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REVIEW

Transient osteoporosis of the hip: review of the literature

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Abstract Transient osteoporosis of the hip (TOH) is a temporary clinical condition of unknown etiology which usually resolves with conservative therapy though may be complicated by fracture or progression to avascular necrosis (AVN). TOH may be slightly more prevalent in men but when it occurs in women, it is most often seen in the latter part of pregnancy. Though fracture is a rare complication of TOH when it occurs, it is most often associated with TOH occurring in pregnancy. Magnetic resonance imaging (MRI) is the best method to diagnosis TOH. Low signal intensity on T1-weighted images, high signal intensity on T2-weighted images, and homogenous pattern of edema (the femoral head and/or neck) with normal subchondral area are in favor of TOH. A shortened course to recovery is reported by use of bisphosphonates, calcitonin, or teriparatide. Based on reported cases, core decompression is not superior to medical therapy. Transient osteoporosis of the hip, which often has no known etiology, usually resolves with conservative therapy but may predispose the patient to fracture or avascular necrosis. Diagnostic method of choice is magnetic resonance imaging. Bisphosphonates, calcitonin, or teriparatide are reported as a useful approach to reduce duration of recovery.

Keywords Bone marrow edema · Transient osteoporosis

Abbreviations

AVN	avascular necrosis
BMD	bone mineral density
BME	bone marrow edema
BMES	Bone marrow edema syndrome
DXA	dual-energy X-ray absorptiometry
PTH	parathyroid hormone
PTH-rP	parathyroid hormone-related peptide
RMO	regional migratory osteoporosis
STIR	short-tau inversion recovery
TOH	transient osteoporosis of the hip

The abstract of part of this study has been presented as an oral presentation at the American Society of Bone and Mineral Research (ASBMR), September 12–14, 2014, Houston, TX [1].

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Introduction

Transient osteoporosis of the hip (TOH) is usually a benign and self-limiting disorder with no obvious etiology [1–3]. It may also be referred to as the primary bone marrow edema syndrome (BMES), a term used to define bone marrow edema (BME) without an obvious cause. TOH is most common in middle-aged men. In women, it is most often seen in the last trimester of pregnancy. TOH may predispose these populations to fragility fractures [2–5]. An insult (such as trauma, infection, inflammation, degenerative process, ischemic injury, neoplasia, surgery, drugs, metabolic, and neurologic disorders) presumably initiates the process, leading to an increase in bone turnover, venous hypertension, and/or microfracture causing edema [5–7]. The differences between primary and secondary TOH in terms of natural course and radiological findings are often unclear; however, it is important to exclude secondary causes of TOH radiologically and clinically.

In 1959, Curtiss and Kincaid reported three cases of TOH who presented with unilateral or bilateral hip pain or thigh pain in the third trimester of pregnancy. Radiology showed spotty demineralization of the femoral head, a small part of the femoral neck, and the acetabulum which recovered spontaneously several months after delivery [8]. In 1988, Wilson et al. used the term "Transient Marrow Edema Syndrome" regarding patients with knee and hip pain who had normal bone or osteopenia on DXA, regional decrease in intensity of signal of the bone marrow on T1-weighted images, and increased signal intensity on T2-weighted images on MRI [9]. In 1993, Solomon introduced the importance of differentiation between BME with and without osteonecrosis: BME with osteonecrosis may cause bone collapse requiring reconstructive surgery, whereas BME without osteonecrosis is a transient and self-limited condition [10].

Information concerning TOH is scattered and in mostly small case series. We performed a literature review using "Bone Marrow Edema" and "Transient Osteoporosis of the Hip" to find articles cited on Pub Med between 1959 and May 2014 (Table 1). Two hundred seventy-four articles were identified, and 97 were selected for review after excluding articles lacking data about age, duration of recovery, tool of diagnosis, modality of treatment or complications, or lacking ability to obtain full text manuscript. Examining the abstract, we selected the articles reporting either cases of transient osteoporosis or bone marrow edema syndrome of the hip with or without other joint involvement. Epidemiology of TOH (age, sex, and reported risk factors), duration of recovery, modality of treatment and complications were assessed and compared. Most cases were diagnosed by MRI, especially articles published after 1990. However, some studies reported cases diagnosed by X-ray, DXA, or bone scans were considered to be TOH on the basis of clinical time course and recovery with pain resolution with or without motion. We found 437 reported cases of TOH (Table 1). Additional weight bearing joints that may be involved include the knee (47 cases), ankle (25 cases), foot (22 cases), spine (2 cases), and shoulder (1 case).

Epidemiology and pathophysiology

TOH is postulated to have three stages. The first stage with acute onset hip pain is due to edema potentially induced by trauma, neurovascular dysfunction, transient hyperemia, or microfracture. The second stage involves increased resorption and demineralization of the bone. The final stage consists of resolution of the process clinically and by radiograph [15, 16].

