SHORT REPORTS

Limited joint mobility and Dupuytren's contracture in diabetic, hypertensive, and normal populations

Limited joint mobility in diabetic hands is obvious when severe, while more subtle abnormalities are defined as the inability to extend to 180° the metacarpophalangeal or interphalangeal joints of at least one finger bilaterally.[1] The prevalence in young diabetics is 18-30%[1-3] compared with less than 5% in controls, and limited joint mobility is associated with an increased incidence of microangiopathy.[1 3]

To examine the possibility that limited joint mobility might represent an accelerated aging process associated with microangiopathy its prevalence was assessed in normal, diabetic, and hypertensive subjects, the last group being exposed to an alternative cause of vascular disease. Dupuytren's contracture, which also occurs more frequently in diabetics,[4] was also assessed.

Subjects, methods, and results

The 168 diabetics (80 men) and 114 hypertensive patients (57 men) were recruited from the diabetic and hypertension clinics of Gartnavel General Hospital and the Western Infirmary, Glasgow. One hundred healthy subjects (34 men) were recruited from friends and relatives of the patients. The mean ages of the groups were 52, 55, and 51 years respectively.

Limited joint mobility was determined using the modified fanned hand to hand method.[1] Dupuytren's contracture[4] in either hand was recorded as a positive finding. The presence or absence of retinopathy was recorded, with no differentiation between background and proliferative retinopathy. Statistical analysis was by χ^2 test and the Mann-Whitney U test.

The table shows the prevalences of limited joint mobility and Dupuytren's contracture in the three groups. The apparently increased prevalence of Dupuytren's contracture in diabetic women and hypertensive men compared with controls of the same sex did not achieve statistical significance ($0{\cdot}05<p<0{\cdot}1$). Limited joint mobility was associated directly with age in controls of both sexes ($p<0{\cdot}01$), diabetic women ($p<0{\cdot}0005$), and men with hypertension ($p<0{\cdot}05$). Dupuytren's contracture was significantly associated with age in all groups (diabetics $p<0{\cdot}0005$; others $p<0{\cdot}01$) except in the male controls. In women with diabetes both limited joint mobility and Dupuytren's contracture were also associated with duration of disease ($p<0{\cdot}01$).

In the diabetic women retinopathy was associated both with limited joint mobility ($p<0{\cdot}05$) and, more strongly, with Dupuytren's contracture ($p<0{\cdot}0001$) but no significant association was shown in the men with diabetes ($0{\cdot}05<p<0{\cdot}1$). Coexistent limited joint mobility and Dupuytren's contracture correlated strongly with retinopathy in both sexes. Retinopathy was present in 63% (17/27) of subjects with both abnormalities but in only 15% (9/59) of those with neither complication ($p<0{\cdot}001$). Insulin dependence was inversely related to limited joint mobility ($p<0{\cdot}02$).

Comment

This survey shows that the prevalence of limited joint mobility is increased not only in diabetes but also in healthy men compared with women and in hypertensive patients. The high prevalence in normal men, which had no discernible relation to occupation, may explain the difficulty in detecting significantly increased prevalences in male diabetics and hypertensives compared with healthy men. Both limited joint mobility and Dupuytren's contracture were related to increasing age and to retinopathy in diabetic patients. This previously noted association of Dupuytren's contracture and retinopathy[3] has been largely neglected but in our present study was highly significant ($p<0{\cdot}0001$). Dupuytren's contracture of diabetes has been suggested to differ from the orthodox disorder in its distribution[4] (fifth fingers rarely affected) and our patients conformed to this pattern (two fifth fingers in 156 fingers affected). The aetiologies of Dupuytren's contracture and limited joint mobility in diabetes may be linked.

