GHbA_{1c}$ during past 5 years) and the presence of shoulder capsulitis (SC) in type I diabetic patients

	Mean age (years (SD))	*Mean duration of diabetes (years (SD))*	*$GHbA_{1c}$ (%)*	*Prevalence of SC, % (number of prevalent cases/all cases)*
Retinopathy				
Normal	28.3 (8.5)	10.7 (7.7)	8.1 (1.7)	4.1 (4/98)
Background	34.7 (9.5)†	21.0 (7.7)†	8.4 (1.5)	9.9 (13/131)
Proliferative	38.9 (9.5)†	26.0 (7.0)†	8.6 (1.4)	21.1 (12/57)‡
Microalbuminuria				
No	32.4 (9.2)	16.9 (9.4)	8.2 (1.4)	8.6 (14/163)
Yes	33.2 (11.0)	20.2 (10.1)*	8.8 (1.8)*	11.4 (5/44)
Macroalbuminuria				
No	32.0 (9.5)	16.3 (9.5)	8.2 (1.5)	8.5 (17/201)
Yes	35.8 (10.0)†	22.3 (8.5)†	8.8 (1.6)†	14.5 (12/83)
Peripheral symmetrical somatic polyneuropathy				
No	29.0 (7.3)	13.9 (8.1)	8.2 (1.6)	3.1 (5/163)
Yes	38.6 (10.3)†	23.9 (8.5)†	8.6 (1.4)*	19.5 (25/128)‡
Autonomic neuropathy				
No	32.1 (9.3)	17.0 (9.1)	8.3 (1.6)	6.8 (16/234)
Yes	37.8 (11.6)†	23.6 (10.2)†	8.5 (1.5)	25.5 (14/55)‡
Hypertension				
No	31.5 (9.4)	16.4 (9.4)	8.3 (1.6)	6.3 (14/221)
Yes	36.0 (8.4)*	23.5 (6.7)†	8.8 (1.3)	21.7 (15/69)‡
History of myocardial infarction				
No	32.8 (9.5)	17.9 (9.4)	8.4 (1.6)	8.8 (25/285)
Yes	54.5 (7.5)†	34.5 (9.6)†	8.5 (0.6)	83.3 (5/6)‡
Coronary heart disease				
No	32.3 (9.2)	17.6 (9.2)	8.4 (1.6)	8.1 (22/270)
Yes	44.9 (12.1)†	28.0 (10.3)†	8.4 (0.8)	35.0 (7/20)‡
Peripheral vascular disease				
No	31.8 (9.7)	17.0 (9.3)	8.4 (1.6)	7.4 (18/244)
Yes	43.1 (10.0)†	27.8 (7.8)†	8.3 (1.6)	28.9 (11/38)‡
Limited joint mobility				
No	30.7 (8.1)	13.9 (8.9)	8.4 (1.7)	8.5 (10/117)
Yes	35.0 (10.7)†	21.2 (9.1)†	8.3 (1.5)	11.5 (20/174)
Dupuytren disease				
No	31.8 (9.2)	16.8 (9.1)	8.4 (1.6)	7.6 (19/250)
Yes	41.9 (10.1)†	27.1 (8.7)†	8.4 (1.5)	25.0 (10/40)‡

‡ $P < 0.01$, *v* diabetic patients without complication or related disease (χ^2 test).
* $P < 0.05$, *v* diabetic patients without complication or related disease (ANOVA).
† $P < 0.01$, *v* diabetic patients without complication or related disease (ANOVA).

