

MAGNETIC RESONANCE IMAGING OF AVASCULAR NECROSIS OF THE FEMORAL HEAD

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Abstract

Avascular osteonecrosis of the femoral head (AVNFH) is an ischemic condition which despite different treatments often leads to collapse of the femoral head and to total hip arthroplasty. However, the magnetic resonance imaging findings predisposing to disease progression and total hip arthroplasty are somewhat elusive. To evaluate the magnetic resonance imaging findings of AVNFH and to assess the patterns of findings which may predict total hip arthroplasty. A retrospective study was conducted with a total of 18 diagnosed AVNFH treated with core decompression combined with intraosseous stem cell treatment. After treatment, magnetic resonance imaging followups were done at three-month and one-year follow-up or until total hip arthroplasty. Association Research Circulation Osseous classification and magnetic resonance imaging findings such as the size and the location of the AVNFH, bone marrow edema in femoral neck, effusion and subchondral fracture were evaluated. Hips advancing to total hip arthroplasty have more often bone marrow edema in femoral neck (90% vs. 0%), adjacent to necrotic lesion (100% vs. 43%) and in acetabulum (90% vs. 14%), but also subchondral fractures (70% vs. 0%), effusion (80% vs. 29%), and synovitis (80% vs. 14,3%). The greater size and the lateral weight-bearing location of the necrotic lesion also predicted future total hip arthroplasty. Hips advancing to total hip arthroplasty have often a combination of pathognomonic AVNFH imaging findings compared to hips not advancing to total hip arthroplast

Keywords: osteonecrosis of the hip joint, diagnosis, radiography, magnetic resonance imaging, Femur, hip.



Introduction

Osteonecrosis of the hip joint is a degenerative-destructive disease, a severe form of pathology of the hip joint, which occurs in young people aged 25-55 and then develops rapidly and leads to disability; in many cases, bilateral localization of the process is characteristic [2, 12, 13]. The onset of osteonecrosis eventually leads to erosion of the load-bearing part of the femoral head, damage to the ankle, secondary damage to the acetabulum, and the development of coxarthrosis. The choice of treatment method for osteonecrosis of the hip joint depends primarily on the stage of the disease, the spread of osteonecrosis, and the clinical appearance at the time of diagnosis [1, 5]. Therefore, early diagnosis of the disease is the key to the success of any treatment method, regardless of whether it is conservative or surgical. In the last 5-7 years, evaluating the structure of hip joint diseases during arthroplasty, 30- Among young people aged 45 years, there was an increase in the incidence of this pathology. Analyzing the characteristics of diagnosis during the first visit of patients at the level of outpatient care, it can be noted that the problem of early diagnosis of necrosis of the femoral head in the early stages of the disease is an unsolved problem today. When patients are in an outpatient setting, diagnostic errors are often made, the disease it is considered as a manifestation of osteochondrosis of the lumbar spine or as a pathology of tendons and ligaments, and the patient's complaints are not taken seriously. Such patients are ineffectively treated in the rheumatology, therapeutic, neurology departments of clinics or are not treated correctly at all. As a result of unsuccessful conservative therapy, the disease develops over several years, and the only treatment option is arthroplasty.

Due to the fact that the incidence of osteonecrosis of the hip joint has been increasing rapidly in recent years, it is appropriate to study the research on this topic as follows: The nature of the changes that occurred at the head of the femur is not fully understood, but the traumatic etiology of the disease is indisputable. According to the origin of non-traumatic or avascular theories of circulatory failure (including atypical variants), Rasulov R.M. conducted research in 2004 on the topographical-anatomical location of the branches of the deep femoral artery. In 2014, Akhtamov I.F. and co-authors studied modern methods of diagnosis of osteonecrosis of the hip joint. The clinical course and diagnosis of avascular necrosis of the hip-pelvic joint were changed by Barskova et al. in 2013. (Sovremennaya rheumatologiya. – 2013.



