was assessed in each patient by two clinicians by ophthalmoscopic examination of the ocular fundi through fully dilated pupils. Statistical analysis was by the χ^2 test.

Proteinuria was found in 53 (14%) of the Asian and 23 (6%) of the white patients (p<0.001). There was no difference in the sex distributions of patients with proteinuria. Proteinuria in the Asians tended to occur with diabetes of shorter duration (<10 years) (p<0.01) and in the absence of clinical retinopathy (p<0.001). No significant differences were noted in glycaemic control or the prevalence of hypertension between the two groups (table). Twenty five of the

Clinical details of patients studied

No studied	Asian		White	
	3	70		368
Men	228		223	
Women	142		145	
Mean (SD) age (years)		52 (12)		58(15)
Mean (SD) duration of diabetes (years)		8(3.6)		12 (4.2)*
No (%) treated with insulin	1	35 (37)		258 (70) [*]
No (%) with proteinuria		53 (14)		23 (6)**
Men	32		14	. ,
Women	21		9	
Without retinopathy	25 (47)		4(17)*	
With hypertension ≥160/95 mm Hg	27 (51)		14 (62)	
Mean (SD) glycosylated haemoglobin (%)		9·5 (2·0)		9·3 (2·5)

*p<0.01, **p<0.001.

228 Asian men had a serum creatinine concentration above normal (>120 µmol/l >1.4 mg/100 ml)), compared with 11 of the 223 white men (p=0.02). No such difference was found in the women.

Comment

Our results indicate that proteinuria is more common in Asian than white diabetics attending the diabetic clinic at Leicester. We believe that this is due to small vessel disease of diabetes as we excluded other causes of renal disease both clinically and radiologically. This difference in renal disease could not be accounted for by variation in glycaemic control or the prevalence of hypertension. Ethnic cultural factors may influence patterns of referral to hospital, but we do not believe that this occurs in Leicester and our view was substantiated by inquiries to general practitioners. Patients with specific diabetic complications are generally not discharged from the clinic, which therefore suggests that the observed difference in renal disease was real and not an artefact.

A larger proportion of Asian patients had proteinuria with diabetes mellitus of less than 10 years' duration and in the absence of clinical retinopathy. The shorter duration of diabetes may partly be because many of the Asians had non-insulin dependent diabetes mellitus, in which the timing of onset may be inexact. It is also possible that differences in immunogenetic influences in populations from different cultures determine the development of diabetic nephropathy, and this aspect requires further study

Asian people in Leicester tend to come originally from the Gujarat region of India, having migrated through east Africa. Women are usually vegetarian, but men are not.² Consumption of animal protein influences the speed of progression of renal disease,³ and this may explain the significantly higher serum creatinine concentration observed in the Asian men.

The higher prevalence of proteinuria in Asian diabetics in this hospital based study should be confirmed by epidemiological work based on the population at large. This is particularly important in view of the high prevalence of diabetes mellitus in Asians in the United Kingdom.45

- 1 Diabetes Drafting Group of the World Health Organisation Multinational Study of Vascular Disease in Diabetics. Prevalence of small vessel and large vessel disease in diabetic patients from 14 centres. Diabetologia 1985;28 (suppl):615-40.
- 2 Leicester City Council and Leicestershire County Council. Survey of Leicester 1983. Leicester: Leicester City Council and Leicestershire County Council, 1983. 3 Williams AJ, Baker FE, Walls J. The effect of dietary protein quality in experimental renal disease.
- Williams AJ, Baker PE, Waits J. The effect of detary protein quality in experimental renardisease. *Proc Eur Dial Transplant Assoc* 1985;22:921-6.
 Mather HM, Keen H. The Southall diabetes survey: prevalence of known diabetes in Asians and Europeans. *Br Med J* 1985;291:1081-4.
 Samanta A, Burden AC. Prevalence of non insulin dependent diabetes mellitus (NIDDM) in Asian *Autor Constants* (2010).
- Indians. Clin Sci 1986;70 (suppl 13):19.