Table 4 Diabetic complications and related diseases according to age, the duration of diabetes, the control of diabetes (mean $GHbA_{1c}$ during past 5 years) and the presence of shoulder capsulitis (SC) in type II diabetic patients

	Mean age (years (SD))	*Mean duration of diabetes (years (SD))*	*$GHbA_{1c}$ (%)*	*Prevalence of SC, % (number of prevalent cases/all cases)*
Retinopathy				
Normal	61.7 (11.8)	9.9 (6.8)	8.6 (1.9)	16.3 (13/80)
Background	64.3 (9.3)	13.5 (6.1)*	8.3 (1.7)	34.4 (11/32)
Proliferative	71.6 (8.1)†	17.5 (6.0)†	9.3 (1.4)	35.3 (6/17)
Microalbuminuria				
No	55.3 (11.3)	10.8 (6.8)	8.4 (1.6)	15.4 (6/39)
Yes	50.3 (9.6)	8.9 (7.9)	9.2 (1.8)	25.0 (2/8)
Macroalbuminuria				
No	62.8 (11.7)	11.3 (7.1)	8.4 (1.6)	20.8 (22/106)
Yes	67.4 (7.5)	12.7 (7.1)	9.6 (2.1)*	28.6 (6/21)
Peripheral symmetrical somatic polyneuropathy				
No	58.5 (11.8)	8.7 (6.6)	8.3 (1.6)	18.6 (11/59)
Yes	66.8 (9.9)†	13.5 (6.9)†	8.8 (1.9)	25.3 (19/75)
Autonomic neuropathy				
No	63.7 (12.0)	11.6 (7.4)	8.5 (1.6)	18.3 (17/93)
Yes	62.5 (9.3)	11.9 (6.6)	8.9 (2.1)	35.5 (11/31)‡
Hypertension				
No	61.5 (11.7)	11.7 (7.2)	8.7 (1.4)	25.8 (17/66)
Yes	65.1 (11.0)	11.6 (7.2)	8.6 (2.0)	19.1 (13/68)
History of myocardial infarction				
No	61.8 (11.6)	11.2 (6.9)	8.4 (1.6)	18.5 (20/108)
Yes	69.1 (8.6)†	13.0 (8.0)	9.5 (2.1)†	38.5 (10/26)‡
Coronary heart disease				
No	58.2 (11.2)	10.0 (7.2)	8.4 (1.5)	20.3 (16/79)
Yes	69.3 (8.3)†	12.9 (6.9)	8.9 (2.0)	25.5 (14/55)
Peripheral vascular disease				
No	62.1 (12.1)	11.2 (7.2)	8.4 (1.6)	20.6 (20/97)
Yes	67.1 (8.5)*	12.9 (7.0)	9.2 (2.0)*	26.5 (9/34)
Limited joint mobility				
No	58.1 (12.5)	8.9 (6.3)	8.2 (1.2)	13.2 (7/53)
Yes	66.7 (9.4)†	13.2 (7.2)†	8.9 (2.0)*	28.4 (23/81)‡
Dupuytren disease				
No	63.1 (12.0)	11.3 (7.4)	8.6 (1.8)	20.0 (23/115)
Yes	65.3 (6.7)	13.1 (5.4)	8.6 (1.4)	36.8 (7/19)

‡ $P < 0.05$, *v* diabetic patients without complication or related disease (χ^2 test).
* $P < 0.05$, *v* diabetic patients without complication or related disease (ANOVA).
† $P < 0.01$, *v* diabetic patients without complication or related disease (ANOVA).

8.1) compared to the subjects without shoulder capsulitis, but no association between autonomic neuropathy and type II diabetes was found. The confounding effects of the duration of diabetes and age were controlled for by logistic regression analysis. In a subsample of 121 type 1 diabetic subjects, shoulder capsulitis was associated with impaired heart rate variation ($P < 0.01$), but this association disappeared when the age and the duration of diabetes were controlled for.

HYPERTENSION, HISTORY OF MYOCARDIAL INFARCTION, CORONARY HEART DISEASE, AND PERIPHERAL VASCULAR DISEASE

The prevalence of hypertension was 24% and 51% in type I and II diabetic subjects, respectively. Twenty two per cent of type I diabetic subjects with hypertension and 6% of patients without hypertension had shoulder capsulitis ($P < 0.01$) (table 3). No association between shoulder capsulitis and hypertension remained when the confounding effect of the age was controlled for. No association between shoulder capsulitis and hypertension was seen in type II diabetic patients (table 4).

