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Femoral neck fracture following avascular necrosis of the femoral head.

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Abstract

Four cases of femoral neck fracture following avascular necrosis of the femoral head were studied histologically. All four patients were women who had received steroid therapy, three of them for systemic lupus erythematosus and the other for idiopathic thrombocytopenic purpura. Two types of fracture were found according to the site and the mechanism of fracture. One was at the junction between the necrotic bone and the repairing bone, and it can thus be regarded as a stress fracture. The other type of fracture commenced at the superior portion of the junction between the femoral head and neck, which was weak due to the repair reaction. The fracture line extended to the inferior cortex of the femoral neck, as often occurs in the elderly. In one patient, the femoral neck fracture was the first sign of avascular necrosis of the femoral head.

KEYWORDS: femoral neck fracture, avascular necrosis, femoral head, mechanism

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Femoral Neck Fracture Following Avascular Necrosis of the Femoral Head

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Four cases of femoral neck fracture following avascular necrosis of the femoral head were studied histologically. All four patients were women who had received steroid therapy, three of them for systemic lupus erythematosus and the other for idiopathic thrombocytopenic purpura. Two types of fracture were found according to the site and the mechanism of fracture. One was at the junction between the necrotic bone and the repairing bone, and it can thus be regarded as a stress fracture. The other type of fracture commenced at the superior portion of the junction between the femoral head and neck, which was weak due to the repair reaction. The fracture line extended to the inferior cortex of the femoral neck, as often occurs in the elderly. In one patient, the femoral neck fracture was the first sign of avascular necrosis of the femoral head.

Key words: femoral neck fracture, avascular necrosis, femoral head, mechanism

Avascular necrosis of the femoral head (ANFH) is usually a progressive condition. In general, the femoral head collapses and eventually secondary osteoarthritis occurs (1, 2). Femoral neck fracture during the course of ANFH is uncommon and there are few reports in the literature (3, 4).

In the present article, we review our experience of four femoral neck fractures associated with ANFH and discuss the pathogenesis of such fractures.

Patients and Methods

From 1972 to 1992, there were seven femoral neck fractures following ANFH out of 220 femoral heads of 140 patients seen at Okayama University Hospital. We histologically examined four cases out of seven femoral neck fractures which had occurred after ANFH.

All four patients were women on steroid therapy; three of them for systemic lupus erythematosus (SLE) (Fig. 1A-F) and the other for idiopathic thrombocytopenic purpura (ITP) (Fig. 2). The average age of onset of ANFH was 43 years (30-62 years). The average age of onset of femoral neck fracture was 44 years (31-62 years).

Four femoral heads were removed at operation. After the macroscopic examination, femoral heads were fixed with 10% buffer formalin and embedded in paraffin. Coronal sections were stained with hematoxylin and eosins (H-E). Serial sections were microscopically examined and the mechanism of these fractures was analyzed.

Results

We found two types of femoral neck fracture following ANFH. Three SLE patients had similar findings and that of ITP patient was different. We first showed the results of the typical case of SLE patients (Case 1) and then the findings of ITP patient (case 4) was presented.

Case 1. A woman developed SLE at 34 years of age and corticosteroid therapy was started immediately. The maximum dose of corticosteroids was prednisolone 10 mg/day and the maintenance dose was 5 mg/day. At 42 years of age, she developed pain in the left hip and visited our clinic. The initial roentgenogram showed flattening of the weight-bearing surface of the left femoral head and a diagnosis of ANFH was made (Fig. 1A). Six months later, left subcapital fracture occurred without any history of injury (Fig. 1B) and femoral head replacement was performed.

