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Mini Review

Autoimmune Diseases: An Overview

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Abstract

These are the diseases that are encountered when the body's own immune system mistakenly attacks the body's tissues. Autoimmune disease affected individuals/animals have autoantibodies and immune cells present in their blood that target their own body tissues, where they can be associated with inflammation. The triggering event for induction of an autoimmune response may primarily be T or B cell mediated or may be both. Whenever IgG antibody production is initiated, help from CD4+ T cells is provided.

Keywords: Antibody Production; Autoimmune Disease; Hypersensitivity; Immunoglobulins; Inflammation

Introduction

In diseases like autoimmune Hashimoto's thyroiditis or insulin dependent diabetes, there is complete and irreversible loss of function of the targeted tissue resulting from the autoimmune aggression. In autoimmune Greaves Basedow disease or myasthenia gravis, the result of an autoimmune aggression is a chronic reaction that leads to either hyperstimulation or inhibition of its function. In SLE there occurs impairment/destruction of several tissues at the same time as the pathogenic events are multiple and complex.

Myasthenia gravis

The disorder of signal transmission that is between the nerves and muscles (neuromuscular transmission) which is characterized by muscular weakness and excessive fatigue constitutes a disorder known as myasthenia gravis. In dogs, cats and humans this disorder is either congenital or acquired (immune mediated). It is defined as a prototype autoimmune disease that is mediated by blocking antibodies. Myasthenic individuals/animals produce autoantibodies that bind to the acetylcholine receptors present on the motor end plates of muscles. Autoantibodies (IgG) cause destruction of the receptors, block the acetylcholine binding sites and trigger compliment mediated lysis of the cells. There is progressive weakness of the skeletal muscles as a result of reduction in the number of available, functional acetylcholine receptors. Clinically it is characterized by abnormal fatigue and weakness after mild exercise. In any breed of Dog, the disease can develop, but German shepherds, Golden retrievers, and Labradors appear to be more susceptible to the disease. There appears to be a breed predisposition for Abyssinians and related Somalis [1].

Clinical signs

Animals are presented with a history of difficulty in swallowing, regurgitation, labored breathing, and generalized weakness. The early signs include drooping eyelids, and inability to retract the corners of the mouth. Weakness is worsened by exposure to heat, infection and stress [1]. Disease can be classified as focal myasthenia gravis and generalized myasthenia gravis. In focal MG, an animal presents with mega esophagus and various degrees of facial paralysis with limb muscle weakness. In generalized MG, limb muscle weakness is associated with facial paralysis and mega esophagus. In acute fulminating MG, the disease rapidly leads to quadriplegia and respiratory difficulty. Aspiration is the main cause of death in myasthenic dogs. Mega esophagus, bark change (usually high pitched), hindquarter weakness, sudden urge to sit down, blind reflex (palpebral reflex) which is defined as a reflex, elicited by touching the eyelid and observing for a blink. This response fatigues and in some cases is absent in myasthenic animals. There is drooping lower lip, drooping tail and lethargy. Moaning noise primarily when lying down. Trouble controlling urine stream or holding squat while defecating. There is difficulty in breathing with aspiration pneumonia. Thymomas are mostly associated with autoimmune disorders. The abnormal neoplastic cells in thymomas express many self like antigens viz AChR- like, titin- like and ryanodine receptor like epitopes [2,3].

Diagnosis

Ice pack test is performed for assessing improvement in ptosis [4], Serological testing and electrophysiological tests, which are of two types RNS (Repetitive nerve stimulation study) and single fiber electromyography (SFE EM). Imaging tests aid to screen possible thymoma or abnormal thymus gland through a chest imaging study (computed tomography or magnetic resonance imaging).

Rheumatoid arthritis

It is an autoimmune disease that causes chronic inflammation of the joints. Immune mediated erosive polyarthritis is a common crippling disease that affects 1% of human population. A very similar disease has been reported in domestic animals (dogs) in which there is neither breed nor sex predilection. Many individuals with RA produce a group of auto antibodies which are called as rheumatoid factors that react with determinants of IgG (Fc region).The classical rheumatoid factor is an IgM antibody with such reactivity. Such autoantibodies bind to normal circulating IgG, forming IgM-IgG complexes. These complexes are then deposited in the joints and result in the activation of the complement cascade, causing type 3 hypersensitive reaction, leading to chronic inflammation of the joints [5].

Clinical signs

Clinical signs in dogs include chronic depression, anorexia, pyrexia, lameness which tends to be more severe after rest. Symmetrical swelling and stiffness (peripheral joints) leading to severe joint erosions and deformities (erosive arthritis). Affected joints may fuse in advanced cases, as a result of the formation of bony ankyloses. Swelling generally involves soft tissues and may be sub chondral rarefaction, cartilage erosion and narrowing of the joint space.

