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TEST YOURSELF: ANSWER

Chronic hip pain

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Diagnosis

Gelatinous marrow transformation (bone marrow starvation syndrome)

Discussion

The MRI images demonstrate diffusely abnormal bone marrow signal in the pelvis and proximal femora. On coronal T1-weighted imaging (Fig. 1), the bone marrow is diffusely hypointense. In the lumbar spine, more profound decreased T1 signal results in disc signal being brighter than bone marrow signal (Figs. 1 and 2). On coronal STIR imaging (Fig. 3), the bone marrow in the pelvis is diffusely hyperintense. The hyperintense signal on STIR is not as pronounced in the spine compared to the pelvis (Fig. 4). There is a paucity of subcutaneous fat. The fat also demonstrates abnormal diffuse T1 hypointense and STIR hyperintense signal. A focal subcutaneous fluid and gas collection is seen along the lateral aspect of the left hip. There is no hip joint effusion to suggest septic arthritis. No pathologic fracture is identified.

The patient's medical history was significant for poorly controlled type 1 diabetes and chronic diarrhea relating to partial colectomy as an infant for necrotizing enterocolitis. He was emaciated and suffering from pressure sores attributed to chronic toilet sitting. Given the clinical history, the abnormal bone marrow signal in the pelvis and proximal femora is compatible with gelatinous transformation of the bone marrow secondary to mobilization of marrow fat stores as a result of chronic malabsorption/malnutrition. More profound decreased T1 signal seen in the lumbar spine is compatible with red marrow reconversion in the setting of chronic illness.

Gelatinous marrow transformation (GMT), also known as bone marrow starvation syndrome, is a rare complication associated with severe under-nutrition. It occurs in a wide range of underlying conditions including anorexia nervosa, malabsorption (celiac disease or inflammatory bowel disease), massive weight loss after bariatric surgery, alcoholism, advanced cancers, and chronic infections such as acquired immunodeficiency syndrome (AIDS) [1–3]. GMT was first identified in autopsy specimens of patients suffering from starvation which led to the alternative term "bone marrow starvation" [2, 4–6]. Histologically, GMT is characterized by hypoplasia of hematopoietic cells, fatty atrophy, and deposition of a gelatin-like substance rich in hyaluronic acid [1, 2, 5]. The pathophysiology of GMT is not well understood. In the early stages of starvation, bone marrow fat paradoxically increases even as other fat stores are being mobilized [7]. Lipolysis of bone marrow adipose tissue does not occur until end-stage starvation, when the body begins breaking down muscle tissue. This is the final stage in which protein wasting can result in death [7]; therefore, when GMT is present, it is indicative of advanced underlying disease.

Patient history and physical examination can help contribute to the diagnosis. Patients are typically cachectic secondary to severe malnutrition. Laboratory studies often show pancytopenia or isolated cytopenias such as anemia or leukopenia [8, 9]. Patients are at increased risk for infection [1]. GMT is also associated with bone fragility and increased risk of stress fractures [1].

The case presentation can be found at doi: 10.1007/s00256-016-2396-5

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MRI commonly leads to diagnosis of GMT before it is considered clinically [1]. The predominate MRI findings are hypointense bone marrow signal on T1-weighted imaging and hyperintense bone marrow signal on fat-suppressed fluid-sensitive imaging, or essentially reversal of normal T1 and T2 bone signal intensity [1]. Fractures of the hips and lower extremities are common [1]. Surrounding subcutaneous tissue may show hypointense T1 signal and hyperintense T2 fat-suppressed signal, possibly due to replacement of adipose tissue by loose vascular tissue [1, 4]. With the administration of intravenous contrast, there is no abnormal enhancement of the bone marrow, helping to distinguish it from neoplastic or infectious causes [1]. It is thought that GMT may be under-diagnosed and under-reported on MRI examinations. A 2015 study of 30 patients with GMT found that 23 % of patients received unnecessary repeat imaging due to misinterpretation of the initial MRI findings as technical error such as failed fat suppression [1].

There is no specific treatment for GMT apart from treatment of the underlying condition and correction of malnutrition. Normalization of nutritional status such as in patients with anorexia nervosa, has been shown to result in reversal of bone signal abnormalities [6].

References

1. Boutin R, White L, Laor T, et al. MRI findings of serous atrophy of bone marrow and associated complications. Eur Radiol. 2015;9: 2771–8. doi:10.1007/s00330-015-3692-5.
2. Osgood E, Muddassir S, Jaju M. Starvation marrow: gelatinous transformation of bone marrow. J Community Hosp Intern Med Perspect. 2014; 4(4). doi:10.3402/jchimp.v4.24811
3. Villate A, Iquel S, Legac E. Gelatinous transformation of the bone marrow: a retrospective monocentric case series of 12 patients. Rev Med Interne. 2015. doi:10.1016/j.revmed.2015.10.349.French.
4. Seaman JP, Kjeldsberg CR, Linker A. Gelatinous transformation of the bone marrow. Hum Pathol. 1978;9(6):685–9.
5. Bohm J. Gelatinous transformation of the bone marrow: the spectrum of underlying diseases. Am J Surg Pathol. 2000;24:56–65.
6. Tavassoli M, Eastlund DT, Yam LT, et al. Gelatinous transformation of bone marrow in prolonged self-induced starvation. Scand J Haematol. 1976;16:311–9.
7. Devlin MJ. Why does starvation make bones fat? Am J Hum Biol. 2011;23:577–85.
8. Lambert M, Hubert C, Depresseux G, et al. Hematological changes in anorexia nervosa are correlated with total body fat mass depletion. Int J Eat Disord. 1997;21:329–34.
9. Sabel AL, Gaudiani JL, Statland B, Mehler PS. Hematological abnormalities in severe anorexia nervosa. Ann Hematol. 2013;92:605–13.