

Transient osteoporosis of the hip, complete resolution after treatment with alendronate as observed by MRI description of eight cases and review of the literature

Yasser Emad · Yasser Ragab · Nashwa El-Shaarawy · Johannes J. Rasker

Received: 13 July 2012 / Accepted: 9 August 2012 / Published online: 30 August 2012
© Clinical Rheumatology 2012

Abstract Transient osteoporosis of the hip (TOH), also referred to as transient bone marrow edema syndrome, is most common in middle-aged men and often after trivial trauma or sport-related injuries. Diagnosis is usually made by eliminating other possible causes of hip pain. Magnetic resonance imaging (MRI) plays an important role in diagnosis and demonstrates a typical pattern of bone marrow edema (BME) in the form of diffuse low signal on T1-weighted images and high signal on T2 fat-suppressed or short T1 inversion recovery images. No consensus exists about the management of TOH, as it may progress to avascular necrosis. We describe eight cases of TOH treated with

alendronate resulting in improvement of pain and function and complete resolution of BME on MRI. The literature is reviewed regarding TOH and the relationship with bone marrow edema syndrome, avascular necrosis of the hip, and regional migratory osteoporosis. To our knowledge, this is the first report describing the improvement of this condition after of alendronate with documented radiological improvement on follow-up MRI.

Keywords Alendronate · Bone marrow edema syndrome · Magnetic resonance imaging (MRI) · Transient osteoporosis of the hip

Y. Emad (✉)
Rheumatology and Rehabilitation Department,
Faculty of Medicine, Cairo University,
Cairo, Egypt
e-mail: yasseremad68@yahoo.com

Y. Emad
Rheumatology and Rehabilitation Department, Dr. Erfan
and Bagedo General Hospital,
Jeddah, Saudi Arabia

Y. Ragab
Radiology Department, Faculty of Medicine, Cairo University,
Cairo, Egypt

Y. Ragab
Radiology Department, Dr. Erfan and Bagedo General Hospital,
Jeddah, Saudi Arabia

N. El-Shaarawy
Rheumatology and Rehabilitation Department,
Faculty of Medicine, Suez Canal University,
Ismailia, Egypt

J. J. Rasker
Rheumatology Department, University of Twente,
Enschede, The Netherlands

Introduction

Bone marrow edema syndrome (BMES) refers to transient clinical conditions with unknown pathogenic mechanisms, including many entities such as transient osteoporosis of the hip (TOH), regional migratory osteoporosis (RMO), and reflex sympathetic dystrophy (RSD). BMES is primarily characterized by bone marrow edema (BME) pattern. The disorder mainly affects the hip, the knee, and the ankle of middle-aged males. Many hypotheses have been proposed to explain the pathogenesis of the disease. Unfortunately, the etiology of BMES remains obscure. The hallmark that separates BMES from other conditions presented with BME pattern is its self-limiting nature.

BME is a general term describing an area of low-signal intensity on T1-weighted and high-signal intensity on T2-weighted and short T1 inversion recovery (STIR) magnetic resonance (MR) images [1]. It is important to note that BME affecting the hip joint is neither a specific magnetic resonance imaging (MRI) finding nor a specific diagnosis. BME of the hip joint cannot only be encountered in TOH but also

in inflammatory arthropathy, early avascular necrosis, occult stress fractures, primary bone neoplasms, myeloproliferative disorders, hemoglobinopathy, and infection. Differentiating between these conditions is crucial, as there are considerable differences in treatment and prognosis [1].

There is no specific laboratory test for TOH, but tests may be necessary to exclude other conditions. Plain radiographs may reveal regional osseous demineralization. MRI is mainly used for the early diagnosis and for monitoring the progression of the disease. Early differentiation from other aggressive conditions with long-term sequelae is essential in order to avoid unnecessary treatment [2].

Acute BME of the hip is a diagnostic challenge for both radiologists and clinicians. Marrow edema is often seen in patients with hip pain and restriction of motion. In patients with acute nontraumatic hip pain, whose radiographs are negative or inconclusive, MRI is the imaging study of choice. MRI is the most sensitive and specific imaging technique for detecting transient osteoporosis and osteonecrosis [3].

