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CASE REPORT

Regional migratory osteoporosis: case report of a patient with neuropathic pain

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Abstract Regional migratory osteoporosis (RMO) is an idiopathic disorder characterized by severe periarticular pain, transient and migratory arthralgia, and osteoporosis. Osteoporosis in this disease may appear in the form of local regional osteoporosis and bone marrow edema or generalized osteoporosis. It occurs most commonly in middle-aged men and late second or third trimester pregnant women. The laboratory findings of the disease are usually normal and do not demonstrate apparent anomalies. The presence of bone marrow edema on MRI is its characteristic finding. RMO can only be separated from transient osteoporosis of hip and avascular necrosis with migration to other joints. Clinically, RMO progresses in three stages: increasing pain and disability, radiological findings (osteopenia), maximalization of symptoms, and finally, the regression of the disease and radiological changes. In this case report, we present a 29-year-old woman whose symptoms had first appeared at the second trimester of pregnancy and migrated both to the other joints in the proximo-distal direction and to the adjacent bones within the same joint. She also had symptoms such as hyperalgesia, hyperesthesia and hypertrichosis along with neuropathic pain, which she described as a burning, biting, and prickling type of pain at the right leg. The neuropathic pain of the patient was resistant to medical treatment. We believe that this case was worth reporting because of the obstinate clinical course of the patient's disease and her severe neuropathic pain that was resistant to treatment.

Keywords Regional migratory osteoporosis · Neuropathic pain · Reflex sympathetic dystrophy · Transient migratory osteoporosis

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Introduction

Regional migratory osteoporosis (RMO) was first described in 1959 by Curtiss and Kincaid in three pregnant women suffering from increasing hip pain. Localized spotty demineralization involving femoral head, neck, and acetabulum was detected by roentgenogram in two of the women right after delivery and in one, 3 months after delivery. All three patients were reported to recover within several months after birth [1]. RMO is an idiopathic disorder [2–5] characterized by severe periarticular pain located mostly in the weight-bearing joints of the lower limb [2–6]. The patients have transient and migratory arthralgia and osteoporosis, which may appear in the form of local regional osteoporosis and bone marrow edema or generalized osteoporosis and may involve the spine [2–6]. The disease occurs most commonly in middle-aged men and late second or third trimester pregnant women [4, 7–9].

Clinical findings are joint tenderness, soft tissue swelling, effusion, muscle atrophy, and restriction in range of motion (ROM) [2, 4, 10]. Typically, RMO starts with acute or gradually increasing joint pain. The pain increases in weeks or months and consequently, the patient becomes unable to walk [2, 3, 6]. The pain can occur at rest and at

night [5]. Multiple joints can be involved [6, 8] and migration within the same joint [6, 11, 12] and to the adjacent bone may occur [6, 11–13]. The disease rarely reoccurs at the same joint [3–6, 14].

Although vasomotor instability and dystrophic changes are not seen in RMO [4, 5], some authors have emphasized that the etiology of RMO resembles that of reflex sympathetic dystrophy (RSD) in terms of the disease's rapid clinical progression, difficulty in weight-bearing, limping, similar osteoporosis without narrowing of the joint space and similar recovery period [15].

The etiopathogenesis of the disease is still elusive. Genetic predisposition [9], local circulatory disturbance [9], Sudeck's atrophy [9, 15], compression of the obturator nerve [1], bone medullary hypertension and small vessel ischemia [14], subperiosteal and subchondral fat embolism [16], chemical and hormonal factors related with pregnancy are accused [8]. Frost [3, 17] introduced the regional acceleratory phenomenon (RAP) to elucidate the pathogenesis of the disease.

Bone marrow edema on MRI is a characteristic finding of the disease. On plain radiograph, the bone may appear normal [6] or focal or diffuse osteoporosis may be observed [2, 3, 13]. RMO is differentiated from avascular necrosis (AVN), the most confluent clinical condition, by the absence of joint space narrowing and damage [13, 18], and the 'double-line' sign on MRI, which is a product of the demarcation zone [18, 19]. Although RMO is clinically and radiologically similar with the transient osteoporosis of the hip (TOH), migration is not seen in TOH. [1, 7, 19, 20].

We present a RMO patient with severe neuropathic pain whose RSD-like symptoms were resistant to the treatment.

Case report

A 29-year-old woman was referred to our outpatient clinic with right hip pain and difficulty in weight-bearing and walking. She reported that her symptoms had begun at the 23rd week of her pregnancy and gradually increased. During the postpartum period, she had been wakened many times by pain.