TOH should be considered in the differential diagnosis of sudden onset hip pain which typically gradually resolves within 6 months (2–12 months) with conservative strategies including minimizing weight-bearing activities, rest, using crutches, wheelchairs, hot packs, ultrasound therapy, and interferential current therapies or analgesics [7, 17, 18]. Pain can be possibly produced by increased intraosseous pressure, venous hypertension, increased focal bone turnover, microfracture, or periosteal irritation (5). Though unlikely, bone marrow edema may progress, causing vascular compression, resulting in ischemic injury to the femoral head and avascular necrosis (AVN) [19].

The distinction between TOH and early-phase AVN is difficult to judge both radiologically and clinically, and may result in needless surgical intervention in patients with only TOH. Furthermore, these two conditions are different in prognosis and treatment. Pain of TOH usually starts spontaneously. Presentation may be varied from insidious vague pain with limping to more severe progressive pain leading to immobility and hospitalization.

Pregnancy is the most common reported risk factor for TOH, though the overall incidence of TOH is higher in men than that of women (Table 1). Occasionally a precipitating event or etiology, such as trauma, alcohol consumption, smoking, corticosteroid, vascular insults, inflammation, drug use, or osteogenesis imperfecta, may be associated (Table 1) [5, 11–14]. The mean age of onset is 40 years of age with cases reported between ages 20 and 80 (Table 1).

Although dual energy X-ray absorptiometry (DXA) shows osteoporosis in some cases, it is not clear if osteoporosis is a risk factor for the development of microfracture and bone marrow edema. On the other hand, microfracture might itself cause the edema and separation of the bone trabeculae, reported as osteoporosis on DXA. Focal bone turnover markers are elevated in aspirates from bone, without any significant detectable change in mean serum concentration of bone turnover markers [5, 20].

Table 1 Characteristics of patients with transient osteoporosis of the hip (TOH) in 97 PubMed indexed articles from 1959 to May 2014

Number of cases	Male	Female	Mean age	Age range	Hip	Risk factors
437	253 (58%)	184 (42%)	39.56	20–79	437	Pregnancy, alcohol, nicotine, corticosteroid, abnormal vascularity, drugs, inflammation, metabolic derangement, mechanical injury, neurologic deficit, and osteogenesis imperfecta [5, 11–14]

Etiology

The etiology of TOH remains uncertain. It is also unclear whether TOH is an isolated entity from or represents the early stage of AVN. The aforementioned insults (trauma, infection, inflammation, degenerative process, ischemic injury, neoplasia, surgery, drugs, metabolic, and neurologic disorders) could be considered as a cause of TOH. Pregnancy is a well-known risk factor of TOH. A similar disorder, migratory osteoporosis, also associated with pregnancy, has been postulated to have increased osteoclast activation called regional acceleratory phenomena [21]. Other risk factors such as alcohol consumption, steroid usage, smoking, hypothyroidism, hypophosphatasia, osteogenesis imperfecta, low testosterone, low vitamin D (25-cholecalciferol), and certain occupations are reported in the literature [1, 5, 14, 22–25].

Diagnosis

Magnetic resonance imaging (MRI) is the best method to demonstrate edema within bone and is sensitive enough to detect TOH as early as 48 h after the onset of symptoms [5, 16]. MRI findings that support TOH include intermediate signal sequences on T1-weighted images (Fig. 1a) and high signal intensity on T2-weighted images (Fig. 1b). Another MRI finding is hyper intensity on contrast enhanced images. Short-tau inversion recovery (or STIR) imaging allows homogeneous and global fat suppression on MRI, which also may be helpful in defining TOH (Table 2 with references [5, 11–16, 26–28]). The delayed peak enhancement of edematous marrow is particularly characteristic of TOH. Findings that favor TOH include a homogenous pattern of enhancement with no clear border, a diffuse pattern of edema with no focal defect, along with the presence of an irregular band of low signal intensity due to stress fracture, and lack of subchondral changes on T2-weighted or contrast-enhanced T1-weighted images [5, 7, 15, 16, 26]. The edema of TOH is usually located at the femoral head and may extend to the femoral neck and intertrochanteric region and is often accompanied by joint effusion [7, 26]. However, there is much dispute about predictive factors and disease progression. While patients differ in the amount, extent of edema and subchondral changes; Malizos (2004) et al. report that there is no correlation between the extent of edema and the duration of symptoms, but TOH with a spared subchondral zone resolves faster clinically. However, the time interval between onset of symptoms and MRI was different for each patient in their study [26]. On the other hand, Ergun (2008) et al. state that duration of clinical recovery correlated with the extent of edema, and the presence and size of subchondral fracture. These investigators find that subchondral fracture itself is also linked to the extent of edema [29]. On the contrary, Klontzas (2015) et al. describe