TOH was first described by Curtiss and Kincaid in 1959 [4] as a syndrome of transient demineralization of the hip in the third trimester of pregnancy. In 1968, Lequesne [5] first used the term in a published report. This rare cause of acute hip pain is still a relatively seldom diagnosed clinical entity. TOH, also referred to as transient BME syndrome, is now known to be most common in middle-aged men [6].

Typically, patients suffering from TOH present with an acute onset of hip pain, antalgic gait, and severe functional disability. In most of the cases, patients with TOH usually present with acute onset of pain which is typically in the front of the thigh, the side of the hip, the buttocks, or the groin with limited range of motion. The pain is constant and intensifies with turning movements and may lessen with rest; the pain gradually increases over a period of weeks or months and may be very intense and disabling. MRI has largely supplanted bone scintigraphy as the first-line

imaging test after conventional radiographs in the setting of TOH and avascular necrosis (AVN) of the hip [7, 8].

Description of the eight cases

All patients diagnosed as TOH after exclusion of other causes of BME were included in this series. MRI was performed at the start of the study and 6 months after initiation of treatment to all patients. MR images were interpreted for the following radiological signs: BME, epiphyseal deformity, femoral head congruity, subchondral changes, synovial enhancement, bone erosions, joint effusion, and cartilage loss. The MR images were read blindly and independently by an experienced radiologist and by an experienced clinician; in case of disagreement, they discussed the case until they both agreed.

Eight patients were seen in our clinics between 2000 and 2012, with established diagnosis of TOH, sharing clinical characteristics as described in details in Table 1. All patients presented with hip pain and limitation of internal and external rotation with positive Farber's test with pain and limited range of motion elicited upon flexion, abduction, and external rotation of the involved hip [9].

All patients filled in visual analogue scale for pain (0–10 cm) at the start of the study and at follow-up. Functional and clinical improvements were defined as improvement of the initially observed limitation of internal and external rotation of the involved hip joint and regaining of normal range of motion.

They were treated during 6 months with weekly dose of alendronate 70 mg/week. All patients were receiving calcium and vitamin D supplementation (calcium carbonate 600 mg/day and 300 IU/day of vitamin D) at the same time and were strictly advised to avoid any weight bearing on the involved limb. One patient (case $n=4$) underwent core decompression surgery; after the surgery, he had persistent BME as observed on MRI and continuing functional

Table 1 Demographic and clinical characteristics of eight patients at their initial evaluation

No.	Age	Gender	VAS	Synovial enhancement	Hip ROM	Unilateral vs. bilateral	Core decompression	Time from initial complaint to MRI study (days)
1	26	M	7	Absent	Limited	Unilateral	Negative	60
2	35	M	7	Absent	Limited	Unilateral	Negative	10
3	32	M	9	Absent	Limited	Unilateral	Negative	21
4	51	M	10	Absent	Limited	Unilateral	Positive	30
5	31	M	8	Absent	Limited	Unilateral	Negative	120
6	35	M	8	Absent	Limited	Unilateral	Negative	20
7	41	M	9	Absent	Limited	Bilateral	Negative	10
8	32	M	10	Absent	Limited	Bilateral	Negative	7

limitations and he was put on alendronate and showed complete resolution of complaints and of BME after 6 months of treatment. All patients had unilateral hip involvement except for two cases who presented with bilateral hip affection (Fig. 1).

In all patients initial MR images showed no evidence of effusion or synovial enhancement. No inflammation or other abnormalities of the sacroiliac joint joints were seen on MR images.

After 6 months, MRI studies were performed in all patients and showed complete resolution of BME pattern compared with the initial MRI study (Figs. 1, 2, and 3).

Discussion

In this report, we describe eight cases with established diagnosis of TOH who were treated with alendronate together with calcium and vitamin D and protective non-weight bearing on the affected limb. The eight patients all showed improvement on both clinical and radiological levels with complete resolution of BME pattern of the affected hips. To our knowledge, this is the first report describing the use of alendronate in patients with TOH showing clinical improvement as well as improvement shown on MRI.