Increased glycosylation of collagen has been suggested as the pathogenesis of limited joint mobility,[2] glycosylation of basement membranes explaining the associated microangiopathy.[2] Tissue glycosylation, however, is no greater in diabetics with limited joint mobility than in other diabetics,[5] and glycosylated haemoglobin correlates with tissue glycosylation[2] but not with limited joint mobility.[1] Our findings are compatible with an alternative aetiology. The association of limited joint mobility and retinopathy may suggest that microvascular disease is a factor in the pathogenesis of limited joint mobility and not solely in diabetes. Limited joint mobility may be a vascular related phenomenon of aging, where women are "protected" in a manner similar to ischaemic heart disease, while diseases which cause vascular damage such as diabetes, hypertension, and possibly scleroderma may accelerate the changes of limited joint mobility.

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Legionnaires' disease cluster and reduction in hospital hot water temperatures

Official guidelines for maintenance of hospital water storage and distribution systems recommend that hot water should be above 50°C at outlets and cold water below 20°C to inhibit growth of *Legionella pneumophila*. In one hospital from 27 November 1980 to 15 July 1983 hot water was held between 55°C and 63°C at outlets, but from 15 July to 15 September 1983 it was considered essential to reduce hot water temperatures by about 10°C (precise data not available) in order to lower the working temperatures in the operating theatres during exceptionally warm weather. Eight weeks later an outbreak of nosocomial legionnaires' disease occurred which terminated just after the reintroduction of higher hot water temperatures.

Prevalences of limited joint mobility and Dupuytren's contracture in diabetic, hypertensive, and normal subjects

	Men			Women		
	No of subjects	No (%) with limited joint mobility	No (%) with Dupuytren's contracture	No of subjects	No (%) with limited joint mobility	No (%) with Dupuytren's contracture
Control	34	9 (26)†	4 (12)	66	6 (9)	10 (15)
Diabetic	80	29 (36)	36 (45)‡	88	38 (43)‡	26 (30)
Hypertensive	57	15 (26)	16 (28)	57	14 (25)†	9 (16)
Total	171	53 (31)	56 (33)*	211	58 (27)	45 (21)

*$p<0{\cdot}05$ compared with women.
†$p<0{\cdot}05$ compared with control women.
‡$p<0{\cdot}01$ compared with controls of same sex.

Outbreak, investigation, and results

The outbreak comprised three definite (cases 1, 2, and 4), one probable (case 3), and one possible (case 5) case of nosocomial legionella pneumonia (table). All patients were receiving steroids or insulin and we estimated the risk of infection for such patients. Patients in hospital from 1 to 14 September 1983, which was considered to be the high risk period, and receiving immunosuppressive or antidiabetic drugs were identified from medical records and hospital pharmacy prescriptions to patients at discharge. Serum samples from these patients taken between 24 November and 2 December were tested by the rapid microagglutination test[1] and (if titres were ≥8) an indirect fluorescent antibody test. Water samples were collected from the cooling tower and the domestic circuit.

No common exposure within the hospital was identified. All patients were in different wards. Only one patient (case 1) showered in the hospital before the onset of symptoms. In addition to the patients in cases 1-5, 101 (6%) out of 1818 patients in the hospital during 1 to 14 September 1983 were in the high risk groups. One of 39 (n=64) patients having immunosuppressive drugs and one of 18 (n=37) diabetic patients were seropositive (cases 6 and 7). One of these (case 6) had had lower respiratory tract symptoms five days postoperatively.

The estimated attack rate for high risk patients between and 1 and 14 September 1983 was 3% (3/106), or 5% (3/61) if non-responders are excluded. The infection rate was between 5% (5/106) and 8% (5/61), or between 6/1000 (3/485) and 10/1000 (5/485) person days exposed.

One shower water sample from near the room of the patient in case 2 yielded *L pneumophila* serogroup 1, which was indistinguishable by monoclonal antibody typing[2] from the two human isolates but differed from the strains isolated from water in the same hospital in 1980 (J Tobin, personal communication).

Comment

The recommendations for control of water temperature do not have unanimous support because of doubts about the effectiveness, cost benefits, and potential hazards such as scalding.[3] This outbreak is circumstantial evidence that the recommended control measures had reduced the risk of infection. Possibly the reduction in hot water temperatures over eight weeks allowed organisms already colonising the water system to multiply to levels infectious for immunocompromised patients. Evidence that the domestic water supply was the source of infection came from the monoclonal typing.

