

Case Report

Chronic Anterior Uveitis: Diagnosing Primary Sjogren's

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Abstract

Chronic anterior uveitis is a rare ocular manifestation in primary Sjogren's syndrome and is not commonly reported. In our case report, we discuss how ophthalmological findings in a patient supported her being diagnosed with primary Sjogren's. Meticulous history taking, Anterior segment photography, FFA, serological examination, aid of a rheumatologist was taken. Following ophthalmic evaluation and extensive laboratory tests, patient was diagnosed as primary Sjogren's. Close monitoring and combination immunosuppressives were relevant in the treatment process.

Delhi J Ophthalmol 2021;32: 82-83; Doi <http://dx.doi.org/10.7869/djo.698>**Keywords:** Autoimmune Complexes, Chronic Anterior Uveitis, , Immunosuppressives, Primary Sjogren's

Introduction

Sjogren's syndrome is characterized by autoimmune inflammation of exocrine glands. The condition is classified as primary when it exists in isolation and secondary when associated with diseases such as rheumatoid arthritis, SLE, systemic sclerosis, mixed connective tissue disease, primary biliary cirrhosis, chronic active hepatitis and myasthenia gravis.¹ Primary Sjogren's syndrome affects females more commonly than males (9:1) and begins in 4th- 5th decade running a course over 8-10 years.²

The cardinal ocular sign in Sjogren's is keratoconjunctivitis sicca (KCS). The ocular manifestations are posterior blepharitis, meibomian gland dysfunction, punctate epithelial erosions in cornea and anterior uveitis. Posterior segment involvement is very rare.

Case report

A 43 year old woman presented with complaints of occasional redness and floaters and history of use of topical steroids since 15 years. She had a systemic history of hypothyroidism (started on medication recently) Her BCVA at presentation was 6/9 (RE) and 6/12 (LE). Her right eye anterior segment revealed granulomatous KP's , heterochromia, AC cells 3+, flare 2+, pseudophakia, no evidence of synechiae or iris nodules (Figure 1,2). She had cells in AVF and mild vitritis and no abnormality in the fundus. She was started on topical steroids , cycloplegics and given a battery of blood investigations. She had raised ESR and a positive ANA (1:1280 speckled pattern).

She was referred to a Rheumatologist who diagnosed her as a possible case of primary Sjogren's. Her SS-A, RO-52 and SSB antibodies were strongly positive. She was started on Deflazacort (6mg), Methotrexate (5mg), Hydroxychloroquine (200mg) and Folic acid and also advised lip biopsy. Her topicals were continued and tapered during relapsing episodes. Her dry eye work up revealed: Schirmers (I &II) <5 mm, corneal/conjunctival staining score, TBUT> 10s, Tear film height < 0.5ml, aqueous deficiency noted. She was started on cycloimmune topical and advised punctual plugging.

During her treatment, she had a recurrent attack after a quiescent phase of 6 months in the same eye. In the following months, Methotrexate was dosed upto 7.5mg/week. She had a relapse after 3 months with severe AC reaction along with mild vitritis. Her fundus during this episode revealed a few hypopigmented spots which showed no leakage on FFA. Her systemic medications at this point were, Hydroxychloroquine (300mg), Methotrexate (15mg), Deflazacort (6mg) and Folic acid. During her treatment at our facility for a period of 18 months, she has had relapsing episodes atleast 4 times, each episode treated for atleast 3 months.

Discussion

Primary symptoms of Sjogren's are a dry mouth and eyes. Other symptoms can include dry skin, vaginal dryness, chronic cough, numbness in the limbs, tiredness, muscle and joint pains and thyroid problems.

Our reported case was not a diagnosed patient of Sjogren's before being examined in our clinics for her ophthalmic condition. On probing into her personal history, she had only to account for frequent gritty sensation in eyes, hypothyroidism, peripheral tingling sensation and myalgia; symptoms which are vaguely suggestive of a possible connective tissue disorder. Our laboratory findings directed us to a provisional diagnosis and finally with the help of the rheumatologist we could give specific treatment to our patient.

Rosenbaum and Bennett described a series of eight patients reporting that in all cases uveitis was bilateral and chronic; anterior and posterior disease (but no chorioretinitis) with posterior synechiae, cataract and pars plana exudation being common.³ These signs are comparable in our patient except for unilaterality. Circulating immune complexes namely cryoglobulins and autoantibodies have been associated with both Sjogren and anterior uveitis. Other manifestations of immune complex-mediated disease include palpable purpura suggestive of leukoclastic vasculitis as described in a case by Bridges and Burns.⁴ In their article they have stressed the efficacy of combination of immunosuppressive therapy in treating acute conditions and close monitoring

of such cases. Cytopenia, uveitis, pericarditis, pleuritis, interstitial lung disease, vasculitis, monoclonal gammopathy of unknown significance and non-Hodgkin lymphoma only occurred in the patients without wide spread pain and high anti-SSB antibodies in primary Sjogren as noted by Borg and Kelder.⁵

Conclusion

Thorough history taking and close monitoring of cases of uveitis are very relevant in treating conditions with autoimmune etiology. Combination of immunosuppressives under proper guidance of a rheumatologist is the key to treating such refractory cases.

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