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Case Report

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MR Imaging Findings in Serous Atrophy of the Bone Marrow: A Case Study

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Abstract

Serous atrophy of the bone marrow, also referred to as gelatinous transformation, is an important diagnostic consideration on MR imaging demonstrating hyperintense signal within the bone marrow on fluid sensitive sequences in conjunction with signal abnormality within the overlying superficial fascia and subcutaneous fat. This diagnosis is associated with a broad range of underlying conditions, such as malnutrition and chronic illnesses including prolonged infection, malignancy, congestive heart failure, and chronic kidney disease. The clinical presentation typically includes weight loss, cytopenia, and occasionally insufficiency fractures secondary to osteoporosis. The diagnosis of serous atrophy of the bone marrow is rarely made in clinical practice, and diagnosis can even be delayed due to initial misinterpretation of the characteristic signal abnormalities as technical error, potentially resulting in unnecessary repeat imaging examinations. Thus, evaluation of clinical history combined with recognition of distinct MRI findings can aid in efficient diagnosis and can avoid unnecessary repeat imaging.

Keywords

Serous atrophy, MR imaging

Introduction

Serous atrophy of the bone marrow (SABM), also referred to as gelatinous transformation of the bone marrow, is a rare disorder characterized by adipose cell atrophy, loss of hematopoietic cells, and extracellular deposition of gelatinous substances in the bone marrow stroma [1]. Though the exact pathogenesis of SABM is poorly understood, the constellation of MR imaging findings is associated with a broad range of conditions including cachexia, malnutrition, and chronic illnesses such as end stage renal disease (ESRD) and congestive heart failure (CHF) [2-6]. SABM may be underdiagnosed and underreported on MRI exams, possibly due to lack of diagnostic suspicion prior to the MRI and possibly due to absence of universal formal diagnostic criteria. Our report of a case of SABM in the left lower extremity reviews the pertinent MR imaging findings to suggest the diagnosis. These imaging findings may be confused with technical scanning errors, and therefore, familiarity with characteristic MR imaging findings of SABM is critical for radiologists for suggesting this diagnosis in patients with a high index of suspicion.

Case Report

A 64-year-old male presented to the hospital for a hypoglycemic episode with a blood glucose of 43 mg/dL. Upon further questioning, the patient reported left lower extremity (LLE) pain for approximately 2 weeks in duration as well as poor appetite over the past few months. The patient's medical history was significant for CHF, ESRD, and type 1 diabetes mellitus. The patient's weight was 155 pounds, decreased from 191 pounds at a hospital admission two months prior. Physical examination revealed tenderness to palpation over the left lateral and posterior thigh. Initial laboratory analysis included complete blood count (CBC), albumin, and point-of-care glucose. CBC was remarkable for WBC of 8.7/L (3.2-9.8 x 19^9/L), hemoglobin of 7.7 g/dL (13.7-17.3 g/dL) and mean corpuscular volume of 73 fL (80-98 fL). The albumin was 1.4 g/dL (3.5-4.8 g/dL). Point-of-care glucose was 46 mg/dL (70-140 mg/dL). Chest x-ray and CT abdomen and pelvis were also ordered and showed large volume ascites and pleural effusions. The patient did not have post-contrast imaging performed due to a depressed GFR of 12 mL/min/1.73m^2 which is below the institution policy for contrast administration.

Additional imaging was ordered to characterize the patient's LLE pain. Plain films of the left lower femur were remarkable for mild swelling (Figure 1). On axial non-contrast CT, the mid- distal femur demonstrated a thin layer of subcutaneous fat (Figure 2). Notably, the muscles appear hypodense, and the bone marrow signal is nearly equal to that of the muscles. In fluid sensitive axial STIR weighted sequences, the marrow signal is bright due to gelatinous transformation (Figure 3A). On axial non-contrast T1-weighted, the marrow loses its normal fat signal, with the marrow being isointense to the adjacent muscle (Figure 3B). The distribution of the abnormal signal is similar in both the T1 and STIR images.

The differential diagnosis for these imaging findings includes malignancy, infection, myeloproliferative disorders, and SABM. No evidence of malignancy or myeloproliferative disorders was found on initial imaging examinations. The patient had a normal WBC count and no clinical signs of infection. The patient's initial cytopenia improved over the course of hospital admission with total parenteral nutrition (TPN) support. The patient's significant recent weight loss, microcytic anemia, low albumin, and clinical history of poor feeding are most