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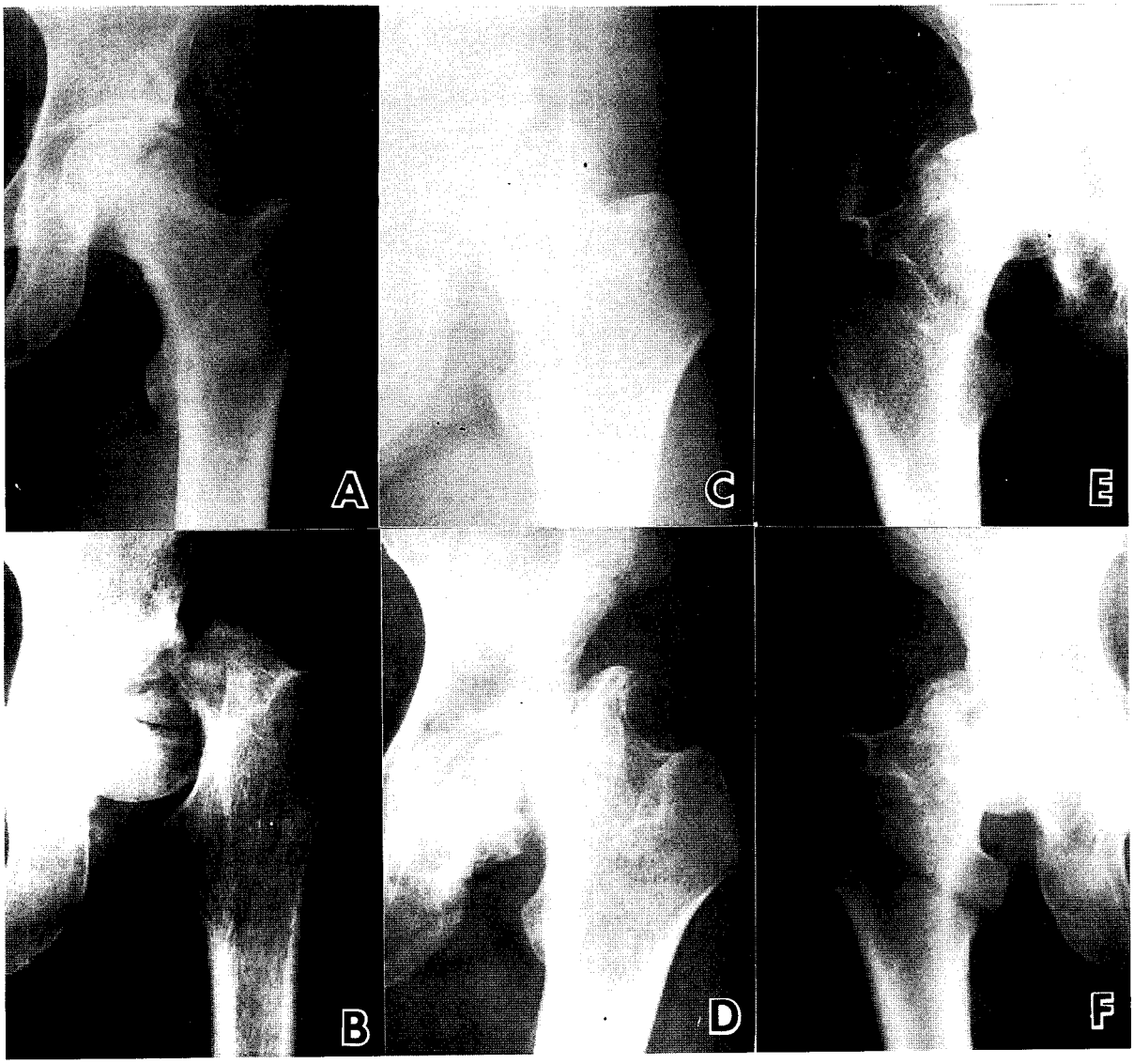

Fig. 1

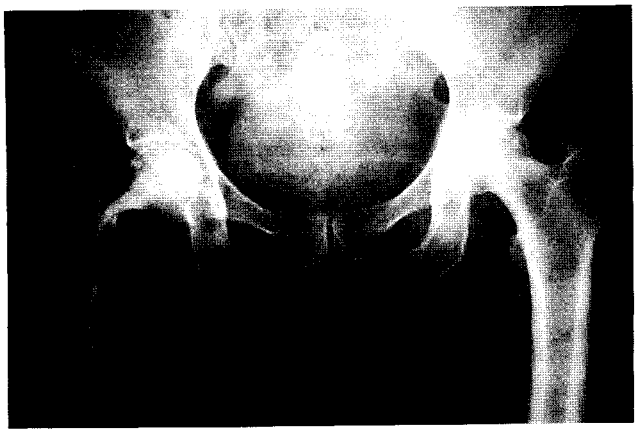

Fig. 1 Roentgenograms of three systemic lupus erythematosus (SLE) patients.
A: Case 1. A 42-year-old woman with SLE on admission.
B: Case 1. Six months later, left subcapital fracture occurred.
C: Case 2. A 38-year-old woman with SLE on admission.
D: Case 2. One year and seven months later, left subcapital fracture occurred.
E: Case 3. A 30-year-old woman with SLE on admission.
F: Case 3. Eight months later, right subcapital fracture occurred.

