SHORT REPORTS

Calcitonin for multiple fractures in Paget's disease

Paget's disease of bone often causes pathological fracture,¹ though Paget made no mention of this complication in his original treatise. Multiple fractures may occur, and some measure of their incidence may be gauged from a series of femoral fractures secondary to Paget's disease described by Barry.² Thirteen out of 73 patients had two femoral fractures, and four had at intervals sustained three fractures of the same femur. We describe here a particularly florid case of Paget's disease with multiple fractures, for which calcitonin was prescribed.

Case report

A 73-year-old woman presented with a week's history of a painful right hip and no history of injury. Radiographs showed Paget's disease of the pelvis and femora with a virtually undisplaced transverse fracture of the proximal right femoral shaft. Within the previous two years she had sustained a left subtrochanteric and a right intertrochanteric fracture secondary to Paget's disease. Both had been treated with internal fixation, with a subsequent incisional infection on the right. The underlying metal implant was removed late, after the fracture had united.

The presenting right femoral shaft fracture was initially treated conservatively by immobilisation, but while lifting herself in bed the patient sustained a pathological mid-shaft fracture of the left clavicle. To improve the patient's mobility the femoral fracture was internally fixed with a compression plate, and at surgery the bone was found to be soft and vascular. A spontaneous supracondylar fracture of the same femur occurred soon after operation, this being the third pathological fracture within three weeks. The leg was immobilised in a plaster-of-Paris cylinder. Serum alkaline phosphatase concentration was raised (1750 IU/l), and because of this florid clinical and biochemical presentation treatment of the Paget's disease was started with salmon calcitonin 100 MRC units daily. The alkaline phosphatase fell dramatically and the subsequent calcitonin dosage was titrated to its fluctuations (see figure). The serum calcium was normal and after a fall at the onset of treatment remained within the normal range. Seven weeks after the femoral fracture the knee was mobilised, and at 10 weeks the patient started full weight bearing.

No further fractures occurred in the next two years, despite the fact that the patient once had a fall that caused an effusion into the right knee. The calcitonin was discontinued after 10 months of treatment, and since that time two further short courses have been given with benefit for pelvic girdle pain.

Changes in serum alkaline phosphatase levels during treatment with calcitonin.

Comment

Calcitonin is advocated in Paget's disease primarily for hypercalcaemia, relief of bone pain³ and nerve compression.⁴ There is one other report of calcitonin being used to prevent further pathological fractures, but this was in a 5-year-old boy.⁵ The beneficial clinical and biochemical responses to calcitonin treatment witnessed in our patient suggest that it may be used to suppress very active Paget's

disease with repeated pathological fractures. The rate of union was also favourable, with full weight bearing 10 weeks after the fracture. In a series of 90 femoral fractures in Paget's disease only one out of every five had similar union within three months.² Rapid union was often associated with abundant callus formation, but in our case little callus was produced with calcitonin treatment. Calcitonin also has the added benefit of preventing bone resorption hypercalcaemia, which is most likely to occur in patients after multiple fractures.

We thank Dr Donald Ward, of the Robert Jones and Agnes Hunt Orthopaedic Hospital, Oswestry, for his interest and help.

¹ Lake, M, *Journal of Bone and Joint Surgery*, 1951, **33B**, 323.

² Barry, H C, *Paget's Disease of Bone*. Edinburgh and London, Livingstone, 1969.

³ Woodhouse, N J Y, *British Journal of Hospital Medicine*, 1974, **2**, 677.

⁴ De Rose, J, et al, *American Journal of Medicine*, 1974, **56**, 858.

⁵ Doyle, F H, et al, *British Journal of Radiology*, 1974, **47**, 9.

(Accepted 13 October 1976)

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Scoop-stretcher "sandwich": method of turning patients with spinal injuries

Those concerned with the immediate care of severely injured people outside hospital are often confronted with cases of suspected spinal injury. When there is also life-endangering cardiorespiratory embarrassment immediate resuscitation supersedes all else. In most cases, however, in which life is not in immediate danger, it is mandatory not to move the casualty more than absolutely necessary. The Ferno-Washington scoop stretcher is an adjustable metal stretcher that separates into two parts along its longitudinal axis. Each half may be introduced beneath the casualty without causing appreciable movement and then clipped together. It is possible and usually advisable to transfer the patient to hospital in exactly the same position as found.

A problem arises with casualties found face-down. If their cardiorespiratory state allows, we believe that they should be transported on a scoop stretcher in the position found. On arrival at hospital, however, they may need to be turned to the supine position for full examination and assessment. Unless performed with extreme caution and control this is often a hazardous and potentially dangerous manoeuvre when traditional methods are used.

"Sandwich" technique

On several occasions we have used what we term the sandwich technique for casualties with spinal injuries. The casualty is kept in the position found and transported to hospital on a scoop stretcher and left on it. When turning becomes necessary a second scoop stretcher adjusted to the same dimensions as the first one is laid on top of the casualty, the two stretchers being secured together with triangular bandages or straps so that the patient is firmly sandwiched between them. When immobility has been ensured the whole unit may be lifted and rotated through 180° without risk of movement of the casualty. If necessary rolled blankets may be laid longitudinally down both sides of the casualty before the straps are applied.

Comment

This technique should be used in accident and emergency departments for turning any unconscious patient with suspected spinal

injury. As an ambulance carries only one scoop stretcher, another has to be obtained. In a busy accident department a second ambulance may often be about from which a scoop stretcher could be borrowed. We, however, believe that a second stretcher should be standard equipment in an accident and emergency department. It could then also be used to move the severely injured from trolley to x-ray table or bed. This is much easier and safer, as well as more comfortable for the casualty, than the traditional straight-lift or poles-and-canvas method.

