AUTO-IMMUNE DISEASES CAUSED BY OVERACTIVE IMMUNE SYSTEM

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ABSTRACT

Immune System is a determining factor for human body to work harmonically. It provides the essential protection people need physically. The innate and adaptive immune system and immune cells decide whether we can survive the diseases and abnormal environments. But when the system goes wrong, the self-consuming situation will occur. And it's destructive and catastrophic, even fatal to the body. Normally the abnormal innate immune system can be roughly divided into overactive immune system and immunodeficiency. In the paragraph below, I will majorly focus on overactive system, and briefly introduce immune system. Many people are suffering from the diseases caused by overactive system, for instance, lupus erythematosus. We will introduce several usual ailments in our paper.

1. INTRODUCTION OF IMMUNE SYSTEM

Although people normally don't view immune system as an essential part like heart or brain for human body, it is actually underestimated by human. Immunity produces large number of cells to keep the whole system functionally. During early childhood, children get their immune cells from the bone marrow.

Majority of these cells need to go through determining secondary education before they are actually passed to the whole body. A professional immunologist will further divide the population categories based on the proteins which are the receptors interacting with the environment on the cells' surfaces. Receptors bind ligands which may be receptors on other cells, or soluble molecules such as cytokines. Normally cells can release hundreds of receptors on the surface in order to adjust the diversity [1]. These receptors assistant the immune system to interrogating the environment with danger like infection, damages, mutants and any other abnormal situation. Immune system and the immune cells can maximize the likelihood of surviving with the minimized casualty and damages.

A microbial infection need the pathogen itself to connect and interact with body cells. But as the first barriers, skins, mucous membranes and fine hair etc. actually increase the difficulty for the pathogen. These physical barriers provide innate protection for human, for instance, the tough overlapping cells of the skin and chemical barriers, and enzymes also limit many bacteria, such as lysozyme in snot and tears and the acid in the stomach [1]. These outward-facing surfaces actually encourage the presence of non-pathogenic microbes. By welcoming and supporting a co-operating microbial population, little opportunity is left for more dangerous relatives to move in. The healthy immune system lives happily with this symbiotic microbial farm, but still reacts when there is a dangerous infection. As our understanding of the ecology of this 'microbiome' grows, it may offer new therapies that can support the exclusion of disease causing organisms.

When pathogens do penetrate these defenses, and seek to live within our bodies and within our cells, they pose many threats, from quiet coexistence to wholesale cell destruction and death. There is wide diversity in pathogens' methods of attachment and entry. For every individual pathogen, this process is tailored to species, to specific cell types and to defined cell-surface receptors. Each infection uses a different door into the cell. Blocking off these routes of entry can stop an infection before it starts. By producing antibodies, the immune system can neutralize an infection before the key to the cell turns in that particular doorway. But this must be carried out one key at a time.

A pathogen that has penetrated the defenses of the skin and mucous membranes and established itself within or between cells, or a cell that has turned into a cancer, can only be eliminated by killing. This is very dangerous, and when the immune system is battling with an infection, it may put the life of the host at risk. Sometimes when it is not infection, but an adverse reaction to a drug or a treatment for cancer which activates the immune system, this leads to critical illness. There is a delicate balance between what is successful and what is sustainable when invoking a full-blown immune reaction.

2. OVERACTIVE IMMUNE SYSTEM AND AUTO-IMMUNE DISEASES

2.1 Hypersensitivity

Sometimes immune system can be really problematic and turn against the own body organs and tissues. This is called auto-immune diseases. Hypersensitivity is a kind of auto-immune diseases.

They are divided into four classes (Type I – IV) based on the mechanisms involved and the time course of the hypersensitive reaction. Type I, which is an immediate and anaphylactic reaction, is normal and easy to understand because it always come along with allergy syndromes [2]. The patients' symptoms can be diverse and different according to

their own immunity, ranging from slightly discomfort to death. Type I hypersensitivity is mediated by IgE, which triggers degranulation of mast cells and basophils when cross-linked by antigen. Type II occurs when antibodies accidentally recognize friendly cells as pathogens and bind antigens to the patients' own body cells [2]. After this connection built, the immune cells and afterward immune actions will destroy body tissues. This is also called antibody-dependent (or cytotoxic) hypersensitivity, and is mediated by IgG and IgM antibodies. Immune complexes deposited in various tissues trigger Type III hypersensitivity reactions [2]. Type IV hypersensitivity, also known as cell-mediated or *delayed type hypersensitivity*, usually takes between two and three days to develop [3]. Type IV is usually involved in many autoimmune and other infectious diseases. These reactions are taken charge by immune cells like T cells, monocytes and macrophages.