Subsequent investigations showed that legionellas had not been eradicated from the hospital water system, and in practice eradication may not be neccessary or possible. Whereas hyperchlorination and temperature regulation have successfully controlled outbreaks of legionnaires' disease elsewhere, legionellas have still been isolated from water samples.[4]

Others have shown that *L pneumophila* is very frequently present in plumbing systems of large buildings which are not associated with human cases of legionellosis.[5] Hence the mere isolation of legionellas from hospital water systems does not prove a causal relation with coincidental nosocomial cases, nor that, in the absence of such cases, there is a serious risk of infection. We emphasise, however, that where legionellas are present in hospital water systems prospective active surveillance of nosocomial respiratory infections is essential.

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Out of depth, out of breath

We describe three patients who were unable to breathe when they stood in water and had difficulty in swimming.

Case reports

Case 1—A 46 year old man first noticed that he could not breathe in water when he dived into the Thames for a swim. Initially he thought that this was because of the coldness of the water but subsequently found that, although he was able to breathe comfortably as he walked into shallow water, he became increasingly dyspnoeic when the level rose above his abdomen. He had also noticed mild dyspnoea when jogging and playing tennis. On examination he was breathless when supine and had paradoxical inward motion of the abdomen on inspiration. Radiological screening showed paradoxical motion of the diaphragm in the supine position. Testing of the respiratory muscles showed weakness of the diaphragm; oesophageal and gastric pressures were measured with balloon catheters attached to differential pressure transducers,[1] and transdiaphragmatic pressure was obtained by an electronic subtraction of gastric pressure minus oesophageal pressure. Transdiaphragmatic pressure was 20 cm H_2O (normal >80 cm H_2O)[2] during a maximal sniff, 10 cm H_2O (normal >25 cm H_2O)[3] during a maximal inspiration to total lung capacity, and 15 cm H_2O (normal >18 cm H_2O)[4] during a maximal static inspiratory manoeuvre from residual volume. No systemic cause for diaphragm weakness was identified.

Case 2—A 26 year old man known to have muscular dystrophy had for 18 months been unable to swim because of shortness of breath. He could paddle in shallow water, but when the level rose above his pelvis he became progressively dyspnoeic. Investigations showed diaphragm weakness. Transdiaphragmatic pressure was 7·5 cm H_2O during a maximal sniff, 2·5 cm H_2O during a maximal inspiration, and 7·5 cm H_2O during a maximal static inspiratory effort.

Case 3—A 54 year old woman had been unable to breathe when she stood in deep water. For 12 months she had been having nightmares of choking at night

Clinical and serological data

Case No	Sex and age	Underlying disease	Admission dates	Onset of legionnaires' disease	Sera	Micro-agglutination titre	Indirect immuno-fluorescence titre	Isolation
1	F 40	Graves' disease treated with dexamethasone and cyclosporin A	1/9/83-	16/9/83	2/9/83	<4	16	
					20/9/83	128	256	
					22/9/83	512	256	Sputum
2	F 75	Insulin depedendent diabetes	31/8/83-7/9/83	8/9/83	15/9/83	<4	<16	
					23/9/83	64	64	
					27/9/83	32	64	
					30/9/83	64	64	
3	M 64	Systemic lupus erythematosus treated with prednisone	26/8/83-2/9/83	12/9/83	29/9/83	4096	4096	
					10/10/83	>4096	4096	
					17/11/83	>512	>512	
4	M 64	Rheumatoid arthritis treated with prednisone; fractured femur fixed on 30/9/83	28/8/83-	5/10/83 (died 9/10/83)	30/9/83	8	16	Lungs
					8/10/83	64	128	(necropsy)
5	M 77	Eczema treated with whole body topical betamethasone. Proctocolectomy 21/9/83	2/8/83-	26/9/83	5/10/83	64	16	
					10/10/83	32	32	
					17/10/83	8	16	
6	M 59	Ulcerative colitis treated with prednisone	29/8/83-5/10/83	?26/3/83	24/10/83	512	1024	
7	M 54	Diabetes, leg amputation	15/8/83-12/9/83		4/11/83	128	256	