One of the most important and landmark studies regarding TOH which can explain the beneficial effects of alendronate among our patients is the study conducted by McCarthy [10] who described biopsy specimens from 19 cases with transient regional osteoporosis at different joints.

Six of these specimens were therapeutic core biopsies, and three were femoral heads removed during total hip replacement. The other patients with osteoporosis in different locations had biopsies to rule out infection or neoplasm. Five of these patients had transient osteoporosis of the knee, three had ankle involvement, and two had involvement of the tibial shaft. Except for one patient who was lost to follow-up, all had resolution of symptoms and radiographic changes [10]. The histological changes in the biopsies were distinctive, although they were present in varying degrees. There was edema and reactive bone formation in the marrow spaces. The most important finding in the study is the presence of osteoclastic bone resorption that was active in 14 out of the 19 cases studied. All but one of the 19 biopsy specimens showed a consistent pattern of histological changes irrespective of the location of the disease. One biopsy, a core specimen from the hip, showed only thin trabecular bone. The other biopsies showed changes in the marrow as well as changes in the bone, although the degree of change varied from case to case. Changes in the marrow were quite distinct. All but one case showed marrow edema. This appeared as a pale eosinophilic material taking up space between marrow fat cells. Occasionally, this material was foamy. In six cases, small lipid cysts were present. Fat necrosis of the marrow was not present in any case [10].

Another common marrow finding, present in 14 cases, was the deposition of thin seams of woven bone in the marrow spaces. These seams were lined by active osteoblasts and were not related to native trabeculae. A final marrow change, present in six cases, was a mild fibroblast

Fig. 1 Axial (a) and coronal (b), STIR images showing hyperintense bilateral bone marrow edema (white arrows) that regressed in follow-up study (c, d)

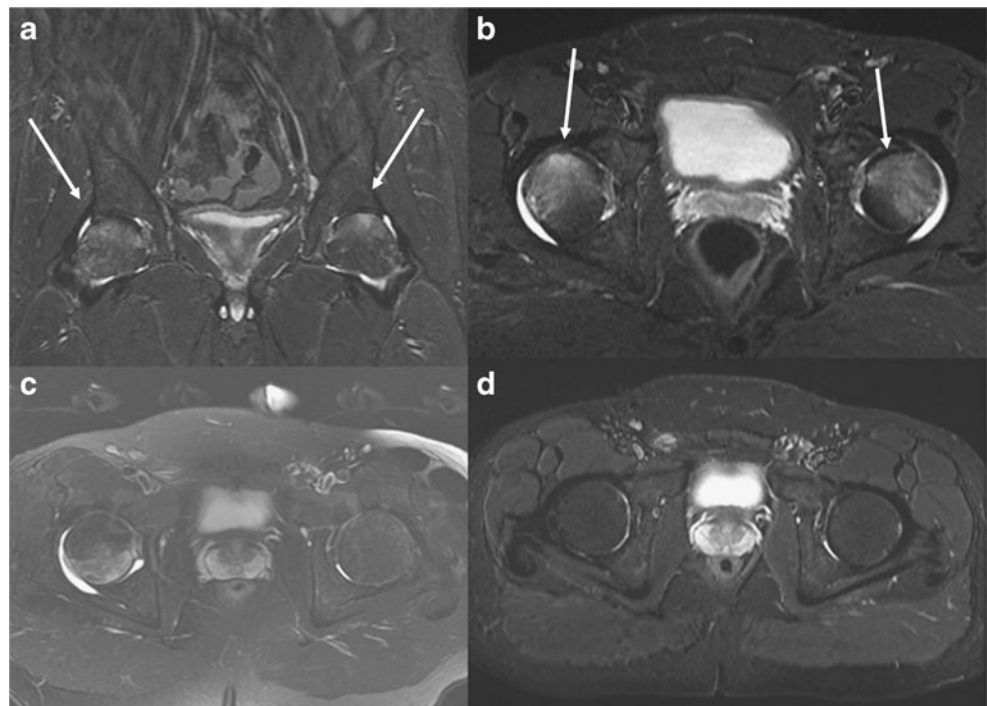
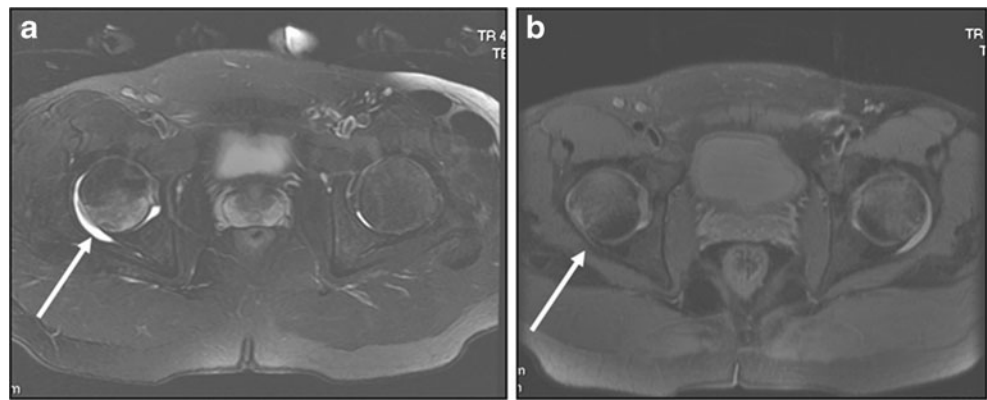