(Accepted 2 June 1986)

Leicester General Hospital, Leicester LE5 4PW

A SAMANTA, MD, MRCP, Pfizer clinical research fellow

A C BURDEN, MD, MRCP, consultant physician J FEEHALLY, MRCP, senior registrar (nephrology)

J WALLS, MB, FRCP, consultant nephrologist

Correspondence to: Dr Burden.

367

Awareness and use of glucagon in diabetics treated with insulin

Recent surveys have shown that 4% of deaths in diabetics under the age of 50 are caused by hypoglycaemia,¹ and 4-10% of patients treated with insulin experience one or more severe episodes each year.²³ Glucagon is an effective drug for such patients and can be given by a relative, friend, or medical attendant. There is little information available about the policies of diabetic clinics in prescribing this drug. We have therefore examined the frequency of hypoglycaemia and the awareness and use of glucagon and its effectiveness in preventing hospital attendance among routine clinic patients.

Patients, methods, and results

Five hundred and four consecutive diabetics treated with insulin routinely attending a clinic over two months were asked about their awareness of glucagon, whether it was kept at home and in date, if it had ever been given, and whether this had prevented hospital attendance. Patients were questioned about the frequency of mild, moderate, or severe hypoglycaemia (severe hypoglycaemia being defined as that aborted by parenteral treatment). They were also asked whether their general practitioner had been called and whether casualty attendance or hospital admission had been required because of hypoglycaemia. Glycosylated haemoglobin concentrations were measured by electrophoresis, the normal range being 5.5.7.9%. Statistical analyses used were Wilcoxon's rank sum test, Spearman rank correlation coefficient, and χ^2 test.

The table shows the clinical details of the patients. Of the 504 patients, 252 (50%) had heard of glucagon and 155 (30%) kept glucagon at home, of whom 114 (23%) were certain it was in date. One hundred and eight patients had received

Clinical details of 504 consecutive diabetics treated with insulin. (Values are means (SD)

Sex (M:F)	249:255
Age (years)	41.0 (16.9)
Body weight (kg)	68.25 (11.4)
Duration of insulin treatment (years)	14.1 (10.3)
Daily insulin dose (units)	52.8 (22.5)
Number of clinic visits in past year	4.3 (2.7)
Total glycosylated haemoglobin concentration (%)	12.0 (3.8)

glucagon at home at least once, of whom 92 (85%) were convinced that casualty attendance or hospital admission had thus been prevented. Of 175 patients whose general practitioner had been called out in the past because of severe hypo-glycaemia, 102 had heard of glucagon. Of 136 attending a casualty department for hypoglycaemia, 61 had heard of glucagon. In the year before the study 67 (13%) had experienced one or more episodes of severe hypoglycaemia, this being more common in younger patients (p<0.001), those with lower glycosylated haemoglobin concentrations (p<0.001), those who visited the clinic more often (p<0.001), those receiving a higher dose of insulin per kg body weight (p<0.01), and those who kept glucagon at home (p < 0.0005).

Comment

Hypoglycaemia continues to be a hazard for diabetics, with 67 (13%) of our patients treated with insulin experiencing one or more severe episodes in the year before the study. Such episodes occurred in younger patients with lower glycosylated haemoglobin concentrations, suggesting that patients with better glycaemic control are more at risk. If improved glycaemic control is the main goal of diabetic management then it is essential that patients and their relatives know how to cope with this problem.

Glucagon is an effective treatment for hypoglycaemia⁴ and this is confirmed by our finding that of 108 patients given glucagon at home or work, only 16 required subsequent hospital attendance. It is clearly a useful first line of defence given subcutaneously at home or work, while in general practice or at the hospital it can be given intramuscularly or intravenously with a quicker effect.

Patients' knowledge of the use of glucagon still seems limited, with half of those interviewed unaware of its existence and only about a third keeping it at home. Those patients with more frequent episodes of severe hypoglycaemia, however, did keep glucagon at home, although, disappointingly, some patients who had been admitted to hospital because of hypoglycaemia before had not heard of glucagon.