– No. 2. – S. 32– 36.) Pathomorphology of osteonecrosis of the hip-pelvic joint by M. T. Mohammadi, L. A. Pashkevich, O. L. Eysmont studied together "Surgery in Eastern Europe". Prilogenia. - 2017. - S. 148–156.) J. Beltran and others compared the methods of magnetic resonance imaging, radionuclide and histological examination in the early clinical stages of this disease. (Med. J (Engl.). - 1998. - Vol. 111(7). – P. 599-602.). Karantanas A N and co-authors studied the importance of Magnetic resonance imaging in early diagnosis of this disease (Semin. Musculoskelet. Radiol. - 2011. - Vol. 15(3). - P. 281-300). M.A. Mont and co-authors studied osteonecrosis of the hip joint of nontraumatic origin (J. Bone Joint. Surg. Am. - 2006. - Vol. 88. - P. 1117-32). GS Huang [et al.] described bone marrow swelling in this disease using Magnetic resonance imaging (Am. J. Roentgenol. - 2003. - Vol. 181(2). - P. 545-549). In recent years, the expansion of radiological diagnostic capabilities has made it possible to achieve significant progress in the early examination of patients with osteonecrosis of the hip joint, including suspected primary diseases, and the sensitivity and specificity of magnetic resonance Bohndorf K. and others, conducted research in 2015. This examination is 98% in the differential diagnosis of the disease, which allows to identify the pathological process in the initial stage (stage 1), while X-ray methods are often important in the diagnosis of the disease in stages 2-3 (according to the international classifications of ARCO, Ficat and Arlet).

Material and Methods

Patients Institutional review board approval was obtained and the requirement for informed consent was waived. This study was conducted according to the World Medical Association Declaration of Helsinki. A retrospective review of 15 consecutive patients (12 males, 3 females) aged 19 to 69 years (mean 54 years) undergoing MRI for AVNFB and treatment with core decompression combined with local intraosseous stem cell therapy between May 2020 and December 2024 was performed. Three patients had both hips affected, so in total 18 hips were included in this study. Accordingly, the MRI studies were reviewed by two radiologists with four and six years of experience: 18 initial MRI studies, 18 three-month follow-up studies, and 10 one-year follow-up studies were evaluated. During the follow-up lasting until November 2018, 10 patients received aTHA and 1 patient did not have



the one-year follow-up MRI performed. Imaging technique and statistical analysis All MRI examinations were performed on 1.5 Tesla MRI scanners (Optima and Signa, General Electric Medical Systems, Milwaukee, Wisconsin, USA) with dedicated coils and routine avascular necrosis protocol including coronal T1- and STIR or T2-weighted fatsaturated, axial T2-weighted fat-saturated and sagittal T1-weighted sequences; additionally, roughly 50% of the MRI scans included an i.v. contrast coronal T1- weighted fat-saturated sequence. The size of the AVNFB lesion was evaluated in T1-weighted coronal plane (both the greatest length and the depth of the lesion, and the freehand drawn area), as well as in the T1-weighted sagittal plane (Fig. 1). The percentage of the volume of the AVNFB was calculated as follows: the areas of the necrotic lesion and the femoral head were freehand drawn from every image in coronal plane and then the sum of the area of the necrosis was divided by the sum of the area of femoral head (Fig. 2). Then, lesions were graded as small (less than 15%), moderate (15–30%), and large (more than 30%). Location of the necrotic lesion in the femoral head was estimated. Additionally, the extent of the necrotic lesion to the weight-bearing area of femoral head was estimated according to Japanese Investigation Committee (JIC) guidelines (Type A lesion occupying medial one-third or less of the weight-bearing portion of the femoral head, Type B lesion occupying medial two-thirds, Type C1 and C2 both occupying more than the two medial two-thirds; Type C2 extending laterally to the acetabular edge whereas Type C1 does not) (Fig. 3). Furthermore, double-line sign, bone marrow edema (BME) adjacent to the AVNFB, BME at femoral neck, subchondral fracture, effusion and synovitis in the hip joint, BME at the acetabulum, and the Association Research Circulation Osseous (ARCO) stage were collected and graded (Fig. 4). Initially, the effusion was graded as normal, small/minimal, moderate (fluid extending around the femoral neck), and severe (bulging of joint capsule), and on the statistical analyses only moderate and severe were treated as significant effusion. Synovitis was considered present if synovial enhancement was seen with i.v. contrast sequence or if clear synovial hypertrophy was present within the joint capsule. Routine anteroposterior and lateral view hip radiographs were also available on all patients; on radiographs, the findings suggestive of AVNFB – crescent sign, femoral head lucency, or distinct necrotic lesion – were evaluated and applied to the ARCO classification, which is a four-tiered grading system for AVNFB: in stage I