Fig. 2 Roentgenogram of case 4 showing right femoral neck fracture and flattening of the weight-bearing surface of the left femoral head.

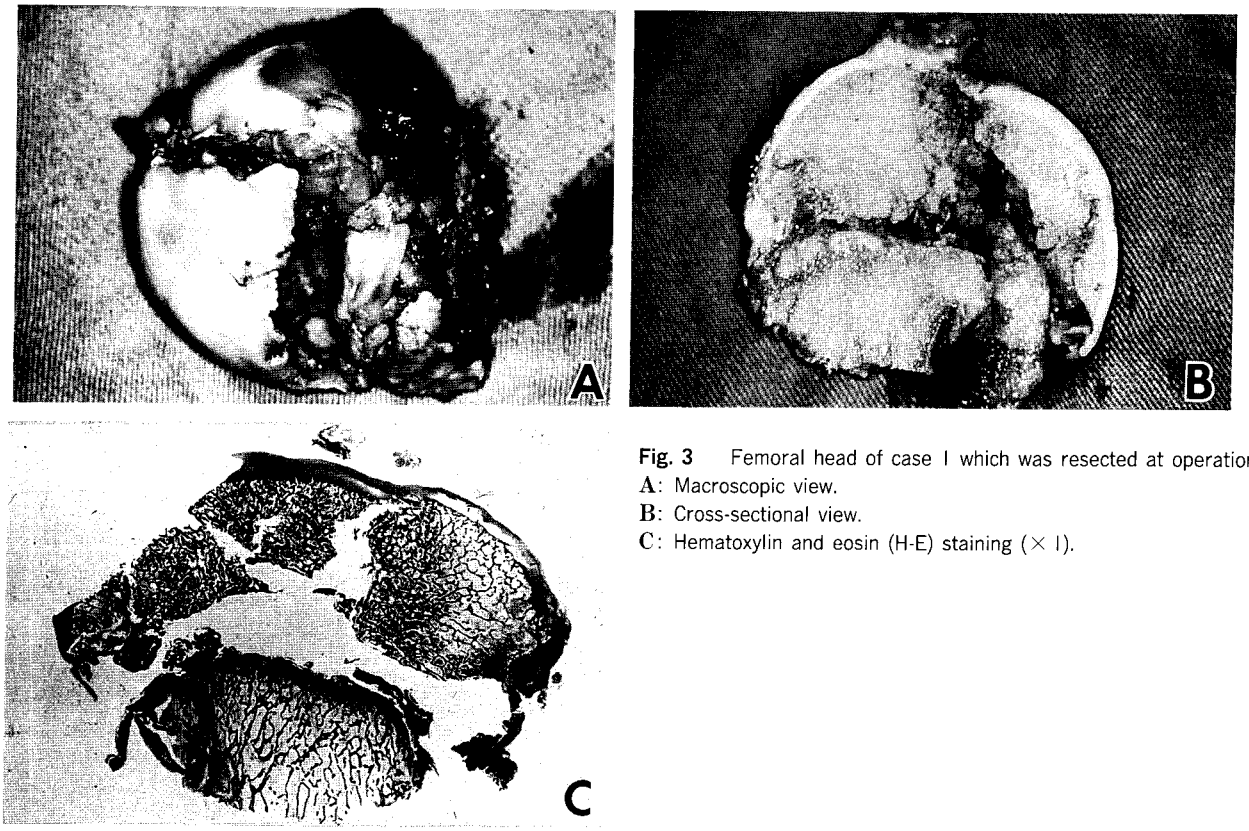

Fig. 3 Femoral head of case I which was resected at operation.
A: Macroscopic view.
B: Cross-sectional view.
C: Hematoxylin and eosin (H-E) staining (× I).

A comminuted fracture was located at the junction of the femoral head and neck (Figs. 3A–C). The femoral head fragment showed complete necrosis of the trabecular bone and marrow (Fig. 4A). The distal fragment showed evidence of a repair reaction, with the formation of woven bone (Fig. 4B) and appositional new bone formation (Fig. 4C). A relatively thick band of fibrous tissue was present between the fragments (Fig. 4D) and the fracture extended along the junction between necrotic bone and the repairing bone.

