Clinical Allergy Tips
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From the Editor: Autoimmune diseases are frequent entities and, consequently, all clinicians should be aware of their clinical recognition and first-line laboratory testing. In this Clinical Allergy Tip Dr. Marianne Frieri provides the basics for clinicians who might be called on to consider the diagnosis of autoimmune diseases.

Rheumatologic disorders
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Allergists/immunologists might evaluate patients with rheumatoid arthritis (RA), systemic lupus erythematosus (SLE), Sjögren’s syndrome (SS), scleroderma (ScI) and Wegner’s in their practice, diseases that can masquerade as allergic conditions. [1] Rheumatologic disorders have prominent clinical manifestations in the upper and lower airway that can be attributed to irritant or allergic reactions. RA causes joint synovitis, subcutaneous rheumatoid nodules, vasculitic skin ulceration, sicca symptoms, pulmonary nodules, interstitial fibrosis, mononeuritis multiplex, and Felty’s syndrome.

The American College of Rheumatology (ACR) criteria for the diagnosis of AR was designed for inclusion in clinical studies and not for routine clinical diagnosis. Approximately 20% of patients have negative rheumatoid factor and anti-cyclic citrullinated peptide antibody test results. IL-1, IL-6, and TNFα, have been linked to RA providing new therapeutic targets. [2]

SLE can present with urticaria or urticarial like lesions before onset. The ANA, using indirect immunofluorescence, is very sensitive so that a negative test result effectively rules out the diagnosis. ELISA testing is not recommended for screening because 10% to 20% of patients might have negative results. Anti-native DNA is associated with aggressive disease and ds-DNA is specific in 30-70% of cases. A high titer of anti-DNA antibodies makes the diagnosis of SLE very likely, although a negative anti-DNA test result does not exclude the diagnosis since 50% of patients never have anti-DNA. Another highly specific assay for SLE is anti-Smith, which reacts...
with one of the soluble extractable nuclear antigens occurring in approximately 25% of patients, and anti ribonucleoprotein can be seen especially with myositis, but is classically associated with mixed connective tissue disease when present in high titers. Complement C3 and C4 can be decreased with active disease. Anti-Ro (SSA) is observed in approximately 25% of patients with SLE, especially those with subacute cutaneous lupus erythematosus and is the best serologic feature along with anti-LA (SSB) for SS. SS may complicate RA and can present with keratoconjunctiva sicca, sinusitis, serous otitis media, bronchitis sicca, urticarial vasculitis and frequent paranasal sinus infections. The increased prevalence of rhinosinusitis and bronchitis often results in recurrent bronchitis. Pharyngeal dryness and hoarseness may masquerade as allergic disease and antihistamines should be avoided.

The allergist/immunologist should be able to identify the subtle early cutaneous and pulmonary manifestations, skin involvement with swelling and Raynaud’s phenomenon of Scl that can masquerade as other autoimmune disorders, in addition to pruritus or gastrointestinal dysfunction with nausea, vomiting, diarrhea, and malabsorption mimicking symptoms of gastrointestinal hypersensitivity. [2] Approximately 80% of patients have a centromere ANA pattern associated with limited disease or nucleolar pattern, anti-Scl70 with diffuse disease and RNA polymerase III antibody associated with renal crisis. Wegener’s presents with prominent ENT manifestations such as rhinosinusitis in almost all patients. Mucosal ulcerations, palatal ulcers, septal erosions and pulmonary involvement may also occur and provide clues to the diagnosis. The most common presentation remains as chronic rhinitis and infectious sinusitis. Early diagnosis and aggressive therapy is important to avoid the serious renal complications Cytoplasmic-ANCA is present in 90% of patients with active disease whereas the p-ANCA is present in 10% but is also present with other vasculitides.

REFERENCES