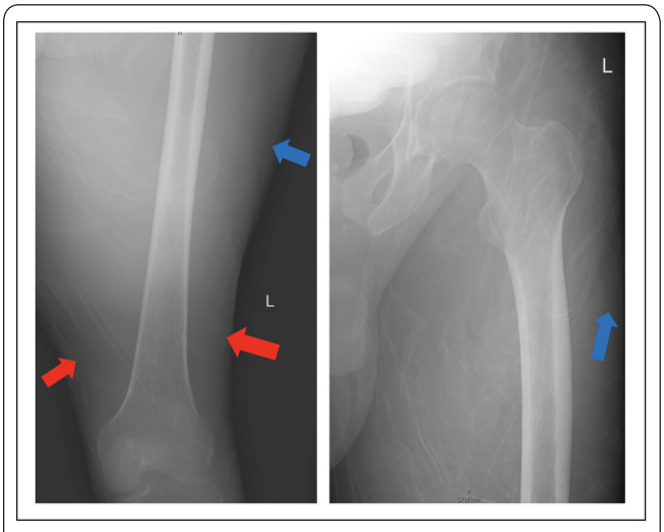

Figure 1: AP radiographs of the left lower extremity show a thin layer of fat (blue arrows) superficial to the muscle tissue. There is some edema interposed within the subcutaneous fat evident by some strand-like hyperdensity in the fat (red arrows). Normal areas of fat have a hypodense, dark appearance. The femur maintains a normal structure and morphology. There is a moderate amount of muscular tissue. Some loose cloth material overlies the mid and proximal femur.

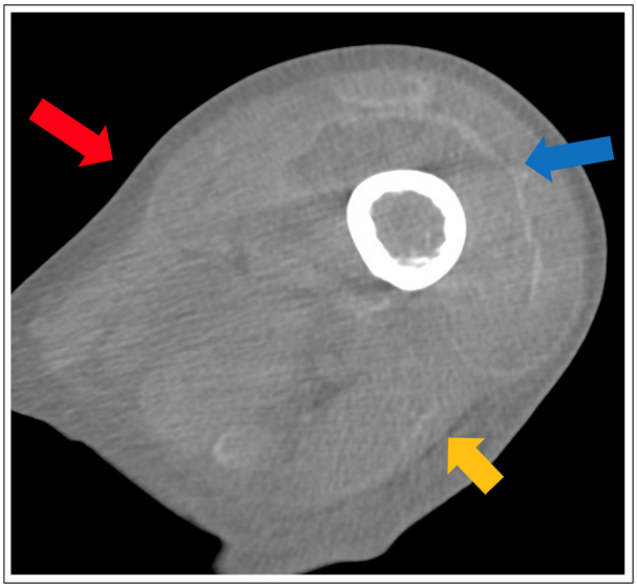

Figure 2: Axial non-contrast CT images of the mid-distal femur demonstrate a thin layer of subcutaneous fat (red arrow). There is some edema within the subcutaneous fat which has a more hyperdense and strand-like appearance (orange arrow). The muscles are somewhat hypodense as evident by the prominent appearance of the myotendinous junction (blue arrow). The hypodense appearance of the muscles is due to the gelatinous deposition within the muscles. Gelatinous material has a higher water content which gives the muscles a hypodense appearance relative to the tendon. The bone marrow signal is nearly equal to that of the muscles suggesting further gelatinous deposition in the bone marrow. The bone cortex maintains its normal density.

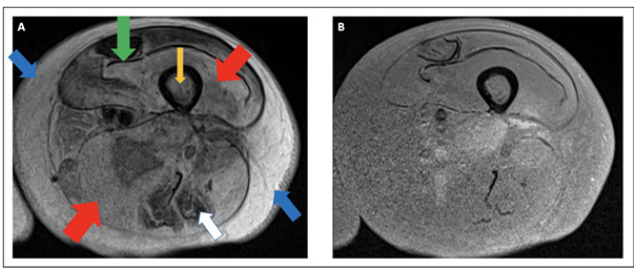

Figure 3: Axial STIR (A) and T1 (B) non-fat-saturated images through the distal thigh. The STIR weighted image demonstrates hyperintense signal throughout the subcutaneous fat (blue arrow), musculature (red arrow), and bone marrow (orange arrow). The tendons (green arrow) maintain their normal STIR and T1 hypointense signal consistent with a lack of gelatinous material deposition in the tendons. There are a few small areas within the distal femur musculature (white arrow) which have a more normal STIR isointense signal consistent with less severe involvement of some of the musculature. The T1 non-fat-saturated image shows nearly diffuse hypointense signal in the subcutaneous fat, musculature, and bone marrow. This is due to the higher water content in the gelatinous deposition within these structures. The distribution of the T1 non-fat-saturated signal abnormality mirrors that of the signal abnormality on the STIR weighted image.

consistent with a diagnosis of malnutrition with subsequent cachexia, and the imaging findings support the diagnosis of SABM. The patient improved with TPN and revised dietary recommendations and was discharged to a skilled nursing facility for further nutritional support and care for their other illnesses.

Discussion

In the setting of other myeloproliferative disorders, malignancy, and infection, a bone marrow aspiration and biopsy has high diagnostic accuracy [7, 8]. Given the patient's malnutrition and clinical improvement with TPN and nutritional support, a bone marrow biopsy and aspiration was not performed. Gelatinous transformation of the bone marrow is seen with etiologies such as malnutrition and cachexia and can be definitively diagnosed by bone marrow aspiration and biopsy. SABM is characterized by bone marrow changes such as adipose cell atrophy and loss of hematopoietic cells with deposition of gelatinous substances in the marrow [1]. Although not fully understood, the pathophysiology of SABM is thought to stem from the effects of malnutrition, cachexia, and hypercatabolic states on the bone marrow and surrounding soft tissue. Prolonged starvation causes mobilization of subcutaneous and visceral fat but with a paradoxical increase in bone marrow fat [9]. This is thought to cause mesenchymal stem cells in the bone marrow to differentiate into adipocytes instead of osteoblasts, leading to bone fragility. In later starvation stages, the fat stores in the bone marrow are finally mobilized, and the extracellular space of the bone fills with gelatinous material made of hyaluronic acid-containing mucopolysaccharide, resulting in the characteristic morphological changes [10].