Fig. 2 **a** STIR sequence showing extensive BME (hyperintense signal) of left femoral head and neck. **b** STIR sequence showing complete resolution



proliferation. This change was focal and was occasionally associated with small clusters of lymphocytes and plasma cells. Changes in cancellous bone were also apparent. All cases showed thinning of the trabeculae, a finding correlating with radiographic osteopenia. In addition, 12 of the 19 cases showed evidence of osteoclastic bone resorption. Osteoclasts were present in resorption tunnels on the trabecular surfaces while necrotic trabeculae were observed [10].

The cause of transient regional osteoporosis is still uncertain. One theory is that edema results from microtrauma of trabecular bone [11]. Another theory suggests that the osteoporosis results from a vasomotor response similar to RSD.

Transient osteoporosis of the hip and osteonecrosis

More recently, an etiologic relationship with osteonecrosis has been proposed [10]. Two observations support this recent theory. First, Turner et al. [12] observed in six patients that the diffuse MRI pattern of transient osteoporosis evolved into a focal pattern consistent with osteonecrosis. Second, several observers have noted marrow fat necrosis in histological samples of transient osteoporosis [13, 14]. One group observed this feature in 7 of 32 core biopsies of transient osteoporosis [13]; they noted that this fat necrosis correlated with the pathologic stage 1 or 2 of osteonecrosis. These observations support early experimental

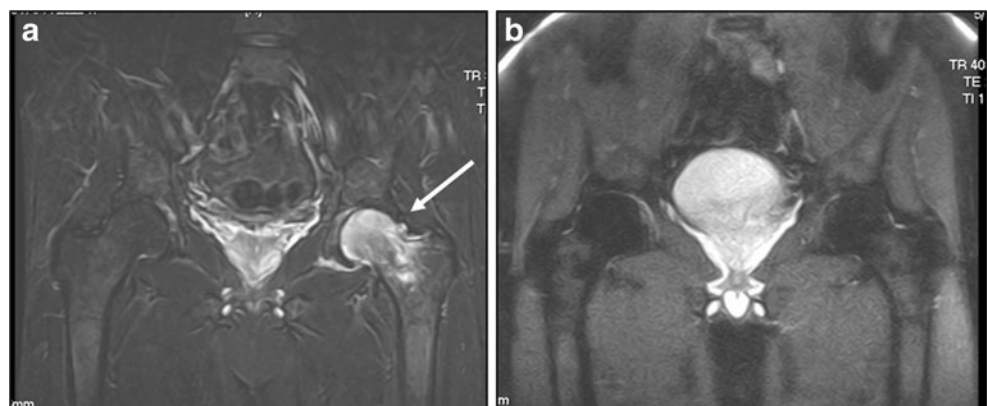
work that modest ischemia produces BME. As a result of these observations, some investigators regard transient osteoporosis as a prodrome to classic osteonecrosis. The fact that many cases of transient osteoporosis resolve spontaneously suggests to these investigators that the ischemic injury is not always severe enough to cause bone necrosis. This theory provides the rationale for treating transient osteoporosis with core biopsy; a treatment used at some institutions for stage 1 or 2 osteonecrosis [10].

