agonists to the management of prostatic cancer but emphasise the importance of further investigation into their combined use with antiandrogens.

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Predominant wrist disease in rheumatoid arthritis associated with high concentration of IgA rheumatoid factor

Both the severity of radiological progression and the pattern of joint disease in rheumatoid arthritis are extremely variable. In a series of patients studied prospectively those with high concentrations of IgA rheumatoid factor in their serum showed a predominance of erosions affecting the wrist.¹ We have therefore studied a larger number of patients to test the hypothesis that such an association exists.

Patients, methods, and results

We studied 46 patients with definite or classical rheumatoid arthritis. Twenty three had raised values of IgA rheumatoid factor in their serum and in the rest these values were normal. Nineteen patients in the first group and 21 in the second had raised IgM rheumatoid factor concentrations. The group with high IgA rheumatoid factor values consisted of six men and 17 women with a mean age of 56.5 years (range 34-78) and a mean duration of disease of 8.7 years (range 2-24), and the group with normal IgA rheumatoid factor values comprised eight men and 15 women with a mean age of 54.6 years (range 35-74) and a mean duration of disease of 7.6 years (range 2-25). Rheumatoid factor was measured by an enzyme linked immunosorbent assay.² Radiographs of hand and wrist were assessed using the defect score described by Sharpe *et al*,³ the scores for erosions in wrist and hand (metacarpophalangeal and proximal and distal interphalangeal joints) being recorded separately. The number of hand erosions was subtracted from the number of wrist erosions to give a score for each patient. Although a ratio of hand to wrist erosions would have been a more satisfactory method of assessment, some patients who had extensive erosions in the wrist had none in the hand. The scores in the two groups were compared by Wilcoxon's rank sum test.

The patients with high IgA rheumatoid factor values had a mean score of 7.1 (range -8 to 36), while the patients with normal values of IgA rheumatoid factor had an average score of -0.4 (range -32 to 18). This difference was statistically significant ($p < 0.05$).

Comment

The aetiology of rheumatoid arthritis remains unknown but it has been suggested that the disease may represent several disorders with different immunogenetic backgrounds and environmental trigger factors. This possible heterogeneity makes the search for aetiological factors very

difficult. It therefore seems reasonable to look for subgroups of disease and that studying these separately may help elucidate the problem. IgA rheumatoid factor has already been associated with a poor prognosis in rheumatoid arthritis.¹ Finding an association with a particular radiological pattern lends further support to the view that patients with rheumatoid arthritis and high values of IgA rheumatoid factor may represent a subgroup of the disease. This association has been shown despite our patients having established disease, for which several had received "specific" or disease modifying agents (gold salts, penicillamine, steroids) which lower IgA rheumatoid factor values (unpublished observations). Burns and Calin have reported a different radiological pattern in patients with seronegative rheumatoid arthritis. (Some patients who are conventionally seronegative have high values of IgA rheumatoid factor in their serum.) They interpreted their findings as suggesting that seronegative rheumatoid arthritis has a different pathological mechanism from seropositive rheumatoid arthritis.⁴ Other studies categorising the radiological progression of rheumatoid arthritis have noted an association between rheumatoid factor titre and severity of erosive disease but have not commented on the pattern of joint erosion.^{3,5}

Though it is unclear why IgA rheumatoid factor should be a marker for a disease subset of rheumatoid arthritis, we believe that IgA rheumatoid factor deserves closer examination in patients with rheumatoid arthritis.

We acknowledge the help of Dr Ingvar Teitsson, who performed the rheumatoid factor assays.

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The neglected hospital wheelchair

Hospital wheelchairs are in constant use and have to withstand considerable wear. We observed that many were in a poor state of repair and that patients had sustained injuries owing to faulty wheelchairs. A previous study identified some faults in hospital wheelchairs,¹ but the prevalence and implications of such faults have not been assessed. We examined this problem.

