Fractured neck of femur and contralateral intracerebral lesions

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SUMMARY Ten cases of fractured neck of femur studied at necropsy were found to have established contralateral intracerebral lesions. These were mainly infarcts, and it is suggested that the presence of hemiparesis predisposes to a fall on to the affected side and that the body weight acts through the hip joint in such a way as to fracture the femoral neck. This may happen irrespective of the magnitude of the patient's trabecular bone volume, but in osteoporotic subjects the fracture will probably be intertrochanteric, whereas in non-osteoporotic patients it will probably be subcapital.

Fractures of the proximal femur represent a considerable threat to the health of our aging population. In the United Kingdom some 40 000 new patients will suffer a fracture of the femoral neck each year, about 70% of these will be subjected to operative treatment with pinning and plating, or the implantation of a prosthesis.¹ It is generally appreciated that without active treatment the outlook is poor and mortality is high. It is perhaps not so widely appreciated that even with active treatment up to 27% of patients will die in the first year and about 50% of survivors remain unable to walk without assistance.²

We noted that a substantial number of cases of fractured neck of femur come to necropsy in our department, and we are currently engaged in a systematic, in depth, prospective study of the healing and tissue events associated with surgical treatment. Particular attention is being paid to the determination of the presence or absence of metabolic bone disease and other risk factors that predispose to the occurrence of the fracture, or which adversely affect the healing process. During the early course of this study we noted several cases in which there had been established intracerebral lesions.

This report looks at certain features of these cases and the role of the intracerebral lesions in the causation of the fractures.

Material and methods

Over 22 months from the beginning of 1984 to the end of October 1985 necropsies were performed on 50 patients who had sustained a fracture of the femoral neck. In 10 (20%) of these established intracerebral lesions were noted, and these cases were examined in

particular detail. In seven cases transiliac bone cores (7 mm diameter) were taken from the left anterior iliac crest. Undecalcified sections (5 μ m thick) were prepared from these and assessed histomorphometrically for the presence of systemic metabolic bone disease.

Results

Six men and four women were included in the series. Table 1 gives some of their details. The combined median age was 78 years (range 62-91), and the median ages of the men and women were 78 (range 62-82) and 81 (range 70-91) years, respectively. There were four established recent intracerebral infarcts and four old infarcts. One case showed an established intracerebellar haemorrhage. In one case there was a grade IV astrocytoma affecting the left parieto-occipital region. Table 2 shows the sites of the intracerebral lesions and Table 3 the type and laterality of the femoral fractures. In each case the intracerebral lesion affected the side of the brain opposite to the side of the femoral neck fracture. Although in some cases more than one intracerebral site was affected, this was always restricted to one side.

The mean time interval from the fracture to death was 22 days with a range from one to 117 days. In five cases the intracerebral lesions were considered, after histological examination, to have occurred at or about the time of the fracture. In the five other cases the morphological features of intracerebral lesions indicated that they preceded the fracture. At necropsy there was no evidence of pulmonary or intracerebral fat embolism. Table 4 gives the causes of death. In all cases these were confirmed by histological examination.

In cases 3 to 10 inclusive the fracture was repaired

Table 1 Clinical and morphological details of cases in series

Case	Age	Sex	Body weight (kg)	Trabecular bone volume (left iliac crest) (%)	Fracture details	Cerebral lesion	Clinically suspected cerebral lesion
1	62	M	68	22.9	Subcapital fracture right femur	Astrocytoma (grade IV) left frontoparietal region	Yes
2	77	M	105	20.4	Subcapital fracture left femur	Recent infarct right parieto-occipital region	Yes
3	81	M	36	13.4	Intertrochanteric fracture left femur	Old infarct right basal ganglia	Yes
4	82	M	54	11.9	Intertrochanteric fracture right femur	Recent infarct left parieto-occipital region	No
5	76	F	49	11.8	Intertrochanteric fracture right femur	Old infarct left temporo-occipital region	No
6	70	F	55	6.1	Intertrochanteric fracture right femur	Recent infarct left cerebellar and left occipital lobes	Yes
7	91	F	34	5.8	Intertrochanteric fracture right femur	Old infarct left internal capsule	Yes
8	80	M	46		Intertrochanteric fracture right femur	Old infarct left parietal region	No
9	86	F	54		Intertrochanteric fracture right femur	Established haemorrhage left parietal region	Yes
10	76	M	47		Intertrochanteric fracture left femur	Recent infarct right parietal region	Yes

by internal fixation. These operations were usually performed several days after admission, and examination of the anaesthetic records showed no untoward events. There were no recorded episodes of intraoperative hypotension.

The trabecular bone volume in the left iliac crest was calculated in seven cases. The mean volume was

14.9% (range 5.8–22.9), and if the two highest values (22.9% and 20.4%) are excluded then the mean value was 9.8% (range 5.8–13.4). There was no histological evidence of osteomalacia or osteitis fibrosa in any of the cases.