One may conclude from these findings that TOH is not as benign as previously thought and may have the potential to progress to AVN resulting in femoral head collapse and functional loss.

In another report, Yamamoto et al. [15] performed bone biopsy on three patients who had been diagnosed as having TOH. The biopsy specimens were studied histopathologically by light and electron microscopy. The authors found that the most characteristic features of TOH were focal areas of thin and disconnected bone trabeculae covered by osteoid seams and active osteoblasts. The surrounding bone marrow tissue showed edematous changes and mild fibrosis, frequently associated with vascular congestion and/or interstitial hemorrhage. No osteonecrotic region was observed in either the bone trabeculae or the bone marrow tissue.

Another similar condition is regional migratory osteoporosis (RMO) which is an uncommon disease characterized by a migrating arthralgia involving the weight bearing joints mainly

Fig. 3 **a** STIR sequence showing extensive BME (hyperintense signal) of left femoral head and neck. **b** STIR sequence showing complete resolution



of the lower limbs. Men in their fifth and sixth decades of life are most commonly affected. The most common presentation is with proximal to distal spread in the lower limb. There are 63 articles that document cases of regional osteoporosis or BME with migratory symptoms. The radiology of RMO is indistinguishable from TOH except for the migratory symptoms and the two conditions are likely to be part of the same spectrum of disease [16].

AVN of the hip is a progressive clinical condition with significant morbidity, and it is characterized by the death of the bone, or part of it, because of insufficient circulation. Under certain pathological conditions, the intraosseous marrow pressure increases and is transmitted to the venules and capillaries within the bone, causing a diminishment of local blood flow, with consequent ischemia. As bone repair occurs, the mechanically weak bone collapses due to the load of weight [17]. The occurrence of collapse or development of collapse greater than 2 mm frequently leads to incapacitating pain or secondary osteoarthritis, making prevention of collapse a fundamental item for obtaining a favorable outcome [18].

Use of bisphosphonates in AVN of the hip

There are few studies in humans, describing the use of bisphosphonates for the treatment of AVN of the hip. Cardozo et al. [19] reviewed the literature regarding the use of bisphosphonates in the management of AVN of the femoral head and found seven articles [17, 20–25]. In their review, article Cardozo et al. [19] reported that all these studies present various limitations, such as: a lack of randomization, control group, and double-blind design with small numbers of patients included, short duration of follow-up and nonstandardization of the type of bisphosphonate, dose used, and time of its use. Furthermore, in the various studies, there is no uniformization with regard to outcome; some use prevention of collapse of the femoral head, and others use pain intensity or articular mobility. Bearing in mind the abovementioned limitations, one observes that the majority of studies suggest a positive result with the use of bisphosphonates in the treatment of AVN [17, 20–22] and one study reports a questionable benefit [23] and another study showed no benefit whatever [25].

Cardozo et al. [19] concluded that the current data are still insufficient for justifying the use bisphosphonates for this indication. On the other hand, noncontrolled studies appear to demonstrate favorable results, particularly in diminishing pain, improving mobility, and lowering the incidence of articular collapse, which justifies new studies being developed in this area.

In our opinion, the prevention of collapse as one single important item should be seriously considered which was reported in two of the studies previously mentioned [21, 22].