2.2 Immediate (Type I) Hypersensitivity

Antigens which cause allergic responses and symptoms are often being regard as allergens. The process of creating allergen-specific IgE is regard as sensitization which is an important prerequisite for the symptoms of immediate hypersensitivity to take place. Allergies and allergic asthma are mediated by mast cell degranulation that is caused by the crosslinking of the antigen-specific IgE molecules on the mast cell surface. The mediators released have various vasoactive effects already discussed, but the major symptoms of inhaled allergens are the nasal edema and runny nose caused by the increased vascular permeability and increased blood flow of nasal blood vessels. As these mediators are released with mast cell degranulation, type I hypersensitivity reactions are usually rapid and occur within just a few minutes, hence the term immediate hypersensitivity.

Most allergens are in themselves nonpathogenic and therefore innocuous. Some individuals develop mild allergies, which are usually treated with antihistamines. Others develop severe allergies that may cause anaphylactic shock, which can potentially be fatal within 20 to 30 minutes if untreated. This drop-in blood pressure (shock) with accompanying contractions of bronchial smooth muscle is caused by systemic mast cell degranulation when an allergen is eaten (for example, shellfish and peanuts), injected (by a bee sting or being administered penicillin), or inhaled (asthma). Because epinephrine raises blood pressure and relaxes bronchial smooth muscle, it is routinely used to counteract the effects of anaphylaxis and can be lifesaving. Patients with known severe allergies are encouraged to keep automatic epinephrine injectors with them at all times, especially when away from easy access to hospitals.

Allergists use skin testing to identify allergens in type I hypersensitivity. In skin testing, allergen extracts are injected into the epidermis, and a positive result of a soft, pale swelling at the site surrounded by a red zone (called the wheal and flare response), caused by the release of histamine and the granule mediators, usually occurs within 30 minutes. The soft center is due to fluid leaking from the blood vessels and the redness is caused by the increased blood flow to the area that results from the dilation of local blood vessels at the site [4].

2.3 Type II and Type III Hypersensitivities

Type II hypersensitivity, which involves IgG-mediated lysis of cells by complement proteins, occurs during mismatched blood transfusions and blood compatibility diseases such as erythroblastosis fetalis (see section on transplantation). Type III hypersensitivity occurs with diseases such as systemic lupus erythematosus, where soluble antigens, mostly DNA and other material from the nucleus, and antibodies accumulate in the blood to the point that the antigen and antibody precipitate along blood vessel linings. These immune complexes often lodge in the kidneys, joints, and other organs where they can activate complement proteins and cause inflammation [4].

2.4 Delayed (Type IV) Hypersensitivity

Delayed hypersensitivity, or type IV hypersensitivity, is basically a standard cellular immune response. In delayed hypersensitivity, the first exposure to an antigen is called sensitization, such that on re-exposure, a secondary cellular response results, secreting cytokines that recruit macrophages and other phagocytes to the site. These sensitized T cells, of the Th1 class, will also activate cytotoxic T cells. The time it takes for this reaction to occur accounts for the 24 hours to 72 hours' delay in development.

The classical test for delayed hypersensitivity is the tuberculin test for tuberculosis, where bacterial proteins from M. tuberculosis are injected into the skin. A couple of days later, a positive test is indicated by a raised red area that is hard to the touch, called an induration, which is a consequence of the cellular infiltrate, an accumulation of activated macrophages. A positive tuberculin test means that the patient has been exposed to the bacteria and exhibits a cellular immune response to it.

Another type of delayed hypersensitivity is contact sensitivity, where substances such as the metal nickel cause a red and swollen area upon contact with the skin. The individual must have been previously sensitized to the metal. A much more severe case of contact sensitivity is poison ivy, but many of the harshest symptoms of the reaction are associated with the toxicity of its oils and are not T cell mediated [4].