In view of these findings and the possible saving in costs incurred when general practitioners are called out and in hospital attendances and admissions, we believe that all diabetics treated with insulin should have glucagon at hand, provided that a relative or friend is available and trained in administering it.

We thank Dr Martin Edwards (Novo UK Ltd) and Mrs Linda McDonald for their help in the preparation of this manuscript.

- 1 Tunbridge WMG. Factors contributing to deaths of diabetics under fifty years of age. Lancet 1981;ü:569-72.
- 2 Casparie AF, Elving LD. Severe hypoglycemia in diabetic patients: frequency, causes, prevention. Diabetes Care 1985;8 (2):141-5.
- Potter J, Clarke P, Gale EAM, Dave SH, Tattersall RB. Insulin induced hypoglycaemia in an accident and emergency department: the tip of an iceberg? *Br Med J* 1982;285:1180-2.
 Elrick H, Witten TA, Arai Y. Glucagon treatment of insulin reactions. *N Engl J Med* 1958;258:
- 5 Mulhauser I, Koch J, Berger M. Pharmacokinetics and bioavailability of injected glucagon: differences between intramuscular, subcutaneous and intravenous administration. Diabetes Care 1985.8(1):39-42.
- (Accepted 20 May 1986)

Diabetic and Dietetic Department, Royal Infirmary, Edinburgh EH3 9YW D M MATTHEWS, MRCP, senior registrar A W PATRICK, MRCP, registrar D A COLLIER, MRCP, registrar H A KELLETT, MRCP, lecturer J M STEEL, FRCPE, associate specialist B F CLARKE, FRCPE, consultant physician

Medical Statistics and Computing Unit, University Medical School, Edinburgh

CCA MACINTYRE, MSC, research associate

Correspondence to: Dr Matthews.

The "last joint" syndrome in ankylosing spondylitis

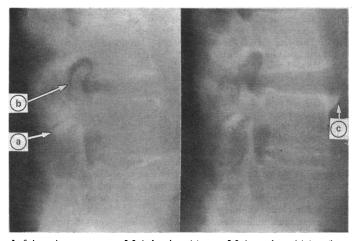
Nerve root compression is a rare complication of ankylosing spondylitis.¹ We describe a case in which bony encroachment into the intervertebral foramina at the only unfused segments in the cervical and lumbar spine (the "last joints") resulted in recurrent nerve root compression. Tomography showed the presence of osteophytes, which were removed surgically, resulting in complete relief of symptoms.

Case report

A 31 year old man with a 10 year history of ankylosing spondylitis was first seen in 1977. He had a rigid thoracolumbar spine with a dorsal kyphosis but still had some painful movement in his neck.

In November 1980 he complained of burning pain in both thighs. There was minor blunting of pain sensation in the anterior aspect of the right thigh. Tomography showed a large posterior osteophyte arising from the posterior articulation and encroaching on the L3-4 intervertebral foramen. This was removed surgically with excellent pain relief. All the lumbar spinal joints other than those at L3-4 had fused.

In October 1981 he was readmitted with pain and sensory loss in the anterior left thigh, weakness and wasting of the left quadriceps, and a diminished left knee jerk. Tomography showed a large osteophyte growing forward into the left L3-4



Left lateral tomograms at L3-4 showing: (a) open L3-4 apophyseal joint; (b large osteophyte arising from the L3-4 apophyseal joint; (c) osteophyte at t anterior margin of the L4 vertebral body.

foramen. At operation the L3 nerve root was found trapped between the lower border of the pedicle laterally and the osteophyte medially. The osteophyte was removed, and the symptoms and signs rapidly resolved.