Most importantly in the study by Lai et al. [21], which is a randomized clinical study (level II evidence) is that 2 out of 29 patients receiving alendronate developed head collapse compared with 19 patients out of 29 in the control group not receiving alendronate. One may conclude that treatment of patients with early AVN before collapse of the femoral with alendronate may offer favorable prognosis. In addition to pain control, it may prevent femoral head collapse and thus play an important role in the management of this serious condition and may postpone surgical options in this clinical setting.

Is TOH self limiting or may it progress to AVN of the hip?

There is still controversy whether TOH represents a distinct self-limiting disease, or reflects only an early, reversible subtype of nontraumatic AVN. The clinical presentation of patients with TOH and AVN is similar with mechanical hip joint pain, AVN risk factors, and a male preponderance. Radiographic, bone scintigraphic, and MRI patterns of TOH are more diffuse while focal in AVN. The histological bone marrow changes are similar in both conditions but with diffuse and sufficient repair in TOH, whereas in AVN only insufficient focal repair at the border of the necrotic lesion occurs [26].

Differentiation of TOH from early cases of AVN of the hip is usually difficult. Radiographs may show diffuse osteopenia of the femoral head and neck in TOH but a localized area of sclerosis in AVN, whereas bone scanning shows homogeneously increased uptake in the femoral head and neck in cases of TOH and a localized area of decreased uptake in AVN. While MRI shows a diffuse edema pattern which is common to both entities, but the absence of focal defects and subchondral changes is highly suggestive of TOH. Early differentiation of TOH from AVN will avoid unnecessary surgical intervention and ensure appropriate treatment [27].

In a recent work, Holzer et al. [28] evaluated 48 patients (54 hips) with sudden hip pain. All patients had radiographs, technetium bone scan, and MRI; in all patients, increased uptake on bone scan and on MRI bone edema in the femoral head and neck were found. Five patients diagnosed with other disease processes were excluded from the study. The remaining (37 men and 6 nonpregnant women; 49 hips) were diagnosed with TOH. All had repeated clinical and MRI investigations until resolution of symptoms with a mean follow-up of 43 months (range, 12–106 months). The authors observed spontaneous resolution of symptoms in all patients, and all were asymptomatic at final follow-up, although one patient had minor restriction of flexion. Despite the presence of crescent lines on initial MRI in 14 patients, none progressed to osteonecrosis, and crescent lines were not apparent on follow-up MRI. The authors

conclude that TOH is a benign disease that does not progress to osteonecrosis, and should be treated conservatively. Crescent lines may initially appear on MRI only to resolve spontaneously. Other diagnoses should be investigated if pain and bone edema persist.

TOH may progress to AVN

In our opinion, TOH has tendency for progression to AVN for many reasons: first, many patients with initial TOH usually pass undiagnosed and receive only symptomatic treatment and the diagnosis is usually not raised at early stages and the patients are not instructed to avoid weight bearing on the involved limb. When symptoms and functional limitations the patients will have MRI performed in a last stage one may find features of early AVN. Second and most important, why do orthopedic surgeons recommend invasive procedures like core decompression to alleviate the increased intraosseous pressure if the condition is that benign in their mind and spontaneously regress. Third, the current available data which documented active osteoclastic being active in bone biopsy of patients with TOH [10, 15] and bone ischemia [13] may be a key player in progression to AVN and in theory may lead to early bone erosions which lead to incongruity of the femoral head with unequal weight distribution on already ischemic femoral head; this may result in an increased intraosseous pressure, more ischemia, and in the progression to AVN.

It is important that BME should be considered a marker for potential progression to advanced AVN, and careful examinations for osteonecrosis are necessary when BME is seen [29]. Subchondral focal lesions are likely to be overlooked on MRI because BME develops at the area of the surrounding living bone, and surrounding BME may obscure the subchondral focal abnormalities on MR images. Vande Berg et al. [30] called the findings on MR imaging of the surrounding BME a “pseudo-homogeneous edema pattern.” Osteonecrosis is likely to be diagnosed after the onset of hip pain, and BME is often observed at the first MRI; therefore, BME could be considered the initial MR finding of osteonecrosis and an early ischemic change [31, 32]. At the stage when BME is detectable on MR images, distinguishing osteonecrosis from transient osteoporosis is difficult but important [33–37].