Her medical history showed that she had low dietary calcium intake due to lactose intolerance along with hyperemesis gravidarum. She had osteopenia, which was coincidentally diagnosed with dual energy X-ray absorptiometry during a clinical research, in which she was a member of the healthy control group. The patient had cesarean section a week ago and has been breast-feeding her baby since then.

Physical examination of the patient revealed that, although her right inguinal area was extremely sensitive upon palpation and her active right hip movements were painful in all directions, passive movements of the hip were painless. She was unable to bear weight on her right hip. Her neurological examination and plain hip radiographs were normal.

The initial diagnosis was iliopsoas tendonitis. Accordingly, cold pack application and moderate stretching exercises were started. At the follow-up visit, 3 days later, there was no response to the therapy. Her pain became worse and was occurring at rest and at night. Hip MRI was requested upon the prediagnosis of AVN and TOH. On MRI, joint effusion and bone marrow edema in right femoral head and linear fracture at the subcapital area were evident (Fig. 1 a, b). Radiological diagnosis was AVN of the right femoral head. The patient was referred to an

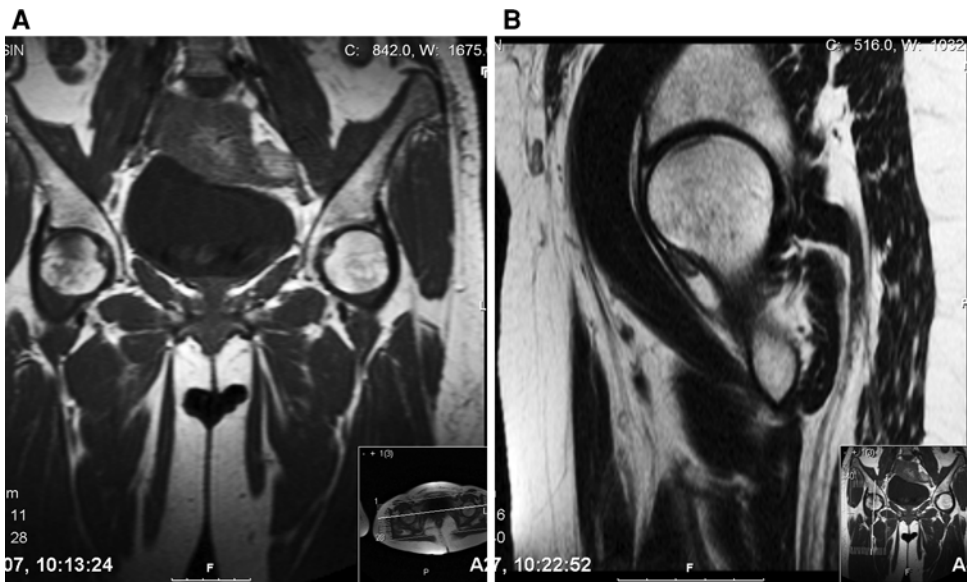

Fig. 1 T1-weighted (**a**) coronal and (**b**) sagittal (right hip) MRI of the hips; bone marrow edema (**a**) in right femoral head and linear fracture (**b**) at the subcapital area is evident

orthopedic surgeon and immobilization for 3 months, non-weight-bearing and passive ROM exercises were advised.

Laboratory tests were carried out. Alkaline phosphatase (ALP), C-reactive protein (CRP), serum calcium, serum osteocalcin, urinary deoxypyridinoline (DPD) and urine protein discharge were mildly high. APTT and PTT were low. Erythrocyte sedimentation rate (ESR) and complete blood count (CBC) were normal. Serum rheumatoid factor and antinuclear antibodies were negative.

In the second week of the patient's follow-up, she had effusion at her right knee and accordingly, cold pack, isometric exercises and ibuprofen 400 mg (3 times a day) were started. At the first follow-up visit, right knee MRI was requested due to the persistence of her complaints and the detection of joint effusion on right knee MRI. At the 6th week of the follow-up, hip MRI revealed clear regression of the bone marrow edema at the right femoral head and it was decided upon that immobilization should be continued for an additional 6 weeks.