Discussion

Osteoporosis is commonly believed to be an important risk factor in patients who suffer fractures of the proximal femur. Not every osteoporotic subject, however, has a fracture nor, indeed, is every person who suffers a fracture an osteoporotic. Published reports dealing with the issue of patients with fractures being appreciably more osteoporotic than age matched controls was recently reviewed by Cummings.³ He concluded that, although the most rigorously designed studies observed less bone mass in patients with fractures, the differences from controls were usually small and overlapping. Aitken⁴ reported that a series of 200 women with a fracture of the femoral neck after minor trauma had bone mass measurements similar to those of a control population of normal women, and 16% were not osteoporotic. It has also been suggested that osteomalacia may be a contributory factor in a large number of cases.^{5–9} In contrast, two more recent Australian studies reported the absence of osteomalacia in patients with fracture of the femoral neck.^{10 11} The idea that diminished bone quantity is the major determinant of femoral neck fracture cannot, therefore, be wholly accepted. It has been recognised that postural instability causing a fall is a common event in relation to the fracture.¹² Aitken⁴ believed that postural instability was the major determinant and that the presence of osteo-

Table 2 Site and side of intracerebral lesions

Site	Right side	Left side
Frontal lobe	0	1
Temporal lobe	0	1
Parietal lobe	2	4
Occipital lobe	1	3
Internal capsule	0	1
Basal ganglia	0	1
Cerebellum	1	0

In five cases more than one anatomical site was affected.

Table 3 Fracture type and laterality in cases studied

Fracture type	Right side	Left side
Subcapital	1	1
Transcervical	0	0
Intertrochanteric	6	2
Total	7	3

Table 4 Causes of death in cases studied

Cause of death	All cases	Men	Women
Bronchopneumonia	5	4	1
Ischaemic heart disease	4	0	4
Calcific aortic stenosis	1	1	0

porosis dictates the type of fracture (usually intertrochanteric) that occurs.

Brocklehurst *et al*¹² performed a clinical study of 384 patients with fracture of the neck of femur and compared them with 226 controls. Forty three per cent of the patients with fracture had neurological disease (12% had a stroke, 12% had the chronic brain syndrome, 6% had parkinsonism, and 13% had other neurological diagnoses). No information was given about the laterality of the lesions and there were no morbid anatomical studies.

Our series of cases is interesting from several viewpoints. There were lateralised intracerebral lesions, and in all instances the fractured femoral neck was on the contralateral side of the body. We believe that there was acute (the majority) or chronic hemiparesis and speculate that the patients fell towards and on to the side of the fracture. We further speculate that there was loss of muscle tone associated with the hemiparesis, allowing the weight of the body to act on the femoral head against the femoral neck, thus causing it to break. In several cases superficial abrasion and bruising of the knee and lower leg on the same side as the fracture had occurred. In case 1 an eyewitness confirmed that the patient had fallen on to the side of the fracture.

The two most noteworthy cases were the men with substantial trabecular bone volumes (22.9% and 20.4%), who cannot be regarded as being osteoporotic and who sustained subcapital fractures. In the remainder in whom the trabecular bone volume was known all had intertrochanteric fractures and were osteoporotic. These observations are compatible with Aitken's findings.

Most of the fractures (7/3) were right sided. We were not able to establish if all of these patients were right handed, although most seem to have been. There is a clear record that the three patients who sustained left sided fractures were left handed. We therefore tentatively suggest that these patients fall on to their preferred side if that is the side of weakness. In three cases the intracerebral lesions (although substantial in an anatomical sense) were not suspected clinically at the time of presentation with the fracture. In two of these (cases 5 and 8), in which the infarcts were clearly old morphologically and preceded the fractures, there were clear histories of clinical episodes of stroke some 12 and 18 months previously with definite, albeit mild, chronic hemiparesis. In the third case (4) the patient was quite ill at presentation, having sustained a recent infarct, and signs of hemiparesis were not detected. Internal fixation was performed after one week and the patient died two days later. It was thought that the cerebral infarct was more likely to have occurred at or about the time of the fracture than later at the time of operation. There is still the possibility, however, that the

infarct occurred between the fracture and the operation. In most cases, however, we feel confident that the intracerebral lesions caused a fall that resulted in fracture and could find no evidence to support the alternative hypothesis that the fracture caused the vascular intracerebral lesions.

Our conclusion is that intracerebral lesions are an important risk factors in fractured neck of femur. When the intracerebral lesion is acute there is loss of protective muscle tone and non-osteoporotic subjects are at risk as substantial body weight may act through the proximal femur in such a way as to cause a fracture. In cases of chronic hemiparesis there may also be a tendency to trip on the affected side, and there is also the possibility that the hemiparetic lower limb may have a lower bone mass than the contralateral limb.

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