Case 4. A woman developed ITP at 61 years of age and was given corticosteroid therapy. The initial dose was prednisolone 60 mg/day and this was progressively reduced over 6 months. At 8 months after the onset of ITP, she fell down and was subsequently unable to walk because of right hip pain. Roentgenogram at her visit showed a right femoral neck fracture as well as flattening of the weight-bearing surface of the left femoral head (Fig. 2). She had no history of any symptoms related to her hips.

Femoral head replacement was performed and a transcervical fracture was found (Fig. 5A). There was no significant collapse of the femoral head, which maintained its spherical shape, but slight kinking of the lateral portion of the cartilage was noted (Fig. 5B, C).

Three-quarters of the femoral head fragment, all but the fovea centralis, showed complete necrosis (Fig. 6A). At the superior portion of the junction between the femoral head and neck, there was invading fibrous connective tissue as well as the resorption of necrotic bone and appositional bone formation, indicating a repair reaction (Fig. 6B–D). Bone resorption had produced structural weakness, increasing the likelihood of fracture at this site.

In this case, complete necrosis with slight deformity of the femoral head was accompanied with repair reaction, and a diagnosis of femoral neck fracture following ANFH was made. The femoral neck fracture was the first sign of

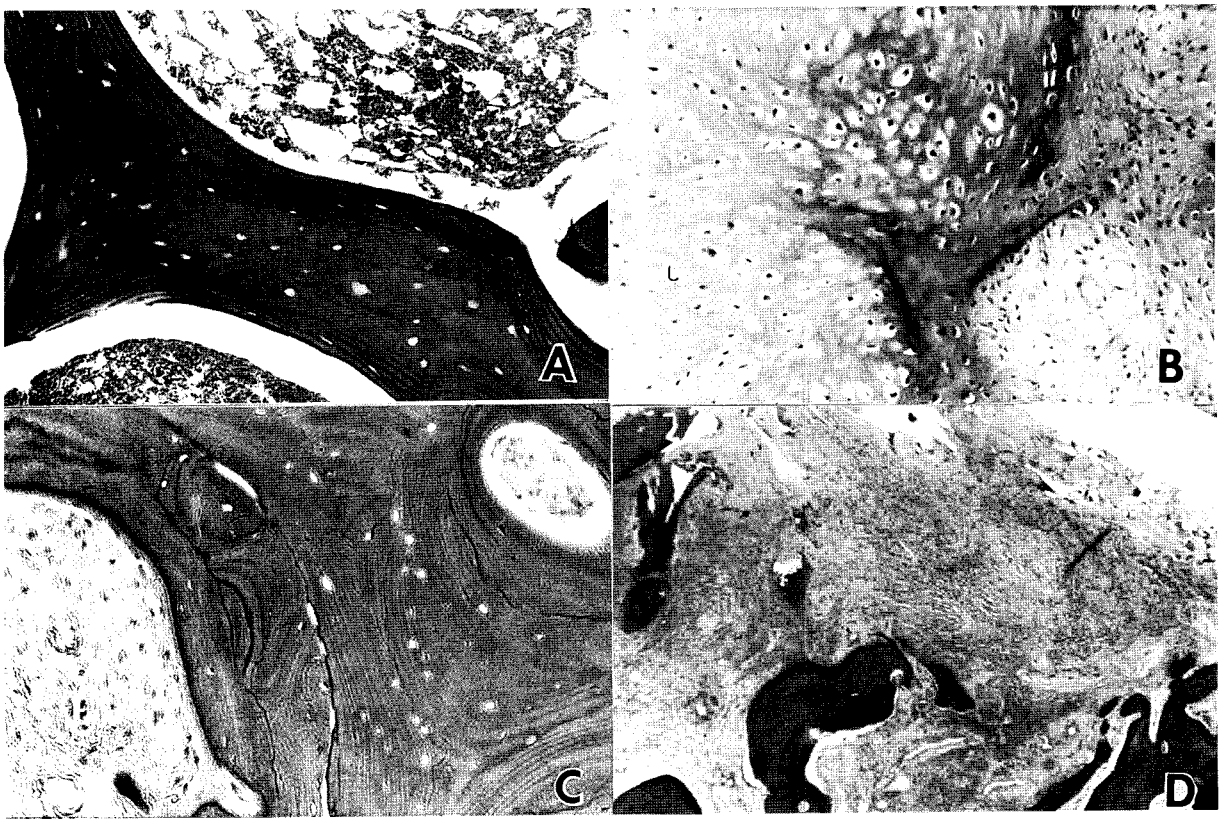

Fig. 4

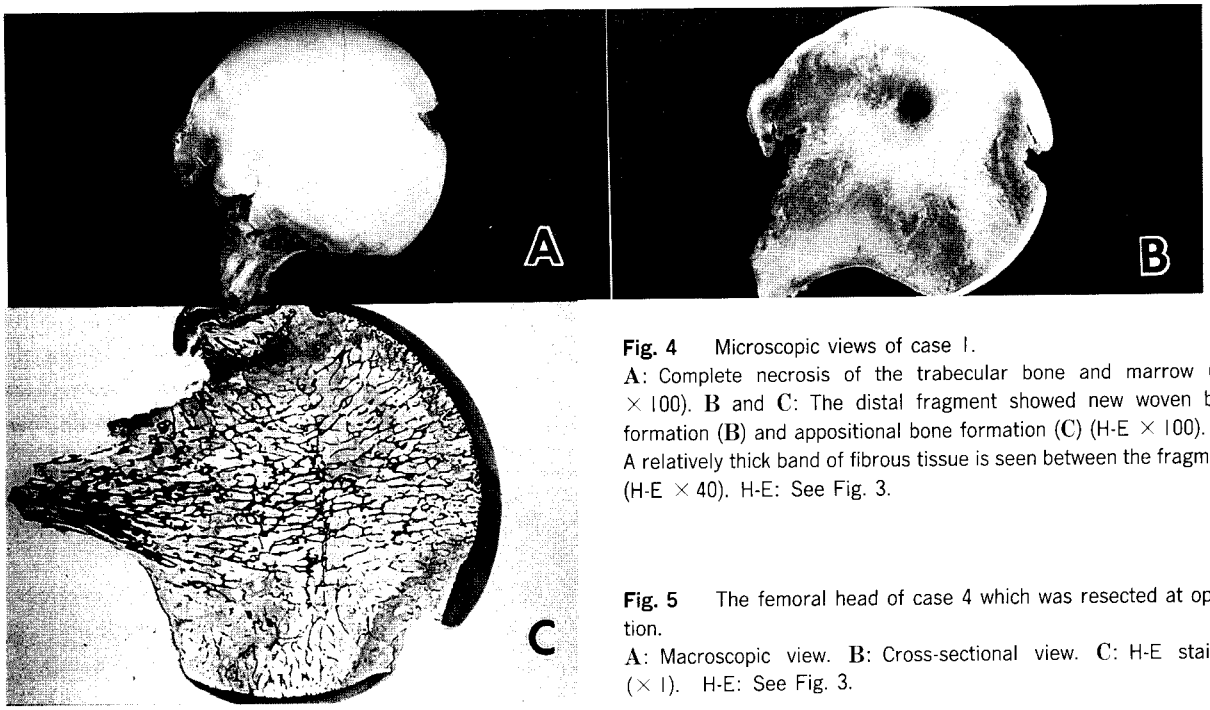

Fig. 4 Microscopic views of case 1.
A: Complete necrosis of the trabecular bone and marrow (H-E × 100). **B** and **C**: The distal fragment showed new woven bone formation (**B**) and appositional bone formation (**C**) (H-E × 100). **D**: A relatively thick band of fibrous tissue is seen between the fragments (H-E × 40). H-E: See Fig. 3.

Fig. 5 The femoral head of case 4 which was resected at operation.
A: Macroscopic view. **B**: Cross-sectional view. **C**: H-E staining (× 1). H-E: See Fig. 3.

