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Incidence and Prevalence of Infectious Uveitis Represents the Prevalence of Endemic Diseases

Katam Bhanupriya*

Department of Ophthalmology, Vignan collage of pharmacy,Hyderabad, India

***Corresponding author:** Katam Bhanupriya, Department of Pharmacy, Vignan collage of Ophthalmology, Hyderabad, India, E-mail: bhanupriya_k@gmail.com

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Description

The uveitides can be caused by inflammation involving primarily uveal tissue. The uvea can be secondarily affected by inflammations of the lens, retina, optic nerve, sclera and cornea. Histologically, the uveitis reveals features of acute non-granulomatous or chronic inflammation of either granulomatous or non-granulomatous. Such histologic changes can reflect underlying immune-pathogenicity of non-infectious and infectious uveitides. These histologic variations are recognized by clinical examinations as well as an initiation of tailored laboratory investigations that can establish the etiologic diagnosis of uveitis in humans.

The uveitides can be of infectious etiology or autoimmune mediated. Globally, uveitis is a significant cause of blindness. In the U.S. it is estimated to be responsible for around 10-15 percent of legal blindness. The estimate of blindness can be higher in developing countries due to infectious causes.

In patients with infectious uveitis, the inflammatory process is also driven by innate and adaptive immunity directed toward eliminating the infectious agent. Such an immune process can prolong ocular inflammation and cause tissue damage as noted in tuberculous uveitis. Release of sequestered tissue antigens from the damaged tissues can initiate autoimmune inflammation. The trigger for the autoimmunity can derive from molecular mimicry of shared antigens of the infectious agent with the tissue antigens, adjuvant effects of the infectious agent and can also be formed by standard activation or epitope spreading.

Use of Immunomodulatory

Such infectious and immune mediated inflammation complicates the treatment of infectious uveitis with antimicrobials to combat the infection, as well as the use of immunosuppressive agents to minimize immune driven inflammation and subsequent tissue damage. Use of immunomodulatory agents can enhance infectious processes, resulting in prolonged/chronic and recurrent inflammation, associated tissue damage and the sequelae of such inflammation, leading to the development of cataract, glaucoma and retinal damage. Thus, balancing elimination of the infectious agent and minimizing or preventing immune mediated prolonged inflammation or autoimmunity is a challenge. A clear understanding in eliminating the infectious agent and minimizing tissue damage from the infectious agent and immune process is required to prevent vision loss in infectious uveitides.

Usually, the incidence and prevalence of infectious uveitis represents the prevalence of endemic diseases of a region. A clear understanding of endemic diseases in a geographic location could enhance early clinical diagnosis and appropriate antimicrobial interventions. Current diagnosis of infectious uveitides requires a big data analysis of endemic diseases and their spread to non-endemic countries. In the United States, a recent big data study using nationwide medical claims revealed hitherto unknown higher incidences of infectious uveitis, stating that overall age increased the risk of infections uveitis significantly for each decade over the age of 18 years. Such data indicates older individuals are more prone to develop infectious uveitides, however, there is a gap in clear understanding of the mechanism for higher incidence of infections in the elderly and these individuals' innate and adaptive immunity against infectious agents.

The Age of Enlightenment

A characteristic of our medical past was that, though well-motivated by a desire to provide better care to our patients, actual therapeutic practice was often driven more by personality and confidence rather than by careful study and appraisal. Thus up to the early 20th century the therapeutic armory of the physician included useless or harmful practices such as bleeding, purging, and widespread provision of tonics, mercuric compounds and hypnotics. Patients with ocular inflammation were similarly provided for. In the 18th century a popular propriety remedy 'Golden Eye Ointment' was a mercury oxide that was normally mixed with hog's lard, and was promoted as being useful for 'all forms of chronic ophthalmia'. In the late 19th century, Savory's Compendium of Domestic Medicine recommended "In inflammation of the eye, originating from cold or accident, it is advisable to apply three or four leeches round the orbit they will always be found to be safe, and generally a successful remedy." Rather more useful was the application of tincture of belladonna to induce pupil dilation. In the 1930s the advent of commercially available antibiotics heralded the start of rational treatment for infectious eye disease, but also gave some indication that much inflammatory eye disease was actually non-infectious in origin. It was recognized then that non-infectious inflammatory eye disease would require an entirely different therapeutic approach.

Most of the immunosuppressants used in ocular inflammatory disease were originally developed for use in transplant medicine, rheumatic disease or other systemic inflammatory diseases. Very few have high level evidence for their use in ocular inflammation, and almost all are used off-label. A recent study which surveyed uveitis experts' approach to a number of clinical scenarios, found that whilst there was a general consensus on the overall approach to immunosuppression (i.e. starting with corticosteroids first line, and subsequent initiation of a steroid-sparing immunosuppressant) there was considerable variation in which second line agent to use and at what dose.

Ocular inflammatory disease is very heterogeneous group. Within uveitis, classification is generally by anatomical grouping and etiology, including the presence or absence of systemic disease. 'Splitting' i.e. defining a very pure cohort (e.g. Posterior uveitis of Birdshot pattern) leads to a very small target population, whereas

'lumping' enables easier recruitment but may be clinically meaningless due to the range of disease included. This is true both for routine clinical practice and for clinical trials. In clinical trials maximization of the signal: noise ratio is critical. An intervention may be highly effective for a disease but will fail to return a statistically significant benefit if it is trialed on too broad a group of patients (e.g. patients who have conditions that look superficially similar but differ fundamentally in etiology).