Conclusions

The current available data documented the presence of osteoclastic activity in bone biopsy specimens of patients with established diagnosis to transient regional osteoporosis; this may explain the improvement of BME pattern in our patients with established diagnosis of TOH after treatment

with a bisphosphonate (alendronate), a drug with main action of inhibiting osteoclastic activity.

Disclosures None.

References

- Ragab Y, Emad Y, Abou-Zeid A (2008) Bone marrow edema syndromes of the hip: MRI features in different hip disorders. *Clin Rheumatol* 27(4):475–482
- Korompilias AV, Karantanas AH, Lykissas MG, Beris AE (2009) Bone marrow edema syndrome. *Skeletal Radiol* 38(5):425–436
- Apostolos K (2007) Acute bone marrow edema of the hip: role of MR imaging. *Eur Radiol* 17(9):2225–2236
- Curtiss PH Jr, Kincaid WE (1959) Transitory demineralization of the hip in pregnancy. A report of three cases. *J Bone Joint Surg Am* 41:1327–1333
- Lequesne M (1968) Transient osteoporosis of the hip. A nontraumatic variety of Sudeck's atrophy. *Ann Rheum Dis* 27:463–471
- Grimm J, Higer HP, Benning R, Meairs S (1991) MRI of transient osteoporosis of the hip. *Arch Orthop Trauma Surg* 110(2):98–102
- Fang C, Teh J (2003) Imaging of the hip. *Imaging* 15:205–216
- Malizos KN, Zibis AH, Dailiana Z, Hantes M, Karachalios T, Karantanas AH (2004) MR imaging findings in transient osteoporosis of the hip. *Eur J Radiol* 50(3):238–244
- Martin RL, Sekiya JK (2008) The interrater reliability of 4 clinical tests used to assess individuals with musculoskeletal hip pain. *J Orthop Sports Phys Ther* 38(2):71–77
- McCarthy EF (1998) The pathology of transient regional osteoporosis. *Iowa Orthop J* 18:35–42
- Beaulieu JG, Razzano CD, Levine RB (1976) Transient osteoporosis of the hip in pregnancy. *Clin Orthop Relat Res* 115:165–168
- Turner DA, Templeton AC, Selzer PM, Rosenberg AG, Petasnick JP (1989) Femoral capital osteonecrosis: MR finding of diffuse marrow abnormalities without focal lesions. *Radiology* 171(1):135–140
- Plenk H Jr, Hofmann S, Eschberger J, Gstettner M, Kramer J, Schneider W, Engel A (1997) Histomorphology and bone morphometry of the bone marrow edema syndrome of the hip. *Clin Orthop Relat Res* 334:73–84
- Potter H, Moran M, Schneider R, Bansal M, Sherman C, Markisz J (1992) Magnetic resonance imaging in diagnosis of transient osteoporosis of the hip. *Clin Orthop Relat Res* 280:223–229
- Yamamoto T, Kubo T, Hirasawa Y, Noguchi Y, Iwamoto Y, Sueishi K (1999) A clinicopathologic study of transient osteoporosis of the hip. *Skeletal Radiol* 28(11):621–627
- Cahir JG, Toms AP (2008) Regional migratory osteoporosis. *Eur J Radiol* 67(1):2–10
- Agarwala S, Jain D, Joshi VR, Sule A (2005) Efficacy of alendronate, a bisphosphonate, in the treatment of AVN of the hip. A prospective open-label study. *Rheumatology (Oxford)* 44(3):352–359
- Nishii T, Sugano N, Ohzono K, Sakai T, Haraguchi K, Yoshikawa H (2002) Progression and cessation of collapse in osteonecrosis of the femoral head. *Clin Orthop Relat Res* 400:149–157
- Cardozo JB, Andrade DM, Santiago MB (2008) The use of bisphosphonate in the treatment of avascular necrosis: a systematic review. *Clin Rheumatol* 27(6):685–688
- Agarwala S, Sule A, Pai BU, Joshi VR (2002) Alendronate in the treatment of avascular necrosis of the hip. *Rheumatology (Oxford)* 41(3):346–347

