

## From the editor's desk — Vasculitis my brief synopsis

Greetings from the desk of your editor. I hope everyone likes the additions in the Indian Journal of Ophthalmology (IJO), its applications on mobiles and e-books, and the new dimension it adds to the usability of our esteemed journal. I hope the readers are making use of it and are finding it helpful while on the move. I have termed it as 'IJO on the go' and this is especially for the youngsters, as you are the torch bearers of this ever-changing and futuristic branch.

I would like to take this opportunity to highlight retinal vasculitis in the Indian context. Vasculitis in its literal terms is inflammation of the retinal vasculature.<sup>[1]</sup> Our classical teachings tell us that either it is infective or autoimmune,<sup>[2]</sup> an isolated ocular syndrome or a component of systemic vasculitis. Evaluation in terms of history taking should be thorough and a review of the organ systems should be carried out. If the detailed history is negative then only a limited number of tests should be ordered. The 'Shotgun approach' is both diagnostically unrewarding and expensive on the patient's pocket. If a specific infectious agent is identified, then a specific antimicrobial therapy is warranted.<sup>[3]</sup>

In India, when we think of vasculitis, tuberculosis (TB) comes to our minds as the first probable diagnosis. Many studies on vasculitis have implicated tuberculosis as a cause of retinal vasculitis. The article by Saurabh *et al.*, in this issue of IJO, has clearly brought out a different presentation and causation of vasculitis in Eastern parts of India. This particular study also points out the Mantoux test positivity in these cases, although diagnosis of TB was not proven, or they were cases of healed TB. The true prevalence of tubercular vasculitis remains a major concern, especially in TB-endemic areas, because of lack of definite diagnostic criteria. TB can affect any part of the eye and patients present with a spectrum of clinical signs. Intraocular tuberculosis unlike pulmonary and gastrointestinal tuberculosis is most likely to be a part of post primary infection, most likely a granulomatous hypersensitivity response that results from persistent intraocular microorganisms that the cell is unable to destroy. It is still speculative whether vasculitis is due to active infection or reflects a granulomatous response.<sup>[2]</sup>

Although it may mimic other clinical entities, a positive tuberculin skin test, healed lesions on chest X-ray, or associated systemic TB, it corroborates the diagnosis of presumed intraocular TB. Administration of anti-tubercular therapy (isoniazid 5 mg / kg / day, rifampicin 450 mg / day if body weight is < 50 kg and 600 mg / day if body weight is > 50 kg, ethambutol 15 mg / kg / day, and pyrazinamide 25 to 30 mg / kg / day initially for three to four months; thereafter, rifampicin and isoniazid are used for another nine to fourteen months, in addition to the standard corticosteroids, significantly reducing the recurrence of vasculitis in these patients. The recently introduced immune-based rapid blood tests [QuantIFERON-TB Gold test (QFT-G) and TSPOT (TB test)] seem to be a significant upgrade of the century-old tuberculin skin test, for diagnosing latent TB infection. QFT-G assay measures the amount of interferon-gamma (IFN- $\gamma$ ) released by the patient's sensitized T-cells, when his whole blood is incubated with two synthetic peptide antigens ESAT-6 and CFP-10. These antigens are present in *Mycobacterium tuberculosis*, but not in the Bacilli-Calmette-Guerin (BCG) or atypical mycobacteria. The advantages of these tests over the routine tuberculin skin test are that these are not affected by the previous BCG vaccination or atypical mycobacteria, the results are available within 24 hours, without any need for a second visit to the hospital, and they are free from any booster effect.<sup>[4,5]</sup> The major limitation of these tests is that they are expensive.<sup>[6]</sup>

Recent studies have speculated that a combination of Mantoux test and QFT-G or TSPOT may be more specific and sensitive. Polymerase chain reaction (PCR) testing in aqueous and vitreous tap has also been shown to diagnose tuberculosis, in many recent reports.<sup>[2]</sup>

Shifting our focus from tuberculosis vasculitis to the management of vasculitis in general and non-infectious vasculitis, we need to determine whether the patient requires interventional therapy. If the patient's visual acuity is near 20 / 40 or better, and is not bothered by vitreous opacities, has mild vascular changes, has none to minimal cystoid macular edema, and the disease process does not appear to be progressing, one may observe. Despite the advancement and use of new immunosuppressives, corticosteroids remain the mainstay of therapy. If vasculitis does not respond to corticosteroids, if the disease becomes resistant, or if unacceptable side effects develop, then one may have to use immunosuppressive drugs such as alkylating agents, antimetabolites or Cyclosporin A. Fulminant cases of retinal vasculitis may require a combination of drugs. Some subset of patients with retinal vasculitis (e.g., Behcets disease, Eales disease) may develop retinal ischemia secondary to capillary destruction. These patients should be closely watched for the development of retinal neovascularization. Panretinal or sectoral photocoagulation may be required in such a setting. Surgical Intervention in the form of vitrectomy, may be required for secondary complications of retinal vasculitis.<sup>[3]</sup> Newer modalities like Intravitreal Dexamethasone implants<sup>[7]</sup> and anti-vascular endothelial growth factors (VEGFs) are being used for complications of retinal vasculitis like neovascularization, with success, but larger studies are needed for a definite insight into the clear-cut management protocols.<sup>[8]</sup>

EDITORS PEARLS: Here I would like to highlight the issue pertaining to plagiarism; in other words use of other individuals published work or unpublished ideas without attribution. As there is such a vast pool of easily accessible data available on the

web, an author tends to use it for enhancing productivity. Sometimes plagiarism is committed by authors whose first language is not English. In that case they should seek help and not resort to using the words of others.<sup>[9]</sup>

Till then, I request everyone to consistently keep IJO as their first choice for publishing.

**S. Natarajan**

Editor, Indian Journal of Ophthalmology,  
Chairman and Managing Director, Aditya Jyot Eye Hospital Pvt. Ltd.,  
Wadala (W), Mumbai, Maharashtra, India.  
E-mail: editor@ijo.in

## References

1. Stanford MR, Graham EM. Systemic associations of retinal vasculitis. *Int Ophthalmol Clin* 1991;31:23-33.
2. Gupta A, Gupta V, Arora S, Dogra MR, Bambery P. PCR –positive tubercular retinal vasculitis: Clinical characteristics and management. *Retina* 2001;21:435-44.
3. George RK, Nussenblatt RB. Treatment of Retinal Vasculitis. *Ophthalmol Clin North Am* 1998;11:637-80.
4. Itty S, Bakri SJ, Pulido JS, Herman DC, Faia LJ, Tufty GT, *et al.* Initial results of QuantiFERON-TB Gold testing in patients with uveitis. *Eye* 2009;23:904-9.
5. Brogden P, Varma A, Backhouse O. Interferon-gamma assay in tuberculous uveitis. *Br J Ophthalmol* 2008;92:582-3.
6. Sudharshan S, Ganesh SK, Biswas J. Current approach in the diagnosis and management of posterior uveitis. *Indian J Ophthalmol* 2010;58:29-43.
7. Myung JS, Aaker GD, Kiss S. Treatment of noninfectious posterior uveitis with dexamethasone intravitreal implant. *Clin Ophthalmol* 2010;4:1423-6.
8. Chanana B, Azad RV, Patwardhan S. Role of intravitreal bevacizumab in the management of Eales disease. *Int Ophthalmol* 2010;30:57-61.
9. Hall GM. In *How to write a paper*. 3<sup>rd</sup> Ed. New Delhi, India: Byword Viva Publishers Private Limited; 2004. p. 153-4.

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