Commentary: Clinical pearls and pitfalls in diagnosing viral anterior uveitis

We congratulate the authors on putting together a comprehensive review article that elaborately covers most aspects of viral anterior uveitis.[1] This manuscript should serve as a "ready reckoner" for all ophthalmologists who wish to gain information regarding diagnosis and management of patients with this enigmatic uveitic entity. We highlight a few points that would add to the vast ocean of knowledge on viral anterior uveitis that this article encompasses:

1. While dealing with various causes of hypertensive anterior uveitis, importance of a thorough slit-lamp examination cannot be underestimated as presence/absence of subtle features helps to clinically elucidate the etiology. Absence of ciliary congestion with high intraocular pressure (IOP) is seen in both Posner–Schlossman syndrome (PSS) and Fuchs uveitis syndrome (FUS). When IOP and corneal edema (generally epithelial) are out of proportion to ciliary congestion in a patient with acute and recurrent unilateral granulomatous/non-granulomatous anterior uveitis with minimal anterior chamber reaction, a diagnosis of PSS can be made with reasonable certainty.[2] FUS has been termed as a microgranulomatous uveitis, with stellate keratic precipitates all over the corneal endothelium and presence of Koeppe's and Busacca's nodules (less commonly). Absence of ciliary congestion, minimal anterior chamber reaction, diffuse iris atrophy, absence of posterior synechiae, early posterior subcapsular cataract formation, and presence of vitreous membranes are characteristic findings which aid in differentiating FUS from other granulomatous anterior uveitic entities that warrant specific workup and targeted management[3]
2. While examining a case of viral anterior uveitis, a dilated fundus examination with an indirect ophthalmoscope is mandatory so as not to miss peripheral lesions of acute retinal necrosis. Necrotizing viral retinopathies may be preceded by episcleritis/scleritis, periorbital pain, and/or anterior uveitis in about one-third cases[4]
3. One of the recently reported causes of viral anterior uveitis with keratitis is mumps. Clinical features include intense photophobia, corneal edema with Descemet's folds, and severe endotheliitis with high IOP. Rapid decrease of corneal endothelial cell density is generally noted after resolution of corneal edema[5]
4. Brimonidine should be used with caution to treat high IOP in patients of viral anterior uveitis. Brimonidine may cause granulomatous anterior uveitis which may mimic recurrence of viral anterior uveitis. This side-effect may present with or without symptoms, usually many months after initiation of treatment. It may occur only in unilaterally and is fully reversible once brimonidine is withdrawn[6]
5. Resistance of herpes simplex virus (HSV) to acyclovir and cytomegalo virus (CMV) to ganciclovir is a recently reported phenomenon. HSV resistance to acyclovir occurs due to specific mutations in the viral thymidine kinase, mutations in the viral DNA polymerase gene, HSV having a deficient thymidine kinase protein or the natural hypervariability of HSV thymidine kinase gene. Acyclovir resistant HSV strains also show some amount of cross-resistance to ganciclovir which has implications in management of these patients. Use of cidofovir and foscarnet is recommended to treat acyclovir resistant HSV anterior uveitis.[7]

Ganciclovir-resistant CMV occurs due to mutation in the UL97 gene that codes for phosphorylation of CMV viral kinase. Management of patients with ganciclovir resistant CMV is recommended with high dose ganciclovir, foscarnet, letermovir, leflunomide, and CMV-immunoglobulin.[8]

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Conflicts of interest

There are no conflicts of interest.

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