radiographs are normal, but either MRI or bone scan is positive; in stage II radiographs are abnormal (subtle trabecular bone changes) without any evidence of subchondral fracture, or femoral head deformity. Accordingly, MRI findings are characteristic with BME, doubleline sign, and necrotic lesion; in stage III fracture in the subchondral or necrotic zone is present on radiographs or MRI, but the joint space is still preserved; in stage IV features of secondary osteoarthritis with associated femoral head deformity, acetabular changes, and joint destruction are present.

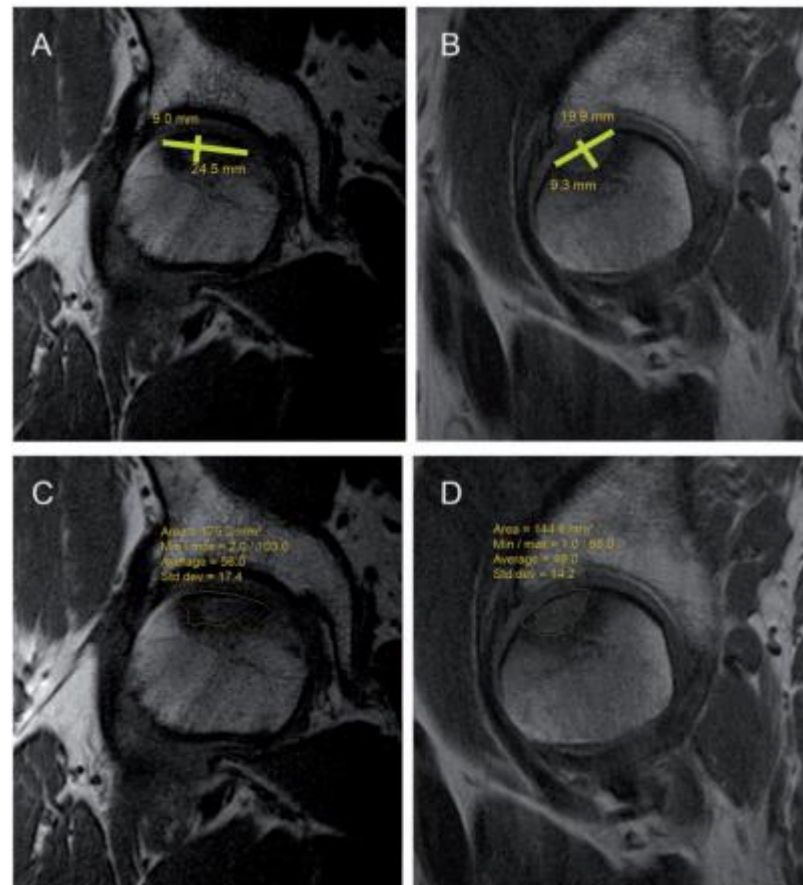


Fig. 1. Maximal measures and area (yellow lines) of the avascular necrosis of the femoral head (AVNFH) were measured from coronal (a, c) and sagittal planes in T1-weighted images (b, d). The surrounding edema was not included.



