Rheumatology : Current Research

Ricardo Ochoa et al., Rheumatology 2013, 4:1
DOI: 10.4172/2161-1149.1000127

Case Report Open Access

Avascular Necrosis of the Spine in a Patient with Systemic Lupus Erythematosus and Antiphospholipid Syndrome

Ricardo Ochoa E[1], Caner Sakin[2], Hareth Madhoun[3], Alan D Rogers[1] and Wael N Jarjour[3]*

[1]*The Ohio State University Medical Center, Department of Radiology, Columbus, Ohio, USA*
[2]*UW/Valley Medical Center, Renton, Washington, USA*
[3]*The Ohio State University Medical Center, Division of Immunology, Columbus, Ohio, USA*

Abstract

Avascular Necrosis (AVN) lesions affecting the long bones, especially the femoral and humeral heads, are observed with increased frequency in patients with lupus and antiphospholipid syndrome. However, AVN involving the spine is exceptionally unusual. We report a case that illustrates the importance of considering the diagnosis of AVN when evaluating lesions involving the spine in those patients and the characteristic findings.

Introduction

Systemic Lupus Erythematosus (SLE) and Antiphospholipid Syndrome (APS) are known risk factors for Avascular necrosis (AVN). AVN commonly occurs in femoral and humeral heads. The imaging findings can mimic what may be in other processes such as infections, malignancy or other marrow replacing processes and may lead to extensive work up. Although rare, AVN can occur in the spine and needs to be considered in the differential diagnosis.3.

Case Report

3 year old caucasian female with an established history of Systemic Lupus Erythematosus (SLE), Antiphospholipid antibody syndrome (APS), deep vein thrombosis on chronic anticoagulation was evaluated for pleuritic type chest pain. A CT pulmonary angiogram was performed (Figure 1). The exam revealed no pulmonary embolus, but did describe two lytic lesions at T9 and T10 which were new since the comparison study from nearly 4 years earlier. Both had an irregular shape with sclerotic margins. The radiologist differential diagnosis included infection and metastatic disease, with the latter considered less likely given the patient's young age.

MRI was later obtained which demonstrated heterogeneous low T1 and high T2 signal within T9, while T10 showed predominantly high T1 signal with T2 signal being high peripherally and low centrally (Figures 2a,2b and 3). Similar findings were also seen in the lumbar spine, particularly at L5 which had appeared abnormal on a lumbar MRI from 4 years prior. Differential diagnoses included a marrow infiltrative process, myelofibrosis, or malignant infiltration, and bone scan was recommended.

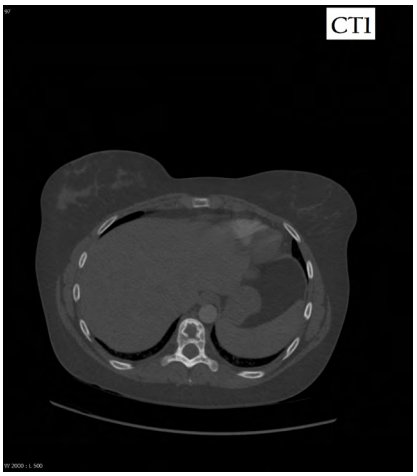

Figure 1: CT demonstrating a T9 lucent lesion with a sclerotic margin.

On Tc 99m MDP bone scan, photopenia was seen in the T9 and T10 vertebral bodies corresponding with the lesions (Figure 4). Heterogeneous uptake in the left proximal femur was suggested to possibly represent an early lytic lesion, and radiographic correlation was recommended.

The patient's pleuritic chest pain resolved and she was asymptomatic in terms of these lesions. She did not report any history of trauma to the spine. She was not on prednisone for at least 6 months prior to her presentation. Based on the imaging studies and patient's clinical history, AVN of the spine secondary to APS / SLE is considered the likely diagnosis. She is being monitored clinically and radiologically, she has not required any escalation of her therapy and is stable at 2 years with minimal progression of her lesions.