The prevalence of a history of myocardial infarction was 2% and 19% and of coronary heart disease, 7% and 41% in type I and II diabetic subjects, respectively. Both type I and type II diabetic subjects with myocardial infarction had shoulder capsulitis more often than those without myocardial infarction, whereas an association between coronary heart disease and shoulder capsulitis was found only in type I diabetic subjects (tables 3 and 4). Because myocardial infarction was strongly associated with the duration of diabetes and age, we used stepwise logistic regression analysis to control the confounding effect of these time related variables. Smoking habits and hypertension were also included in the model. The odds ratio for myocardial infarction in type I diabetic subjects was 13.7 (95% CI, 1.3 to 139.5) when the confounding effects of the age, the duration of diabetes, hypertension, and smoking habits were controlled. The correlation remained significant when serum total cholesterol was included in the model. In type II diabetic subjects no correlation was found between shoulder capsulitis and myocardial infarction when the time related variables were controlled for. No association between shoulder capsulitis and coronary heart disease was found when the confounding effects of the duration of diabetes or the age were controlled for in type I or II diabetic subjects.

The prevalence of peripheral vascular disease was 13% and 26% in type I and II diabetic patients, respectively. Type I diabetic patients with peripheral vascular disease had shoulder capsulitis more often than those without peripheral vascular disease ($P < 0.01$) (table 3), but this association was fully explained by the age and the duration of

diabetes. No association was found between peripheral vascular disease and shoulder capsulitis in type II diabetic subjects (table 4).

LIMITED JOINT MOBILITY AND DUPUYTREN DISEASE

The prevalence of limited joint mobility was 60% in type I and II diabetic patients. No association between limited joint mobility and shoulder capsulitis was found in type I diabetic subjects (table 3). Type II diabetic subjects with limited joint mobility had shoulder capsulitis more often than those without (table 4), but this association disappeared when the confounding effects of the duration of diabetes and the age were controlled for.

The prevalence of Dupuytren disease was 14% in both type I and II diabetic subjects. Twenty five per cent of type I diabetic subjects with Dupuytren disease had shoulder capsulitis compared to 8% without Dupuytren disease ($P < 0.01$). This association disappeared when the confounding effects of time related variables were controlled for. No significant association between Dupuytren disease and shoulder capsulitis was found in type II diabetic subjects (table 4).

Discussion

This report shows that shoulder capsulitis is a common disorder in both type I and type II diabetic subjects, which is in line with previous studies.[14 18 21] The presence of shoulder capsulitis was highly dependent on the age and the duration of diabetes in type I diabetic subjects, whereas age was the most important factor explaining shoulder capsulitis in type II diabetic subjects. The prevalence of shoulder capsulitis increased after the age of 40 and 50 years in type I and II diabetic patients, respectively. The prevalence of shoulder capsulitis did not increase until after the duration of diabetes had exceeded 20 years in type I diabetic patients. The reason for the high prevalence of shoulder capsulitis after a short duration of diabetes in type II patients may be explained by the fact that the known duration of diabetes is probably an inaccurate marker of the true duration of the disease in many patients with type II diabetes. Two previous studies have shown an association between shoulder capsulitis and the duration of diabetes,[16 21] but these studies showed no association between shoulder capsulitis and the age of diabetic subjects. Sattar and Luqman have shown no difference in the prevalence of shoulder capsulitis in insulin treated and non-insulin-treated diabetic subjects.[21] The higher prevalence of shoulder capsulitis in type II diabetic subjects seen in our study was most probably explained by the higher age of these subjects compared to type I patients. In addition to the different age and the duration of diabetes, type II diabetic subjects differed from type I in that they had a higher BMI, which is, however, unlikely to explain higher prevalence of shoulder capsulitis because no association was found between shoulder capsulitis and BMI.

Smoking has been shown to contribute to microvascular complications in diabetic subjects.[30] Eadington *et al* found that smoking associated with limited joint mobility but not with Dupuytren disease,[31] whereas Gamsted *et al* found no association between smoking and these hand abnormalities.[32] In the present study, shoulder capsulitis was associated with previous smoking in type I subjects, but this association was fully explained by the fact that subjects who had previously smoked were older than non-smokers or present smokers. Shoulder capsulitis was associated with present smoking in type II diabetic subjects and this was not explained by the age of the patients, by the duration of diabetes, or by the control of diabetes. The association may be explained by the fact that smoking causes vasoconstriction, which may also underlie the development of shoulder capsulitis.

