The prevalence and clinicopathological appearance of extension of osteonecrosis in the femoral head

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In about 50% of cases, osteonecrosis of the femoral head is known to occupy more than one site. There is controversy as to whether a single focus may increase in size. We have reviewed 606 consecutive femoral heads which had been surgically removed for osteonecrosis. Extension of osteonecrosis was observed in only two (0.3%) and was confirmed histopathologically by the enlargement of the necrotic segment beyond the repair zone formed for the primary necrosis into the adjacent, previously uninvolved bone. In both cases, the necrotic regions were wedge-shaped and occupied over 80% of the femoral head.

It appears that an increase in size is extremely rare and that osteonecrosis is due to a single event. Our findings may be of value in assessing the use of joint-salvage procedures for osteonecrosis of the femoral head.

Materials and Methods

From 1993 to 1997 we examined 606 consecutive femoral heads which had been removed from 547 patients for non-traumatic ON of the femoral head. There were 314 women and 233 men with a mean age of 41 years (14 to 87). We reviewed the gross photographs, specimen radiographs, and microscopic sections in each case.

Immediately after removal, all femoral heads had been photographed and fixed in 10% formalin solution. In each, a 5 mm thick mid-coronal section had been obtained using a band saw, and the section both photographed and radiographed using low voltage X-rays (Faxitron, Buffalo Grove, Illinois) and fine-grain films (Kodak, Rochester, New York). After decalcification in 5% nitric acid solution for three days, the sections had been processed, embedded in paraffin and full slab sections 5 µm thick had been prepared. They had been stained with haematoxylin and eosin.

Histopathological definition of the extension of osteonecrosis. In ON, repair tissue is formed at the junction of the dead and living bone. There is an initial inflammatory response with an accumulation of macrophages, followed by the formation of vascular-rich granulation tissue, infiltration of fibrous scar, and creeping bone substitution.3-5,10,11 These histological characteristics have been commonly used for the determination of a time sequence after the ischaemic events even in advanced ON. In our
study, the diagnosis of extended ON was based on the enlargement of the osteonecrotic segment beyond the primary lesion to involve both the peripheral repair tissue, formed for the primary necrosis, and the adjacent previously uninvolved cancellous bone (Fig. 1). Focal necrosis of the repair tissue around an area of fracture-collapse was not considered to be extended ON, since it is more likely to have resulted from the collapse and the resultant injury than from a recurrent ischaemic event.

The histopathological examinations were performed independently by three pathologists with a primary interest in orthopaedic pathology.

Results

Based on both gross and microscopic examination, extension of ON was identified in only two of the 606 femoral heads (0.3%). In these, evidence of extended ON was reconfirmed by three-dimensional assessment of the necrotic area on serial 5 mm cuts through the entire femoral head.

Based on the specimen radiographs and microscopic examinations, 597 of the 606 femoral heads (98.5%) had undergone collapse secondary to fracture; the remaining nine (1.5%) showed no evidence of collapse. Microscopic evidence of focal necrosis of the repair tissue was observed around the fracture site in 590 (98.8%) of the collapsed femoral heads.

Illustrative case reports

Case 1. A 58-year-old man had complained of pain in his left hip for approximately one year. There was no history of corticosteroid intake, alcohol abuse, or other related conditions. The range of movement in the left hip was 120° flexion, 20° abduction, 0° adduction, 20° external rotation and 5° internal rotation. Radiographs showed slight flattening of the superior portion of the femoral head and an obvious subchondral fracture (crescent sign) on the lateral view. A curvilinear sclerotic rim was seen defining the anterosuperior one-third of the femoral head (Figs 2a and 2b). Serial radiographs were not available and MRI had not been performed.

The excised femoral head showed a slightly flattened superior surface with a flap of articular cartilage and subchondral bone, 3 × 3 cm in size, which was partially attached at its margin. On the cut section, over 80% of the head showed the opaque yellow colour of osteonecrosis. Two distinct areas were apparent (Fig. 2c). The first, located in the upper quarter of the head, represented the primary focus, and was bordered by a well-defined dense sclerotic margin, which corresponded to the curvilinear sclerotic rim seen in both the clinical and specimen radiographs (Fig. 2d). The second necrotic area, at least four times the size of the primary area, totally encompassed the primary focus and had an irregular border which extended from the superolateral articular surface down into the medial part of the femoral neck. The margin of the extended area of infarction was not discernible on the clinical radiographs, but on the specimen film an osteopenic area was observed at the junction between the secondary necrosis and the viable area, especially at the superolateral portion of the femoral head (Fig. 2d).

Microscopically, the boundary between the primary and secondary infarcts consisted of necrotic repair tissue, including fibrous tissue and prominent lamellar bone layered on the surfaces of the pre-existing trabeculae (Fig. 2e). The boundary between the secondary osteonecrosis and the adjacent viable bone consisted of vascular granulation tissue and fibrous tissue with only a small amount of appositional bone (Fig. 2f). Microscopic differences between the appearance of the necrotic bone or marrow tissue in the primary and secondary regions were not discernible.

