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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

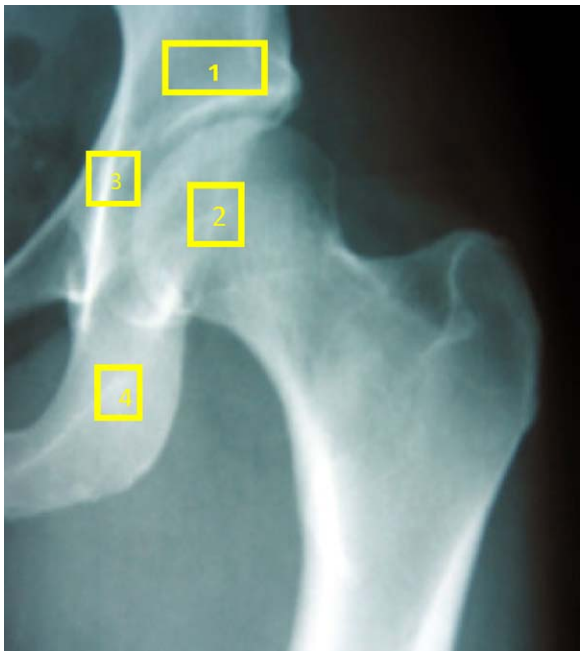

Fig. 1. The regions selected as regions of interest (ROI) for measuring BMD are shown in the figure; area 1 is 2 cm^2 and area 2, 3 and 4 are all 1 cm^2.

Table 1
Overview of patients included.

	Gender[a]	Age (years)	Harris hip score at two years	Dislocation[b]	Time from dislocation to reduction in minutes	Fracture type	Mechanism of trauma	Mean age adjusted *T*-score[c]
1	M	27	80	2	580	Undislocated thin Pipkin fracture type 1	MVA	0.20
2	M	56	100	0		Posterior column	Fall from height	0.94
3	M	18	100	1	36		MVA	−1.75
4	M	37	100	2	60		MVA	1.69
5	M	22	100	1	124		MVA	−0.22
6	M	18	100	1	303		Sports accident	−0.43
7	M	58	96	0		Anterior column posterior hemitransverse	Fall from height	−1.50
8	M	19	100	1	320		MVA	−0.86
9	F	29	100	1	107		Bicycle accident	0.57
10	F	18	100	0		Anterior wall of acetabulum	Fall from height	0.35
11	F	35	96	0		Transverse with small posterior wall	Sports accident	−1.16
12	M	38	100	0		Anterior column	Sports accident	0.69
13	M	20	100	1	111		Sports accident	0.20

MVA, motor vehicle accident.
[a] M, male and F, female.
[b] Dislocation: 0 = No dislocation, 1 = posterior, and 2 = anterior dislocation.
[c] Mean adjusted *T* score for the patients in healthy side at first examination.

anterior part. Two patients with posterior dislocation had type 3 bone bruise changes, one patient with anterior luxation had type 3 changes, four of the dislocations had type 2 changes and one patient had type1 changes, all according to Costa Paz' classification.

MRI changes with time: The bone bruise lesions tended to resolve within 42 weeks (Table 2); the bigger the lesions, the longer the time to resolution. Only in three patients did the bone bruises persist; the first had segmental avascular necrosis (AVN) of the femoral head (Fig. 3), the second had 1–3 mm spots of bone bruises in the femoral head and the third had <1 cm lesions in both femoral head and acetabulum. One patient (number 13) did not attend the last MRI examination. He had small residual bone bruise lesions left on his MRI after 42 weeks, but a Harris hip score (HHS) of 100 points and normal radiographs at the final follow-up. No patients had arthritic changes on MRI or radiographs at the final follow-up.

Muscle damage: All seven patients with a posterior hip dislocation had affection of the small external rotator muscles; three had ruptures and the rest oedematous muscles. One patient with an anterior hip dislocation also demonstrated ruptured small external rotators, whereas none of the included fractures had ruptured rotators. The oedema had completely disappeared at 42 weeks. No patients had ruptures of any of the gluteal muscles, only oedema, which had disappeared at 17 weeks.

Function: Patient 1 developed AVN of a small Pipkin fragment. He was the only one who had not returned to the same work as before trauma at the 2-year follow-up. He had an HHS of 80 points partly due to a slight reduction of muscle strength after a cervical spine fracture. Ten patients had excellent hip function with an HHS of 100 points; two had HHS of 96 points.