2.5 Lupus erythematosus

SLE (systemic lupus erythematosus) occurs in the reproductive age of women, more common in the 15 to 45 age group, with female: male ratio of 7 to 9: 1. SLE epidemiology in the United States, according to multi-regional survey report, the prevalence rate of 14.6 ~ 122/10 million people. SLE has complex and diverse symptoms and aftermaths. Most of the cases have the performance of mild arthritis, rash, occult nephritis, thrombocytopenic purpura, some patients have long-term stability in the sub-clinical state or light lupus, some patients can be symptom-free at first and suddenly become severe within days. SLE's natural course of the disease shows more aggravated and relieved. SLE can cause nausea, vomiting, abdominal pain, diarrhea or constipation, which is more common diarrhea, and may be associated with protein loss of enteritis, and cause hypoproteinemia. Active SLE can occur mesenteric vasculitis. SLE often causes anemia, leukopenia and thrombocytopenia. Some patients with the related lighter symptoms only have migraine, personality changes, memory loss or mild cognitive impairment; severe ones, however, can express cerebrovascular accident, coma, epilepsy status and so on. Often there will appear symmetrical polyarticular pain, swelling, usually not causing bone destruction. SLE skin lesions include light-sensitive, hair loss, and erythema, discoid erythema on hands and feet. SLE has no cure, but appropriate treatments can make the majority of patients to achieve complete remission of the disease. The immune system usually deals with dangerous infections and bacteria to keep your healthy. When the immune system attacks the body, it will occur autoimmune diseases, because it will confuse foreign things. There are many autoimmune diseases, including systemic lupus erythematosus (SLE). The term lupus has been used to identify many similar clinical manifestations and laboratory characteristics of immune diseases, but SLE is the most common type of lupus. People usually refer to SLE when they say lupus. SLE is a chronic disease whose symptoms are worsening and accompanied by mild symptoms. Most people with SLE can cure normal life. According to the American Lupus Foundation, at least 1.5 million Americans have diagnosed lupus. The Foundation believes that the number of people actually sick is much higher, and many cases are not diagnosed.

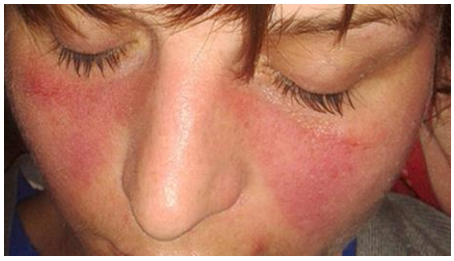

Figure 1: System Lupus erythematosus [3]

Symptoms can vary and unpredictable. Common symptoms

include [3]:

- severe fatigue
- joint pain
- joint swelling
- headaches
- a rash on the cheeks and nose, which is called a "butterfly rash"
- hair loss
- anemia
- blood-clotting problems
- fingers turning white or blue and tingling when cold, which is known as Raynaud's phenomenon

2.6 Rheumatoid arthritis

Rheumatoid arthritis is a chronic, etiology of inflammatory synovitis-based systemic disease. It is characterized by the multi-joint symptom, symmetry, invasive joint inflammation on hand and small joints, which is often associated with external organ involvement and serum rheumatoid factor positive. It can lead to joint deformity and loss of function.

The symptoms of this disease are:

- The stiffness in the morning when the joint activity is not flexibly subjective feeling. It is a non-specific manifestation of arthritis. Its duration is proportional to the severity of inflammation.
- multi-joint involvement was symmetrical polyarthritis. Involved joints are hand, foot, wrist, ankle and temporomandibular joint, the other can also have elbow, shoulder, cervical, hip, knee and so on. Cervical involvement may have neck pain, neck weakness and difficult to maintain its normal position, atlantoaxial subluxation, the corresponding spinal cord compression and vertebrobasilar insufficiency of the performance.
- General performance on the outside of the joints may have fever, rheumatoid nodules, rheumatoid vasculitis and lymph nodes Swollen. Heart involvement may have pericarditis, pericardial effusion, epicardium, myocardium and valve nodules, myocarditis, coronary artery disease, aortitis, conduction disorders, chronic endocarditis and heart valve fibrosis and other performance. Respiratory system involvement may have pleurisy, pleural effusion, pulmonary arteritis, interstitial lung disease, nodular lung disease. Nervous system in addition to the symptoms of peripheral nerve compression, but also induced neurological diseases, myelopathy, peripheral neuropathy, secondary to vasculitis in ischemic neuropathy, muscle hypertrophy and drug-induced neurological disease. Anemia is the most common manifestations of RA, are chronic diseases, anemia, often mild to moderate. Digestive system may be due to RA vasculitis, complications or drug treatment caused.

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