Scleritis And A Positive Anti-Cyclic Citrullinated Peptide

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Citation

Abstract
Scleritis can be caused by a variety of systemic inflammatory diseases such as rheumatoid arthritis (RA), Wegener's granulomatosis, polyarteritis nodosa, sarcoidosis, inflammatory bowel disease and infectious diseases such as syphilis and tuberculosis. The incidence of RA in patients presenting with scleritis is 33%. We describe a patient with scleritis who presented with no signs or symptoms of RA but with a positive anti-cyclic citrullinated peptide (anti-CCP) antibody.

CASE REPORT
A 44-year-old woman was seen by the ophthalmologist in January 2004 with ocular pain and redness in the right eye. The patient was diagnosed with marginal keratitis with ulceration and treated with moxifloxacin eye drops and prednisolone eye drops. Redness, light sensitivity and tearing in the right eye persisted and scleritis was diagnosed by slit-lamp examination. The patient was treated with oral steroids and required up to 80mg of prednisone daily. A rheumatologic work up revealed a positive rheumatoid factor of 658 IU/ml (normal, < 14 IU/ml), which prompted referral to rheumatology. Work up was as follows. The patient denied any history of alopecia, rashes, bowel abnormalities, morning stiffness, swollen and painful joints, back pain, dry eyes or mouth, mouth or nose sores, easy bruising, respiratory or cardiac symptoms. Her past medical history was significant for Grave's disease treated with propylthiouracil and propranolol. Family history showed no rheumatoid arthritis (RA). Physical exam revealed a well appearing young woman with a normal skin and joint exam. She had erythema of the nasal aspect of the right eye, thyromegaly and poor dentition. Laboratory testing revealed a C-reactive protein of 0.33 mg/dl (normal, <0.80 mg/dl), strong positive CCP antibody, IgG of 148 Units (normal, <20 Units), and angiotensin-converting–enzyme (ACE) of 73 U/liter (normal <67 U/liter). Anti nuclear antibody (ANA), anti neutrophil cytoplasmatic antibody (ANCA), cryoglobulins and Lyme disease ELISA were negative. Chest and hand x-rays were normal. In conjunction with ophthalmology azathioprine was added as a steroid-sparing agent. The patient's symptoms improved and prednisone was gradually tapered.

DISCUSSION
Our patient presents with no joint symptoms and no clinical or radiographic evidence of joint disease with a positive rheumatoid factor and anti-CCP antibody. The anti-CCP antibody appears to be a specific diagnostic marker of RA with a reported sensitivity and specificity of 47.4% and 97.4% respectively. The anti-CCP antibody and rheumatoid factor can predate the onset of RA by several years. Furthermore the combination of anti-CCP and IgM RF increased the ability to predict erosive and progressive disease. The patient will require close clinical follow up and institution of early aggressive treatment if rheumatoid arthritis develops.

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References

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