DXA measurements: Patient 1 did not complete the first DXA measurement due to a surgically treated, unstable cervical spine fracture. The other patients had normal age-adjusted BMD (*T*-scores) measured in the healthy hip at the first exam (Table 1). When comparing BMD in the different ROIs from the first DXA measurement with the second, we found no statistically significant group differences, and we also did not find any differences when comparing the injured and uninjured sides (Table 3).

Table 2
Bone bruise changes in the femoral head, femoral neck and acetabulum. The three first columns show maximum size in diameter of the bone bruises. The last column shows the classification of bone bruise in the femoral head according to Costa-Paz during the two years follow-up. The posterior dislocations are no longer outlined in grey.

ID	Dis-location[a]	Bone bruise, size[b] Femoral head					Bone bruise, size[b] Femoral neck					Bone bruise, size[b] Acetabulum					Costa-Paz type 0–3[c] Femoral head				
		MR1	MR2	MR3	MR4	MR5	MR1	MR2	MR3	MR4	MR5	MR1	MR2	MR3	MR4	MR5	MR1	MR2	MR3	MR4	MR5
2	0	0	0	0	0		0	0	0	0		3	1	0	0		0	0	0	0	
7	0	1	1	1	1	0	1	0	0	0	0	3	3	2	2	1	1	1	2	1	0
10	0	1	0	0	0		0	0	0	0		3	2	0	0		1	0	0	0	
11	0	0	1	0	0	0	0	1	0	0	0	3	1	0	0	0	0	1	0	0	0
12	0	1	2	1	0	0	1	2	1	0	0	3	3	1	0	0	2	2	1	0	0
3	1	1	0	0	0		1	0	0	0		1	0	0	0		1	0	0	0	0
5	1	3	0	0	0		2	0	0	0		1	1	0	0		3	0	0	0	
6	1	3	2	1	0		2	1	1	0		2	0	0	0		2	1	1	0	0
8	1	3	1	1	0	0	0	0	0	0	0	2	2	1	1	0	3	1	0		
9	1	2	3	0	0	0	2	3	0	0	0	1	1	0	0	0	2	3	0	0	0
13	1	2	2	1			2	1	1			2	0	0			2	2	2		
1	2	2	2	2	1	1	2	0	0	0	0	2	0	0	0	0	3	3	3	3	3
4	2	2	2	0	0		2	1	0	0		1	2	0	0		2	2	0	0	

Mean time in weeks from trauma to MRI1 = 1, to MRI2 = 17, to MRI3 = 42, to MRI4 = 82, to MRI5 = 97.
[a] Dislocation: 0 = no dislocation, 1 = posterior, and 2 = anterior dislocation.
[b] Size of bone bruise: 0 = no bone bruise, 1 = small spots 1–3 mm, 2 = area less than 1 cm, 3 = bigger areas 1–4 cm and 4 = the whole bone.
[c] Costa Paz' bone bruise classification: Type 1: diffuse signal with change of the medullary component, often reticular and distant from the subjacent articular surface, Type 2: localized signal with contiguity to the subjacent articular surface, and Type 3: disruption or depression of the normal contour of the cortical surface often associated with a Type 2 lesion.

