OPEN ACCESS Freely available online

Immunological Disorders and Immunotherapy

Perspective

The Relationship Between Auto-immune Disease and Common Variable Immunodeficiency

Richard Moore*

Department of Microbiology, University of Pennsylvania, Philadelphia, USA

DESCRIPTION

Autoimmunity is the immune system that an organism uses to attack its own healthy cells, tissues, and other normal bodily parts. An "autoimmune disease" is any illness that develops as a result of this type of immune reaction. The autoimmune illness is a sickness that develops when the immune system reacts abnormally to a healthy bodily part. An organism's immune system reacts in a certain way in order to protect itself from outside intruders. These microorganisms, which include viruses, bacteria, parasites, and fungi, are among the many invaders that could seriously harm the host organism's health if they are not removed from the body [1].

All people, including those in a state of normal health, have autoimmune disease, which is defined as the presence of antibodies or T cells that respond to self-protein. If self-reactivity can result in tissue damage, it creates autoimmune disorders. An antibody (a type of protein) that is created by the immune system and is directed against one or more of the person's own proteins is known as an autoantibody. Such antibodies are linked to numerous autoimmune disorders. T cells are a particular kind of lymphocyte [2]. One of the crucial immune system's white blood cells, T cells are essential to the adaptive immune response. T-Cell Receptors (TCRs), which are present on the cell surface of T cells, allow them to be recognized from other lymphocytes [3]. All proteins that are produced endogenously by DNA-level transcription and translation within a target organism are referred to as self-proteins.

This excludes proteins produced as a result of viral infection, but it may also contain proteins produced by commensal bacteria in the intestines. Proteins that enter the body of the target organism through the circulation, a break in the skin, or a mucous membrane but are not produced there may be classified as "non-self" and then targeted and destroyed by the immune system. For optimal health, the body must be able to tolerate self-proteins; if it mistakenly views them as "non-self," the immune reaction against endogenous proteins that follows could result in the onset of an autoimmune illness [4].

Immunodeficiency and autoimmunity

Immunodeficiency syndromes that exhibit clinical and biochemical signs of autoimmunity are widespread. These patients' impaired immune systems' capacity to fight off infections may be to blame for the ongoing immune system activation that leads to autoimmunity. One illustration is Common Variable Immune Deficiency (CVID), a condition in which several autoimmune diseases are present, including autoimmune thyroid disease, autoimmune thrombocytopenia, and inflammatory bowel disease. Low antibody levels, particularly in Immunoglobulin (Ig) types IgG, IgM, and IgA, and recurring infections are two features of the immunological condition known as Common Variable Immune Deficiency (CVID) [5]. The high sensitivity to foreign invaders, persistent pulmonary illness, and gastrointestinal inflammation and infection are among the typical symptoms.

Another illustration is the autosomal recessive primary immunodeficiency known as familial hemophagocytic lymphocytosis. These people frequently exhibit pancytopenia, rashes, swollen lymph nodes, and enlargements of the liver and spleen. It is believed that the cause is the presence of several uncleared viral infections caused by a perforin deficiency. An excessive number of histiocytes are referred to as histiocytosis in medicine [6]. The term is also frequently used to describe a number of rare disorders that share this symptom. The term "histiocytosis" is occasionally and perplexingly used to refer to specific illnesses.

Additionally, Pancytopenia is a medical disorder in which practically all blood cells have significantly decreased numbers. A condition known as lymphadenopathy or adenopathy causes the lymph nodes to grow or behave abnormally. The most prevalent type of lymphadenopathy is lymphadenitis, which results in swollen or enlarged lymph nodes. Lymphadenopathy and lymphadenitis are typically treated as synonyms in clinical practise, with very few exceptions. Hepatosplenomegaly, on the other hand, is the simultaneous enlargement of the spleen and liver.

Correspondence to: Richard Moore, Department of Microbiology, University of Pennsylvania, Philadelphia, USA; E-mail: moorerichard03@gmail.com

Received: 15-Mar-2022, Manuscript No. IDIT-22-20104; **Editor assigned:** 17-Mar-2022, Pre-QC No: IDIT-22-20104 (PQ); **Reviewed:** 01-Apr-2022, QC No: IDIT-22-20104; **Revised:** 07-Apr-2022, Manuscript No: IDIT-22-20104 (R). **Published:** 15-Apr-2022; DOI: 10.35248/2593-8509.22.7.114

Citation: Moore R (2022) The Relationship Between Auto-immune Disease and Common Variable Immunodeficiency. Immunol Disord Immunother. 07: 114

Recurrent bacterial and fungal infections, as well as persistent lung and gut inflammation, are also observed in patients with Chronic Granulomatous Disease (CGD), according to the study. The neutrophils produce less Nicotinamide Adenine Dinucleotide Phosphate (NADPH) oxidase, which is what causes CGD. Patients with midline granulomatous disease, an autoimmune condition frequently seen in people with granulomatosis with polyangiitis and NK/T cell lymphomas, are found to have hypomorphic RAG mutations. The patients with Wiskott-Aldrich Syndrome (WAS) also frequently exhibit lymphoma, eczema, and autoimmune symptoms. The additionally, autoimmunity and infections coexist in autoimmune polyendocrinopathy-candidiasis-ectodermal dystrophy, including organ-specific autoimmune symptoms (such as hypoparathyroidism and adrenocortical failure) and chronic mucocutaneous candidiasis. And last, autoimmune and atopic disorders can occasionally be brought on by IgA deficiency.

REFERENCES

1. Wells CR, Pandey A, Ndeffo Mbah ML, Gaüzère BA, Malvy D, Singer BH, et al.The exacerbation of Ebola outbreaks by conflict in the Democratic Republic of the Congo. Proc Natl Acad Sci U S A 2019; 116:24366–72.
2. Kuhn JH, Bavari S. Asymptomatic Ebola virus infections-myth or reality? Lancet Infect Dis 2017; 17:570–1.
3. Halfmann PJ, Eisfeld AJ, Watanabe T, Maemura T, Yamashita M, Fukuyama S et al. Serological analysis of Ebola virus survivors and close contacts in Sierra Leone: a cross-sectional study. PLoS NeglTrop Dis 2019; 13:e0007654.
4. Leroy EM, Baize S, Volchkov VE, Fisher-Hoch SP, Georges-Courbot MC, Lansoud-Soukate J, et al. Human asymptomatic Ebola infection and strong inflammatory response. Lancet 2000; 355:2210–5.
5. Ploquin A, Zhou Y, Sullivan NJ. Ebola immunity: gaining a winning position in lightning chess. J Immunol 2018; 201:833–42.
6. Pigott DM, Golding N, Mylne A, et al. Mapping the zoonotic niche of Ebola virus disease in Africa. eLife 2014; 3:e04395.
7. Kuhn JH, Bavari S. Asymptomatic Ebola virus infections—myth or reality?. The Lancet Infectious Diseases. 2017 Jun 1;17:570-1.