CASE REPORT

Does the Necrosis Develop Simultaneously in Patients with Bilateral Hips Necrosis? A Case Report

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Introduction

Osteonecrosis of the femoral head (ONFH), also known as avascular necrosis of the femoral head, is a debilitating disease that affects relatively young and active patients. It is defined as the cell death of marrow and mineralized bone, and represents the final pathway of several disease conditions. Among various known causes, steroid therapy is responsible for a significant majority of these cases. More than half of corticosteroid-induced osteonecrosis affects both femoral heads¹⁻³; bilateral ONFH is considered to develop simultaneously⁴. Only one case study has reported development of contralateral ONFH after increasing the dose of corticosteroid⁴. To our knowledge, no reports have focused on the time course over which this condition develops in both hips after brief pulse treatment with steroids. We here describe a case of ONFH after steroid therapy in which serial MRI imaging revealed asynchronous development of bilateral lesions. The study was approved by the institutional review board (IRB) and informed consent was obtained from the patient.

Case Report

A 35-year-old man presented with onset of right hip pain one month after receiving steroid therapy for three days (dexamethasone, 10 mg per day) for idiopathic deafness. The pain was continuous, exacerbated by walking and not completely relieved by non-steroidal anti-inflammatory drugs. The patient's height was 175 cm, weight 72 kg and his body mass index normal (23.5). Initial examination revealed tenderness in the right inguinal region and the range of motion of the right hip was significantly limited. Physical examination of the left hip was normal. Laboratory examinations such as erythrocyte sedimentation rate, serum C-reactive protein concentration and other routine blood tests were normal. Bone mineral density was not measured.

At his first visit, which was two days after the initial onset of hip pain, an anteroposterior radiograph of both hips revealed no abnormalities (Fig. 1). The findings on MRI of the right hip on axial T1-weighted and coronal T2-weighted images were normal (Figs. 2A, B). However, an indistinct, discontinuous high signal band on axial and coronal short time inversion recovery (STIR) images was observed in the right hip. A Grade 2 effusion⁵ was also observed. No abnormality was found in the left hip (Figs. 2C, D).

Eight days later, another MRI was performed to investigate the progression of the condition. Axial T1-weighted and coronal T2-weighted images still showed normal signal intensity (Figs. 3A, B). On STIR images, a continuous high signal band with bone marrow edema was observed in the right femoral head and an obscure high signal band had started to appear on STIR images in the left femoral head (Figs. 3C, D).

Fifty-two days later, a third MRI examination was performed. Osteonecrosis in the right femoral head (Figs. 4A, B),

Fig. 1 Anteroposterior radiograph of both hips showing normal bony structure two days after onset of right hip pain.

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Fig. 2 The first MRI. (A) Axial T1-weighted image and (B) coronal T2-weighted image show no abnormality in either hip. (C) Axial STIR and (D) coronal STIR images show discontinuous high signal bands in the right femoral head and normal signals in the left femoral head.

Fig. 3 The second MRI performed eight days after the first. (A) Axial T1-weighted and (B) coronal T2-weighted images show no abnormality in either hip. (C) Axial STIR and (D) coronal STIR images show continuous high signal bands with bone marrow edema in the right femoral head and obscure high signals in the left femoral head.

Fig. 4 The third MRI performed 52 days after the first imaging. Typical osteonecrosis is apparent in both hips and there is bone marrow edema in the right femoral head. (A) Coronal T1-weighted image. (B) Coronal T2-weighted image.

which had shown progression of the condition on the previous MRI, was confirmed. There was subchondral collapse and evidence of flattening of the femoral head, as well as significant edema around the femoral head and surrounding soft tissue. In addition, there were typical low signal intensity bands on T1-weighted images and a high signal band on the corresponding STIR images of the left femoral head, indicating early ONFH.

Discussion

The known causes include trauma, steroid intake, alcohol abuse and several other conditions. Corticosteroid administration is one of major reasons for osteonecrosis; however, though the exact mechanism(s) has yet to be fully understood^{2,3,6}. Various theories have been proposed, including compartment syndrome, occlusion of blood vessels, fat embolism and fat cell hypertrophy. In 50%–70% of cases in which the osteonecrosis has developed secondary to steroid intake, both hips are affected and the lesions are believed to develop simultaneously¹. Only one case study has reported development of contralateral ONFH after increasing the dose of corticosteroids⁴. The current case is the first report to document a temporal difference in the development of osteonecrosis in both after brief pulse treatment with steroids. Recurrence of osteonecrosis has also seldom been reported. Kim *et al.* reported three ONFH cases in which MRI scans showed a progressively increasing extent of necrosis⁷. In a histological study of 606 necrotic femoral heads removed surgically, only two cases (0.3%) showed evidence of progression of osteonecrosis⁸. However, the study also advised caution in classification of these lesions as recurrence or extension of focal necrosis because an increase in the necrotic area caused by collapse at the junction between the lesion and the adjacent viable bone should not be so classified⁸. The rarity of recurrence or newly developed osteonecrosis in the contralateral hip indicates that osteonecrosis is generally the result of a single episode of injury and is not an ongoing, progressive disease. Many studies have shown that osteonecrosis usually develops within six months of commencing steroid therapy, typically within two to three months^{6,9–11}. However, how long the pathological process continues and the interval between occurrence of necrosis and

steroid intake has not been reported. In this case, we observed a time lag between the appearance of ONFH on MRI images from right and left hips. This was particularly interesting because the patient had received only a short course of steroid treatment and had no other identifiable risk factors such as alcoholism, history of trauma or sickle cell disease. This suggests that, although the short course of steroids had caused the ONFH, the development of osteonecrosis in the two hips was asynchronous.