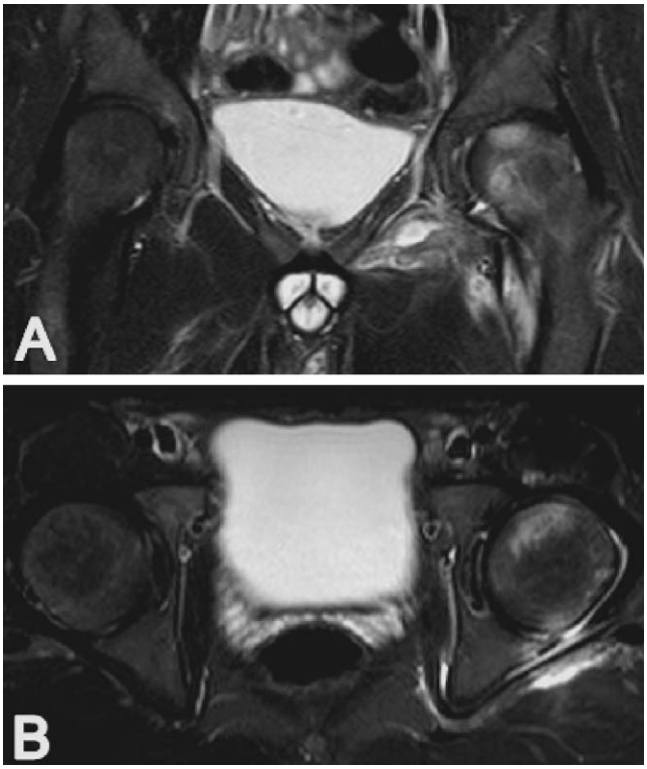

Fig. 2. Type 2 bone bruise in patient number 6s left hip ((A) coronal images and (B) axial) after a posterior luxation, both STIR weighted Images 1 week after trauma. The cartilage is not affected, the crescentic bone bruise lesions are adjacent to the cartilage.

Discussion

In this study, to our knowledge the first evaluating bone bruise and BMD changes after high-energy trauma to the hip joint, the degree of bone bruise was modest. We saw no BMD changes, and there were no visible arthritic changes evaluated by radiographs and MRI in the hips after 2 years. Only one patient developed segmental AVN. A noticeable reduction of the changes was observed from the first MRI to the second MRI at 4 months, but most changes did not disappear until 10 months. Only three patients had bone bruise type 3 changes at the first MRI, but except for patient 1, with AVN of a Pipkin fragment, these were gone or reduced to type 1 at 4 months.

Experimental animal studies have shown that acute subchondral fractures predict degenerative changes in the knee within 6 months after trauma.[17] Costa Paz et al.[15] found that all type 1 lesions and 91% of type 2 lesions disappeared after 2 years in ACL-reconstructed human knees, but all type 3 lesions had evidence of persistent cartilage thinning or cortical depression on MRI. In Vellet et al.'s[3] work, 14 of 21 patients had persistent osteochondral changes, including cartilage thinning, and cortical impaction in the knee 1 year after injury. Other authors show that the bone bruise changes resolve within a few months after trauma.[9,18,19] All of these studies are based on examinations of knees, not hips. In the ankle, Alanen et al.[9] found bone bruise changes in 27% of 95 patients with inversion injuries and no fracture. They concluded that bone bruises had very little clinical significance after inversion injuries of the ankle, although the study had a weakness of a very short follow-up time. Damage to articular cartilage is considered a particular risk factor for later degenerative changes in a joint. Bone bruises with subchondral or osteochondral injuries may persist years after trauma and might present possible precursors of post-traumatic arthritic changes, at least in the knee. Our findings in the hip in a small sample size in relatively young patients do not directly support this. Influence of the articular cartilage of the femoral head in the hip dislocations was only seen in three patients. All the acetabular fractures had affected articular cartilage, hence a type 3 change according to Costa Paz, but even so, most lesions were resolved at the third MRI (42 weeks), and no patients developed OA in 2 years.

The importance of treating a dislocated hip as an emergency has long been established, to avoid complications such as AVN and secondary OA.[20–27]There is, however, some debate on the time