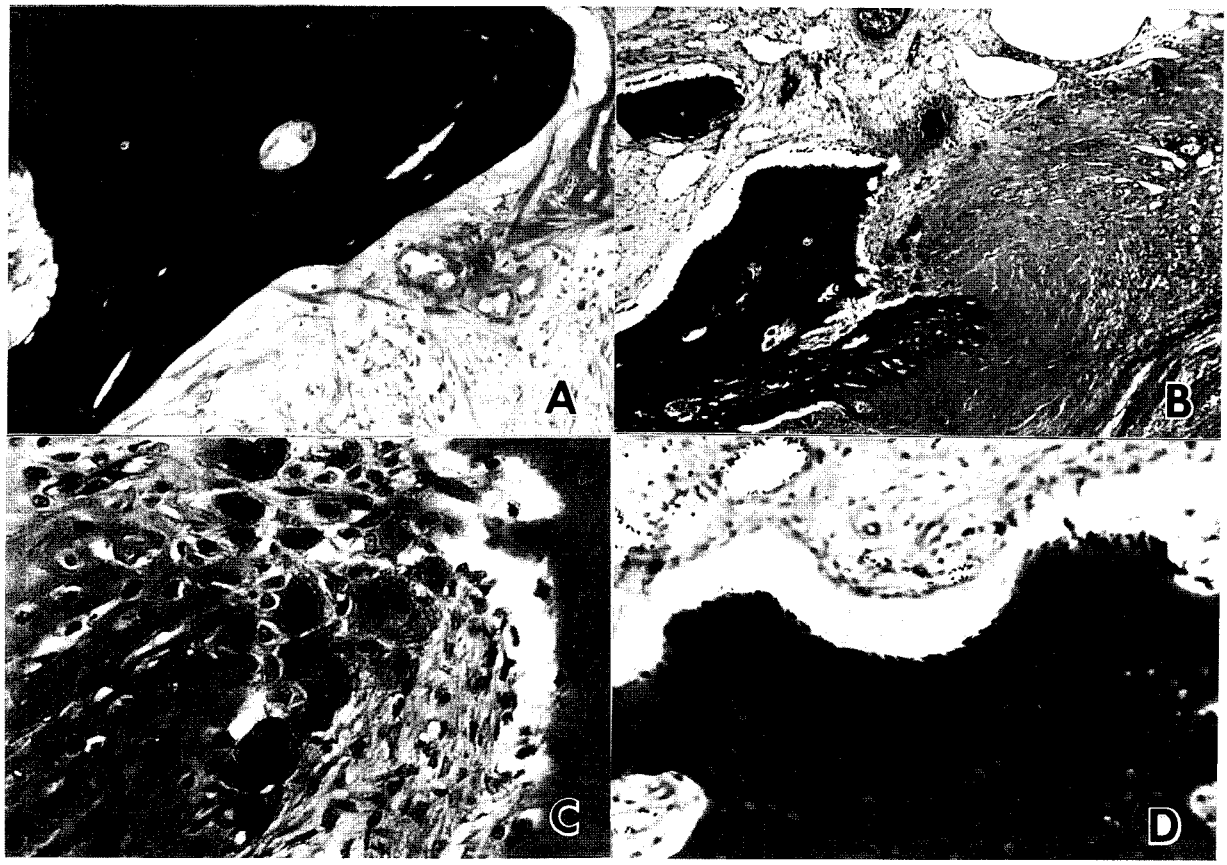

Fig. 6 The microscopic views of case 4.
A: Complete necrosis of the trabecular bone and marrow (H-E × 100). **B**–**D**: Invasion of fibrous connective tissue (H-E × 40) (**B**); resorption of necrotic bone (H-E × 200) (**C**); and appositional bone formation (H-E × 100) (**D**) were seen in the superior portion of the femoral head.

ANFH.

Discussion

In patients with avascular osteonecrosis following subcapital fracture, a fracture within the femoral head and neck was observed by Glimcher and Kenzora (5), but such fractures are rare in ANFH. In 1978, Tagawa first reported four cases of femoral neck fracture following steroid-induced ANFH and Hasegawa reported two such patients in 1986 (3, 4). At Okayama University Hospital, there were seven femoral neck fractures following ANFH out of 220 femoral heads of 140 patients seen, and four cases were studied histologically.

Regarding the mechanism of these fractures, Tagawa stated that corticosteroid administration produced secondary osteoporosis and structural weakness of the femoral neck, increasing the likelihood of fracture without any precipitating injury (4).

