

Dry Eye Disease In Primary Sjogren's Syndrome

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Sjogren's syndrome (SS) is a chronic systemic autoimmune disease characterized by hypofunction of exocrine glands (mainly the lacrimal and salivary glands) with a wide spectrum of extraglandular manifestations. Dry eye diseases and their complications may be the first presenting symptoms of SS and are commonly seen in ophthalmology practices. This conference provides a brief overview with an update on primary SS (pSS) and dry eye disease (epidemiology, etiopathogenesis, clinical manifestations, classification criteria, evaluation and management guidelines of dry eye disease) from the perspective of ophthalmology and internal medicine. The most commonly used classification criteria have been the American-European Consensus Group (AECG) criteria. In 2012, new classification criteria developed using the NIH-funded Sjogren's International Collaborative Clinical Alliance (SICCA) registry were published. Developments within the fields of medical specialty and biological science have contributed considerably to the understanding of the pathologic process of Sjogren syndrome. One report in 1985 showed human white cell substance DR (HLA-DR) expression in animal tissue cells of duct gland diagnostic test specimens taken from patients with Sjogren syndrome. The predominant leucocyte infiltrate consisted of helper T cells. herpes virus genotype analysis by PCR disclosed that solely kind I herpes virus genomes may well be detected in thirty fifth of traditional lacrimal glands. herpes virus genotype analysis by PCR disclosed that solely kind II herpes virus nuclear antigens (EBNA-2-deleted) sequence sequences were amplified from traditional lacrimal glands. In distinction, kind I herpes virus genomes (but not EBNA-2-deleted herpes virus sequences) were amplified from duct gland diagnostic test specimens obtained from patients with Sjogren syndrome. All of this data suggests that persistent herpes virus infection plays a task within the duct gland pathology of Sjogren syndrome. Human T-cell lymphotropic virus type-1 (HTLV-1) has conjointly been involved.

Tear Production/Secretion Augmentation Augmentation of tear production/secretion has been tried with medications like bromhexine and 3-isobutyl 1-methylxanthine (IBMX). These are tried outside of the us, though neither rose geographic area staining nor ocular discomfort improved with the employment of bromhexine. The mode of action is to extend duct gland secretion directly. Agents to stimulate muscarinic receptors (pilocarpine and cevimeline) are approved by the U.S. Oral alkaloid

has been incontestible to extend the amount of goblet cells and to boost the general health of the mucosa epithelial tissue in Sjogren syndrome as proved by impression biological science. Immunomodulators general immunological disorder agents is also necessary to boost tear production and to resolve severe inflammation in recalcitrant primary or secondary Sjogren syndrome. For the general element further as for the treatment of native immune effects, modification of the response with amethopterin, antimalarials, cyclophosphamide, anti-TNF compound, or tumour sphacelus issue (TNF) protein has been advocated. A pilot study and 1-year follow-up open trial with anti-TNF compound, a antibody to TNF-alpha, incontestible improvement all told tested objective and subjective measures of Sjogren syndrome sickness activity. However, a follow-up randomised, double-blind, placebo-controlled trial with anti-TNF compound and Enbrel, a person's TNF-alpha-p75 receptor, incontestible no profit. additional studies of TNF-alpha antibodies area unit thus required to work out therapeutic impact.

Cyclosporin A Cyclosporin A, a strong suppressor of T-cell operate, has been evaluated for treatment of Sjogren syndrome. Though this agent has been used orally to suppress T-cell operate in patients World Health Organization have had organ transplants, in Sjogren syndrome, its use is experimental and reports haven't been encouraging. Cyclosporin a tenth or two in liquid or ointment kind has been used locally following the initial report that its use raised duct gland operate in dogs. In one study, though oral cyclosporin A (5 mg/kg/d) crystal rectifier to subjective improvement of xerotes symptoms compared with placebo in most patients with primary Sjogren syndrome, solely two hundredth of patients noted improvement in ocular irritation. No distinction in liquid tear production evaluated by Schirmer testing was rumored between the two teams. A pilot trial of 1 Chronicles cyclosporin A ophthalmic ointment showed marked subjective improvement of symptoms when put next with placebo. Patients treated with topical cyclosporin A had less ocular surface rose geographic area staining than management subjects; but, there was no distinction in Schirmer take a look at values or tear break-up times between the two teams. Topical two cyclosporin an answer has been rumored to with success treat paracentral tissue layer ulcers in patients with autoimmune disease and secondary Sjogren syndrome. Cyclo-

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sporin A functions as a secretagogue for the duct gland and conjointly inhibits T-cell activation, thereby limiting lymphocyte-induced cell death of acinar cells. Apoptosis-related markers were found to decrease in mucosa epithelial tissue when vi months of treatment with topical cyclosporin A. Recently, a new approach has been developed by the American College of Rheumatology and European League against rheumatism (2016 ACR/EULAR Classification Criteria for Primary Sjogren's Syndrome). Those diagnostic criteria use two dry eye signs: Schirmer's test of 4 or ocular staining score >5). The assessment of dry eye requires multiple tests including the Schirmer's test, the tear breakup time, the lissamine green staining test, and the corneal staining with fluorescein dye. Many diagnosis and treatment guidelines have been developed, including the Delphi (the Dry Eye Preferred Practice Patterns of the American Academy of Ophthalmology and the International Task Force Delphi Panel on Dry Eye) panel treatment recommendations for dysfunctional tear syndrome (2006), the International Dry Eye Workshop (DEWS) (2007), the Meibomian Gland Dysfunction (MGD) Workshop (2011), and the updated Preferred Practice Pattern guidelines

from the American Academy of Ophthalmology pertaining to dry eye and blepharitis (2013).

Recent Publications: 1. Del Papa N and Vitali C (2018) Management of primary Sjögren's syndrome: recent developments and new classification criteria. *Ther Adv Musculoskelet Dis*.10(2):39–54. 2. Shiboski C H, et al. (2017) 2016 American College of Rheumatology/European League against rheumatism classification criteria for primary Sjögren's syndrome: A consensus and data-driven methodology involving three international patient cohorts. *Annals of the Rheumatic Diseases* 76:9–16. 3. Baer A N and Walitt B (2017) Sjögren syndrome and other causes of Sicca in older adults. *Clinics in Geriatric Medicine* 33:87–103. 4. Foulks G N, et al. (2015) Clinical guidelines for management of dry eye associated with Sjögren disease. *The Ocular Surface* 13:118–132. 5. Shiboski S C, et al. (2012) American College of Rheumatology classification criteria for Sjögren's syndrome: a datadriven, expert consensus approach in the SICCA cohort. *Arthritis Care and Research (Hoboken)* 64:475–487.

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