Commentary: Posterior scleritis: Nuances to discern and handle effectively!

Kumar *et al*. present the clinical profile of patients with posterior scleritis from eastern India in this issue of the Indian Journal of Ophthalmology.[1] In this commentary, I describe a few additional nuances to diagnose and manage this condition effectively.

Posterior scleritis as defined by Watson is inflammation of the sclera posterior to ora serrata that is severe enough to cause abnormalities in the normal anatomy of the posterior segment of the eye.[2] It has considerable overlap with the inflammation on the adjacent structures, namely periscleritis, sclerotenonitis, and pseudotumor. Pathophysiologically, the thickening and inflammation of the sclera causes impermeability to transscleral outflow leading to the signs observed in it.

When posterior scleritis is bilateral, it can be potentially misinterpreted with Vogt Koyanagi Harada's disease,[3] which classically presents as a panuveitis. However, associated anterior segment involvement is not uncommon in the posterior scleritis and does not rule the diagnosis out. Work up of the symptoms such as periocular pain that characteristically increases with ocular movements, blurred vision, headache, increased myopia, decreased hyperopia, and the subtle signs of congestion near the fornices of the conjunctiva are helpful to the astute clinician to suspect the diagnosis of posterior scleritis. The differentials of posterior scleritis such as malignant melanoma, choroidal hemangioma, uveal metastasis, pseudotumors, and orbital neoplasm can be lucidly delineated using the clinical and imaging techniques. In particular, the differentials of annular ciliochoroidal detachment and exudative macular detachment merit attention. In intraconar tumours,the chorioretinal folds are directed radial from the optic nerve. In extraconal tumours the folds are directed in a concentric fashion with convex side towards the optic nerve. But radial chorioretinal folds are seen in choroidal neovascularizations too.

The B-scan findings of posterior scleritis are retinochoroidal scleral thickening, T-sign, subtenon's fluid. They are not sine qua non for its diagnosis. Since posterior sclera is not easily clinically visualized, adjunct imaging modalities such as computerized tomogram and magnetic resonance imaging (MRI) are also helpful to rule out the differentials. MRI findings[4] includes the direct signs such as scleral enhancement, thickening, periscleral cellulitis ("ring sign" of Chaques) as well as the indirect signs such as choroidal/retinal detachments and the effusions in the suprachoroidal space needs clinical focus. It adds real value in delineating the mimics such as pseudotumors, melanoma, and lymphomas which reveals a restricted diffusion in diffusion-weighted imaging technique. Although the findings are not very unique of posterior scleritis, it is the fundus fluorescein angiogram and not indocyanine green angiogram that is recommended[5] in bilateral cases to rule out central serous chorioretinopathy and choroidal hemangiomas.[6] In the current scenario, enhanced depth imaging techniques of optical coherence tomograms (OCT) adds distinct topographical patterns and intralesional patterns[7] to delineate the differentials.

Once the diagnosis of posterior scleritis is established, evaluation of the associated systemic autoimmune conditions that had set the stage is vital to treat the eye as a part of the body systems. This helps in preventing the recurrences as well as to prognosticate it. The commonly associated autoimmune conditions are rheumatoid arthritis, systemic lupus erythematosus, and granulomatosis with polyangiitis. On the flip side, the clinician should understand that the rarity of the other autoimmune etiologies and the miscellaneous causes (such as viral infections, drugs, malignancies, monoclonal gammopathy, and myeloma) should never be a license to overlook them. When systemic steroids are initiated early, before ordering the workup of autoimmune conditions, the resultant laboratory values can potentially yield falsely negative results. Hence, ordering autoimmune workup should ideally be done before adding the steroids. This needs to be kept in mind before ruling them out as idiopathic. It is important to realize that autoimmune conditions is have a distinct spectrum of involvement in different stages of evolution[8] and a sound clinician should not hesitate to repeat the tests especially during recurrences.

Sainz De La *et al*.[8] proposed the degree of involvement of scleritis both anterior and posterior as an excellent prognosticator. It merits considering in practice. This can be subjectively done based on the accurate documentation of the quadrants of involvement of the scleritis, both anterior and posterior. However, the objective anterior segment OCT based grading of scleritis[9] adds real value to the anterior scleritis. The various armamentaria of treatment of noninfectious scleritis are nonsteroidals, steroids in various routes, immunosuppressants (sans mycophenolate[3] due to higher recurrences) to biologicals[3] now. At the onset, very aggressive conditions can be tackled by adding parenteral steroids with monitoring of comorbid conditions such as diabetes and hypertension. Before embarking on the accurate titration of inflammation, ruling out the common infectious etiologies as tuberculosis, syphilis is crucial. In the developing world for cases of tubercular scleritis, when oral steroids are given without anti-tubercular drugs can potentially worsen it. Most of these oral immunosuppressants have their onset of action after a lag period of establishing an equilibrium in the body system after 3–6 weeks. Hence, the titrated addition of steroids in the different routes in the initial period is a fundamental concept in the management plan.

Early diagnosis of posterior scleritis is essential for it has a higher potential to be misdiagnosed with its differentials. Accurate titration of inflammation promptly using appropriate anti-inflammatory medications is the key element for treatment success. As the treatment is simpler than the diagnosis, ruling out the differentials using adjunct imaging modalities is crucial. What is intriguing is that during follow-up, the retinal striae may rarely persist[6] despite clinical resolution!

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