Recently a strong relation between long term glycaemic control and diabetic microvascular complications has been definitely established.[33] It would be important to know if other diseases closely related to diabetes are also related to the long term glycaemic control. Bridgman investigated 800 diabetic patients (23% of them were insulin dependent) and he stated that shoulder capsulitis correlated with the severity of diabetes because 36% of the affected (shoulder capsulitis) patients were insulin dependent.[14] Mavrikakis *et al* have also stated that shoulder capsulitis is associated with the poor control of diabetes, because type II diabetic subjects who had shoulder capsulitis were more often insulin treated than those without shoulder capsulitis.[16] We found no difference in the prevalence of shoulder capsulitis in type II diabetic subjects treated with different regimens. According to present knowledge the type of regimen is not good marker of the control or severity of diabetes. In the present study, type II diabetic patients with poor control of diabetes ($GHbA_{1c} > 9\%$) seemed to have more shoulder capsulitis than patients with better control (figure). However, no significant association between shoulder capsulitis and the control of diabetes was found in type I or II diabetic patients. The reason for not finding a significant association might be that there were very few patients whose control was as good as in the intensive therapy group of the DCCT study.[33] It would be important to know the effect of control of diabetes during the first years of the disease on the development of shoulder capsulitis.

The correlation between limited joint mobility and microvascular complications of diabetes has been well documented,[20 22] but there are also two studies showing an association between shoulder capsulitis and retinopathy.[19 21] Our study also showed an association between shoulder capsulitis and proliferative retinopathy in type I patients, but the correlation was fully explained by age and by the duration of diabetes. This indicates that diabetic subjects with shoulder capsulitis do not have a higher risk of microvascular complications, such as limited joint mobility, than those without the condition.

In some patients shoulder capsulitis may precede, accompany, or follow diffuse swelling, coldness, erythema, tenderness, and hyperhidrosis of the hand.[4] After weeks or months, the tenderness, swelling, and vasomotor dysfunction completely resolve, with residual atrophic or dystrophic changes, finger contractures, and occasionally shoulder capsulitis. This has been called shoulder-hand syndrome or reflex sympathetic dystrophy. Because of a possible association between reflex sympathetic dystrophy and shoulder capsulitis, autonomic neuropathy may help us to understand the pathogenesis of shoulder capsulitis as previously discussed by Steinbrocker and Thompson.[13 34] Our study supports this, because we observed about a fourfold and a threefold risk of autonomic neuropathy in type I and II diabetic subjects with shoulder capsulitis, respectively, compared to those without shoulder capsulitis when the confounding effects of age and duration of diabetes were controlled for. A subsample of 121 type I diabetic patients also showed that shoulder capsulitis was associated with impaired heart rate variation, but this association was fully explained by the time related variables. The association between shoulder capsulitis and autonomic neuropathy seen in the present study must be interpreted with care, because our criteria for autonomic neuropathy were "soft" and the heart rate variation test or other neurophysiological testing were not performed in all subjects.

Many investigators have shown a higher prevalence of shoulder capsulitis in diabetic patients and in patients with a history of myocardial infarction.[6 14] On the other hand, diabetes is associated with higher rates of cardiovascular disease.[35] Previously Johnson has suggested that vasoconstriction of the peripheral arteries of the hand resulting from cardiac pain, pre-existing arteriosclerotic narrowing of the vessels of the hands, and anoxia of varying duration and intensity resulting from the myocardial infarction were responsible for the production of local changes in the hands and fingers.[36] Mavrikakis *et al* have shown that diabetic subjects with shoulder capsulitis had higher cholesterol and triglyceride levels than diabetic subjects without shoulder capsulitis or shoulder capsulitis control group.[16] These results suggest that the higher prevalence of shoulder capsulitis in diabetic patients could be explained by atherosclerotic changes in vessels, leading to changes in local blood flow and producing altered physiology in tendons, with resultant shoulder capsulitis. We studied clinical features which could help us to understand the possible pathogenic associations between shoulder capsulitis, diabetes, and myocardial infarction. Type I diabetic patients with shoulder capsulitis seemed more frequently to have had a history of myocardial infarction than those without shoulder capsulitis, independent of the age of the patient, the duration of their diabetes, hypertension, or smoking habits. The limited number of type I diabetic subjects with a history of myocardial infarction in the present study does not allow us to draw final conclusions, but our data suggest an increase in the prevalence of shoulder capsulitis in patients with a history of myocardial infarction. An association between myocardial infarction and shoulder capsulitis in type II diabetic patients was fully explained by the time related factors. Thus the correlation between shoulder capsulitis and myocardial infarction does not explain the high prevalence of shoulder capsulitis in type II diabetic patients.