Based on histological data, Glimcher and Kenzora have proposed the following mechanism (5). Following avascular osteonecrosis, the resorption of necrotic bone and the invasion of fibrous tissue as the repair reaction proceeds causes structural weakness. As a result, stress is concentrated at the junction between the necrotic bone and the repairing bone due to differences in the elastic modulus and compliance of each type of bone. Accordingly, microfractures tend to occur at this junction. The cyclic loads produced by daily activities gradually increase the number of microfractures and eventually result in a macrofracture that is propagated along this junction. In case 1, the mechanism of the fracture was similar to that

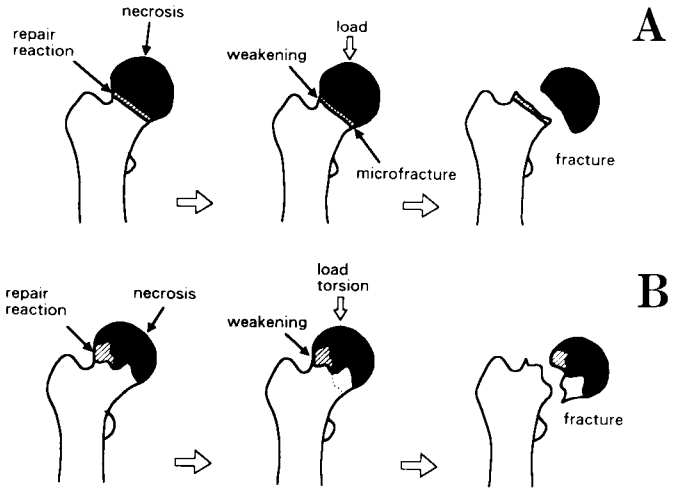

Fig. 7 Schematic illustration of the pathogenesis of femoral neck fracture following avascular necrosis of the femoral head (ANFH). A: Microfractures at the junction between the necrotic bone and the repairing bone accumulated until a macroscopic fractures was produced. B: The superior portion of the junction between femoral head and neck was weakened, and the load and torsion may have caused a fracture at this site extending to the inferior cortex of the femoral neck.

proposed by Glimcher and Kenzora. Microfractures accumulated at the junction between the necrotic bone and the repairing bone until a macroscopic fracture was produced (Fig. 7A). There was no history of injury, so this is very likely a kind of stress fracture. This appears to be the usual type of the femoral neck fracture associated with ANFH.

However, case 4 had a different clinical course and type of fracture. The onset was abrupt as is usually seen following femoral neck fracture induced by injury or trauma, while the onset of ANFH is usually insidious. The pattern of the fracture was also different, since it did not run along the junction between the necrotic and repairing bone but instead extended beyond the junction to the inferior cortex of the femoral neck. The superior portion of the junction between the femoral head and neck was weakened structurally by bone resorption and the invasion of fibrous connective tissue. Thus, the load and torsion produced by falling down may have caused a fracture at this site and it may have extended to the inferior cortex of the femoral neck (Fig. 7B). This mechanism appears to be similar to that of an ordinary femoral neck fracture in the elderly, where the superior portion of the junction between the femoral head and neck is weakened by the progression of osteoporosis (6).

In conclusion, there appear to be two types of femoral neck fracture following ANFH: the insidious stress fracture and the ordinary trauma-induced femoral neck fracture.

References

1. Merle d'Aubigne R, Postel M, Mazabraud A, Massias P and Gueguen J: Idiopathic necrosis of the femoral head in adults. J Bone Joint Surg (1965) **47**B, 612-633.
2. Patterson RJ, Bickel WH and Dahlin DC: Idiopathic avascular necrosis of the head of the femur. J Bone Joint Surg (1964) **46**A, 267-284.
3. Hasegawa I, Masuda T, Matuno T, Sugana O and Yamaguchi H: Two cases of secondary femoral neck fracture following aseptic necrosis of the femoral head. Rinsho Seikei Geka (1986) **21**, 1171-1175 (in Japanese).
4. Tagawa H, Ninomiya S, Miyanaga Y and Okutsu I: Clinical features of the steroid-induced necrosis of the femoral head; in Annual Report of the Japanese Investigational Committee for Avascular Necrosis of the Femoral Head. Ministry of Health and Welfare, Tokyo (1978) PP 19-24 (in Japanese).
5. Glimcher MJ and Kenzora JE: The biology of osteonecrosis of the

human femoral head and its clinical implication. Clin Orthop (1979) **140**, 273-312.
6. Garden RS: Low-angle fixation in fractures of the femoral neck. J Bone Joint Surg (1961) **43**B, 647-663.

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