Methods and results

We inspected all the transit and self propelled wheelchairs in a geriatric hospital (215 beds) and all those in the medical wing of a teaching hospital (204 beds). The type of tyre (pneumatic or solid) was recorded, and the backrest, seat, armrests, and footrest plates were examined for defects. The pneumatic tyres were inspected for wear and state of inflation. They were recorded as worn if the central part of the tread was indistinct, soft if there was insufficient air to support a person sitting in the wheelchair and excessive bulging of the tyre occurred, and deflated if there was clearly no air in the inner tube. The deflated tyres were pumped up, their valves tested, and the tyres later re-examined to determine if a puncture was present. Steering and brakes were tested with a person sitting in the wheelchair. Each brake was applied separately and recorded as defective if the wheelchair could still be moved easily.

To evaluate injuries to patients caused by defective wheelchairs we prospectively investigated for two months all accidents on our geriatric unit in which wheelchairs played a part and that required the completion of a hospital accident report.

There were 93 wheelchairs in the geriatric hospital and 30 in the medical wing. Only 21 and seven, respectively, were free from faults. The table gives details of the faults found. Wheelchair arms were termed dangerous when their foam covering had been torn away, exposing the sharp ends of the bolts inside. Spikes on the footrest plates were sharp vertical projections that remained on the

footplate when the canvas heel loops were torn away and the protective plastic caps lost. Two wheelchairs had a brake mechanism missing on one wheel, and on two others the footplates had been tied with string because they were loose.

Faults found in 123 hospital wheelchairs

Fault	No (%) of wheelchairs	Fault	No (%) of wheelchairs
Defective pneumatic tyres	56 (46)*	Defective footrest plates	39 (32)
Soft	37	Missing	27
Punctured	16	Spikes	6
Leaking valve	2	Loose or stiff	6
Worn	1	Defective arms	14 (11)
Defective brakes	75 (61)	Dangerous	10
Torn seat or backrest	10 (8)	Missing	4
Defective steering	2 (2)		

*57% of the 98 wheelchairs with pneumatic tyres.

During the two months 11 accidents related to wheelchairs were reported. In five of these a defective wheelchair was partly or completely responsible for the patient's injury. One patient received a laceration to her hand from an exposed bolt on the armrest, and another sustained a large shin laceration when a loose footrest plate dropped down during transfer. Three patients fell while getting up from wheelchairs with defective brakes.

Comment

The hospital wheelchairs inspected in this study were in an unsatisfactory state. Many were unsafe, and some resulted in injury to the patient. Only 28 (23%) were safe and in good working order. The most common problems—defective brakes, soft or punctured tyres, and absent or defective footrest plates—were simple faults that could easily have been corrected. Although the danger of exposed spindles on the footrest plate was specifically highlighted in a circular from the Department of Health and Social Security (safety information bulletin No 14, 1984), six wheelchairs had exposed spindles.

The results of this study support the suggestion of a recent report that hospitals should organise regular inspection and maintenance of wheelchairs.² Most hospital staff have no special knowledge of problems connected with wheelchairs, and better instruction and information about wheelchairs and their use are required.

At present many hospital wheelchairs are in a poor state of repair; this neglect should not continue.

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Macroamylasaemia: how common is it?

In macroamylasaemia a macromolecular complex circulates in the plasma and usually causes benign hyperamylasaemia. Possibly these findings may be interpreted incorrectly as due to acute pancreatitis. We therefore studied the extent of unrecognised problems arising from macroamylasaemia in a British teaching hospital.

Present series

Serum specimens from all 74 patients with hyperamylasaemia seen over three months were assayed by a recently described simple polyethylene glycol

precipitation test.¹ Two patients were found to have macroamylasaemia. This was confirmed by gel filtration chromatography, which distinguishes the macromolecular complex from the usual molecular mass amylase; urinary amylase activity assay, because low activity in the presence of high serum activity suggests low renal clearance if glomerular function is not impaired; and serum lipase activity assay, because a high serum amylase activity without raised lipase activity suggests that the former is not due to pancreatic damage.