Shoulder capsulitis was associated with Dupuytren disease in type I and with limited joint mobility in type II diabetic patients in the present study. Because Dupuytren disease and limited joint mobility were strongly related to the age and duration of diabetes, it is important to control for this confounding effect. After using a stepwise logistic regression analysis, the correlations between shoulder capsulitis and Dupuytren disease and limited joint mobility disappeared. Fisher *et al* showed an association between shoulder capsulitis and limited joint mobility in 57 diabetic patients.[18] Another study of 49 type I and 60 type II diabetic patients showed no correlation between limited joint mobility and shoulder capsulitis.[15] Both of these studies may be misleading, because Fisher *et al* eliminated half of their original patient population on the basis that their joint contractures were obviously due to Dupuytren disease, osteoarthritis, or flexor tenosynovitis, and Pal *et al* made no attempt to distinguish the causes of the limitation in finger joint mobility.[15 18]

In conclusion, this study shows that shoulder capsulitis is a common disorder in type I and II diabetic patients. It is associated with age in type I and II diabetic patients and with the duration of diabetes in type I patients, which explains most of its correlations with diabetic complications. Independent associations were found between shoulder capsulitis and autonomic neuropathy in type I and II diabetic subjects and with the history of myocardial infarction in type I only.

1 Duplay S. De la péri-arthrite scapulo-humérale et des raideurs de l'épaule qui en sont la conséquence. *Arch Gen Med* 1872;**20**:513–42.
2 Lundberg BJ. The frozen shoulder. *Acta Orthop Scand* 1969; suppl 119.
3 Codman EA. Rupture of the supraspinatus tendon and other lesions in or about the subacromial bursa. In: *The shoulder*. Boston: Thomas Todd Co, 1934:216.
4 Rosenbloom AL. Connective tissue disorders in diabetes. In: *International textbook of diabetes mellitus*, vol 2. New York: John Wiley & Sons, 1992:1415–31.
5 Charnley J. Periarthritis of the shoulder. *Postgrad Med J* 1959;**35**:384–8.
6 Steinbrocker O, Argyros TG. The shoulder-hand syndrome: present status as a diagnostic and therapeutic entity. *Med Clin North Am* 1958;**42**:1533–53.
7 Askey JM. The syndrome of painful disability of the shoulder and hand complicating coronary occlusion. *Am Heart J* 1941;**22**:1–12.
8 Minter WT. The shoulder-hand syndrome in coronary disease. *J Med Ass Ga* 1967;**56**:45–9.
9 Swan DM. Shoulder-hand syndrome following hemiplegia. *Neurology* 1954;**4**:480–2.
10 Johnson JTH. Frozen shoulder syndrome in patients with pulmonary tuberculosis. *J Bone Joint Surg Am* 1959; **41**:877.
11 Engleman RM. Shoulder pain as a presenting complaint in upper lobe bronchogenic carcinoma: report of 21 cases. *Conn Med* 1966;**30**:273–6.
12 Oldham BE. Periarthrosis of the shoulder associated with thyrotoxicosis. *N Z Med J* 1959;**58**:766–8.
13 Thompson M. Shoulder-hand syndrome. *Proc R Soc Med* 1961;**54**:679–81.
14 Bridgman JF. Periarthritis of the shoulder and diabetes mellitus. *Ann Rheum Dis* 1972;**31**:69–71.