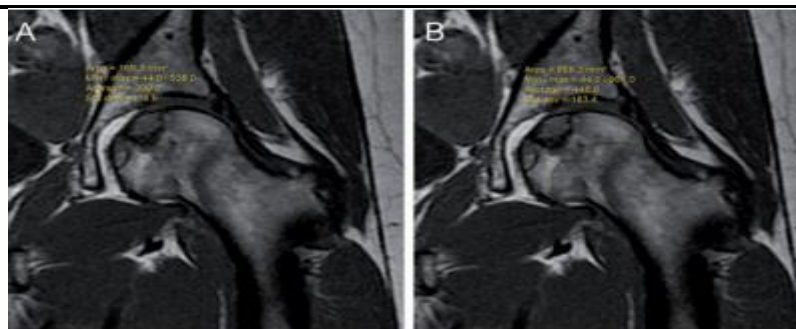


Fig. 2. Avascular necrosis lesion and femoral head (yellow lines) (a, b) were measured from every coronal plane to calculate the percentage of the volume of the necrosis. Femoral head volume was measured along the epiphyseal line. For statistical analyses, paired Student's T-test was applied to test statistical significance between the groups. Statistical software (SPSS Inc., version 24.0, Chicago, IL) was used for the analysis.

Results Two groups were formed of the hips advancing and not advancing to THA. The group advancing to THA consisted of seven hips undergoing THA after three-month follow-up and three hips undergoing THA after one-year follow-up. From THA the MRI findings were separately evaluated retrogradely and then combined forming findings in the two last MRI scans before THA. Another group (8 hips) which did not advance to THA had initial, three-month and one-year followup MRI findings evaluated as a control. One ARCO stage I hip was lost after three-month follow-up for patient being completely symptomless and subsequent one-year follow-up MRI was not performed.

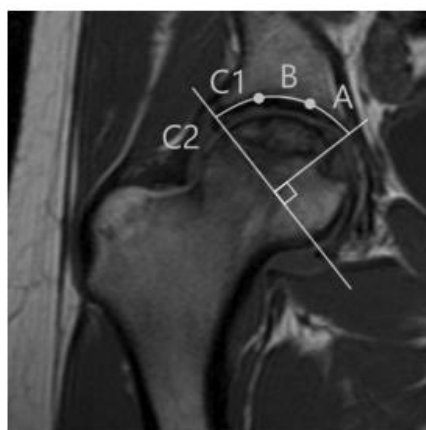


Fig. 3. The extent of the avascular necrosis of the femoral head (AVNFH) to the weight-bearing region of the femoral head was evaluated according to Japanese

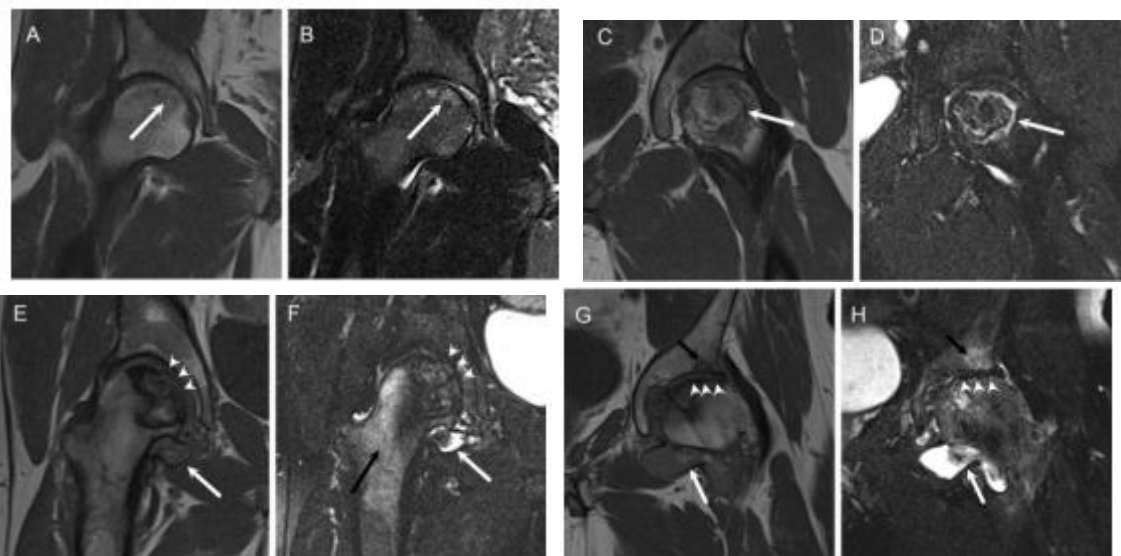


Investigation Committee (JIC) classification. This shown AVNFB extends to lateral third of the weight-bearing area representing JIC type C1 lesion.