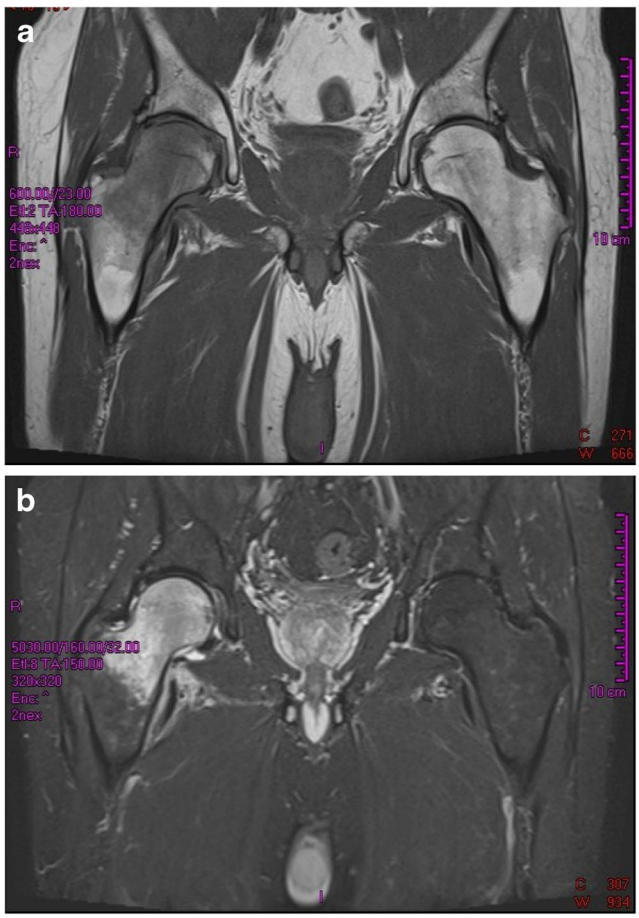

Fig. 1 Coronal T1-weighted (**a**) and T2-weighted (**b**) MR images of the right hip revealed extensive bone marrow edema in the right femoral head and neck, mild joint effusion without signs of fracture (subchondral, subcapital, or femoral neck). Diffuse pattern of edema with no focal defect, fracture, and subchondral changes is in favor of bone marrow edema or TOH

Table 2 Characteristic MRI findings and risk factors of TOH and AVN [5, 11–16, 26–28]

Disease	TOH	AVN
MRI	- Low signal intensity on T1-weighted - High signal intensity on T2-weighted	- Femoral head deformity - Crescent sign (subchondral radiolucency) - Single line sign with edema on T1 - Double line sign with edema on T2
MRI with contrast	- Hyper-intensity on contrast enhanced - Homogenous pattern of enhancement - No clear border and diffuse pattern of edema - No focal defect - No subchondral changes - Delayed peak enhancement of the edematous marrow	- Low signal intensity of subchondral area, at least 4 mm thick on T2 or contrast-enhanced T1-weighted image [15] - Subtle focal contour deformity of the subchondral bone plate - Filling defect
MRI of stress fracture	Irregular band of low signal intensity	Fracture line and filling defect
Risk factors	Pregnancy, alcohol, nicotine, corticosteroid, abnormal vascularity, drugs, inflammation, metabolic derangement, mechanical injury, neurologic deficit, and osteogenesis imperfecta [5, 11–14]	- Vascular disruption (fracture or hip dislocation) - Vessel occlusion or endothelial dysfunction (sickle cell aggregations, clots or lipid thrombi, hyperhomocysteinemia, or cytotoxic drugs) - Increased intraosseous pressure or vessel compression (lipid deposition and adipocyte hypertrophy, corticosteroid, or alcohol intake) - Other conditions: Gaucher disease, hyperlipidemia, hyperuricemia, pancreatitis, leukemia or lymphoma, and hypertriglyceridemia, pregnancy, radiation, bone marrow transplantation, metastatic malignancies, inflammatory diseases, such as systemic lupus erythematosus (SLE), rheumatoid arthritis, polymyositis/dermatomyositis, granulomatosis with polyangiitis, and inflammatory bowel diseases and psoriatic disease [27, 28]

that the duration of symptoms correlates statistically with the extent of edema, but not subchondral fracture [30]. In contrast, MRI findings of AVN may reveal femoral head deformity, subchondral radiolucency (crescent sign) (Figs. 2 and 3), single line sign with edema on T1-weighted image and double line sign with edema on T2-weighted image (Table 2).