This stretcher has proved popular with the ambulance service and with our general-practitioner accident unit on many occasions. We have found its versatility of great value, even on wet and uneven ground.¹ We have successfully used it to remove a casualty with spinal injuries lying face down at the bottom of a narrow, 2-m-deep trench and on many occasions at the road-side and on building sites in all weathers.

We thank Mr R Drucker and his colleagues, of the London Ambulance Service, for their help and co-operation, and F W Equipment and Co, Bradford, for the loan of the stretchers.

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¹ Robertson, B, *Journal of the Royal Army Medical Corps*, 1976, **122**, 21.

(Accepted 2 November 1976)

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Chromosome 1 heteromorphism in patients with malignant disease: a constitutional marker for a high-risk group?

Polymorphism for the heterochromatic regions of the No 1 chromosomes is common and has no apparent phenotypic effect.^{1,2} My attention was drawn, however, to a possible association with malignancy by the observation that the normal No 1 chromosomes in the aneuploid tumour cells of two ovarian carcinomas and a seminoma were heteromorphic for their C-band-positive regions. I could not ascertain whether the heteromorphism was present in the patients' normal cells but decided to investigate the incidence of heteromorphism in a series of patients with malignant disease at these and other sites.

Methods and results

Normal cells from 58 patients with malignant disease were studied, either in blood cultures (47 cases) or in direct preparations or cultures of lymphoid or tumour tissue. Seventeen patients had received chemotherapy or radiotherapy. Peripheral blood from 12 normal people and from 36 patients sampled at random from among those referred to the haematology laboratory for blood tests was studied. Cultured lymph nodes from five patients, spleen from one patient, and a fibroblast culture from a normal fetus were also studied. All these control patients had non-malignant conditions. Chromosome preparations made by standard procedures were stained by a C-banding technique.³ Heteromorphism was considered to be present when the areas of the heterochromatic regions of the No 1 chromosomes differed on average by at least 25% (measurements were made on photographs at a final magnification of $\times 4500$; in most cases, however, the presence or absence of heteromorphism could easily be assessed by direct microscopic observation). All assessments and measurements were made "blind."

Thirty-two of the 58 patients with malignant disease showed the heteromorphism compared with 17 of the 55 controls (see table). This difference was statistically significant ($\chi^2 = 6.74$ with Yates's correction; $P < 0.01$). Nine of the 32 positive patients had received treatment. No difference was discovered between the "malignant" and control groups in factors that might

Incidence of heteromorphism in 58 patients with malignant disease and 55 controls

	Heteromorphism present	Heteromorphism absent
<i>Patients with malignant disease</i>		
Tumour site or type:		
Ovary	12	13
Testis	4	3
Hodgkin's disease	4	4
Other lymphomas	6	2
Various carcinomas	5	4
Melanoma	1	0
Total	32 (15 men)	26 (7 men)
Mean ratio of areas of greater:lesser heterochromatic regions	1.50 (1.27-2.01)*	
Mean age (years)	51.5	51.1
<i>Controls</i>		
Healthy people:		
Men	1	3
Women	3	6**
Patients with non-malignant conditions:		
Men	6	8
Women	7	21
Total	17	38
Mean ratio of areas of greater:lesser heterochromatic regions	1.50 (1.30-1.63)†	
Mean age (years)	44.5	43.2‡

*Based on 207 metaphases. †Based on 61 metaphases. **Fetus included. ‡Fetus not included.

have caused an artefactual difference in C-band areas—for example, a difference in the lengths of the two No 1 chromosomes due to unequal spreading on the slide.

Comment

It seems reasonable to assume that there is a relation between the amounts of heterochromatin on the No 1 chromosomes and susceptibility to at least some forms of malignant disease. A common heritable chromosomal variant—a marker whose presence can be determined by a simple technique—can serve as a guide to the risk of cancer. (Although usually traceable to one or other parent, variant chromosomes may sometimes arise de novo during a lifetime, as suggested by the occurrence of mosaicism for the variant⁴; however, mosaicism was clearly present in only one normal man in this present study.)

The nature of the association with cancer is unclear. The heteromorphism might be related to a general instability of the chromosomes or more directly to the No 1 chromosomes' susceptibility to undergo structural changes. Such changes are common in the aneuploid tumour cells of ovarian carcinomas and lymphomas, at least (N B Atkin and M C Baker, unpublished data). It is interesting that the frequency of the association of the heterochromatic regions of the No 1 chromosomes with acrocentric chromosomes is positively correlated with the extent of the regions on the No 1 chromosomes.⁵

Studies are in progress to try to determine whether the association with malignancy is limited to variation affecting the No 1 chromosome or also encompasses the other chromosomes that are subject to similar variation, such as Nos 9 and 16.

This work was supported by a grant from the Cancer Research Campaign.

¹ Craig-Holmes, A P, Moore, F B, and Shaw, M W, *American Journal of Human Genetics*, 1973, **25**, 181.

² McKenzie, W H, and Lubs, H A, *Cytogenetics and Cell Genetics*, 1975, **14**, 97.

³ Sumner, A T, *Experimental Cell Research*, 1972, **75**, 304.

⁴ Craig-Holmes, A P, Moore, F B, and Shaw, M W, *American Journal of Human Genetics*, 1975, **27**, 178.

⁵ Schmid, M, Vogel, W, and Krone, W, *Cytogenetics and Cell Genetics*, 1975, **15**, 66.

(Accepted 26 October 1976)

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