During the follow-up, she had left hip pain and a burning, biting, and prickling pain at the right leg (from the hip to the foot). Moreover, hyperalgesia, hyperesthesia, and hypertrichosis were added to her symptoms. Allodynia, hyperpathy, extreme sweating, edema and coldness were present at both legs. Transcutaneous electrical nerve stimulation (TENS) for the knee and hip pain and electrical stimulation (ES) for the quadriceps atrophy were started. At the 12th week of her treatment, hip MRI revealed regression of the bone marrow edema at the right hip. However, there was minimal bone marrow edema at the left femoral head and effusion at the left hip. Due to the improvement observed on MRI of the right hip, mobilization was advised. However, the patient was able to walk only a few steps by the help of a walker.

Due to an increase in the patient's knee pain, a knee MRI was performed. On MRI, bone marrow edema was observed in both femoral medial condyles (more prominent in right one) (Fig. 2). Based on the patient's clinical and radiographic findings, she was diagnosed with RMO.

The patient was referred to a hydrotherapy and fitness program. Although this program helped increase her endurance, there was no decrease in her hip pain. After she gave-up breast-feeding, nasal salmon calcitonin 200 IU/day for RSD symptoms and pregabalin 300 mg/day for neuropathic pain were prescribed. Her pain and RSD-like symptoms did not respond to the calcitonin therapy and since she had ulcerative hemorrhagic rhinitis, calcitonin was stopped after 3 months. There was improvement in her pain with pregabalin and she has been using the drug to date.

At the 6th month, MRI scanning of the hip demonstrated a decrease in the joint effusion and edema was no more evident in the right hip (Fig. 3). Although a decrease was observed in the bone marrow edema in the femoral condyles, a new bone marrow edema was detected in the right tibial plateau (Fig. 4). Our case is interesting since migration within the same joint is a rare phenomenon [2, 5, 6, 11–13].

Fig. 2 T2-weighted fat suppressed MRI of the right knee; bone marrow edema in medial femoral condyle is seen

Fig. 3 T1-weighted control hip MRI; no more edema is evident in right hip

The patient began to mobilize using two canes, with weight-bearing as tolerated weight shifting exercises were started; TENS for knees and ES for the quadriceps muscles were continued. Tramadol 100 mg/day was added to the

Fig. 4 T2-weighted fat suppressed MRI of the right knee; decrease in the bone marrow edema at the medial femoral condyle is evident. A new bone marrow edema at the right tibial plato is seen

Fig. 5 T2-weighted fat suppressed MRI of the right ankle; diffuse bone marrow edema is seen at the distal end of the tibia, talus and calcaneus

medical treatment for pain control. In literature, oral and i.v. biphosphonates are described to be effective in resolving symptoms and bone marrow edema within months [3, 4, 10, 21–23]. Initially, we did not use biphosphonates due to the age and fertility status of the patient. However, persistent symptoms and the migration pattern necessitated the addition of oral biphosphonates to the therapy. Oral alendronate sodium 70 mg/once a week and 1,000 mg calcium and 880 IU D3 vitamin once a day were prescribed. There was poor response to the alendronate treatment and it was stopped after 3 months due to the patient's age and fertility status.

At the 9th month of the follow-up, she had pain and swelling in her right ankle while MRI revealed diffuse bone marrow edema at the distal end of the tibia, talus, and calcaneus (Fig. 5). Cold pack, whirlpool, and analgesic electrotherapy were added to the treatment. Oral alendronate sodium 70 mg/once a week was added to the therapy for 3 months. Her symptoms resolved within 3 weeks with alendronate.

At the 12th month of follow-up, knee MRI (Fig. 6) and ankle MRI revealed a decrease in bone marrow edema. Her symptoms calmed down with incomplete recovery and she returned to work and mobilized with a cane.

At the 19th month of the follow-up, her foot pain increased and on the MRI of the right foot; bone marrow edema was evident in the proximal portion of the first metatarsal bone (Fig. 7).

Fig. 6 T2-weighted fat suppressed MRI of the right knee; completely normal

At the last follow-up visit, she has been mobilizing without any assistive device but she has been using pregabalin 150 mg 2 × 1 for her neuropathic pain and also paracetamol and non-steroidal anti-inflammatory drugs for pain relief when needed.