While no radiographic changes are noted initially, 3–6 weeks from the onset of symptoms, diffuse osteopenia of the femoral head or periarticular demineralization can be appreciated on X-ray, yet the femoral head usually remains intact

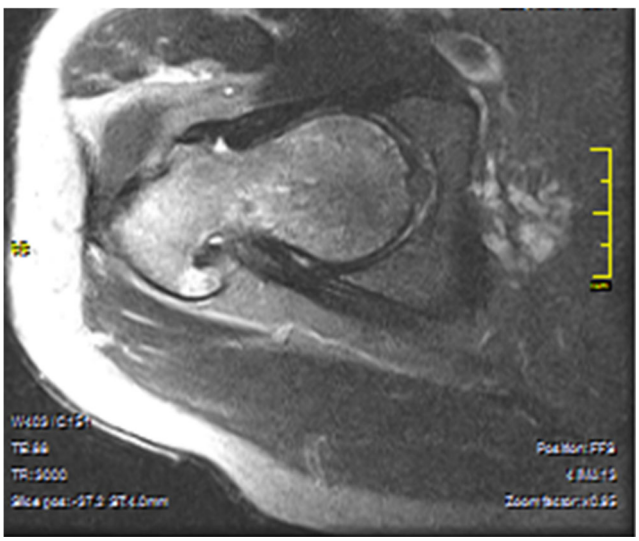

Fig. 2 Repeated MRI 8 weeks following Fig. 1 revealed no femoral head collapse and persistent but improved edema with no fracture lines present, most consistent with TOH

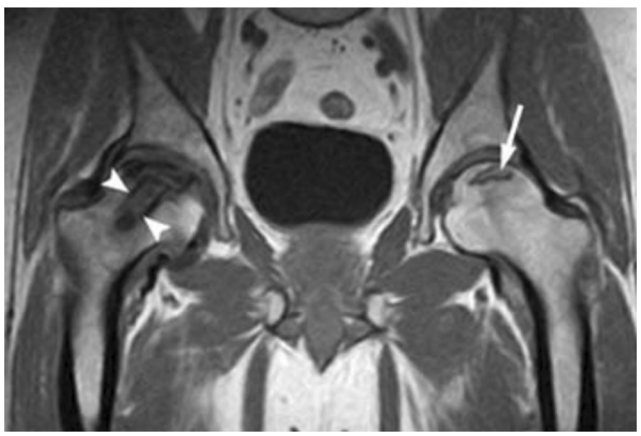

Fig. 3 A coronal T1-weighted image of the hip with avascular necrosis which received core decompression (the right hip, *arrowheads*) and occult AVN (the left hip, *arrow*) with subchondral radiolucency, crescent sign. [David S. Levey. MRI Web Clinic, AVN of the Hip. November 2005]

Table 3 Comparison of different modalities of treatment listed in order of duration of recovery in different studies which reported a case of TOH or bone marrow edema syndrome of the hip

