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Does Altered Biomechanics Cause Marrow Edema?[1]

PURPOSE: To determine if altered weight bearing causes the appearance of marrow edema on magnetic resonance (MR) images.

MATERIALS AND METHODS: Twelve volunteers underwent MR imaging with a short inversion time inversion-recovery (STIR) sequence at 1.5 T. The hips, knees, ankles, and feet were evaluated before and 2 weeks after altered weight bearing achieved with overpronation of one foot. Three volunteers underwent imaging a third time, 2 weeks after overpronation was stopped. Two observers assessed the images for evidence of marrow edema.

RESULTS: Changes were seen on images in 11 volunteers; the overpronated side only was affected in 10. Most changes occurred in the foot followed by the tibia and the femur. Most changes were a diffuse increase in marrow edema. In two volunteers, the changes resembled those of stress fractures.

CONCLUSION: Altered weight bearing should be added to the list of causes of increased medullary signal intensity (ie, marrow edema) on MR images.

Index terms: Bone marrow, abnormalities, 48.49 • Bone marrow, MR, 48.121413 • Bones, 40.49

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Described causes of increased intramedullary signal intensity on T2-weighted magnetic resonance (MR) images include avascular necrosis (1), primary and metastatic tumors (2), osteomyelitis (3), fractures, bone bruises (4), and transient osteoporosis (5). Although little histologic evidence is available, several authors refer to increased intramedullary signal intensity as marrow edema (1,2). To determine if altered weight bearing causes the appearance of marrow edema on MR images, we imaged volunteers at baseline, after an alteration in weight bearing, and after return to normal ambulation.

MATERIALS AND METHODS

After informed consent was obtained, the hips, knees, ankles, and feet of 12 asymptomatic volunteers (six women, six men; age range, 19–41 years; mean age, 30 years) were examined with MR imaging at 1.5 T by using a commercially available imager (Signa; GE Medical Systems, Milwaukee, Wis). To obtain the baseline images, a fast spin-echo short inversion time inversion-recovery (STIR) sequence was used (6). The sequence was performed in the knees and hips in the coronal plane and in the feet and ankles in the sagittal plane (repetition time msec/echo time msec/inversion time msec = 6,000/48/150, echo train length of 8, 256 × 256 matrix, two signals acquired, 4-mm section thickness, 1-mm intersection gap). The hip and knee images were obtained with a body coil and 45- and 28-cm fields of view, respectively. The foot and ankle images were obtained with both feet in a head coil and a field of view of 28 cm.

Three sets of images were obtained: before (baseline) and 2 weeks after altered weight bearing, and in three volunteers, 2 weeks after removal of the pad and return to normal ambulation. The alteration in weight bearing was accomplished by placing an extra-large, 9/16-inch (1.4-cm) longitudinal metatarsal arch pad (Hapad, Bethel Park, Pa) underneath the lateral aspect of one foot to increase pronation. This orthotic was placed in the shoe, but the volunteers did not undergo casting with the foot in this position. Therefore, movement was altered somewhat voluntarily. Volunteers were instructed not to alter their daily or recreational activities in any way other than that caused by the pronation. Volunteers were given an adequate number of pads for all pairs of their shoes. The pads were placed unilaterally to minimize discomfort.

After 2 weeks of altered weight bearing, MR images of the both lower extremities were obtained with a protocol identical to that for the baseline images. Three randomly selected volunteers were imaged a third time, 2 weeks after removal of the pads, to determine if the marrow edema had resolved. At all times, it was emphasized to the volunteers that they should not alter the type or intensity of their usual activities.

All three sets of images were obtained with identical window and level settings. All images subsequently were assessed by the authors for areas of high signal intensity in marrow on the altered-weight-bearing images only. The observers were blinded only to the side on which the arch pad was placed. Signal intensity was compared with that of adjacent muscle as the standard. All volunteers were followed up clinically 1 day, 1 week, and 1 month after pad removal for the presence of pain to exclude true traumatic injuries.

RESULTS

MR images in 11 of the 12 volunteers demonstrated changes. The overpronated side only was affected in 10 volunteers. One of the volunteers with medial involvement had the findings only on the nonoverpronated side. These changes were seen most frequently in the foot (eight phalanges, four metatarsals, two calcanei, one medial cuneiform bone). Changes were predominant laterally in six volunteers. The tibia was affected in three volunteers (one diffusely, one proximally, one distally) (Fig 1), and in three volunteers, the

Abbreviation: STIR = short inversion time inversion recovery.

