Serous Atrophy: A Rare and Potentially Baffling Imaging Diagnosis

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ABSTRACT

Serous atrophy of the bone marrow (SABM) is a hematologic disorder characterized by progressive reduction in both fat cells and hematopoietic cells followed by deposition of gelatinous material. These findings are most commonly associated with cachectic disease states secondary to anorexia nervosa, chronic infection, or malignancy. Once manifested, these compositional changes of the marrow are detectable through MR imaging as diffuse signal abnormalities with an alteration of T1 and STIR signal characteristics. Here, we present a 37-year old male with leukopenia and generalized musculoskeletal pain with imaging features consistent with SABM and review the literature on this uncommon condition.

CASE REPORT

INTRODUCTION

Serous atrophy of the bone marrow (SABM), commonly referred to as gelatinous transformation of bone marrow (GTBM), is a rare hematologic disorder that is characterized by progressive reduction of both fat cells and hematopoietic cells of the bone marrow and with subsequent deposition of a gelatinous matrix composed of hyaluronic acid mucopolysaccharides in the subcutaneous tissues [1, 2]. Though the exact mechanism of these pathologic changes has not been elucidated, these pathologic changes most commonly present during states of malnourishment or cachexia, notably with anorexia nervosa, but can also be seen with AIDS, Systemic Lupus Erythematosus (SLE) and malignancy. In rare cases endocrine disorders, myeloproliferative disease, and bariatric surgery have also been reported in association with these findings [2-6].

Serous atrophy is an uncommon but potentially reversible disease entity most commonly seen in anorexia nervosa prompting an early diagnosis and treatment. Diagnostic imaging features of serous atrophy may be misleading with interpretation as a technical scan error which is a relatively common pitfall. This is due to the "flip-flop" nature of T1-weighted and STIR imaging sequences where bone marrow is inversely hypointense on T1 and bright on STIR [3]. Here, we present a case of serous atrophy of the bone marrow in a patient with leukopenia and recent weight loss.

CASE REPORT

A 37-year-old male presented to our medical center with generalized fatigue and chronic knee, hip and lumbar spine pain. He has a past medical history of IBS-C on Amitiza, hypogonadism, and previous history of leukopenia being followed by the Hematology/Oncology service. He denied "B" symptoms. Of note, at time of presentation, the patient's BMI was 19.2, with a height of 5 feet 10 inches and weight of 134 lbs. Upon further chart review, he had previously weighed over 200 lbs. He endorsed feelings of body consciousness but denied abnormal dietary habits. He otherwise had no remarkable findings on physical exam.

Screening for Hepatitis B, Hepatitis C, HIV, and autoimmune diseases was performed and results for all tests were negative. A panel for leukemia/lymphoma was performed and was also negative.

Initial MR imaging of his knees was performed for workup of generalized joint pain (See Figure 1A & B). It was noted on interpretation that the imaging sequences for T1 and STIR appeared inverted with diffusely low signal on T1 and high signal on STIR images particularly of the bone marrow. The knee exam was repeated with the assumption of a technical error during the prior imaging acquisition and yielded a similar result (data not shown). Subsequently, the patient underwent MR imaging of the lumbar spine and the pelvis for generalized

joint pain with similar inversion of the T1 and STIR sequences (Figure 1C-F). It was noted that the subcutaneous fat was nearly non-existent and a diagnosis of Serous Atrophy was then made. As of this writing, the patient was being treated for nutritional imbalance.

DISCUSSION

SABM can be attributed to a heterogeneous group of pathologies, though it has been most commonly described as a sequela to cachectic disease states, chronic infection, endocrine-driven catabolism, and radical malnutrition [2, 7]. The diversity of associated disease suggests that the biomechanical processes of SABM are related to the extreme exacerbation of a baseline regulatory function [2]. Indeed, in the setting of starvation there is initially fatty deposition within the marrow. Lipolysis of these reservoirs only occurs with exhaustion of other sources as the malnutritional state progresses [8].

In the presented case, a definite causative pathologic process has yet to be determined. As of initial presentation, the patient did not meet diagnostic criteria for anorexia nervosa, nor did he endorse eating behaviors consistent with this disease. Previous documentation, however, did report 64 lbs. of weight loss along with a BMI near threshold for diagnosis at 19.3. In the context of this patient's self-reported history, non-pathologic weight loss was to be considered only after exclusion of more severe underlying disease. HIV, Hep B, and Hep C were ruled out. Though uncommon, myeloproliferative disorders have also been associated with SABM [4], and must be ruled out as was the case with our patient.

MR Imaging of serous atrophy is easily misinterpreted as technical error with "flip-flop" signal changes in T1 and STIR weighted imaging. However, marrow infiltrative processes must be ruled out before making a diagnosis of serous atrophy. Initial findings of signal abnormalities for SABM notably first present in the yellow marrow of the peripheral skeleton, as opposed to the majority of dysfunctional marrow disorders which predominate in the axial skeleton [1, 7]. This pattern of progression can be helpful in further differentiating SABM from infiltrative marrow disease. Additionally, bone marrow in SABM is not expected to enhance, while infiltrative malignancies or infectious processes may show post-contrast enhancement [9]. Additionally, post-radiation treatment change may show diffusely low marrow signal on T1-weighted imaging, while STIR imaging may be hyperintense [10]. A review of the patient's history can rule out this as a possible etiology with similar imaging findings. In at least 1 case report however, it was shown that imaging features of post-radiotherapy and SABM may overlap further confounding the diagnosis [11].