21. Lai KA, Shen WJ, Yang CY, Shao CJ, Hsu JT, Lin RM (2005) The use of alendronate to prevent early collapse of the femoral head in patients with nontraumatic osteonecrosis. A randomized clinical study. *J Bone Joint Surg Am* 87(10):2155–2159
22. Nishii T, Sugano N, Miki H, Hashimoto J, Yoshikawa H (2006) Does alendronate prevent collapse in osteonecrosis of the femoral head? *Clin Orthop Relat Res* 443:273–279
23. Nguyen T, Zacharin MR (2006) Pamidronate treatment of steroid associated osteonecrosis in young patients treated for acute lymphoblastic leukemia-two-year outcomes. *J Pediatr Endocrinol Metab* 19(2):161–167
24. Wang CJ, Wang FS, Yang KD, Huang CC, Lee MS, Chan YS, Wang JW, Ko JY (2008) Treatment of osteonecrosis of the hip: comparison of extracorporeal shockwave with shockwave and alendronate. *Arch Orthop Trauma Surg* 128(9):901–908
25. Ramachandran M, Ward K, Brown RR, Munns CF, Cowell CT, Little DG (2007) Intravenous bisphosphonate therapy for traumatic osteonecrosis of the femoral head in adolescents. *J Bone Joint Surg Am* 89(8):1727–1734
26. Hofmann S, Kramer J, Schneider W, Plenk H (1997) Transient osteoporosis may represent a reversible early form of avascular necrosis of the hip joint. *Curr Orthop* 11(3):164–172
27. Balakrishnan A, Schemitsch EH, Pearce D (2003) Distinguishing transient osteoporosis of the hip from avascular necrosis. *Can J Surg* 46(3):187–192
28. Holzer I, Snir N, Ben-Galim P, Maman E, Rosenblatt Y, Dekel S (2009) Transient osteoporosis of the hip: long-term outcomes in men and nonpregnant women. *Current Orthopaedic Practice* 20(2):161–163
29. Iida S, Harada Y, Shimizu K, Sakamoto M, Ikenoue S, Akita T, Kitahara H, Moriya H (2000) Correlation between bone marrow edema and collapse of the femoral head in steroid-induced osteonecrosis. *AJR Am J Roentgenol* 174(3):735–743
30. Vande Berg BE, Malghem JJ, Labaisse MA, Noel HM, Maldague BE (1993) MR imaging of avascular necrosis and transient marrow edema of the femoral head. *RadioGraphics* 13:501–520
31. Li KCP, Hiette P (1992) Contrast-enhanced fat saturation magnetic resonance imaging for studying the pathophysiology of osteonecrosis of the hips. *Skeletal Radiol* 21:375–379
32. Hofmann S, Engel A, Neuhold A, Leder K, Kramer J, Plenk H Jr (1993) Bone-marrow oedema syndrome and transient osteoporosis of the hip. *J Bone Joint Surg Br* 75(2):210–216
33. Turner DA, Templeton AC, Selzer PM, Rosenberg AG, Petasnick JP (1989) Femoral capital osteonecrosis: MR finding of diffuse marrow abnormalities without focal lesions. *Radiology* 171:135–140
34. Hayes CW, Conway WF, Daniel WW (1993) MR imaging of bone marrow edema pattern: transient osteoporosis, transient bone marrow edema syndrome, or osteonecrosis. *RadioGraphics* 13:1001–1011
35. Guerra JJ, Steinberg ME (1995) Distinguishing transient osteoporosis from avascular necrosis of the hip. *J Bone Joint Surg Am* 77(4):616–624
36. Richardson ML, Can MR (1994) Imaging distinguish between transient osteoporosis of the femoral head and osteonecrosis? *AJR Am J Roentgenol* 162(5):1244
37. Trepman E, King TV (1992) Transient osteoporosis of the hip misdiagnosed as osteonecrosis on magnetic resonance imaging. *Orthop Rev* 21(9):1089–1091, 1094–8