Systemic Lupus Erythematosus (SLE) Analysis

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Introduction

We are exposed to an enormous number of microorganisms and foreign agents all the time. Our immune system safeguards us against these foreigners as they may be detrimental to our body's health. The immune system is empowered with the capacity to discriminate between self and non-self. For non-self-substances may include toxins, microbes, etc., and the term self refers to our own body cells, tissues and organs etc. immune system is an intricate multi-component system that has the capacity to retaliate and hit back at any foreign substance that may cause harm to the human body.

Discussion

Imagine if the custodian of the body's health perpetrates, repels and runs riot and starts killing the body's self-tissues, organs and cells, leading to serious complications. Yes! It happens and is termed "auto-immune disease". Lupus erythematosus, commonly known as Lupus, is a chronic and lifelong auto-immune disease in which the immune system runs wild, destroying cells, tissues and organs if left untreated. This results in inflammation of the site where the immune system exerts its deleterious effects. Lupus is a lifelong disease and does not subside with time, unlike most diseases, which are acute and symptoms resolve after some time. Inflammation is not site-restricted but generalized and seen in multiple locations, for example, joints, blood, lungs, skin, heart, brain, and kidney and it can be anywhere in the body.

Most people develop mild disease, but serious symptoms and complications have been seen in large proportions of Lupus cases. The whole gamut of anomalies prevails, including an increase in body temperatures, fatigue, joint and mussel's pain, body aches, chest pain, headache, shortness of breath, chronic dry eyes, skin lesions, confusion, loss of memory, rashes, including a butterfly on the face.

Great work has been done and is still underway to investigate the cause of the disease, the factors that influence chances of getting auto-immune disease are environmental, genetic, harmones, infections and medications. Thus, Lupus is a multifactorial disorder. Among the environmental factors, ultraviolet rays, certain medications, traumas, viruses, and physical and mental stem have been reported to be important facilitators of Lupus or any other auto-immune disease.

Lupus of four types has been identified: cutaneous Lupus (only confined to the skin), systemic lupus (SLE is the most common type of Lupus), erythematosus, DIL (drug-induced) and neonatal (is rare and seen in neonates bone to diseased mothers). In this essay, we'll focus on SLE, as it happens to be the most common type of Lupus.

As mentioned earlier, SLE is a chronic disease and it effects various organ systems, it happens primarily due to the deposition and formation of autoantibodies and the complexities of immune system that leads to ultimate organ damage. The production of hyperactive B cells that results from T-cells and antigen stimulation is increased by the antibodies against antigens, which are uncovered on the surface of apoptotic cells. The antigens triggering T-cell and B-cell stimulation in patients with SLE can be accredited to the improper disposal of apoptotic skin cells. Along the way of cellular death, bits of material form on the surface of the perishing cell. Antigens which were absent on the cellular surface are now present on the surface, they are normally embedded within the cellular material. Anionic and nucleosome phospholipids are common examples of antigens that potentially trigger an immune response and have been recognized in patients with SLE.

The impaired functioning of phagocytic cells compromises the removal of these apoptotic cells, consequently resulting in the disposal of antigen recognition in patients with SLE. The development of SLE is thought to be associated with T-lymphocyte when it is introduced to an antigen-presenting cell (APC). The main histocompatibility complex (MHC) portion of the APC hemmed in with the T-cell receptor leads to cytokine release, B-cell stimulation and inflammation. The tissue damage in SLE is caused by the stimulation of B-cell division and the production of immunoglobulin G (IgG) autoantibodies. Autoantigen-specific T cells and B cells interact and produce injurious autoantibodies in unhealthy individuals. Antinuclear antibodies (ANAs) are among the many autoantibodies identified in SLE that directly attack the nuclear components of the cells. In the process of diagnosis the detection of antinuclear antibodies is an essential element. There might be positive results in patients for more than one ANA.

The most extensively tested ANAs are the anti-double-stranded DNA antibodies in SLE. SLE-induced kidney and skin diseases are linked with these antibodies and are present in a significant number of patients. Among other examples of ANAs are the anti-La antibodies and anti-Ro antibodies; these antibodies are commonly detected during pregnancy and are linked with fatal heart damage. Others are anti-Smith antibodies, which are marked with kidney disease. There is another group of autoantibodies that attacks the phospholipid moiety of the prothrombin activator complex and also the cardiolipin that can lead to the loss of pregnancy and abnormal clotting (Maidhof and Olga, 2012). In summing up, the production of autoantibodies is due to the presence of hyperactive B cells along with the harmful exclusion of apoptotic cellular material that results in the formation of immune complexes.

The diagnosis of SLE is made on symptoms and observed signs, diagnostic testing and laboratory testing adapted to each patient. According to The 1997 Update of the 1982 American College of Rheumatology (ACR) Revised Criteria for Classification of Systemic Lupus Erythematosus (ACR), if the

patient exhibits four or more of the 11 criteria, the diagnosis can be made with % sensitivity and 95% specificity (Hochberg, 1997). Nevertheless, according to a study conducted in 2003, a higher sensitivity to the weighted criteria has been seen as compared to the ACR criteria referred to above (Sanchez et al., 2003). Therefore, diagnostic testing varies depending on symptoms and signs affecting each patient. For instance, radiography, renal ultrasound, chest radiography and electrocardiography are used to assess different pains in the body.

As far as the treatment is concerned, the approaches vary depending on the severity and kind of disease. Recommendations for every patient include proper diet, nutrition, sun protection, appropriate immunization, exercise, cessation of smoking and stability of comorbid conditions. NSAIDs, antimalarial medications, steroids, immunosuppressive agents, monoclonal antibodies, and rituximab are among the medications applied in different situations depending on the symptoms and signs (Bertsias et al., 2008). The use of stem-cell transplantation has interested many researchers due to its power to rebuild the immune system by introducing healthy cells into the body. It has been studied that rituximab decreases the number of B cells, while DHEA is believed to help in regulating sex hormones (Thatayatikom and Andrew, 2006).

Conclusion

Although scientific research has been done on Lupus for many decades, it continues to present questions that cannot be answered. There is no cure that has been discovered for this disease; the medications listed above are only to control the flares, to manage symptoms and to maintain remission. However, healthcare professionals and Pharmacists can perform a crucial role in treatment by enlightening people. In order to increase the value of life for all and the increased rate of survival, current research is underway to kindle hope for many patients affected by SLE every year.

Works Cited

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