Commentary

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# Systemic Autoimmune Disorders of Neurology

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## Description

Systemic autoimmune diseases like Systemic Lupus Erythematosus (SLE) and Rheumatoid Arthritis (RA) are highly heterogeneous both at the clinical and pathogenic levels. Genetic, environmental, hormonal, epigenetic, and immunoregulatory factors are involved within the expression of systemic autoimmune diseases. Recent advances have identified numerous autoimmune-predisposing genomic loci and genes. However, it remains to be elucidated how each gene is involved within the development of disease. Animal models for spontaneous autoimmune disorder have contributed enormously to the identification of genes that are important in disease pathogenesis. Moreover, study of gene-manipulated animals that develop systemic autoimmune diseases has provided insights into the mechanisms of maintenance and breakdown of self-tolerance. Although animal modes don't represent with fidelity human disease, they need provide significant insights into the abrogation of self-tolerance, the development of autoimmunity, and related organ damage. Animals that developed autoimmunity spontaneously or after gene engineering or after the injection of a stimulant serve the preclinical studies which seek to work out the clinical efficacy of rationally developed drugs or biologics .

Systemic inflammatory diseases that regularly affect both the central and peripheral nervous systems and may begin with neurological symptoms. These diseases cross traditional boundaries between neurology and rheumatology, and diagnosis and treatment require familiarity with the spectrum of neurological involvement with interdisciplinary communication. We reviewed a number of the more common neurological manifestations of selected autoimmune diseases like systemic lupus erythematosus, Sjogren syndrome, rheumatoid arthritis, scleroderma, sarcoidosis, primary angiitis of the central nervous system, systemic vasculitis syndromes, and antiphospholipid syndrome.

We discuss several systemic diseases that have an immune-mediated pathogenesis. Although most of those diseases are infrequent and infrequently affect the neurological system, clinicians should remain cognizant of their existence. While an in-depth encounter with each of the subsequent diseases is out of the scope of this chapter, we cover the foremost encountered and also the most prominent neurological and clinical manifestations to assist guide the clinician into identifying, diagnosing, and treating these illnesses.

Remarkable discoveries over the last twenty years have elucidated the autoimmune basis of several, previously poorly understood, neurological disorders. Autoimmune disorders of the system may affect any part of the system including the brain and medulla spinalis (central systema nervosum, CNS) and also t he peripheral nerves, synapse, and muscle (peripheral system, PNS). This comprehensive overview of this rapidly evolving field presents the factors which can trigger breakdown of self-tolerance and development of autoimmune disorder in some individuals. Then the pathophysiological basis and clinical features of autoimmune diseases of the systema nervosum are outlined, with a stress on the features which are important to acknowledge for accurate clinical diagnosis. Finally, the most recent therapies for autoimmune CNS and PNS disorders and therefore their mechanisms of action and the most promising research avenues for targeted immunotherapy are discussed. The past decade has seen a dramatic increase within the discovery of novel neural antibodies and their targets. Many commercial laboratories can now test for these antibodies, which function diagnostic markers of diverse neurologic disorders that occur on an autoimmune basis. Some are highly specific certainly cancer types, and also the neural antibody profiles may help direct the physician's cancer.

The diagnosis of an autoimmune neurologic disorder is aided by the detection of an objective neurologic deficit (usually subacute in onset with a fluctuating course), the presence of a neural autoantibody, and improvement within the neurologic status after a course of immunotherapy. Neural autoantibodies should raise concern for a paraneoplastic etiology and will inform a targeted oncologic evaluation (eg., N-methyl-D-aspartate [NMDA] receptor antibodies are related to teratoma, antineuronal nuclear antibody type 1 [ANNA-1, or anti-Hu] are related to small cell lung cancer). MRI, EEG, functional imaging, videotaped evaluations, and neuropsychological evaluations provide objective evidence of neurologic dysfunction by which the success of immunotherapy could also be measured. Most treatment information emanates from retrospective case series and expert opinion. Nonetheless, early intervention may allow reversal of deficits in many patients and prevention of future disability.