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Rheumatology: Current Research

Commentary

Pathogenesis and Environmental Triggers of Sjogren Syndrome

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ABOUT THE STUDY

Sjogren syndrome, sometimes known as Sjogren's syndrome, is a chronic autoimmune disease that affects the body's moisture-producing glands (lacrimal and salivary), as well as the lungs, kidneys, and nervous system. Dryness (dry mouth and dry eyes), discomfort, and exhaustion are the main symptoms. Other signs and symptoms may include dry skin, dry vagina, a persistent cough, numbness in the arms and legs, fatigue, pain in the muscles and joints, and thyroid issues. Additionally, there is a 15% greater risk of lymphoma among those affected.

While the specific etiology is unknown, it is thought to be a result of a genetic predisposition along with an environmental factor, such as exposure to a virus or bacterium. Primary Sjögren's syndrome and secondary Sjögren's syndrome are two different types of the connective tissue illness that might result in it. Other autoimmune conditions including Rheumatoid Arthritis (RA), Systemic Lupus Erythematosus (SLE), or systemic sclerosis may also be linked to Sjögren's syndrome. The ensuing inflammation gradually harms the glands. Blood tests are used to detect certain antibodies and moisture-producing gland biopsies are used to diagnose. On biopsy, the glands often contain lymphocytes.

Despite being one of the most prevalent auto-immune disorders, Sjögren's syndrome has no specific, non-invasive diagnostic tests, and therapy focuses on controlling the patient's symptoms. Artificial tears, anti-inflammatory drugs, punctual plugs, or surgery to close the tear ducts can all be used to treat dry eyes.

Chewing gum, ideally sugar-free, drinking water, or using a saliva substitute can help with dry mouth. Ibuprofen may be used by those who have joint or muscle pain. Antihistamines and other medications that can make person dry out may also be stopped. The most accurate diagnosis currently possible requires a lip biopsy.

PATHOGENESIS

Due to incomplete elucidation of the pathogenetic pathways underlying Sjögren's disease, there is a paucity of pathophysiological knowledge regarding the treatment of this autoimmune exocrinopathy. Despite the difficulty in determining the precise genesis and causation of this disease due to the myriad of contributing elements, significant advancements over the past ten years have led to a postulated series of pathogenic events that take place before the diagnosis of Sjögren's syndrome.

The exocrine glands, specifically the acinar and ductal cells, are thought to be lost in a unique, self-sustaining, immune system-mediated condition known as Sjögren's syndrome. This explains the more pronounced symptoms (such as the absence of saliva and lacrimal fluid), but it does not account for the more pervasive systemic consequences noticed.

Environmental and hormonal factors, specifically CD4+ T cells, B cells, and plasma cells, are thought to be able to cause the infiltration of lymphocytes, specifically CD4+ T cells, B cells, and plasma cells, leading to glandular dysfunction in the salivary and lacrimal glands in the presence.

Increased levels of IL-1RA, an interleukin 1 antagonist, are found in Cerebrospinal Fluid (CSF) in people with Sjögren's syndrome. This shows that the disease starts with an uptick in interleukin 1 system activity, followed by an auto regulatory uptick in IL-1RA to decrease interleukin 1's ability to bind to its receptors. Although increased IL-1RA is seen in the CSF and is associated with greater fatigue through cytokine-induced illness behavior, interleukin 1 is most likely the marker for fatigue.

However, IL-1RA levels in saliva are known to be low in Sjögren's syndrome, which may be the cause of mouth irritation and dryness. In addition to displaying secondary Sjögren's syndrome symptoms, patients with main rheumatic diseases such systemic lupus erythematosus, rheumatoid arthritis, or systemic sclerosis frequently also do. Environmental triggers

Epithelial cells may be prompted by environmental events, such as glandular viral infection, to activate the innate immune system via toll-like receptors. Although a number of infectious, exogenous agents, including the Epstein-Barr Virus (EBV), human T-lymphotropic virus 1, and hepatitis C virus, have been linked to the development of Sjögren's syndrome, their connection to the condition is tenuous.

Although EBV is found in the salivary glands of healthy people, Sjögren's patients have a significant incidence of EBV reactivation due to elevated quantities of EBV DNA. This suggests that in Sjögren's syndrome, viral reactivation and lymphoid infiltrates' incapacity to regulate EBV replication result in the onset or maintenance of an immune response in target organs. However, it is still unclear how exactly EBV is reactivated in lesions of individuals with Sjögren's syndrome and whether precise molecular processes are involved in the process.

Programmed cell death

Although its significance in Sjögren's syndrome is debatable, dysregulation of apoptosis (programmed cell death) is thought to contribute to the pathophysiology of a number of autoimmune illnesses. While expression of BCL-1, which is known to suppress apoptosis, was shown to be dramatically reduced in acinar and ductal epithelial cells of Sjögren's patients compared to healthy individuals, both the Fas and Fas ligand proteins are overexpressed in primary Sjögren's patients.

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Received: 04-Oct-2022, Manuscript No. RCR-22- 20242; **Editor assigned:** 07-Oct-2022, PreQC No. RCR-22- 20242 (PQ); **Reviewed:** 21-Oct-2022, QC No. RCR-22- 20242; **Revised:** 28-Oct-2022, Manuscript No. RCR-22- 20242 (R); **Published:** 04-Nov-2022, DOI: 10.35841/2161-1149.22.12.317

Citation: Austen Y (2022) Pathogenesis and Environmental Triggers of Sjogren Syndrome. Rheumatology (Sunnyvale). 12: 317.