Case 2. A 76-year-old man had complained of pain in the right hip for two months. There was no history of corticosteroid intake, alcohol abuse or other related conditions. Left total hip arthroplasty had been performed in another institution ten years previously for osteoarthritis but the histological findings were not available. The range of movement in the right hip was 90° flexion, 10° abduction, 25° adduction, 15° external rotation, and 5° internal rotation. Radiographs showed joint-space narrowing and migration centromedially, suggesting medial osteoarthritis. A sclerotic rim was seen extending from the superolateral
The articular surface to the medial edge of the femoral head. In the lateral view, a region of collapse was visible on the anterior surface. When the films were reviewed again for this study it was evident that within the area surrounded by the sclerotic rim there was a second poorly-defined wedge-shaped area which was also subtly seen as a sclerotic line in the anterosuperior region of the head (Figs 3a and 3b).

On gross section, necrosis was seen in an area occupying more than 75% of the femoral head. Within this necrotic area, a second wedge-shaped area could be seen sub-articularly as a patchy poorly-defined area (Fig. 3c). On the specimen radiograph, this wedge-shaped area was also observed in the upper quarter of the femoral head and was surrounded by a patchy osteolytic area. Beyond this osteolytic zone, an obvious sclerotic rim was seen separating the necrotic area from the viable bone (Fig. 3d). The radiological and gross appearance of this second wedge-shaped region suggested the possibility of extension, and this was confirmed by the microscopic examination.

Microscopically, the boundary between the necrotic and viable areas consisted of granulation tissue and a prominent amount of appositional bone, which corresponded to a sclerotic rim on the radiographs. Another zone of dead repair tissue comprising a minimum amount of appositional bone and fibrosis was observed around the second wedge-shaped area (Figs 3e and 3f), which was considered to have been the boundary of the primary osteonecrosis. The patchy osteolytic area around the second wedge-shaped region consisted of thin disconnected bone trabeculae.

**Discussion**

In a histological study of 40 cases of ON, 83% were said to show evidence of recurrent episodes. Sequential MRI studies, however, have not demonstrated the presence of recurrent ON. There have been many reports of a success rate of around 70% to 80% for joint-salvage procedures in this condition. These paradoxes may be resolved by con-
Considering the definition of ‘recurrence’. In our opinion, the diagnosis of recurrent ON should not include the focal necrotic regions caused by collapse at the junction between the infarcted and the adjacent viable area. Such focal necrosis is not the result of reinfarction but is caused by the injury consequent to the collapse itself. All fractures give some degree of focal tissue necrosis. Histological findings of ON after collapse are often confusing because of the disruption of the repair tissue caused by the further collapse and repair of this damaged tissue. In our study focal necrosis of both the repair tissue and the damaged tissue was observed in about 98% of the collapsed femoral heads at the site of collapse. An extended area of ON caused by a recurrent ischaemic episode was extremely rare.

In the two cases of extended ON which we recognised, the final area of infarction occupied over 80% of the femoral head, while the primary necrosis was less than one-quarter of the final lesion. The area of primary osteonecrosis was totally encompassed by the second lesion. Such extension of the necrotic area is similar to that seen in recurrent necrosis in myocardial infarction, and can be explained by the extension of the circulatory deficiency.

While extension of the area of infarction is relatively common in the heart it seems to be exceedingly rare in the femoral head.

The histopathological characteristics of the repair tissue for primary ON are useful in assessing the time delay between the first and recurrent ischaemic episodes. This
tissue has been considered to occur at the junction between the necrotic and viable area in the following order, accumulation of macrophages, granulation tissue, fibrous tissue and formation of appositional bone.3-5,10,11 The prominent formation of appositional bone observed between the primary and secondary osteonecrosis in case 1 may indicate the relatively long period, several months, between the two ischaemic episodes. By contrast, little or no apparent evidence of a sclerotic rim radiologically, and minimal but definite formation of apposition bone microscopically between the primary and secondary osteonecrosis in case 2, would indicate a shorter period between the two episodes.

A sclerotic rim on the radiograph has generally been used as the marker of the boundary between ON and the viable area, since histopathologically it represents the formation of appositional bone to the necrotic bone trabeculae at the reactive interface. In case 1, the sclerotic rim on the radiographs was the boundary between the primary and secondary ON. Radiography could not detect the most recent boundary between the necrosis and viable area because of the small amount of formation of new bone. Therefore a sclerotic rim on conventional radiographs does not necessarily correspond to the marker of the boundary between necrosis and the viable area. MRI will detect the repair process before conventional radiographs7,8 and thus would be useful for evaluating the necrotic area and detecting these rare cases of extended ON.

Our study shows that extension of ON is extremely rare and that a single ischaemic event is the most common cause of ON. Our findings may be useful not only for considering the pathogenesis of ON and evaluating the risk of collapse but also in terms of the use of joint-salvage procedures for this disease.

No benefits in any form have been received or will be received from a commercial party related directly or indirectly to the subject of this article.

References