Diagnosis

Rheumatoid nodules, presence of serum rheumatoid factor which is measured by reliable tests, and characteristic radiographic changes in the wrists/hands.

Equine recurrent uveitis

The most common cause of blindness in horses is recurrent uveitis which is also known as periodic ophthalmia/moon blindness. The inciting factors in horses include microbial agents such as *Leptospira* spp. Microbial factors as well as genetic predisposition to the disease may provide clues as why horse appears to be the most susceptible host [5]. Inter-photoreceptor retinoid binding protein is the major autoantigen involved, with subsequent epitope spreading to the S. protein. Affected animals (horses) have circulating antibodies to *L. interrogans*, recurrent attacks of uveitis, retinitis, and vasculitis. They have blepharospasms, lacrimation and photophobia in severe cases. The eye lesions are infiltrated with Th-1 cells and neutrophils with extensive fibrin and C3 deposition. An autoimmune attack on the ocular tissue may also be responsible, which might be due to the result of molecular mimicry with *L. interrogans*. Other cases may be due to infectious agents like *Borrelia burgdorferi* and *Onchocerca cervicalis.*

Clinical signs

Clinical signs include catarrhal conjunctivitis, ocular pain, blepharospasms (squinting), lacrimation, photophobia, edema of cornea, hypopyon (pus in the eye), myosis which progresses to keratitis and iridocyclitis.

Diagnosis

Clinical diagnosis is based on the presence of characteristic clinical signs and a history of recurrent episodes of uveitis.

Autoimmune haemolytic anaemia (AIHA)

The auto-antibodies that are produced against red blood cell antigens provoke their destruction and cause a disease known as AIHA. It is well recognized in humans, dogs, cattle, horses, cats, mice, rabbits. The destruction of red blood cells may result from either intra vascular hemolysis that is mediated by compliment or by the removal of antibody coated red cells by the macrophages of spleen and liver. The cause of red cell destruction is the alteration in red cell surface antigens that is induced by drugs and viruses. The autoantibodies are produced against red cell glycophorins, cytoskeletal protein spectrin and the membrane anion exchange protein (CD 233 or band 3) in case of dogs. Its onset may be associated with other immunological abnormalities, with stress (vaccination), viral diseases, or with hormonal imbalances (pregnancy or pyometra).

Classification

Classification of AIHI is based on the identification of particular antibody involved, the optimal temperature at which such antibodies react and nature of hemolytic process which occurs.

Class I

Both immunoglobulins (IgG, IgM) are involved in this class, which is caused by autoantibodies that agglutinate the red cells at body temperature. As IgG is not able to activate compliment effectively, hence intra vascular hemolysis is not a characteristic feature of this class. The red blood cells are destroyed by phagocytosis in the spleen. Extensive erythrophagocytosis by neutrophils and monocytes are reflected on blood smears in severe cases.

Class II

It is mediated by IgM auto antibodies that act at a body temperature. As IgM is responsible for compliment activation efficiently, intravascular haemolysis is the cause of red blood cell destruction leading to haemoglobinaemia, haemoglobinuria, jaundice and anemia. The Kupffer cells in the liver or in the lymph nodes remove red cells that have compliment on their surfaces, hence such animals develop lymphadenopathy as well as hepatomegaly.

Class III

Class III of AIHA is mediated by IgG1 and IgG4. These auto antibodies bind to red blood cells at 37ºC. Neither these auto-antibodies activate compliment nor cause agglutination of red cells. These auto antibodies on these cells opsonize them and make them susceptible to phagocytosis by mononuclear phagocytes mainly in the spleen, as a result of this, splenomegaly is a consistent feature of this class.

Class IV

Some IgM auto antibodies are capable of agglutinating red cells, provided the blood is chilled. Such auto antibodies are termed as cold agglutinins. As the blood circulates through the extremities, it may be cooled sufficiently allowing agglutination of erythrocytes within the capillaries leading to stasis in the vessels, ischemia of tissues and finally necrosis.

Class V

This class is usually mediated by IgM auto antibodies. These auto antibodies combine with red blood cells when the blood is chilled to 4ºC but will not cause agglutination of them hence resulting in the activation of compliment and eventually leading to intravascular hemolysis.

Diagnosis

Anaemia and a regenerative response by the bone marrow are revealed from the hematology of affected animals. Blood smears show spherocytes (resulting from the partial phagocytosis of auto antibody coated red cells). The number of spherocytes in the blood smear is a measure of intensity of red blood cell destruction. Type 2, 3, 5 direct agglutination test are used to detect the presence of non-agglutinating/incomplete antibodies.

Conclusion

Autoimmune disease affected individuals or animals have autoantibodies and immune cells present in their blood that target their own body tissues, where they can be associated with inflammation.

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