The conditions associated with SABM are broad and typically include nutritional or chronic illness-related disorders. Nutritional associations include malnutrition (e.g. anorexia nervosa), malabsorption, and alcoholism. Chronic illnesses associated with SABM include CHF, ESRD, malignant tumors, and chronic infection [10].

The clinical manifestations of SABM are nonspecific, making it difficult to establish a diagnosis on initial presentation. Most commonly, clinical findings include weight loss and anemia. Complications result from decreased function of normal bone marrow. For instance, loss of hematopoietic cells can cause leukopenia, which leads to increased susceptibility to infection. Decreased stem cell differentiation into osteoblasts can increase bone fragility leading to greater fracture risk [11]. SABM is not a common diagnosis, but despite this, the condition may be underdiagnosed and under-reported on MRI exams due to the absence of universal diagnostic criteria. However, there are characteristic imaging findings that can point to the diagnosis. For instance, hypointense marrow on T1-weighted sequences, hyperintense marrow on fluid-sensitive sequences, and abnormal signal intensity and thickness of adjacent subcutaneous tissues suggest a diagnosis of SABM.14 The MR imaging findings in SABM can easily be mistaken for technical malfunction given the abnormal signal intensities in the bone marrow and surrounding subcutaneous tissue. Suggestion of SABM can be supported by clinical history of cachexia or malnutrition. Additionally, it can be useful to perform fat-suppressed T2 and STIR sequences to avoid misinterpreting SABM signal abnormality as failed fat suppression [10].

The differential for the imaging findings in this case include malignancy, infection, myeloproliferative disorders (e.g. myelodysplastic syndrome, acute myeloid leukemia, myelofibrosis), and SABM. There was no evidence of primary tumors on imaging workup. The infiltration of the bone marrow by infection, malignancy, or myeloproliferative disorders has a similar appearance with hypointense T1 signal and hyperintense STIR signal. Infection can show a geographic area of low signal intensity on T1-weighted images often with concomitant cortical destruction, which can help differentiate it from other lesions. Malignancy is characterized by patchy areas of hypointense T1 signal, well defined area of bone marrow abnormality and possibly extension into the cortex and periosteal soft tissues. In neoplastic processes such as metastasis, other bones and organ systems may be involved, indicated by parenchymal involvement and enlarged lymph nodes. Marrow infiltration can show multiple punctate lesions and sclerosis in some cases [12-15].

Given the overlap in the imaging findings of multiple diseases of the bone marrow, clinical evaluation, laboratory assessment, and tissue analysis are helpful for further differentiation of underlying disease. Contrast enhancement is less useful in making this differentiation as both red marrow and underlying disease such as infection, inflammation and tumors can enhance.7 In this case, further imaging delineation with contrast-enhanced imaging was deferred in accordance with hospital policy due to the patient's GFR of 12 mL/min/1.73m^2.

Treatment of SABM is aimed at addressing the underlying cause. Previous studies have shown that treatment of the underlying cause can help replete hematopoietic abnormalities [16]. In the setting of further complications, treatments such as packed red blood cells and hematopoietic growth factors have been shown to help patients achieve sustained hematopoietic recovery [17].

In this case, the patient presented with a 2-week history of leg pain. The gelatinous transformation of the bone marrow, muscle and subcutaneous fat causes a higher water content in these tissues. This is evident on CT imaging by the hypodense appearance of the musculature. The T1 non-fat-saturated MR images demonstrate hypointense signal in the bone marrow, subcutaneous fat and musculature. Similarly, the STIR MR images demonstrate hyperintense signal in the bone marrow, musculature, and subcutaneous fat, consistent with increased water content. Given these imaging results along with the clinical context of numerous chronic illnesses, including CHF and ESRD, as well as more recent history of cachexia and laboratory findings significant for anemia, a diagnosis of SABM was made with a high index of suspicion. The patient subsequently improved with TPN and dietary recommendations to address the underlying cause of

malnutrition and was discharged to a skilled nursing facility for further nutritional support and care of their other illnesses

Conclusion

SABM is an important consideration on MR imaging when mildly hypointense marrow on T1- weighted and hyperintense marrow on fluid-sensitive sequences are present with abnormal signal within the overlying soft tissues. Imaging can support this diagnosis in the setting of a clinical history of cachexia, malnutrition, chronic illness, or cytopenias. Early recognition of SABM as a diagnosis is necessary to avoid repeat imaging examinations due to suspected technical error.

Conflict of Interest

The authors declare no conflict of interest.

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