Fig. 7 T2-weighted fat suppressed MRI of the right foot; bone marrow edema at the proximal portion of the first metatarsal bone is evident

Discussion

Regional migratory osteoporosis is a rare disease [3, 4]. Typically, it begins with acute or gradually increasing joint pain, increases within weeks or months and eventually, the patient becomes unable to walk [2, 3, 6]. The pain usually begins with weight-bearing and increases with joint movements, and can occur at rest and during sleep [5]. The patient may have to use crutches or a wheelchair [5, 7, 8]. As in our case, joint tenderness, soft tissue swelling, effusion, muscle atrophy, and restriction in ROM can be seen [2, 4, 10]. Commonly, a history of trauma is absent [2], and similarly to our case, the hip joint is the most frequently involved joint [4, 5].

The symptoms become evident in a few months and usually calm down in 6–12 months. The disease may end spontaneously with complete recovery or migrate to another joint [4, 5, 12].

More than one joint may be involved at the same time and migration in the same joint and to the adjacent bone may be observed [6, 8, 11, 13]. In our case, the two knees were involved simultaneously. Migration to the adjacent bone (from medial condyles to the tibial plateau) in the same joint pattern was seen in the right knee.

The symptoms of our patient became evident in 2 months. Initially, the right hip was involved. The disease migrated to the right knee on the 2nd week, to the left hip on the 10th week and to both femoral distal ends on the 15th week. On the 24th week, proximal tibial end, on the 38th week distal tibia end, talus and calcaneus and finally on the 59th week, metatarsal bones were involved. Generally, it tends to migrate from the proximal joints to the distal ones, as in our case [2–4, 6]. It always migrates ipsilaterally, and the involved sites may sometimes overlap [2, 3]. Migration occurs usually within the first year. Rarely, it may be reported after many years [4].

Neurological deficits or atrophic skin changes are not seen in RMO [5]. It can reoccur in the same joint [3, 5, 14, 24] and may involve the spine [2, 5]. RMO has three clinical phases:

1. Gradually increasing pain and disability;
2. Radiographic osteopenia and maximalization of the symptoms;
3. Regression of the disease and radiographic changes [8, 15].

Spontaneous recovery is the rule and radiographic remineralization may occur in a period of 2 months to 2 years [2, 20].

Low calcium dietary intake (<400 mg/day), smoking and osteogenesis imperfecta are known to be the risk factors for the disease [3, 24]. Our patient had low dietary calcium intake and was osteopenic.

Laboratory findings are usually insignificant, and high levels of ESR [5, 8] and hypercalciuria [2, 3] may be observed. Elevation of ALP [10, 21], bone-specific ALP [19], and osteocalcin [19] may also be observed. ALP, osteocalcin, CRP, blood calcium, DPD, and urine protein discharge levels of our patient were mildly high, while APTT and PTT were low, and ESR and CBC were normal.

Upper limb involvement is rarely seen in RMO [5] while vasomotor instability and dystrophic changes are not observed [4, 5]. On the other hand, permanent joint damage is not expected [8] and neuropathic pain has not been reported to date.

However, in our case, neuropathic pain, sensorial (hyperalgesia, allodynia, hyperpathy), sudomotor (extreme sweating and edema), vasomotor (coldness) instability, and dystrophic changes (hypertrichosis) were significant and pregabalin, calcitonin, and tramadol were used to relieve these symptoms.

Etiopathogenesis of the disease has not been clarified yet. RMO has been described as a coxo-femoral variation of Südeck's atrophy in some studies [9, 15]. Lesquesne [15] stressed the disease's similarity with RSDS in terms of etiology and emphasized that the two diseases have similar characteristics such as rapid clinical progression, difficulty in weight-bearing, limping, osteoporotic appearance, and recovery period. RMO is considered as different from RSD because skin atrophy, sensory–motor alterations, and contractures are not seen in RMO. It has a good prognosis with

benign and self limiting behavior without treatment [2, 3, 19]. RSD is usually preceded by trauma and commonly involves upper extremities [2].

Genetic predisposition [9], local circulatory disturbance [9], compression of the obturator nerve [1], bone medullary hypertension and small vessel ischemia [14], subperiosteal and subchondral fat embolism [16], chemical and hormonal factors related with pregnancy are accused [8]. The potential negative calcium balance, which may appear during pregnancy and lactation, does not seem to be a valid cause, since it can not explain selective demineralization [14]. Bone loss is commonly associated with estrogen-deficiency states. Thus, it is interesting that bone loss occurs in RMO patients during pregnancy, in the course of which plasma oestradiol levels remain high [7, 8, 14]. Axt-Fliedner et al. [7] suggested that low dose sequential estrogen–gestagen therapy should be used during puerperal period unless the normal menstrual cycles begin.