15 Pal B, Anderson J, Dick WC, Griffiths ID. Limitation of joint mobility and shoulder capsulitis in insulin- and non-insulin-dependent diabetes mellitus. *Br J Rheumatol* 1986; **25**:147–51.
16 Mavrikakis ME, Sfikakis PP, Kontoyannis SA, Antoniades LG, Kontoyannis DA, Moulopoulou DS. Clinical and laboratory parameters in adult diabetics with and without calcific shoulder periarthritis. *Calcif Tiss Int* 1991;**49**:288–91.
17 Crisp AJ. Diabetes mellitus and the rheumatologist. *Br J Rheumatol* 1986;**25**:135–40.
18 Fisher L, Kurtz A, Shipley M. Association between cheiroarthropathy and frozen shoulder in patients with insulin-dependent diabetes mellitus. *Br J Rheumatol* 1986; **25**:141–6.
19 Morén-Hybbinette I, Moritz U, Scherstén B. The clinical picture of the painful diabetic shoulder—natural history, social consequences and analysis of concomitant hand syndrome. *Acta Med Scand* 1987;**221**:73–82.
20 Rosenbloom AL, Silverstein JH, Lezotte DC, Richardson K, McCallum M. Limited joint mobility in childhood diabetes indicates increased risk for microvascular disease. *N Engl J Med* 1981;**305**:191–4.
21 Sattar MA, Luqman WA. Periarthritis: another duration-related complication of diabetes mellitus. *Diabetes Care* 1985;**8**:507–10.
22 Arkkila PET, Kantola IM, Viikari JSA. Limited joint mobility in type 1 diabetic patients: correlation to other diabetic complications. *J Intern Med* 1994;**236**:215–23.
23 National Diabetes Data Group. Classification and diagnosis of diabetes mellitus and other categories of glucose intolerance. *Diabetes* 1979;**28**:1039–57.
24 Noble J, Heathcote JG, Cohn H. Diabetes mellitus in the aetiology of Dupuytren's disease. *J Bone Joint Surg Br* 1984;**66**:322–5.
25 Murray A, Ewing DJ, Campbell IW, Neilson JMM, Clarke BF. RR interval variations in young male diabetics. *Br Heart J* 1975;**37**:882–5.
26 Ewing DJ, Clarke BF. Diagnosis and management of diabetic autonomic neuropathy. *BMJ* 1982;**285**:916–8.
27 Koskinen P. Nontransferability of C-peptide measurements with various commercial radioimmunoassay reagents. *Clin Chem* 1988;**34**:1575–8.
28 Kouri TT, Viikari JSA, Mattila KS, Irjala KMA. Microalbuminuria. Invalidity of simple concentration-based screening tests for early nephropathy due to urinary volumes of diabetic patients. *Diabetes Care* 1991;**14**:591–3.
29 Dixon WJ, ed. *BMDP statistical software*. Los Angeles: University of California Press, 1990.
30 Muhlhauser I. Smoking and diabetes. *Diabet Med* 1990; 7:10–15.
31 Eadington DW, Patrick AW, Frier CB. Limited joint mobility, Dupuytren's contracture and retinopathy in type 1 diabetes: association with cigarette smoking. *Diabet Med* 1989; **6**:152–7.
32 Gamstedt A, Holm-Glad J, Ohlson C-G, Sundström M. Hand abnormalities are strongly associated with the duration of diabetes mellitus. *J Intern Med* 1993;**234**:189–93.
33 The Diabetes Control and Complications Trial research group. The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. *N Engl J Med* 1993;**329**:977–86.
34 Steinbrocker O, Neustadt D, Bosch SJ. Painful shoulder syndromes. Their diagnosis and treatment. *Med Clin North Am* 1955;**39**:563–85.
35 Wilson PW, Anderson KM, Kannel WB. Epidemiology of diabetes mellitus in the elderly: the Framingham study. *Am J Med* 1986;**80**:3–9.
36 Johnson AC. Disabling changes in the hand resembling sclerodactylia following myocardial infarction. *Ann Intern Med* 1943;**19**:433–56.