Discussion

Avascular necrosis is reported in association with many entities including excessive alcohol use, glucocorticoid use, SLE and APS [1-6]. AVN has been reported in 3 to 30 percent of patients with SLE [4,5]. 20 percent of patients were found to have asymptomatic AVN in patients with primary APS based on a study by Tektonidou et al. [3]. AVN of the spine is an uncommon disease in general [7] and in the setting of SLE and APS. Only one case in this setting was reported based on a search of the English literature [8]. The case presented above reflects some of the characteristic radiological findings that are seen with AVN using various imaging modalities and the varied manifestations of AVN based on location and phase. For example, the characteristic sclerotic line surrounding a bone infarct occurs late in the disease course [9,10]. While the appearance is often very characteristic, differential diagnoses

***Corresponding author:** Wael N Jarjour, MD, FACP, Rm S2056 Davis Medical Research Center, 480 Medical Center Dr, Columbus, OH 43210, USA, Tel: 614-571-1242; Fax: 614-293-5361; E-mail: Wael.Jarjour@osumc.edu

Received October 28, 2013; **Accepted** November 28, 2013; **Published** December 04, 2013

Citation: Ricardo Ochoa E, Sakin C, Madhoun H, Rogers AD, Jarjour WN (2013) Avascular Necrosis of the Spine in a Patient with Systemic Lupus Erythematosus and Antiphospholipid Syndrome. Rheumatology 4: 127. doi:10.4172/2161-1149.1000127

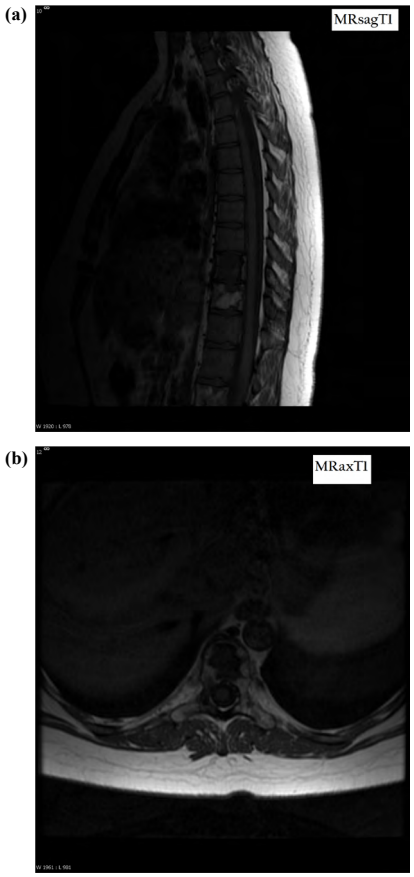

Figure 2: a & b: MRI, T1 sagittal and axial images demonstrating a predominantly low T1 signal lesion within T9, while a T10 lesion was predominantly high signal with a low signal margin. An L1 lesion with similar characteristics to that at T10 is partially visualized.

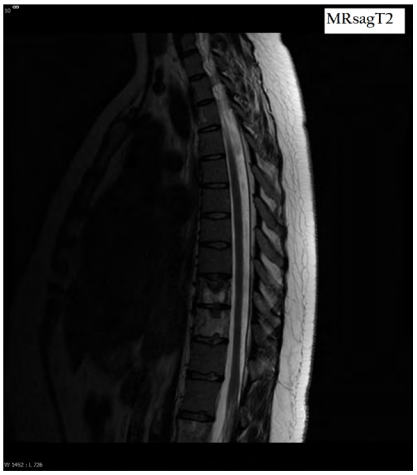

Figure 3: MRI, T2 sagittal and axial images demonstrating high T2 signal lesions at T9, T10, and L1, with low signal margins corresponding to the sclerosis.

may include an involuting fibroxanthoma or enchondroma. When occurring in a subchondral location, subchondral cystic changes of osteoarthritis and osteochondral defects may be considered [11]. Earlier in the evolution, the sclerotic rim is absent with the lesion

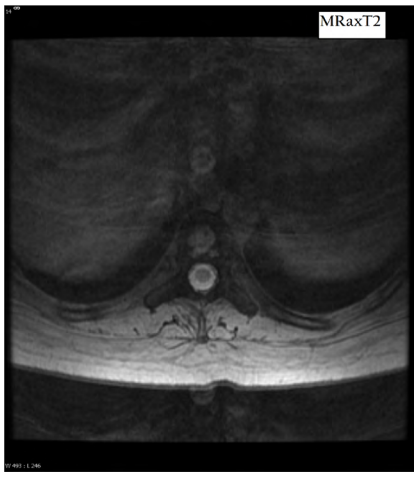

Figure 4: Tc 99m MDP bone scan demonstrating photopenic defects at T9 and T10 corresponding with the lesions.

appearing either normal or as a lucency, typically in the subchondral bone [12]. When the infarct is predominantly an ill-defined lucency, osteomyelitis or neoplastic etiologies such as myeloma and metastasis can also enter the differential along with insufficiency fracture.