Treatment	Clinical recovery	Radiological recovery	Patients and method of diagnosis	Author/year
Alendronate 10 mg/day postpartum	Within a few days		1 case (36 yo pregnant) Dx by X-ray	Samdani A 1998 [38]
Calcitonin and alendronate	Within 2 days after surgical repair of low-energy trauma-induced left hip fracture		1 case (79 yo M) Dx by X-ray, bone scan, DXA and MRI	Guardiano SA 2004 [39]
Teriparatide 20 μg daily	4 weeks	8% increase in left hip BMD	1 case (62 yo M left hip pain) Dx by MRI and DXA but had normal X-rays	Fabbriciani G 2012 [40]
Zoledronate 5 mg	4 weeks		1 case (46 yo M) Dx by X-ray, MRI, and bone SPECT	Seok H 2011 [41]
Calcitonin nasal spray for 6 weeks and alendronate (Fosamax 70 mg tab once weekly) for 3 months	1 month		1 case (30 yo M) Dx by MRI and X-rays	Suresh S 2009 [42]
Ibandronate 4 mg IV	1 month		1 (50 yo M, 18 years AS, left knee pain) Dx by MRI	Straten VH 2009 [43]
Alendronate 70 mg/week	3 weeks (5 month therapy), 6 weeks (4 months therapy), 2 months (4 weeks 60% improvement)		3 cases Dx by MRI: 50 yo W (knee pain) 45 yo M (hip pain) 57 yo W (hip pain)	Kibbi L 2008 [44]
Pamidronate 45 mg IV, 3 doses, every 3 days	2 months for 14 cases (2 cases minimal pain)	After 2 months (+10.9% TH, +12.3% FN) After 4 months (+1.8% TH, +2% FN)	16 cases Dx by X-ray, bone scintigraphy, MRI	Varenna M 2002 [45]
Clodronate 300 mg IV × 10 days	8–10 weeks		3 cases Dx by X-ray	Varenna M 1996 [46]
Clodronate 300 mg IV × 10 days	2 months asymptomatic (2 weeks pain reduction)	+16% BMD at the left hip	1 case Dx by X-ray	Schapira D 2003 [47]
Neridronate sodium 25 mg/month IM for 6 months, Ca + vitamin D	2 months asymptomatic (3 weeks improvement)		1 (36 yo pregnant) Dx by X-ray, bone SPECT, DXA, and MRI	La Montagna G 2005 [48]
Alendronate 70 mg once in 2 weeks	8 weeks		1 case (27 yo M) Dx by MRI	Anand A 2010 [49]
Alendronate 70 mg/once a week + ca and vitamin D	Poor response after 3 months (hip pain) 3 weeks (ankle pain)		1 case (29 yo W, 23 GA) Dx by MRI	Kartal E 2011 [50]
Pamidronate 60 mg IV weekly, then every 2 weeks (4 infusions), and 6 months alendronate 70 mg/week +1000 mg calcium with 800 IU vitamin D 3 times a day	3 months (pain reduction in 2 weeks)		1 case (26 yo, GA 18, right ankle, GA 30 bilateral knee pain, GA 36 right hip) Dx by X-ray, bone scan and MRI	Rozenbaum M 2011 [51]
Alendronate 70 mg/week and vitamin D for 3 months	3 months		1 case (35 yo W, last trimester) Dx by MRI and X-ray (fracture)	Spinarelli A 2009 [52]
Alendronate (35 mg/week) and calcitriol (0.5 μg/day)	5 months postpartum		1 case Dx by MRI and DXA	Anai T 2013 [53]
alendronate 70 mg/week Ca-carbonate 600 mg/day + vit D	6 months		8 cases Dx by MRI	Emad Y 2012 [4]
Ibandronate IV 4 mg and 2 mg after 3 months Daily calcium (1 g) and vitamin D	Pain reduction: 1 month 43.3% 3 months 78.4% 6 months 94.3%	Mean increase in LS BMD 4% Change in TH BMD + 7.5%	12 cases Dx by MRI	Ringe JD 2005 [54]
Bisphosphonates once a week + calcium and vitamin D	9 months postpartum		1 case Dx by X-ray (fracture) (29 yo W, 40 GA)	Lamarca M 2009 [55]
Calcitonin for 1 month then followed by alendronate 70 mg/week for 6 months	6 months postpartum		1 case Dx by MRI (34 yo W at 30 GA, postpartum started treatment)	Ergin T 2010 [1] [56]
Calcitonin	1–8 months (median 4.7 months)		6 cases (42–54 yo, M) Dx by MRI, bone scan and X-ray	4 articles[2] [18, 46, 57, 58]
Calcitonin	Few days to 9 weeks in 4 cases One case few M One case 6 M One case 12 M One case no response after 3 months[4]		8 cases (28–42 yo, W) During pregnancy or postpartum Dx by MRI, DXA, X-ray	7 articles[3] [50, 59–64]

Table 4 Comparison of different modalities of treatment in order of time to recovery of 275 cases who had TOH or bone marrow edema syndrome of the hip

Treatment	Clinical recovery	Radiological recovery	Increase in BMD	Patients and method of diagnosis	Author/year
Conservative therapy	6 months (2–12)			222 cases Dx by X-ray, bone scintigraphy, CT or MRI	97 articles
Alendronate	3 months (within days to 9 months)			10 cases	Table 2a
Alendronate	6 months	6 months		8 cases Dx by MRI	Emad Y/2012[5]
Ibandronate	6 months	6 months	LS + 4% (−0.8 to 7.7%) FN + 10.1	12 cases Dx by MRI	Ringe JD/2005[6]
IV clodronate	8–12 weeks			4 cases Dx by X-ray	Varenna M/1996 & Schapira D/2003[7]
IV clodronate	2–6 months			3 cases 2 cases Dx by X-ray, bone scintigraphy and MRI 1 case Dx by X-ray	Trevisan C/2002[8]
Pamidronate	2 month (14 cases) & 2 cases minimal pain after prolonged weight-bearing	3 months	After 2 months +10.9% TH, +12.3% FN After 4 months + 1.8% TH, + 2% FN	16 cases Dx by X-ray, bone scintigraphy, MRI	Varenna M/2002[9]
IV zoledronate	4 weeks	6 months		1 case Dx by X-ray, MRI, and bone SPECT	Seok H/2011[10]
Teriparatide	4 weeks	2 months	2 months (8% increase)	1 case Dx by MRI and DXA but had normal X-rays	Fabbriciani G/2012[11]
Calcitonin and bisphosphonate together	1 month			1 case Dx by MRI and X-rays	Suresh S./2009[12]

AS ankylosing spondylitis, *Dx* diagnosis, *DXA* dual energy X-ray absorptiometry, *FN* femoral neck, *GA* gestational age per week, *LS* lumbar spine, *M* Man, *TH* total hip, *W* woman, *yo* years old

Clinical recovery was considered when pain disappeared completely, and no discomfort could be elicited on passive motion as reported in each study