Most of the lesion size and area measurements showed minor growth in both groups, but most statistically significant increases in size were seen in the group advancing to THA. Also, the size and area measurements of the AVNFB lesion were generally greater in the group advancing to THA (Table 1). In the group not advancing to THA the volume of most AVNFB was less than 15% of the femoral head, whereas in the group advancing to THA most AVNFB were greater than 15% (Table 2). There was not apparent difference in the location of the necrotic lesion. In the group advancing to THA 70% and in the group not advancing to THA 75% of the necrotic lesions located in the central and anterolateral portion of the weight-bearing femoral head. Instead, the extent of the lesions differed between the groups. In the group advancing to THA 90% of the AVNFBs extended more laterally than medial two-thirds of the weight-bearing femoral head (JIC types C1 and C2). In the group not advancing to THA the extent to weight-bearing femoral head of the lesions was more varied (Table 3).

Fig. 4. The different ARCO stages on coronal T1-weighted (a, c, e, g) and T2-weighted fat-saturated (b, d, f, h) MR images. The ARCO I stage avascular necrosis of the femoral head (AVNFB) shows only subtle bone marrow edema in the subchondral bone (white arrow) (a, b), while plain radiograph findings are still normal (not shown). The ARCO II stage AVNFB demonstrates characteristic double-line sign (white arrows) surrounding the lesion (c, d). Plain radiographs show subtle changes but no findings of subchondral fracture (not shown). The ARCO III stage AVNFB depicts subchondral fracture i.e. irregularity of the bony contour (white arrowheads), effusion with synovitis (white arrows) and bone marrow edema (black arrow) spanning to the neck of the femur (e, f). The ARCO IV stage AVNFB shows deformity of the femoral head paired with full-thickness cartilage loss (white arrowheads), acetabular bone marrow edema (black arrows) and effusion with synovitis implying secondary osteoarthritis





In the group advancing to THA, eventually every hip had BME adjacent to the AVNFH. Nearly all (80–90%) had BME in the femoral neck, effusion, and synovitis. Out of the 10 hips, the presence of acetabular BME increased from six to nine and the presence of subchondral fracture from five to seven. Interestingly, the pathognomonic double-line sign was seen in 5 hips out of 10 in the second last MRI scan and it was seen only in three in the last MRI scan before the hips advanced to THA.

Table 1. AVNFH lesions of hips advancing to THA are generally larger.

| | Non-THA group | | THA group | |
|--------------------------------|---------------|----------|--------------|----------|
| | Initial | 12 month | 2nd last MRI | Last MRI |
| TI cor width (mm) | 22.4 | 26.4* | 28.2 | 30.2 |
| TI cor depth (mm) | 13.7 | 14.9 | 13.2 | 13.8 |
| TI cor area (mm ²) | 246.7 | 277.9 | 265.5 | 297.5* |
| TI sag length (mm) | 28.2 | 27.6 | 29.1 | 32.8* |
| TI sag depth (mm) | 11.4 | 13.6 | 12.9 | 14.3* |
| TI sag area (mm ²) | 248.0 | 286.0 | 276.5 | 347.0* |
| Volume of femoral head (%) | 17.4 | 19.5 | 18.4 | 21.3* |

MRI: magnetic resonance imaging; cor: coronal; sag: sagittal; THA: total hip arthroplasty.

*Statistically significant change ($p < 0.05$).

