Commentary: Presumed tubercular posterior scleritis - What is our understanding so far?

In this issue of Indian Journal of Ophthalmology, Agarwal and Majumdar have discussed a challenging case of a 47-year-old female who presented with optic disc edema, suprachoroidal and subretinal fluid, and multiple choroidal lesions involving one eye. Based on a positive Mantoux test, and computerized chest tomography (CT-scan) findings of enlarged mediastinal lymph nodes, tubercular etiology was considered, and anti-tubercular therapy (ATT) with corticosteroids were initiated.[1] Prior to this case report, only four cases of posterior scleritis with presumed tubercular etiology have been published, which have been eloquently reviewed by the authors.[2-5]

There are several similarities in the index case and the previously published cases (total 5 published cases now). Totally, 4 out of 5 eyes had presence of disc edema, choroidal folds/fluid, and suprachoroidal fluid (there was no view of the posterior segment in one case). While these features are usual in all cases of posterior scleritis, certain findings such as presence of deep choroidal lesions, subretinal abscess, intraocular mass were very unique in these cases.[1] As ophthalmologists who face challenging cases every day, we can assume that these clinical features if present in our future patients with posterior scleritis, could be likely indicators of tubercular etiology. An extensive search for tuberculosis may be warranted in these cases with atypical features. In order to determine these pathological manifestations, it is imperative to perform good quality imaging using fluorescein as well as indocyanine green angiography, ultrasonography, optical coherence tomography (especially enhanced-depth imaging), and other such tools.[6]

Another interesting aspect of tubercular posterior scleritis is the high rate of positive test results for detection and isolation of acid-fast bacilli albeit from a remote tissue (such as cervical and mediastinal lymph nodes) (one case had positive acid-fast bacilli from an enucleated sample). Thus, 3 out of 5 cases had tissues amenable to biopsy and histopathology and all of them demonstrated mycobacteria conclusively. So, the question is – do patients with infective scleritis (tubercular in this context) have a high systemic mycobacteremia? The series of presumed tubercular scleritis from Japan may support this hypothesis, since their patients responded to ATT alone without the need for immunomodulatory therapy.[7] Thus far, there is no literature that quantifies the systemic mycobacterial load and correlates the findings with clinical manifestations including scleritis, simply because of the rarity of the disease. A learning message from this analysis is that in patients with posterior scleritis with high suspicion of tuberculosis, an aggressive search for acid fast bacilli, or mycobacterial DNA using nucleic acid amplification techniques could prevent misdiagnosis and

help in timely initiation of ATT. This is exemplified by the report from Velasco de Cruz *et al*. whose patient did not improve with corticosteroids and 8 years of immunosuppression, until finally enucleated specimen demonstrated mycobacteria.[5]

Immunological tests such as Mantoux and interferon release assay provide strong evidence of tubercular etiology in patients with intraocular tuberculosis. Imaging of the chest (preferably contrast-enhanced CT scan rather than plain chest radiography) are extremely important for evaluating presence of primary Ghon's focus. In addition, radiological tests provide an opportunity to assess lymph nodes and/or pleura/pleural fluid that may be amenable to advanced laboratory testing.[8,9]

In summary, posterior scleritis is a rare uveitic entity and there are numerous challenges in reaching the correct diagnosis. The ophthalmologist has to consider various possibilities and collaborate closely with internists, pulmonologists, general surgeons, radiologists, pathologists, and immunologists to be able to manage these patients successfully. The authors, Agarwal and Majumdar,[1] must be congratulated for highlighting this case that adds value in the literature. Their multimodal imaging approach with fluorescein and indocyanine green angiography, and ultrasonography was extremely relevant to the case for understanding the true extent of ocular involvement.

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