[1] Ergin T, et al. (2010) 34-year-old woman with right hip TOH was treated with calcium 1000 mg, calcitriol 0.5 mcg, and salmon calcitonin 200 IU/day after delivery for 1 month then changed to alendronate 70 mg/week for 6 months

[2] Leyes Vence M 1996, Varenna M 1996, Ribera Zabalbeascoa J 1999, Trevisan C 2002

[3] Arayssi TK 2003, Marczak D 2006, Laktasic-Zerjavic N 2007, Ververidis AN 2009, Chalouhi GE 2010, Truszczyńska A 2012, Kartal E 2011

[4] Kartal E, et al. (2011) 29-year-old pregnant woman with RMO (right hip involvement and then 2 weeks later the right knee and 6 weeks later left hip and then left knee and right tibia, ankle, and foot). The right hip improved after 12 weeks of conservative therapy. The left hip had no significant improvement after conservative therapy and 3 months intranasal calcitonin (200 IU/day). Knee and leg pain had no significant improvement following additional 3 months of alendronate and calcium/D

[5] Emad Y, et al. (2012) Weekly alendronate 70 mg/week (6 months) + calcium & vitamin D (calcium carbonate 600 mg/day and 300 IU/day of vitamin D)

[6] Ringe JD, et al. (2005) in an open-label, prospective, observational study (6 months), 12 patients with localized transient osteoporosis (LTO; bone marrow edema syndrome) were initially received ibandronate 4 mg IV, a second IV injection of 2 mg was optional after 3 months (3 patients received). Daily calcium (1 g) and vitamin D (800 IU) supplements were provided. Decrease in mean (SD) pain score was 43.3% (15.5%) after 1 month, 78.4% (19.2%) after 3 months, and 94.3% (7.6%) after 6 months. The mean baseline lumbar spine BMD T-score was −1.6 (range − 3.4 to +0.1). Effect on BMD after 6 months, mean lumbar spine BMD increased by 4% (range − 0.8 to 7.7%) relative to baseline. In patients with LTO of the hip, the mean difference in BMD between the affected and unaffected sides of the femoral neck decreased from 10.1 to 2.6% at 6 months

[7] Varenna M (1996) Three cases were treated with IV clodronate for 10 consecutive days; and Schapira D (2003)

[8] Trevisan C, et al. (2002) reported three cases of regional migratory osteoporosis that had recurrent episodes and received calcitonin and bisphosphonate (alendronate and clodronate) at different times

[9] Varenna M, et al. (2002) Pamidronate 45 mg IV, every 3 days, 3 doses

[10] Seok H (2011) A 46-year-old man treated with intravenous zoledronate (5 mg) and conservative therapy such as using crutches, wheelchair, minimizing weight bearing, hot pack, and ultrasound and interferential current therapies were performed on the hip

[11] Fabbriciani G (2012) A 62-year-old man treated for 4 weeks with teriparatide at a daily dose of 20 μg and cholecalciferol 800 IU daily. Protected weight bearing was recommended

[12] Suresh S, et al. (2009) A 30-year-old man was given calcitonin nasal spray and alendronate (Fosamax 70 mg tab once weekly), calcitonin for 6 weeks, and alendronate for 3 months. The symptoms disappeared within 1 month of starting the antiresorptive therapy

[7, 26, 31]. The absence of subchondral changes is an indicator of TOH [22]. The increase in blood flow and capillary permeability causes an augmentation in radionuclide uptake, thus a positive bone scan may be seen in all three phases of TOH and persist for weeks after clinical improvement [15, 26, 31]. While regional migratory osteoporosis (RMO) is similar in that it is also usually transient; it is defined by its asymmetric involvement and in its movement from proximal to distal, progressing unilaterally from the hip to knee and ankle [32]. Since RMO is sometimes associated with changes in bone mineral density, serial bone density measurements may demonstrate the progression of both bone loss and subsequent recovery of affected areas [21].

Course and treatment

Transient osteoporosis, especially TOH, probably happens in the setting of microfractures and bone marrow edema causing separation of the bone trabeculae. During the recovery process, if the primary insult is removed and adequate reparative mechanisms proceed, then as edema and hypervascularity resolve, clinical and radiological improvement will follow. Conservative therapy, suppression of bone resorption, or induction of bone formation may reduce further damage, promote recovery, or shorten duration of recovery. However, if the patient with TOH does not follow these recommendations or, if for other reasons, TOH does not reverse, a fracture may ensue, confusing the diagnosis with AVN. Hayes et al. in a review in 1993, report that TOH improved after several weeks to months with conservative therapy. With the lack of histologic or radiographic evidence of bone necrosis in TOH patients, they surmise that risk factors for AVN determine the distinction between resolving TOH and further progression to osteonecrosis [33]. Otherwise, continued injury to the bone and further increase in edema may lead to necrosis, bone collapse, articular distortion, and fracture of the bone [5]. However, TOH and AVN have some common risk factors such as alcohol consumption, nicotine or corticosteroids usage, and coagulation disorders [5, 11, 14]. Debatably, TOH might be considered as an early-stage precursor of AVN. However, while TOH generally resolves without sequelae, AVN is usually an irreversible and progressive disease, resulting from interruption of blood supply to the femoral head, sometimes also leading to permanent joint failure [7].

