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The natural history of bone bruise and bone remodelling in the traumatised hip A prospective 2-year follow-up study of bone bruise changes and DEXA measurements in 13 patients with conservatively treated traumatic hip dislocations and/or fractures

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ABSTRACT

Introduction: The purpose of this study was to assess the natural history of bone bruise and bone mineral density (BMD) after traumatic hip dislocations and conservatively treated acetabular fractures. Our hypothesis was that poor bone quality can influence degree of bone bruise and, in time, cause degenerative changes.

Materials and methods: Eight consecutive patients with traumatic hip dislocations and five patients with conservatively treated fractures in the femoral head and/or acetabulum were included. Magnetic resonance imaging (MRI) was obtained after 1, 17, 42, 82 and 97 weeks. Dual-emission X-ray absorptiometry (DXA) measurements were made after 10 days and 2 years. Sizes of bone bruise lesions were measured and classified. At the 2-year follow-up, Harris hip score (HHS) was calculated and signs of radiological osteoarthritis (OA) registered.

Results: The bone bruise changes were small and all changes resolved within 42 weeks in all, except for three patients; one with a small Pipkin fracture had segmental avascular necrosis (AVN) of the femoral head, one had persisting1–3 mm small spots of bone bruises in the femoral head and the third had <1 cm lesions in both the femoral head and the acetabulum. The lesions were bigger in the femoral head in the hip dislocations and more pronounced in the acetabulum in the fractured acetabuli. We found no significant changes in BMD in four regions of interest (ROIs) after 2 years. No patients developed OA, and all had excellent HHS except for the one patient with AVN.

Conclusion: The post-traumatic bone bruise changes in the dislocated hips and the fractured acetabuli were small and transient compared to findings of other authors examining traumatised knees. The patients had excellent function and no OA after 2 years if they did not develop AVN. In our small sample of relatively young patients with normal age-adjusted BMD, no post-traumatic osteopenia was observed. This might differ in the elderly with poorer bone quality; further studies are needed to assess that.

Introduction

Despite anatomic fracture reduction and stable fixation, some traumatised joints develop secondary osteoarthritis (OA). The aetiology of this development remains largely unknown. In the knee, focus has been centred on the amount of bone bruise measured by magnetic resonance imaging (MRI) and poor bone quality measured by dual-emission X-ray absorptiometry (DXA) as predictors of OA, whereas these associations have been sparsely evaluated in the traumatised hip joint.

Bone bruise was first described in MRI after knee injuries by Yao and Lee,[1] and is usually the result of trauma to cancellous bone. The changes in bone are occult, detectable by MRI, but not by conventional radiographs.[2,3] During arthroscopy, the changes

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may be hidden by a normal-looking overlying cartilage.[4] Histopathology and cryosections of human bone bruise lesions have shown microfractures of cancellous bone and weight-bearing trabeculae, oedema and bleeding of fatty marrow, which correlate with the lesions detected on MRI.[5] Many studies conducted on traumatic bone bruise lesions have focussed on the knee joint; that is, bone bruise occurs in 80% of patients with *anterior cruciate ligament* (ACL) rupture of the knee.[2,4,6] Bone bruise is frequently present after severe ankle sprains or wrist trauma.[7–9] Damage to articular cartilage is considered a major risk factor for later degenerative joint changes, as cartilage has very limited healing potential.[3,10,11] According to a review on the natural history of bone bruise lesions in the knee, the percentage of complete resolution of the lesions ranges from 88% after 11–16 months to 100% after 2–12 months of follow-up.[12]

The bone structure, form and density of the hip are different from that of metaphyseal, cancellous bone of the knee. The hip joint has a ball and socket shape and might be less susceptible to bone bruise changes. Our hypothesis was that poor bone quality may influence the degree of bone bruise and, hence, in time, cause degenerative changes. Thus, in the present work we studied the natural history of bone bruise changes and bone remodelling in patients with traumatised hip joints acutely and 2 years after trauma.

Material and methods

Eight consecutive patients with traumatic hip dislocations and five patients with conservatively treated fractures affecting the femoral head or acetabulum were included prospectively at our level one trauma centre at Oslo University Hospital, Ullevål, Norway, from August 2007 to February 2009. Standard radiographs and computed tomography (CT) scans of the pelvis and injured hip were performed at admittance. Dislocations were reduced as soon as possible. Our post-reduction and conservative fracture treatment protocol consisted of early active and passive range of motion exercises and hip dislocation precautions, toe-touch weight bearing for 8 weeks, before full weight bearing. MRI and DXA measurements of both hips were performed within 10 days after trauma. Radiographs and MRI were repeated after 4 months and then every 6 months until 2 years after trauma. DXA measurement of both hips was repeated at the 2-year follow-up. Time from dislocation to hip joint reduction was calculated.