One possible explanation for asynchronous development is that other unidentified preexisting factors such as thrombophilia, hypofibrinolysis, lipid metabolism abnormalities and cell toxicity may play a role in the development of ONFH, resulting in the detrimental effect persisting beyond the actual time when the insult, in this case steroid administration, took place. The time course of abscess development in pyosepticemic patients provides a possible analogy. The etiopathogenesis and presentation of ONFH differs from that of other ischemic diseases, such as myocardial infarction or cerebral infarction, which typically present as acute episodes, or sometimes as recurrent attacks. Interruption of blood supply to the bone is the final pathologic process resulting from an unknown mechanism.

Another possible explanation may lie in the fact that the repair capacity of different body parts after bone necrosis varies; only when the marrow has been infiltrated by significant numbers of abnormal plasmacytes or lymphocytes can ONFH be detected by MRI¹². In our patient, the first abnormal signal was not the classical “band sign” on T1-weighted images or “double band sign” on T2-weighted images or high signal band sign on STIR images^{1–3,13}, but discontinuous high signal bands on STIR images. At the subsequent MRI examination, continuous and high signal band signs were present on T1-weighted and STIR images. This suggests that discontinuous, obscure high signals on STIR images may be the first detectable MRI evidence of ONFH. The continuous low signal band on T1 weighted images is a relatively later indication of osteonecrosis, possibly indicating that the area of necrotic bone has been separated from healthy tissue by fibrosis and sclerotic bone. A more sensitive exam, such as STIR, or a repeated MRI should be performed when there is a suspicion

of avascular necrosis of bone, especially after steroid therapy. Early intervention may be effective and timely during the period prior to formation of fibrous tissue and sclerotic bone.

We documented asynchronous development of osteonecrosis in both hips after steroid therapy in this case. Repeated

MRI exams were performed a few weeks apart, as they commonly are in patients at high risk of bilateral osteonecrosis. The findings may have an impact on future research in this area, especially with regards to developing protocols for screening, diagnosis and prophylactically treatment of the second hip affected by ONFH.

References

1. Fukushima W, Fujioka M, Kubo T, Tamakoshi A, Nagai M, Hirota Y. Nationwide epidemiologic survey of idiopathic osteonecrosis of the femoral head. *Clin Orthop Relat Res*, 2010, 468: 2715–2724.
2. Mont MA, Hungerford DS. Non-traumatic avascular necrosis of the femoral head. *J Bone Joint Surg Am*, 1995, 77: 459–474.
3. Mont MA, Jones LC, Hungerford DS. Nontraumatic osteonecrosis of the femoral head: ten years later. *J Bone Joint Surg Am*, 2006, 88: 1117–1132.
4. Sonoda K, Yamamoto T, Motomura G, Yamaguchi R, Karasuyama K, Iwamoto Y. Contralateral osteonecrosis of the femoral head newly developed after increasing the dose of corticosteroids. *J Orthop Sci*, 2014, Feb 10. [Epub ahead of print].
5. Chan WP, Liu YJ, Huang GS, Jiang CC, Huang S, Chang YC. MRI of joint fluid in femoral head osteonecrosis. *Skeletal Radiol*, 2002, 31: 624–630.
6. Lafforgue P. Pathophysiology and natural history of avascular necrosis of bone. *Joint Bone Spine*, 2006, 73: 500–507.
7. Kim YM, Rhyu KH, Lee SH, Kim HJ. Can osteonecrosis of the femoral head be recurrent? *Clin Orthop Relat Res*, 2003, 406: 123–128.
8. Yamamoto T, DiCarlo EF, Bullough PG. The prevalence and clinicopathological appearance of extension of osteonecrosis in the femoral head. *J Bone Joint Surg Br*, 1999, 81: 328–332.
9. Guo KJ, Zhao FC, Guo Y, Li FL, Zhu L, Zheng W. The influence of age, gender and treatment with steroids on the incidence of osteonecrosis of the femoral head during the management of severe acute respiratory syndrome: a retrospective study. *Bone Joint J*, 2014, 96: 259–262.
10. Sugano N, Nishii T, Shibuya T, Nakata K, Masuhara K, Takaoka K. Contralateral hip in patients with unilateral nontraumatic osteonecrosis of the femoral head. *Clin Orthop Relat Res*, 1997, 334: 85–90.
11. Shibata M, Fujioka M, Arai Y, et al. Degree of corticosteroid treatment within the first 2 months of renal transplantation has a strong influence on the incidence of osteonecrosis of the femoral head. *Acta Orthop*, 2008, 79: 631–636.
12. Vande Berg BC, Lecouvet FE, Galant C, Maldague BE, Malghem J. Normal variants and frequent marrow alterations that simulate bone marrow lesions at MR imaging. *Radiol Clin North Am*, 2005, 43: 761–770. ix.
13. Zhao FC, Li ZR, Zhang NF, et al. Lesion size changes in osteonecrosis of the femoral head: a long-term prospective study using MRI. *Int Orthop*, 2010, 34: 799–804.