The sclerotic line seen radiographically correlates to a low signal line on T1 weighted MRI, which on T2 sequences demonstrates low signal with an adjacent hyperintense inner border known as the double line sign. A pseudomass appearance may result, with preservation of internal marrow fat distinguishing the infarct from tumor [13,14]. During the acute phase of infarction associated edema results in increased T2 signal. Differential diagnoses for the MR appearance include the same entities as radiographically.

On Tc99 MDP bone scan, bone infarcts can appear normal or result in a photopenic defect [15]. This is a nonspecific finding, with differential diagnoses including myeloma, metastatic disease, some sarcomas, postradiation changes, and artifact from overlying metal or orthopedic hardware. In the acute phase, there may be increased tracer uptake.

The imaging findings in AVN can mimic many other etiologies and may lead to extensive work up. Although rare, AVN can occur in the spine and needs to be considered in the differential diagnosis.

Acknowledgement

We are grateful for the expert help of Mikelle Oelker in preparation of this manuscript.

References

1. Vreden SG, Hermus AR, van Liessum PA, Pieters GF, Smals AG, et al. (1991) Aseptic bone necrosis in patients on glucocorticoid replacement therapy. Neth J Med 39: 153-157.
2. Matsuo K, Hirohata T, Sugioka Y, Ikeda M, Fukuda A (1988) Influence of alcohol intake, cigarette smoking, and occupational status on idiopathic osteonecrosis of the femoral head. Clin Orthop Relat Res : 115-123.
3. Tektonidou MG, Malagari K, Vlachoyiannopoulos PG, Kelekis DA, Moutsopoulos HM (2003) Asymptomatic Avascular Necrosis in Patients with Primary Antiphospholipid Syndrome in the Absence of Corticosteroid Use: A Prospective Study by Magnetic Resonance Imaging. Arthritis Rheum 48: 732-736.
4. Abeles M, Urman JD, Rothfield NF (1978) Aseptic necrosis of bone in systemic lupus erythematosus. Relationship to corticosteroid therapy. Arch Intern Med 138: 750-754.

5. Dimant J, Ginzler EM, Diamond HS, Schlesinger M, Marino CT, et al. (1978) Computer analysis of factors influencing the appearance of aseptic necrosis in patients with SLE. J Rheumatol 5: 136-141.

6. Sayarlioglu M, Yuzbasioglu N, Inanc M, Kamali S, Cefle A, et al. (2012) Risk factors for avascular bone necrosis in patients with systemic lupus erythematosus. Rheumatol Int 32: 177-182.

7. Yu CW, Hsu CY, Shih TT, Chen BB, Fu CJ (2007) Vertebral osteonecrosis: MR imaging findings and related changes on adjacent levels. AJNR Am J Neuroradiol 28: 42-47.

8. Mok MY, Isenberg DA (2000) Avascular necrosis of a single vertebral body, an atypical site of disease in a secondary APLS. Ann Rheum Dis 59: 494-495.

9. Pope TL (2008) Imaging of the Musculoskeletal System, Volume 1. Saunders/ Elsevier, Philadelphia.

10. Andrews CL (2000) From the RSNA Refresher Courses. Radiological Society of North America. Evaluation of the marrow space in the adult hip. Radiographics 20 Spec No: S27-42.

11. Norman A, Steiner GC (1983) Radiographic and morphological features of cyst formation in idiopathic bone infarction. Radiology 146: 335-338.

12. Munk PL, Helms CA, Holt RG (1989) Immature bone infarcts: findings on plain radiographs and MR scans. AJR Am J Roentgenol 152: 547-549.

13. Zurlo JV (1999) The double-line sign. Radiology 212: 541-542.

14. Mitchell DG, Rao VM, Dalinka MK, Spritzer CE, Alavi A, et al. (1987) Femoral head avascular necrosis: correlation of MR imaging, radiographic staging, radionuclide imaging, and clinical findings. Radiology 162: 709-715.

15. Stoller DW, Tirman PF, Bredella MA (2004) Diagnostic Imaging: Orthopaedics. Amirsys, Salt Lake City.