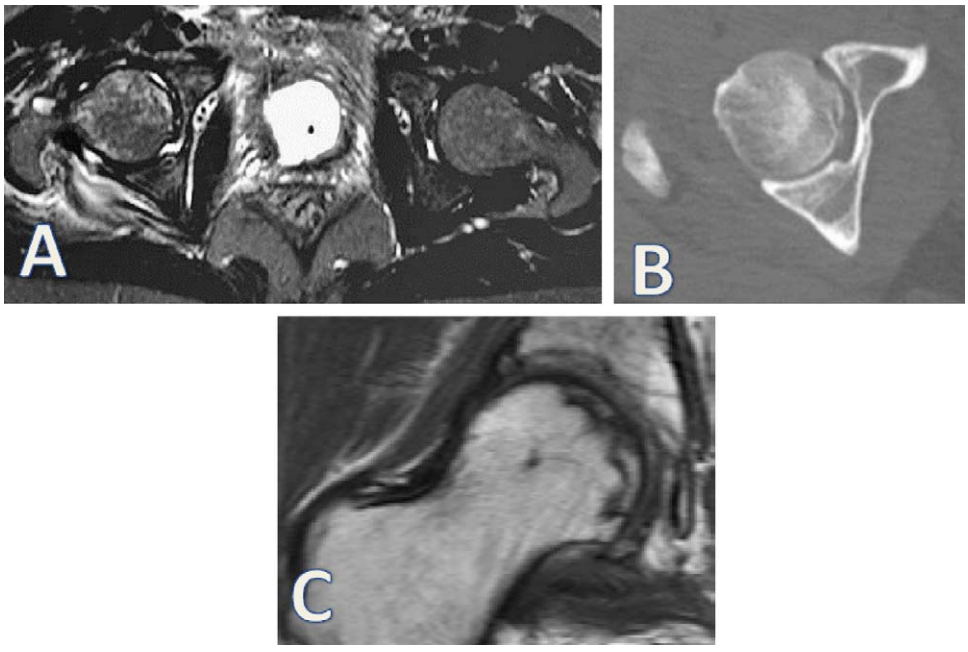

Fig. 3. (A) STIR MRI of patient 1 a week after trauma, the cartilage is affected with adjacent crescentic bone bruise (white arrow), hence a Type 3 lesion. (B) CT scan that illustrates a small Pipkin fracture (black arrow) in the right hip. (C) The T1 weighted MRI picture shows the injured hip at two year follow-up, it is still a type 3 lesion according to Costa Paz', but the bone bruise is smaller in size.

Table 3
BMD in the regions of interest (ROIs) at 1st and 2nd exam in all patients.

ID		BMD 1st exam	BMD 2nd exam	BMD 1st exam	BMD 2nd exam
		Injured side		Healthy side	
2	ROI 1	1.485	1.358	1.342	1.291
	ROI 2	1.458	1.553	1.995	1.679
	ROI 3	1.241	1.281	1.359	1.552
	ROI 4	0.856	0.884	0.982	1.067
3	ROI 1	1.750	1.655	2.472	1.678
	ROI 2	1.465	1.358	2.189	1.512
	ROI 3	2.010	1.949	1.825	1.863
	ROI 4	1.574	1.196	1.484	1.163
4	ROI 1	1.777	1.471	1.180	1.308
	ROI 2	1.816	1.905	1.924	2.116
	ROI 3	1.235	1.134	1.246	1.332
	ROI 4	0.877	1.084	0.885	0.917
5	ROI 1	1.811	1.315	1.878	1.767
	ROI 2	2.023	2.244	2.172	2.242
	ROI 3	1.661	1.695	2.046	1.946
	ROI 4	1.311	1.188	1.288	1.181
6	ROI 1	1.304	1.325	1.159	1.241
	ROI 2	1.848	1.787	1.756	2.100
	ROI 3	1.953	1.949	1.749	1.709
	ROI 4	1.017	1.150	1.383	1.008
7	ROI 1	1.640	1.432	1.846	1.771
	ROI 2	1.878	1.608	1.947	2.167
	ROI 3	1.607	1.372	1.947	1.794
	ROI 4	1.028	1.085	1.055	0.954
8	ROI 1	1.618	1.578	1.618	1.578
	ROI 2	2.185	1.839	2.185	1.839
	ROI 3	1.210	1.585	1.210	1.585
	ROI 4	1.126	1.490	1.126	1.490
9	ROI 1	1.547	1.462	1.618	1.578
	ROI 2	1.952	1.859	2.185	1.839
	ROI 3	1.263	1.265	1.210	1.585
	ROI 4	0.889	0.915	1.126	1.490
10	ROI 1	1.844	1.742	1.844	1.742
	ROI 2	1.978	1.881	1.978	1.881
	ROI 3	1.666	1.775	1.666	1.775
	ROI 4	1.061	0.943	1.061	0.943
11	ROI 1	1.971	2.519	1.612	1.656
	ROI 2	1.964	2.043	1.893	1.650
	ROI 3	.959	0.953	1.216	1.323
	ROI 4	1.030	1.146	1.161	1.160
12	ROI 1	1.107	1.099	1.310	1.217
	ROI 2	1.876	1.792	1.844	1.826
	ROI 3	1.056	1.051	1.276	1.513
	ROI 4	0.829	1.085	0.819	0.798
13	ROI 1	1.318	1.505	1.895	1.840
	ROI 2	1.958	2.455	2.380	2.285
	ROI 3	1.413	1.607	1.730	1.437
	ROI 4	1.114	1.282	1.281	1.314

Patient 1 did not attend DXA measurements.