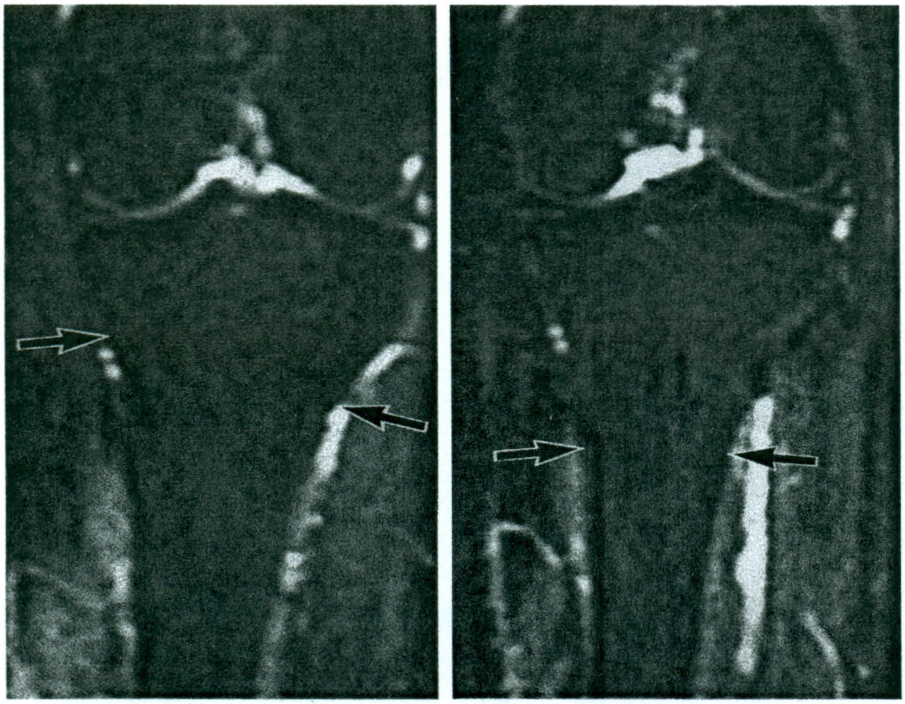


Figure 1. STIR images (2,500/48/150) of the left tibia in a 29-year-old woman. **(a)** Image obtained before altered weight bearing shows tibial marrow is hypointense to muscle (arrows). **(b)** Image obtained 2 weeks after altered weight bearing shows a diffuse change in appearance of tibial marrow to slightly hyperintense to muscle. Subtler changes also are present in the femur.

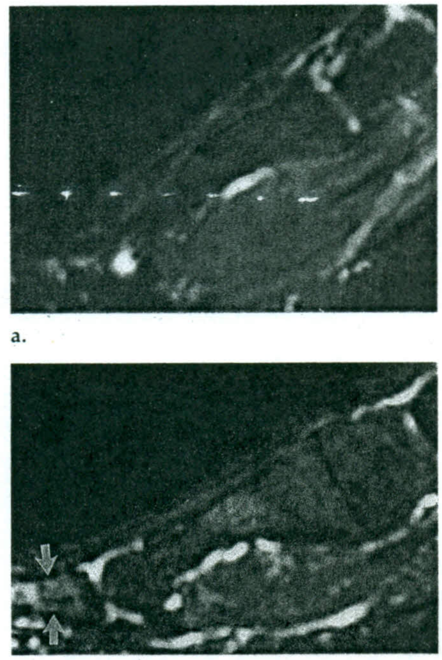


Figure 2. STIR images in the foot of a 31-year-old man. **(a)** Baseline image (2,600/48/150). **(b)** Image obtained after altered weight bearing shows a marked increase in marrow signal intensity in the first proximal phalanx (arrows) in comparison with **a**. The first metatarsal and medial cuneiform bone also are affected to a somewhat milder degree. An effusion has developed in the metatarsophalangeal joint.

femur was affected (one proximally, two distally). Multiple bones were affected in four volunteers (Fig 2). The changes usually consisted of a diffuse increase in marrow edema (nine volunteers), often intense (Fig 1), but occasionally the edema was more subtle (Fig 2). Less frequently, the changes appeared similar to those of stress-type fractures (two volunteers) (Fig 3).

At MR follow-up, in two of the three volunteers, the MR images returned completely to normal, but in the third volunteer, the MR images demonstrated minimal persistent edema. This residual edema was decreased in intensity and was more diffuse (Fig 3). All volunteers were completely asymptomatic immediately after pad removal and at clinical follow-up. Most volunteers, however, described discomfort while wearing the orthotic.