A histopathologic diagnosis of SABM can be confirmed via bone marrow biopsy and would be expected to show increased amounts of gelatinous tissue and corresponding decrease in hematopoietic and fat cells [7]. Some institutions have made use of T2-weighted Turbo Spin Echo (TSE) Dixon sequences to confirm as both the in-phase and opposed-phase images are nearly identical in SABM [11]. This technique may be a useful adjunct when questions of technical error versus SABM arise. Finally, MR spectroscopy may show a small lipid peak and large water peak in SABM and may be useful in difficult cases [3].

Serous atrophy remains an uncommon diagnosis with many different causes however, it is most commonly associated with cachexia and severe weight loss. The "flip-flop" nature of T1 and STIR sequences on MRI may present a diagnostic conundrum for the interpreting radiologist who may be unaware of this disease entity. Awareness of SABM, a good clinical history and the use of adjunct techniques will prove useful in the correct and timely diagnosis of SABM.

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FIGURES

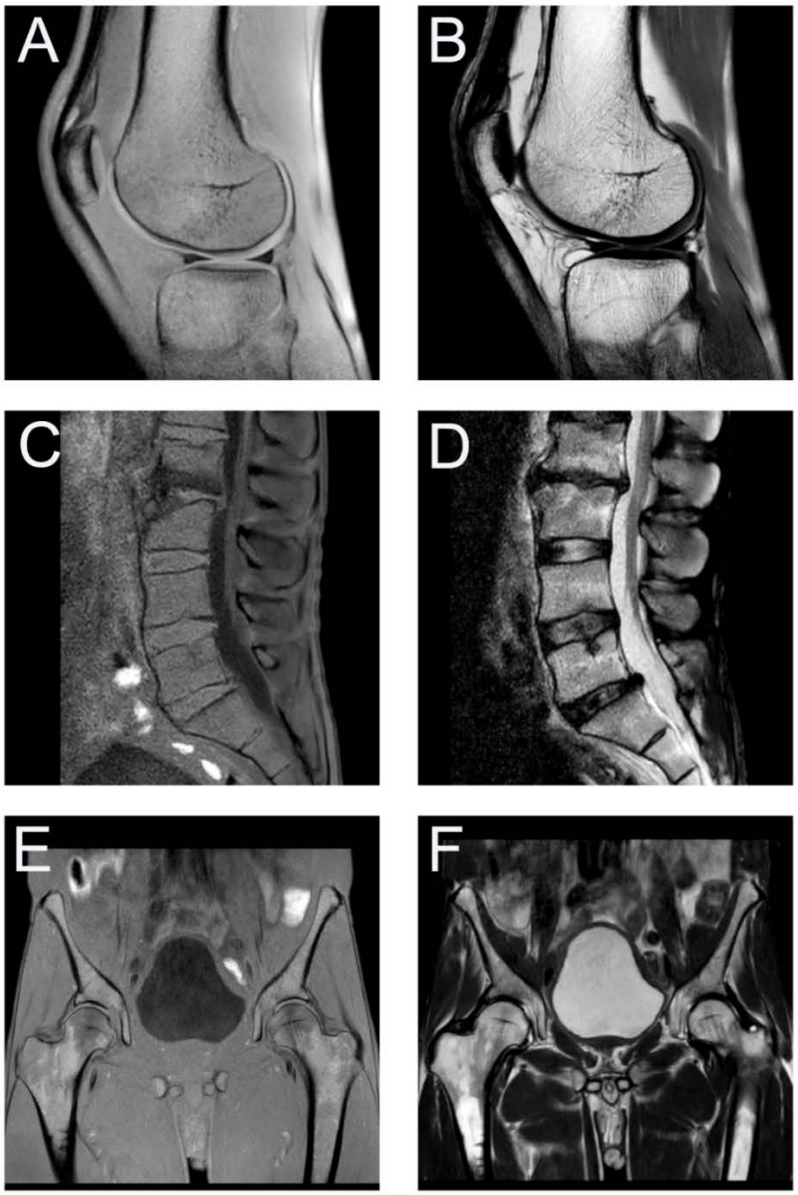

Figure 1: A) Sagittal T1 and B) Sagittal STIR images of the right knee was performed showing inversion of signal characteristics (the subcutaneous fat and bone marrow on the T1-weighted image are diffusely low signal and STIR images are diffusely high signal). This was repeated due to presumed technical error with same result. C) Sagittal T1-weighted and STIR image D) show similar result in the lumbar spine and showing minimal subcutaneous fat. E) Coronal T1-weighted and STIR image F) showing inversion of signal and very little subcutaneous fat.

KEYWORDS

Serous Atrophy, Gelatinous Transformation of the Bone Marrow, Musculoskeletal, T1 and STIR MRI, Cachexia