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Commentary

Systemic autoimmune diseases

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INTRODUCTION

Systemic autoimmune diseases (often referred to as "connective tissue diseases") are enormous and clinically diverse cluster of disorders. They share in common inappropriate targeting of normal tissues by immune and inflammatory mechanisms resulting in self-injury. Although variety of those conditions predominantly affects one target tissue, all of them share the potential to cause disease in multiple organ systems. Systemic autoimmune diseases are thought to be linked because they're going to occur together within an equivalent patient and are often associated with overlapping patterns of autoantibodies. The lack of molecular-level understanding of etiology, sensitive and specific tests to help in diagnosis, and diverse clinical features render systemic autoimmune diseases a challenging but extraordinarily interesting group of diseases to diagnose and treat.

Whereas organ-specific autoimmune diseases target specific organs or tissues, systemic autoimmune diseases are more generalized, targeting multiple organs or tissues throughout the body. Some of the examples of systemic autoimmune diseases include MS, myasthenia, psoriasis, rheumatism, and systemic lupus erythematosus.

MULTIPLE SCLEROSIS

Multiple Sclerosis (MS) is an autoimmune central nervous system disease that affects the cerebrum and spinal cord. Lesions in multiple areas inside the central nervous system are a sign of multiple sclerosis and are caused by infiltration of immune cells across the blood-brain barrier. The immune cells include T cells that promote inflammation, demyelination, and neuron degeneration, all of which disturb neuronal signaling. Symptoms of Multiple Sclerosis (MS) include visual disturbances; muscle weakness; difficulty with coordination and balance; sensations like numbness, prickling, or "pins and needles"; and cognitive and memory issues. Myasthenia Gravis Autoantibodies coordinated against acetylcholine receptors within the synaptic cleft of neuromuscular junctions end in myasthenia. Anti-AChR antibodies are high-affinity IgGs and their synthesis requires activated CD4 T cells to interact with and stimulate B cells. Once produced, the anti-AChR antibodies affect neuromuscular transmission by a minimum of three mechanisms:

• Complement binding and activation at the myoneural junction

• Accelerated AChR endocytosis of molecules cross-linked by antibodies

• Functional AChR blocking, which prevents normal acetylcholine attachment and activation of, AChR despite the mechanism, the effect of anti-AChR is extreme muscle weakness and potentially death through respiratory arrest in serious cases.

PSORIASIS

Psoriasis may be a disease of the skin that causes irritated or sore patches of thick, red skin with silvery scales on elbows, knees, scalp, back, face, palms, feet, and sometimes other areas. Some people with psoriasis additionally get a type of arthritis called psoriatic arthritis, in which the joints can become inflamed. Psoriasis results from the complicated exchange between keratinocytes, dendritic cells, and T cells, and the cytokines produced by these different cells. In a process called cell turnover, skin cells that grow deep within the skin rise to the surface. Normally, this process takes a month. In psoriasis, as a result of cytokine activation, cell turnover occurs in only a couple of days. The thick inflamed patches of skin that are characteristic of psoriasis develop because the skin cells rise too quickly.

SYSTEMIC LUPUS ERYTHEMATOSUS

The damage and pathology of Systemic Lupus Erythematosus (SLE) is caused by type III hypersensitivity reactions. Autoantibodies produced in Systemic Lupus Erythematosus.

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SLE is directed against nuclear and cytoplasmic proteins. Antinuclear antibodies are present in more than 95% of patients with SLE,16 with extra autoantibodies including anti-double–stranded DNA (ds-DNA) and anti-Sm antibodies (antibodies to small nuclear ribonucleoprotein). Anti-ds-DNA and anti-Sm antibodies are unique to patients with SLE; in this manner, their presence is included in the classification criteria of SLE. Cellular cooperation with autoantibodies prompts to nuclear and cellular destruction, with components released after cell death leading to the development of immune complexes. Because autoantibodies in SLE can target a large variety of cells, symptoms of SLE can occur in many body locations. However, the most common symptoms include fatigue, fever with no other reason, balding, and a sunlight-sensitive "butterfly" or wolf-mask (lupus) rash that is found in about half of the individuals with SLE. The rash is frequently seen over the cheeks and bridge of the nose, but is often widespread. Other different symptoms may appear depending on affected regions. The joints may be affected, prompting to arthritis of the fingers, hands, wrists, and knees. Effects on the brain and nervous system can lead to cerebral pains, numbness, shivering, seizures, vision problems, and personality changes. There may additionally cause stomach pain, nausea, vomiting, arrhythmias, shortness of breath, and blood within the sputum. Effects on the skin can result in additional areas of skin lesions, and vasoconstriction can cause color changes within the fingers once they are cold (Raynaud phenomenon). Effects on the kidneys can result in edema within the legs and weight gain. A diagnosis of SLE depends on identification of 4 of 11 of the foremost common symptoms and confirmed production of an array of autoantibodies unique to SLE. A positive test for ANAs alone isn't diagnostic.