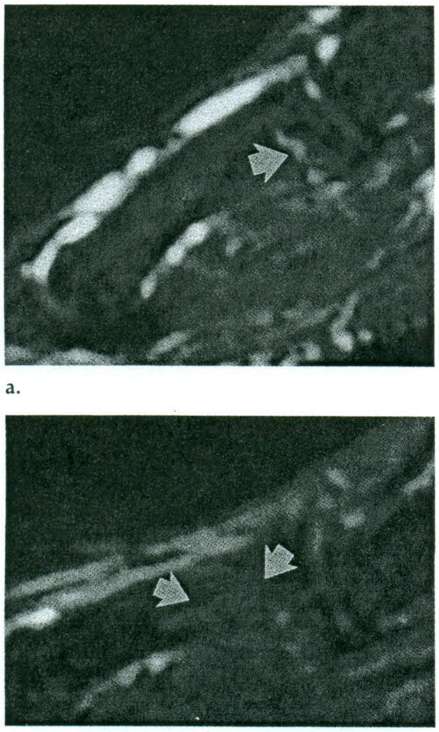
DISCUSSION

Bone is dynamic, undergoing hypertrophy in response to stress (7,8). Alternatively, after immobilization from casting (9) or paralysis (10) or in a gravity-free environment (11), bone atrophy occurs. Remodeling occurs as a response to these changes in stress. Because remodeled bone is known to contain microfractures (12), it may demonstrate abnormal uptake at scintigraphy (13). This physiologic response is associated with pain (14). Although, to our knowledge, marrow changes on T1-weighted MR images have never been proved, conjectures regarding their presence have been made (14). Because remodeled trabecular bone demonstrates both microfractures (12) and medullary edema (15) at histologic examination, not surprisingly we found that the MR imaging appearance of this response often mimicked that of a more diffuse bone bruise. The question arises whether all lesions that fulfill the previously described MR criteria for bone bruise (16,17) are truly traumatic. Since internal derangements cause altered biomechanics, perhaps a fraction of these marrow changes are a response to altered weight bearing.

The MR changes visualized in this study were sometimes similar to those described in stress fractures (18,19). These MR changes may represent the conjectured "stress response" or subclinical stress fracture (14,19). We agree with other prior authors that there is a continuum from physiologic response to stress fracture that represents a relative imbalance or uncoupling between bone resorption and bone formation (16,20). The MR imaging appearance depends on the patient's place in this continuum. Although it has been stated anecdotally that these changes can be painful, all volunteers in our study were free of symptoms immediately after removal of the pads and at 1-week and 1-month follow-up. In addition, the MR imaging appearance rapidly returned close to baseline. Therefore, although these changes sometimes had the appearance of stress fractures on MR images, they clearly were not fractures clinically.

The major stress in ambulation is on the metatarsals and the calcaneus (10). The load on the metatarsals during ambulation is closer to the stress threshold than that of any other part of the skeleton (21). This explains why the changes were seen most prominently in the feet, in particular, the metatarsals. We found the proximal phalanges to be affected even more frequently than the metatarsals. The explanation for this is overpronation excessively stresses the first ray in the toe-off phase of walking. Marrow changes, often subtle, were also seen in the hips, knees, and ankles.

This study was limited by the relatively small number, small age range,



a.

b.

Figure 3. STIR images (2,100/48/150) in the foot of a 49-year-old woman. The base of the first metatarsal was normal on the baseline image (not shown). **(a)** Image obtained after altered weight bearing shows a linear region of high signal intensity (arrow) has developed. **(b)** Image obtained 2 weeks after pad removal shows the marrow edema has diminished and become more diffuse (arrows).

and lack of symptoms of the volunteers. Many changes were subtle and did not occur in all volunteers. Part of this subtlety and lack of universality may be related to suboptimal compliance of the volunteers owing to the discomfort caused by the pads. In addition, the 2-week period selected was relatively arbitrary. Bone remodeling is relatively slow up to 2 weeks and peaks at 6–7 weeks (22,23). Perhaps some changes may have been more obvious after longer periods of altered weight bearing. However, because the pads were uncomfortable, the maximal time for even reasonable compliance was 2 weeks. Another limitation was that no specific relaxation time was obtained, which hindered quantitative analysis. In addition, STIR sequences are somewhat variable in signal intensity characteristics, potentially limiting reproducibility. Magnetic field inhomogeneity also may lead to poor fat suppression with STIR sequences. All these technical difficulties may have added to the subtlety of the changes seen. An additional limitation was that the reviewers were aware of which images were baseline, raising the theoretic possibility of bias in image interpretation. A final limitation was the lack of pathologic proof of our findings. Although we found "marrow edema" to be associated with altered biomechanics, we have no proof of what this MR imaging appearance would represent at histologic examination.

