

Multiple Myeloma Presenting with Clinical and Serological Features of Systemic Lupus Erythematosus: A Case Report

Arthur E. Brawer, M.D.¹ and Sindhuja Korem, M.D.^{2*}

¹Department of Medicine, Division of Rheumatology, Monmouth Medical Center, Long Branch, New Jersey USA.

²Department of Medicine, Monmouth Medical Center, Long Branch, New Jersey USA.

*Correspondence:

Sindhuja Korem, M.D., Department of Medicine, Monmouth Medical Center, 300 Second Avenue, Long Branch, New Jersey 07740, USA.

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ABSTRACT

Features of multiple myeloma may mimic autoimmune conditions, resulting in erroneous diagnoses and incorrect treatment. We present an unusual case of a 62-year-old male who presented with clinical and laboratory phenomena highly suggestive of systemic lupus erythematosus, but who was correctly identified as having multiple myeloma.

Keywords

Multiple myeloma, Systemic lupus erythematosus, Antinuclear antibodies.

Background

Multiple myeloma (MM) is a hematologic malignancy characterized by abnormal proliferation of plasma cells and monoclonal immunoglobulins. Systemic lupus erythematosus (SLE) is an autoimmune condition that occasionally can be accompanied by a monoclonal gammopathy of undetermined significance (MGUS) and rarely can be complicated by the development of MM [1,2]. The underlying mechanisms for this secondary disease evolution are not completely understood, but it has been suggested that chronic hyperactivity of B cells eventually leads to their escape from normal regulatory mechanisms. The primary occurrence of MM, where its initial presentation is accompanied by a wide assortment of autoantibodies typically associated with SLE, has not been previously reported.

Case Report

A 62-year-old white male presented with nine months of myalgias, fatigue and intermittent fevers to 101 degrees Fahrenheit, all of which were transiently responsive to several short courses of antibiotics. Over the next three months he developed symmetrical arthralgias in small and large joints. Exhaustive infectious disease evaluations failed to uncover a definitive cause for his complaints. Further laboratory testing revealed a Westergren sedimentation rate (ESR) of 38, a positive antinuclear antibody (ANA) in a titer of 1:640 (diffuse pattern), and positive antibodies to RNP, Sm and

double stranded DNA. Normal results were obtained for CBC, urinalysis, renal function, liver tests, muscle enzymes and serum complement levels. No other connective tissue disease symptoms or signs were present. Specifically, there was no evidence of Sjogren's syndrome, rheumatoid arthritis, nor vasculitis.

A three-month trial of hydroxychloroquine 400mg daily did not produce any consistent clinical improvement, after which ESR had increased to 119, hemoglobin had declined to 10.5, and total protein had increased from 7.2 to 11.0. Skeletal x-rays and a chest x-ray were normal. Serum protein electrophoresis, quantitative immunoglobulins and serum immuno-electrophoresis revealed an IgG monoclonal gammopathy with low levels of serum IgA and IgM. Bone marrow examination revealed 50% plasma cells (IgG lambda), without free light chains in the urine. A diagnosis of multiple myeloma was made, and treatment instituted with lenalidomide, dexamethasone, allopurinol, colchicine, and aranesp. One year later total protein was 6.2, hemoglobin 13.8, and all rheumatologic symptoms had resolved. Lupus serologies at that time were not repeated.

Discussion

The concurrent association of SLE and MM is an uncommon event that can give rise to considerable diagnostic dilemmas. Most reports comment on the possible predisposition of the former leading to pathologic evolution of the latter, typically occurring in patients with well-established SLE [2-4]. In these reports the mean age at diagnosis of MM was 54 years, which is ten years earlier than the mean age for MM diagnosis alone. Equally confusing is

the converse, i.e., when MM presents with clinical and serological “lupus-like” manifestations that may occur at the evolutionary onset and progression of MM [5-7].

Although a positive ANA test is not uncommon in this scenario, our patient had stronger and more misleading serological evidence for SLE encompassing the presence of four autoantibodies (including “specific” antibodies to double-stranded DNA via the crithidia kinetoplast assay). In totality, however, his clinical features and overall disease course made it unlikely that he had a primary inflammatory autoimmune process which then predisposed him to MM. A stronger and more plausible assumption is that his MM process produced multiple abnormal laboratory features that masqueraded as SLE.

This case report reinforces prior observations that rheumatologic symptoms may appear at the onset of MM, and it simultaneously provides evidence that a wide variety of positive serologic autoantibody tests common to SLE may also be seen at the onset of MM. In such situations, unresponsiveness to initial anti-inflammatory therapy can also raise suspicions for an alternative diagnosis such as MM. Clinicians should routinely consider requesting a serum protein electrophoresis in patients presenting with clinical and serological rheumatologic phenomena, regardless of how “specific” such phenomena may appear.

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