Several classification systems have been developed for grading and locating bone bruise lesions.[1,3,10,13–15] These classification systems focus mostly on localisation and proximity to the joint and cartilage damage, and less on the size of the lesions. Most are designed especially for evaluating changes in the knee. We classified the bone bruises in the femoral head according to Costa Paz et al.[15]: type 1 has a diffuse MRI signal with change of the medullary component, often reticular and distant from the adjacent articular surface. Type 2 is a localised signal with continuity to the adjacent articular surface, usually crescentic lesions with variable thicknesses (Fig. 2). Type 3 lesions involve disruption or depression of the normal contour of the cortical surface often associated with a type 2 lesion (Fig. 3). Hence, an acetabular fracture will always be a type 3 lesion. MRI examinations were done at the following intervals: 1 week (MRI 1), 17 weeks (MRI 2), 42 weeks (MRI 3), 82 weeks (MRI 4) and 97 weeks (MRI 5). The maximum sizes of all the bone bruise changes and oedema and/or rupture of the muscles of the hip were measured on all MRIs. The MRIs were obtained using a 1.5 Tesla Philips Gyroscan ACS-NT (Best, the Netherlands), with a body surface coil. Turbo spin echo images were obtained: T1 weighted and short TI inversion recovery (STIR) in the axial and coronal plan with proton density fat saturation (PDFS) weighted images of the affected hip in the sagital plane. The maximum width and location of bone bruise changes were evaluated in all scans using the STIR images.

DXA measurements were performed approximately 1 week after trauma with a Lunar Prodigy machine (GE Healthcare, Madison, USA, software version enCore 11.4), with the patient supine and both hips rotated inwards, with a fixed distance from bench to scanner. The measurements were repeated after 2 years. Two measurements were performed of both hips, the mean value was calculated and the healthy side served as control. We selected four regions of interest (ROIs) in the acetabulum and femoral head with a fixed areal for bone mineral density (BMD) measurements (Fig. 1), and compared the results. Because of the small selection of patients, the non-parametric Wilcoxon's test was used to calculate correlation.

The patients were included and followed up prospectively by the authors AKBW or SØ, according to our well-established protocol for acetabular fractures as previously described.[16] The study was approved by the South-Eastern Regional Ethical Committee for Medical and Health Research in Norway in 2007 (acceptance number 1.2007.51) and the patients gave written informed consent to the work.

Results

There were 10 male and 3 female patients, with a mean age of 32.1 (18–58) years. Mean time from accident to reduction of dislocation was 3.4 h (range 36–580 min) (Table 1). Patient 1 differed from the rest of the group; he was involved in a car accident, severely intoxicated and trapped in the car wreck in a remote place for approximately 8 h before he was found and cut loose. It took 10 h from accident to reduction of his anteriorly dislocated hip. Mean follow-up time was 25.2 months (range 23.7–29.3 months).

Size and localisation of bone bruise changes: The bone marrow lesions were mainly small in diameter; thus, a volumetric measurement was considered unreliable and unsuitable (Table 2, Figs. 2 and 3). The anterior dislocations had changes posterior in the femoral head, and the posterior dislocations had changes in the

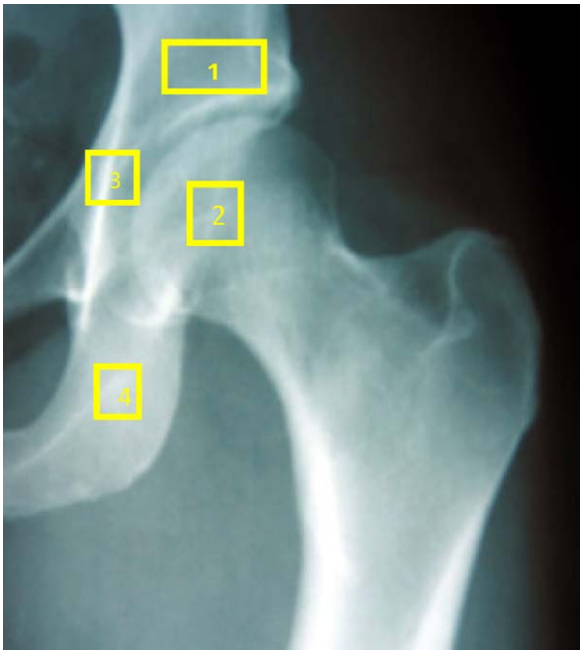

Fig. 1. The regions selected as regions of interest (ROI) for measuring BMD are shown in the figure; area 1 is 2 cm^2 and area 2, 3 and 4 are all 1 cm^2.