Frost [3, 17] introduced RAP for the pathogenesis of RMO. He suggested that the ordinary biological regional processes such as blood flow, cell metabolism and turnover, and tissue remodeling are greatly accelerated under noxious tissue stimuli. RAP normally occurs after a fracture, artrodesis or osteotomy and provides 2–10 times faster recovery. However, a prolonged or exaggerated RAP can activate a large number of bone turnover focuses and can cause transient osteoporosis. Bone microdamage is supposed to be the most common noxious tissue stimulus [3, 17]. When microdamage accumulation is too fast or repair processes are impaired, RAP is activated [3]. In a study by Banas et al. [2], the authors obtained transiliac bone biopsies from patients and determined an increase in the number of osteoclasts per mm^2 of the medullary bone space and in the cellular rate of mineralization, which proves accelerated bone remodeling. Trevisan et al. [3] argued that, in some cases, osteopenia, a relative overload due to the increased body weight, and the hormonal status of pregnancy may combine to determine microdamage accumulation, RAP induction and transient osteoporosis. Our patient gained 20 kg during pregnancy.

Focusing on the etiology of the intramedullary pressure highness, Hofmann et al. [25] performed core decompression in 10 patients whose pain did not respond to conservative treatment. They stated that RMO may be an early phase of AVN and the repair capacity of the femoral head determines the exact clinical picture. Common MRI findings reported in patients with RMO are bone marrow edema, effusion in the joint, and linear subchondral lesions (trabecular insufficiency fractures). On MRI, the bone marrow edema was seen as a reduced signal on T1-weighted images and an increased signal on T2-weighted images [4, 11, 18, 20, 26]. Since these findings were similar with AVN, our initial diagnosis was AVN. However, after the resolution of the bone marrow edema and migration to another focus, our definitive diagnosis was RMO.

In differential diagnosis, AVN, rheumatoid arthritis, osteoarthritis, gout, infectious arthritis, osteomyelitis, malignancy, calcium storing diseases, multiple myeloma, tuberculosis, demineralizing variety of synovial chondromatosis must all be taken into consideration [2, 12, 15]. The most important disease in the differential diagnosis of RMO is AVN. Some authors believe that RMO is a reversible stage of AVN [3, 25]. The radiological findings of RMO are indistinguishable from AVN. When the radiological findings of a patient with clinical AVN imply a change and the symptoms seem to migrate to another joint, the diagnosis is changed to RMO [4]. In radiological investigations, RMO can only be separated from TOH and AVN with the presence of migration.

Some pregnant women with RMO cannot have vaginal delivery due to pain and ROM restriction in the hip. Elective cesarean section is recommended to such patients.

Funk et al. [20] recommended against breast-feeding because of the rapid BMD increase after the end of lactation. Our patient had cesarean section on the 39th week of her pregnancy, and after 6 months, lactation was stopped.

Non-weight-bearing (to decrease microscopic trabecular fractures) and analgesics are effective in medical treatment [4, 24]. Infusion of iloprost (Prostacyclin analog) has been reported to help resolve the symptoms within 3 days to 3 months (reduction of capillary permeability with vasodilatation and inhibition of platelet aggregation), and to bring out complete resolution of bone marrow edema within 6 weeks [4, 27, 28]. Oral and i.v. biphosphonates are also effective in resolving symptoms in 1 month and are accompanied by complete resolution of bone marrow edema within 3 months [3, 4, 10, 21–23, 29]. In our case, iloprost was not administered to our patient because she was in the early postpartum period and was using anticoagulants for protection against pulmonary embolism. At the beginning, biphosphonates were not used due to the patient's age and fertility status. However, after obstinate clinical symptoms were observed, oral biphosphonates were added to the therapy. Although our patient's symptoms could be controlled with this medical therapy, she still uses pregabalin for her neuropathic pain.

In conclusion, our case is worth presenting due to the patient's severe, persistent, and neuropathic pattern of pain and the obstinate clinical course of her disease. Although RMO has a self-limited nature, reoccurrence, and prolonged duration of symptoms may complicate the process. Moreover, due to its unknown etiopathogenesis, a symptomatic approach is followed and there is no consensus on treatment approaches. Early differentiation from other aggressive diseases with sequelae and poor prognosis is crucial to avoid unnecessary treatment. Pain in RMO may

be very severe and persistent and have a neuropathic pattern, as in our case. Furthermore, the disease may cause disability and affect the quality of life.

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