The mechanism explaining bone fragility in TOH is not completely understood. Histopathology findings from patients with TOH show increased intramedullary pressure and enhanced bone formation [5]. Studies report evidence of osteoclastic bone resorption, increased resorption lacunae filled with osteoclasts, signs of microfractures, and thin bone trabeculae [15, 34–37].

Medical treatment with bisphosphonate therapy, calcitonin, or teriparatide is reported to shorten recovery time for TOH in small, non-randomized, and non-controlled case studies. We have summarized the outcomes of TOH patients with these various treatments (Tables 3, 4, 5). Conservative therapy, including minimizing weight-bearing activities, rest, using crutches, wheelchairs, hot packs, analgesics, ultrasound therapy, or interferential current therapies is commonly recommended [7, 17, 18]. Intravenous pamidronate in 15 cases shows an average clinical recovery of 2 months, comparing favorably to a historical control recovery of 6 months [45]. However, alendronate oral therapy in 8 cases shows an average clinical recovery of 6 months, similar to that of the controls (Table 3) [4]. Calcitonin use in 6 cases shows a shortened mean clinical recovery of 4.7 months (Table 3). Combination of calcitonin and bisphosphonate therapy, zoledronate, or teriparatide in different reports shows almost a 1 month recovery time (Tables 3, 4) [40–42]. Nasal calcitonin, strontium ranelate, teriparatide, and bisphosphonates are reported to treat pregnancy-associated osteoporosis during pregnancy or lactation. Bisphosphonate therapy may improve bone density in pregnancy-associated osteoporosis [65, 66]. However, exposure to bisphosphonate before conception or during pregnancy might affect fetal skeletal mineralization, shorten gestational age, cause low birth weight, transient hypocalcaemia of the neonate, and spontaneous abortion and/or anomalies [66, 67]. Calcitonin does not cross the placenta, appears safe during pregnancy, and has no known deleterious effects on the fetus [68, 69]. Using calcitonin during pregnancy, as reported in some cases, may reduce duration or recovery [50, 59–64].

Core decompression therapy, which is recommended for AVN, does not appear to improve outcomes any more than medical therapy (duration of recovery was between 1 week and 8 months, medium 1.5–2 months) (Table 5). However, the data are limited to very few case reports, and further study of TOH therapy is needed before these agents can be recommended routinely.

Complications and prognosis

Subchondral, femoral neck, and subcapital fracture are rare but potential complications of TOH. While men and women have the same incidence of subchondral fracture, femoral neck, and subcapital, fracture are seen more frequently in female patients (Table 6). Of the two male cases, one occurred in a man with osteogenesis imperfecta and the other in a 79-year-old man (Table 6).

Of the 24 reported cases of TOH who developed femoral neck or subcapital fracture, 22 are women who fractured during pregnancy or after delivery. In one study, only one patient had a true femoral head fracture, as opposed to a femoral head stress fracture,

Table 5 Reported recovery in 107 cases who received core decompression as a treatment

Treatment	Core decompression 25 cases[1]	Core decompression 43 cases[2]	Core decompression 6 cases[3]	Core decompression 38 cases[4]
Clinical recovery	1.5 months (1 week to 7 M)	2 M	1.5 M (0.5 to 8 M)	3.9 M
Radiological recovery	3–12 months (in 4 cases)		3 M	

[1] Hofmann S 1993, Hayes CW 1993, Moran MC1995, McCarthy EF 1998, Yamamoto T 1999, Toms AP 2005 Apr, Emad Y 2012 Nov, and Uzun M 2013 Jun. One patient with severe osteopenia developed femoral neck fracture after decompression and biopsy. (Moran MC 1995, 33-year-old man)

[2] Hofmann S, et al. (2000) reported a retrospective study of 43 cases who received core decompression therapy for BMOES (bone marrow oedema syndrome) without surgical complications

The average duration of recovery was lower with core decompression (2 months) in comparison to that of conservative therapy (6 months)

[3] Calvo E et al. (2000) reported 12 symptomatic hips in 9 male patients:

7 core decompression and recovered clinically after 1.5 months (0.5–8 M) and radiologically after 3 months

5 conservative therapy and improved clinically after 6 months (4–8 M)

[4] Radke S, et al. (2003) reported a study of retrospective evaluation of 43 cases of BME

38 core decompression, 24 cases recovered in less than 6 months, and 11 cases recovered after 6 months