Table 2. Volumes of AVNFH in hips advancing and not advancing to THA.

| | Non-THA group (N = 8) | THA group (N = 10) |
|-----------------------|--------------------------|-----------------------|
| Volume | | |
| Small (less than 15%) | 6 | 3 |
| Moderate (15–30%) | 0 | 4 |
| Large (more than 30%) | 2 | 3 |

THA: total hip arthroplasty.

Table 3. The extent to weight-bearing femoral head of AVNFH in hips advancing and not advancing to THA (according to JIC guidelines).

| | Non-THA group (N = 8) | THA group (N = 10) |
|-----------|--------------------------|-----------------------|
| Type | | |
| A | 2 | 0 |
| B | 3 | 1 |
| C1 and C2 | 3 | 9 ^a |

THA: total hip arthroplasty.

^aOnly one type C2 lesion existed in the study.

Table 4. The prevalence of MRI findings in hips advancing and not advancing to THA.

| | Non-THA group (N = 8) | | | THA group (N = 10) | |
|----------------------|-----------------------|-----------|--------------------------------|--------------------|-----------|
| | Initial | 3 month | 12 month (N = 7 ^a) | 2nd last MRI | Last MRI |
| Double-line sign | 7 (87.5%) | 6 (75%) | 5 (71.4%) | 5 (50%) | 3 (30%) |
| BME AVN lesion | 3 (37.5%) | 5 (62.5%) | 3 (42.9%) | 9 (90%) | 10 (100%) |
| BME femoral neck | 0 | 2 (25%) | 0 | 9 (90%) | 9 (90%) |
| Subchondral fracture | 0 | 0 | 0 | 5 (50%) | 7 (70%) |
| BME acetabulum | 0 | 1 (12.5%) | 1 (14.3%) | 6 (60%) | 9 (90%) |
| Effusion | 4 (50%) | 1 (12.5%) | 2 (28.6%) | 8 (80%) | 8 (80%) |
| Synovitis | 1 (12.5%) | 1 (12.5%) | 1 (14.3%) | 9 (90%) | 8 (80%) |

THA: total hip arthroplasty; MRI: magnetic resonance imaging; BME: bone marrow edema; ARCO: Association Research Circulation Osseous; AVN: avascular necrosis.

^aOne ARCO stage I hip was lost during follow-up for being symptomless and one-year MRI was not done.

Table 5. ARCO classification of hips advancing and not advancing to THA.

| ARCO | Non-THA group (N = 8) | | | THA group (N = 10) | | |
|-----------|-----------------------|----------------|--------------------------------|--------------------|---------|--------------------------------|
| | Initial | 3 month | 12 month (N = 7 ^a) | Initial | 3 month | 12 month (N = 3 ^b) |
| Stage I | 2 | 1 ^a | 0 | 2 | 0 | 0 |
| Stage II | 6 | 7 | 7 | 3 | 3 | 1 |
| Stage III | 0 | 0 | 0 | 4 | 4 | 1 |
| Stage IV | 0 | 0 | 0 | 1 | 3 | 1 |

THA: total hip arthroplasty; ARCO: Association Research Circulation Osseous.

^aOne ARCO stage I hip was lost during follow-up for being symptomless and one-year MRI was not done.

^bSeven hips advanced to THA after 3-month follow-up; two stage II, two stage III and three stage IV hips.



Hips not advancing to THA had at three-month follow-up increased BME adjacent to necrotic lesion and in femoral neck, but at one-year follow-up BME on both regions had decreased. Also, initially the presence of BME was less common than in hips advancing to THA. In contrast to hips advancing to THA there were not any subchondral fractures and almost no acetabular BME. At the initial MRI scan, four out of eight hips had effusion, and at one-year follow-up only two of the hips presented effusion. In hips not advancing to THA, the presence of double-line sign also became rarer. Table 4 summarizes the MRI findings in both groups. ARCO classification was initially more severe and showed clear progression in hips advancing to THA. All ARCO stage III and IV hips and total of three stage II hips eventually underwent THA during the followup. In hips not advancing to THA, the ARCO classification was less severe and only one hip progressed from stage I to stage II (Table 5)