He remained well until March 1983, when he developed shooting pains down both arms, brought on by neck movement, and parasthesiae in the medial three fingers of the left hand. Oblique films of the cervical spine showed bilateral encroachment by an osteophyte on the C3-4 intervertebral foramina, with the left side affected more than the right. Severe osteoarthritic change was evident at the apophyseal joints, which were open. All other levels in the cervical spine except the occipitoatlantic joint were fused. An anterior cervical discectomy with fusion resulted in rapid and complete relief of symptoms.

In October 1984 he had a recurrence of neurological signs in the right thigh. Tomograms and a computed tomography scan again showed open apophyseal joints at L3-4, with a large osteophyte compromising the intervertebral foramina (see figure). Surgical removal of the osteophyte gave immediate relief.

Comment

Although pain in a nerve root distribution is sometimes encountered in ankylosing spondylitis, corresponding neurological abnormalities are rare and evidence of structural lesions affecting the nerve roots is usually lacking.² Bony ankylosis of intervertebral foramina is not a feature of the disease ² and root lesions are usually attributed to inflammatory changes in related structures or to vascular insufficiency.1

In the case described here four instances of nerve root compression occurred, one in the cervical spine and three at the same level in the lumbar spine. Pain and parasthesiae in the nerve root distribution were the presenting features and were accompanied by neurological deficit. Large osteophytes arising from the posterior apophyseal joint and protruding forward into the corresponding intervertebral foramina were shown in each instance. The corresponding apophyseal joints at the two levels affected were unfused, in contrast to extensive ankylosis above and below.

We believe that prominent osteophytes arose from the apophyseal joints of the unfused segment as a result of the abnormal stresses imposed during movement. The osteophytic spurs were distinct from the spondylitic process.1 Similarly, the bony protruberances at the anterior margins of the corresponding intervertebral joints appeared to be osteophytes rather than syndesmophytes.

Destructive changes at unfused segments in late ankylosing spondylitis have been described,3-5 and massive osteophytosis may occur during the healing of these areas.5 Nerve root compression due to encroachment on intervertebral foramina at unfused levels has not, however, been reported. There was no evidence of fracture or destructive lesions in our patient.

Marcos and Freiberger³ suggested immobilisation and promotion of complete ankylosis as the treatment of choice in destructive vertebral lesions. Our patient has had no recurrence of cervical nerve root compression since fusion, but he has had further problems at the L3-4 level, which was not fused surgically and is the last joint to remain mobile in his spine.

- 1 Thomas DJ, Kendall MJ, Whitfield AGW. Nervous system involvement in ankylosing spondylitis. Br Med 7 1974;i: 148-50.
- 2 Matthews WB. The neurological complications of ankylosing spondylitis. J neurol Sci 1968;6: 561-73.
- 3 Marcos R, Freiberger RH. Vertebral destruction at unfused segments in late ankylosing spondylitis. Radiology 1969;93:251-6. 4 Cawley MID, Chalmer TM, Kellgren JH, Ball T. Destructive lesions of vertebral bodies in
- anyklosing spondylitis. Ann Rheum Dis 1972;3:345.
 5 Dunn N, Preston B, Jones KL. Unexplained acute backache in longstanding ankylosing spondylitis. Br Med J 1985;291:1632-5.

(Accepted 21 May 1986)

University Department of Medicine, Bristol Royal Infirmary, Bristol BS2 8HW

K VEERAPEN, MB, MRCP, clinical assistant in rheumatology P A DIEPPE, MD, FRCP, senior lecturer in rheumatology

Department of Neurosurgery, Frenchay Hospital, Bristol BS16 1LE R VEERAPEN, FRCS, SN, registrar in neurosurgery H B GRIFFITH, FRCS, FRCP, senior consultant neurosurgeon

Correspondence to: Dr Paul Dieppe.

Correction

Prevalence of antibody to HTLV-III in haemophiliacs in the United Kingdom

In the Patients, methods, and results section of this short report (19 July, p 175) it was stated that "20 (60%) of 324 patients with haemophilia B who were tested were positive...." This should have read "20 (6%) of 324 patients with haemophilia B who were tested were positive." We apologise for this error.