frame. Some recommend a 6-h limit,[20,27] while others state a 12-h limit.[25] Hougaard found AVN in 4.8% of 98 patients with traumatic posterior dislocation reduced within 6 h and 52% AVN in those reduced after 6 h. Mean follow-up time was 5 years.[20] Dreinhofer et al.[26] examined 38 posterior and 12 anterior traumatic dislocations reduced within approximately 3 h and found partial AVN in only two patients, mild OA in seven patients and moderate OA in two patients after 8 years. Yang et al.[22] studied 96 patients with hip dislocations retrospectively after 7.5 years; 58% had excellent or good function, and an early reduction gave better results. Our eight hip dislocation patients had their hips reduced within a mean of 3.5 h, and the clinical and radiological results in these patients, except number 1, were all excellent. Two years is, however, a fairly short follow-up time and AVN and OA may occur after several months, even years.[20,28,29]

The proper post-reduction rehabilitation has been debated. Hougaard and Thomsen recommended maximum non-weight bearing at 6 weeks at an early stage.[20] This is similar to our treatment protocol. Our good clinical and radiological results and the findings of modest BMD and bone bruise changes, despite a limited amount of patients, support this rehabilitation protocol. Considering the current knowledge on the probability of OA development in other joints, one could consider a prolonged non-weight-bearing period for older patients with widespread bone bruise changes and low BMD until further studies have been conducted. There are, to our knowledge, no studies that show a direct aetiological connection between weight bearing and cartilage degeneration and/or the development of AVN in the hip joint.

According to the World Health Organization (WHO) criteria, osteoporosis is defined as a BMD 2.5 standard deviations or more below the average value[30] (a *T*-score of <-2.5 standard deviation (SD)). Our study indicates no significant changes of BMD in the chosen ROIs 2 years after trauma, and also no difference between the injured and the healthy side. Some authors found postoperative osteopenia after surgically treated fractures. Neander et al.[31] examined bone loss in displaced femoral neck fractures, measuring BMD with quantitative CT scans after 18 months. They compared patients treated with open reduction and internal fixation (ORIF) and patients treated with arthroplasty. The authors found a 9% decrease in bone mass and muscle volume of the middle femur in the fractures, and the distal femur and proximal tibia showed an even more marked osteopenia. There was no difference in bone mass between the two groups. Karlsson et al.[32] controlled BMD in 102 displaced femoral neck fractures at 10 days, 4 months and 12 months after trauma. There was significantly more bone loss in the fractured hip than in the uninjured hip. Finsen et al.[33] studied BMD after femoral shaft fractures treated with nailing or plating, and observed a considerable loss of bone mineral in the distal femoral metaphysis and a moderate loss in the tibia 31–55 months after injury. They[34] also studied 57 surgically treated ankle fractures and found a significant decrease in BMD on the injured side, independent of extremity weight bearing, unloaded or immobilised in a cast for 6 weeks. The amount of early weight bearing and active exercises after ankle fracture did not modify the post-traumatic osteopenia.

We found no change in BMD in our chosen ROIs after 2 years in our small sample of quite young patients. A possible transient osteopenia may have been missed, as our examinations were obtained with a 2-year increment. However, according to the studies above, some residual bone loss would likely be apparent after a year or two, which we did not find. The studies above were committed to surgically treated fractures, and the same post-traumatic osteopenia may not apply to conservatively treated fractures. In addition, our patients were young compared to the proximal femur fracture patients in the Neander (86 years, range 79–94) and Karlsson (mean age 74 years for men and 79 years for women) studies. A more significant bone loss might be expected after trauma in older patients with a lower BMD in the first place. These considerations need further examination.

Conclusion and clinical implications

Our study demonstrated mainly small and transient areas of bone marrow oedema in the hips and no changes in BMD in the chosen ROIs in 2 years. The dislocated hips were reduced after a mean time of 3.5 h and they had excellent functional outcomes and no arthritic changes on MRI or radiographs after 2 years, except one patient with a small Pipkin fracture who developed AVN, where the

reduction was delayed for 10 h. This finding supports the importance of a quick reduction of a dislocated hip. Our patients were relatively young with normal bone densities, and none was treated surgically. These factors may explain the lack of post-traumatic osteopenia after 2 years. We find no reason to change our post-reduction protocol of 8 weeks of toe-touch weight bearing for patients with dislocated hips or conservatively treated acetabular fractures.

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