Case 1—A 62 year old man with severe abdominal pain and jaundice was admitted and diagnosed clinically as having acute cholecystitis. Serum bilirubin concentration was 55 $\mu\text{mol/l}$ (3.2 mg/100 ml; reference range 0-17 $\mu\text{mol/l}$; 0-10 mg/100 ml), alkaline phosphatase activity 190 U/l (reference range 20-120 U/l), and γ -glutamyltransferase activity 145 U/l (reference range 7-42 U/l). Investigations showed a serum amylase activity of 5772 U/l (reference range 70-300 U/l) and acute pancreatitis was therefore added to the diagnosis. Pancreatic ultrasonography was performed twice but the pancreas was poorly visualised; there were no abnormalities. Intravenous cholangiography showed a small stone in the common bile duct. At laparotomy a perforated gall bladder was found and cholecystectomy performed. Serum amylase activity fell to within the reference range after massive blood loss and transfusion but later rose again to between 3920 and (on discharge) 7590 U/l. This was considered to be worthy of suspicion but no further biochemical investigations were initiated. The results suggested a pancreatic pseudocyst but two further ultrasound studies failed to confirm this. A serum specimen was submitted for assessment of amylase activity during our study when the patient was seen three months after discharge; macroamylasaemia was found and confirmed by gel filtration, urinary amylase activities of 114 and 228 U/l (reference range 800-2000 U/l), and serum lipase activity of 75 U/l (reference range up to 190 U/l). Renal impairment was not evident—serum urea concentration 5.7 mmol/l (34.3 mg/100 ml; reference range 3.3-6.6 mmol/l; 19.9-39.8 mg/100 ml), creatinine 110 $\mu\text{mol/l}$ (1.2 mg/100 ml; reference range 44-150 $\mu\text{mol/l}$; 0.5-1.7 mg/100 ml).

Case 2—A 65 year old woman presented with a three day history of persistent, severe abdominal pain radiating across the abdomen, urinary and faecal incontinence, nausea, and haematemesis. She had had angina pectoris since 1970 and two episodes of myocardial infarction, the most recent in 1981, complicated by axillary vein thrombosis. She also had congestive cardiac failure and chronic respiratory disease. Serum amylase activity assay was requested and was 564 U/l. A clinical diagnosis of ischaemic colitis or mesenteric infarction was made. At laparotomy extensive small bowel infarction due to a superior mesenteric artery embolus was discovered and partial bowel resection undertaken. Her stormy postoperative course in the intensive care unit was complicated by aspiration pneumonia. Macroamylasaemia was found by us during her stay in the unit and confirmed by gel filtration and, early during hospitalisation, urinary amylase activities of 58 and 89 U/l and a serum lipase activity of 17 U/l; serum urea concentration was 18.5 mmol/l (111.4 mg/100 ml) and creatinine 196 $\mu\text{mol/l}$ (2.2 mg/100 ml). Serum amylase activity remained high and was 800 U/l on discharge after 13 days.

Comment

Review of case 1 showed that the abdominal pain was due to acute cholecystitis and obstruction of the common bile duct which culminated in gall bladder perforation. Initial treatment may have been delayed unnecessarily, since the clinical diagnosis appeared to us to have been modified after finding a raised serum amylase activity. Macroamylasaemia did not cause confusion in case 2, since the symptoms led to a clinical diagnosis. Serum amylase activity was, however, determined to exclude pancreatitis; this was certainly included in the differential diagnosis.

It is well known but not always remembered that hyperamylasaemia is not diagnostic of pancreatitis.² Macroamylasaemia, in particular, is an important potential source of diagnostic confusion.

In the short term we suggest that the polyethylene glycol precipitation test for macroamylase should be introduced by hospital service laboratories. We agree with recent reports that efforts should be made to replace amylase activity assays with alternative methods such as assay of lipase activity.³ Macroamylasaemia would thus be eliminated as a cause of diagnostic confusion.

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