5 conservative therapy, 4 patients improved after 6 months, and 1 patient improved before 6 months

and also suffered a subchondral fracture [70]. In pregnancy, mechanical overload and metabolic changes, such as an increase in 24-h urinary excretion of calcium, low PTH levels during the first trimester, increase in parathyroid hormone-related peptide (PTH-rP) levels around the mid-second to third trimester, and upregulation of calcitriol (1,25 $(OH)_2$ vitamin D) may increase susceptibility to microfracture and fracture [17, 96–98]. Increase in bone resorption markers and decrease in bone mineral density are found during pregnancy and lactation. During lactation, suckling and prolactin reduce estradiol and progesterone by suppressing the

Table 6 Fracture data for femoral neck, subcapital, and subchondral fracture and time of detection in 437 reported cases with transient osteoporosis of the hip [25, 26, 29, 39, 49, 52, 55, 63, 70–95]

	Number	Age (age range)	Time of detection of fracture
Femoral neck or subcapital fracture in female[1]	15	31 (22–37)	11 cases before starting treatment
			3 cases during conservative therapy
			1 case during calcitonin & conservative therapy
Femoral neck or subcapital fracture in male	2	40[2] (OI) & 79[3]	Both of them during conservative therapy
Femoral head fracture female	1	30[4]	Before starting treatment
Subchondral fracture female	9	49 (27–74)	8 cases before starting treatment
			1 case during conservative therapy
Subchondral fracture male	9	41 (27–54)	8 cases before starting treatment
			1 case during conservative therapy

[1] We found 7 more cases with TOH and femoral neck or subcapital fracture (without complete data about time of fracture and age) during or after pregnancy

[2] Young SD, et al. (2008) reported two case of osteogenesis imperfecta, one of them complicated by femoral neck fracture

[3] Guardiano SA, et al. (2004) reported a 79-year-old man with a long history of relapsing and remitting lower extremity joint pain who developed left ankle and hip pain. MRI showed BME. The patient tripped and fell and the low-energy trauma resulted in hip fracture, and so surgical repair, calcitonin, and alendronate were prescribed

[4] Sarah Steib-Furno, et al. (2007) reported 12 cases of gestational or early postpartum hip disease and 6 of them had TOH (26–36 y/o). Four cases (27–31 y/o) had femoral head (stress) fracture, and 1 of them had subchondral and femoral head fracture

hypothalamic-pituitary-ovarian axis. Suckling, prolactin, low estradiol, and calcium-sensing receptor increase production of PTH-rP from the mammary tissue and placenta [66, 99]. Most fractures (femoral neck, subcapital, femoral head, and subchondral fracture) occur before treatment, but some happen during conservative therapy (Table 6). As a clinical point, therapy to shorten recovery time might be helpful with regards to reducing risk of fracture.

Progression of TOH to AVN or osteonecrosis is another rare but potential complication of TOH. Five articles report the development of AVN in 9 cases of TOH [100–104]. Differentiation of AVN and TOH is important. Szwedowski et al. (2014) outlines that in AVN, T1-weighted imaging shows fibrotic connective tissue around necrotic bone with a weak signal. Bone marrow edema of AVN occurs in more advanced stages of necrosis and has correlation with pain progression and fracture of the femoral head [16]. Signs on MRI that indicate osteonecrosis include femoral head deformity, crescent sign (seen as subchondral radiolucency on MRI), single-line sign with edema on T1-weighted images, double-line sign with edema on T2-weighted images, low signal intensity of subchondral area, or the presence of subchondral area of low signal intensity at least 4 mm thick on T2 or contrast-enhanced T1-weighted images (Table 2) [31, 105].

Summary

Transient osteoporosis, especially TOH, usually presents with the acute onset of pain. A primary insult causing microfractures and vascular disturbance may result in bone marrow edema with separation of the bone trabeculae. Transient marrow edema causes temporary bone loss. While TOH usually affects the hip, it may migrate to other joints, especially those with weight bearing. MRI is the best method of diagnosis of TOH and to rule out traumatic injury, fracture, degenerative processes, inflammatory diseases, ischemic injury, infectious, and neoplasia. Differentiating AVN from TOH on MR imaging is important to prevent needless surgical intervention. Based on reported cases in the literature, medical therapies (including teriparatide, zoledronic acid, or combination of alendronate and calcitonin) may reduce duration of symptoms. Core decompression is not better than medical therapy in TOH alone. Pregnant women are at risk of femoral neck fracture, yet most treatments are contraindicated. In pregnancy, calcitonin, might shorten the duration of recovery which may prevent this complication. However, further investigation and randomized clinical trials are needed to determine the best method to reduce pain and improve bone health simultaneously, in order to decrease risk of complications such as AVN, fracture and joint degeneration [106].

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Compliance with ethical standards

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