Discussion

In this study we show that a cavalcade of typical MRI findings exists on hips where the AVNFB tends to progress. Earlier it has been represented that the band-like pattern is the initial finding of the necrotic lesion¹¹ and later the developing BME in the femoral head and neck is associated with the onset of the symptoms and considered a marker for potential progression of the AVNFB and collapse of the femoral head.¹² Several studies have also found an association between the BME and AVNFB – however, it must be noted that BME is not pathognomonic for AVNFB.^{13–15}

Also, lately it was reported that BME adjacent to necrotic lesion indicates a subchondral fracture, which may not be visible on MRI.¹⁶ Our study favors this finding as nearly every hip advancing to THA had initially BME adjacent to necrotic lesion and 7 out of 10 hips also in the femoral neck. In hips not advancing to THA, the presence of BME in femoral neck and adjacent to the AVN lesion was less common. Initially, subchondral fracture was present in five hips and during follow-up two new subchondral fractures became visible. In these five hips and in hips with not visible subchondral fracture before THA, there were nearly always BME but also effusion, synovitis and secondary BME in the acetabulum suggestive of intra-articular pathology. Similar with BME, majority of patients with AVNFB have



shown present with a hip effusion.^{15,17–19} Iida et al.¹² mention that in the presence of BME, 92% of the symptomatic hips also had joint effusion and most of these hips showed progression of the AVNFB. In another study it was demonstrated that BME associates with pain and the effect of BME was enhanced in the presence of effusion; accordingly, the findings were most pronounced in ARCO stage III hips – i.e. in hips with subchondral fractures. In the same study effusion was seen in 72% of hips with AVNFB compared to 10% of healthy controls and effusion was seen most in ARCO stage III disease (92%) and less in lower stages than in more advanced necrotic lesions.¹² In our study joint effusion was strongly associated with hips with more advanced AVNFB lesions as in the group advancing to THA effusion was seen initially in 80% of the hips. In our study, AVNFB lesions kept slightly increasing in size. Most size increases were very minor, which could be included also in measurement bias. However, most of the statistically significant increases in size were in the group advancing to THA. According to the previous literature, the collapse commonly occurs within two years in 32%–79% of patients having symptomatic AVNFB,²⁰ and untreated asymptomatic AVNFB progresses to symptomatic disease or collapse in approximately 60% during a maximal 20-year followup.²¹ On MRI, it has been demonstrated that the greater size and the weight-bearing location of the AVNFB is a risk factor for further collapse of the femoral head.^{22–24} Our study indicates similar results as hips advancing to THA had greater mean size and volume of the AVNFB compared to cases not advancing to THA. Similarly, the lateral weight-bearing location of the AVNFB seemed to predict future THA. On the contrary, medial location of AVNFB seemed to be a protective factor as none of these hips underwent THA; similar results were recently suggested by Takashima et al.²⁵ Previously, Ito et al.²⁶ demonstrated that large necrotic volume of 30% or more of the femoral head may predict worsening of hip pain and the BME was strongly associated with the volume. In our study, 15% seemed to be the crucial volume of the AVNFB to predict future THA. Our study has several limitations. First, the sample size in our study is too small and follow-up time rather short to draw solid conclusions. There was not histopathological confirmation of osteonecrosis and diagnoses of avascular necrosis were only dependent on clinical symptoms and specific imaging findings. There was no sub-grouping of hips according to known risk factors of AVNFB. Moreover, an isotropic 3D MRI



sequence would have been useful to assess the integrity of the articular surface of the femoral head. Lastly, on one patient the one-year follow-up MRI scan was not done for patient being symptomless. In conclusion, our study suggests hips advancing to THA have a combination of pathognomonic MRI findings such as BME adjacent to necrotic lesion and in femoral neck but also subchondral fracture, effusion, synovitis, and secondary acetabular edema. Also, the greater size and the lateral location of the AVNFH seem to be predictive factors for THA.

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