The concept that stress can cause MR imaging alterations, although anecdotally described (16), has not been previously evaluated scientifically, to our knowledge. Increased intramedullary signal intensity is a nonspecific finding. Although increased signal intensity is present in stress fractures and "bone bruises," our results indicate that increased signal intensity also may be the result of bone response to stress without fracture and even without pain. On the basis of this study, we believe that altered biomechanics should be added to the list of causes of increased intramedullary signal intensity on T2-weighted images. ■

References

1. Vande Berg BE, Malghem JJ, Labaisse MA, et al. MR imaging of avascular necrosis and transient marrow edema of the femoral head. RadioGraphics 1993; 13:501–520.
2. Kroon HM, Bloem JL, Holscher HC, et al. MR imaging of edema accompanying benign and malignant bone tumors. Skeletal Radiol 1994; 23:261–269.
3. Morrison WB, Schweitzer ME, Bock GW, Mitchell DG, Hume EL. Diagnosis of osteomyelitis: utility of fat-suppressed contrast-enhanced MR imaging. Radiology 1993; 189:251–257.
4. Robertson PL, Schweitzer ME, Bartolozzi AR, Ugoni A. Anterior cruciate ligament tears: evaluation of multiple signs with MR imaging. Radiology 1994; 193:829–834.
5. Hayes CW, Conway WF, Daniel WW. MR imaging of bone marrow edema pattern: transient osteoporosis, transient bone marrow edema syndrome, or osteonecrosis. RadioGraphics 1993; 13:1001–1011.
6. Hilfiker P, Zanetti M, Debatin JF, McKinnon G, Hodler J. Fast spin-echo inversion-recovery imaging versus fast T2-weighted spin-echo imaging in bone marrow abnormalities. Invest Radiol 1995; 30:110–114.
7. Lanyon LE. Functional strain as a determinant for bone remodeling. Calcif Tissue Int 1984; 36:56–61.
8. Maffulli N, King JB. Effects of physical activity on some components of the skeletal system. Sports Med 1992; 13:393–407.
9. Whedon GD. Disuse osteoporosis: physiological aspects. Calcif Tissue Int 1984; 36:146–150.
10. Minaire P, Edouard C, Arlot M, Meunier PJ. Marrow changes in paraplegic patients. Calcif Tissue Int 1984; 36:338–340.
11. Whedon GD, Lutwak L, Reid J, et al. Mineral and nitrogen metabolic studies on Skylab orbital space flights. Trans Assoc Am Physicians 1974; 87:95–110.
12. Carter DR. Mechanical loading histories and cortical bone remodeling. Calcif Tissue Int 1984; 36:19–24.
13. Roub LW, Gumerman LW, Hanley EN Jr, Clark MW, Goodman M, Herbert DL. Bone stress: a radionuclide imaging perspective. Radiology 1979; 132:431–438.
14. Daffner RH, Pavlov H. Stress fractures: current concepts. AJR 1992; 159:245–252.
15. Johnson MW. Behavior of fluid in stressed bone and cellular stimulation. Calcif Tissue Int 1984; 36:72–76.
16. Mink JH, Deutsch AL. Occult cartilage and bone injuries of the knee: detection, classification, and assessment with MR imaging. Radiology 1989; 170:823–829.
17. Deutsch AL. Traumatic injury of bone and osteonecrosis. In: Deutsch AL, Mink JH, Kern R, eds. MRI of the foot and ankle. New York, NY: Raven, 1992; 75–109.
18. Steinbronn DJ, Bennett GL, Kay DB. The use of magnetic resonance imaging in the diagnosis of stress fractures of the foot and ankle: four case reports. Foot Ankle Int 1994; 15:80–83.
19. Umans H, Pavlov H. Stress fractures of the lower extremities. Semin Roentgenol 1994; 2:176–193.
20. Jaworski ZFG. Coupling of bone formation to bone resorption: a broader view. Calcif Tissue Int 1984; 36:531–535.
21. Alexander RMN. Factors of safety in the structure of mammals. Sci Prog 1981; 67:168–203.
22. Meyer SA, Saltzman CL, Albright JP. Stress fractures of the foot and leg. Clin Sports Med 1993; 12:395–413.
23. Yeh CK, Rodan GA. Tensile forces enhance prostaglandin E synthesis in osteoblastic cells grown on collagen ribbons. Calcif Tissue Int 1984; 36:67–71.