

Acute Retinal Necrosis After Intravitreal Triamcinolone Acetonide For Aphakic Macular Edema- A Case Report

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ABSTRACT

Triamcinolone acetonide is used in the form of intravitreal injection to treat ocular diseases like uveitis, vasculitis, proliferative vitreoretinopathy, macular degeneration etc. But complications have been reported after injection like elevated intraocular pressure and cataract. Even though rare, acute retinal necrosis is a risky complication after intraocular Triamcinolone acetonide injection, which may cause potential blindness. Here we report a case of acute retinal necrosis following intravitreal injection of Triamcinolone acetonide and review the relevant literature.

Keywords: Acute retinal necrosis, Intraocular injection, Triamcinolone acetonide.

Introduction

Triamcinolone Acetonide (TA), a water-insoluble corticosteroid is widely used in the field of ophthalmology. It can be injected either under tenon's capsule, into the retrobulbar space or directly into the vitreous. Machermer used for the first time intravitreal injection Triamcinolone Acetonide (IVTA) to prevent cellular proliferation after retinal detachment surgery[1]. Recent studies showed the use of IVTA in various ophthalmologic conditions like uveitis, vasculitis, proliferative vitreoretinopathy, macular degeneration, clinically significant diabetic macular edema (CSDME), pseudophakic/aphakic cystoids macular edema etc. These effects are due to the anti-inflammatory, anti-angiogenic, and anti-permeability properties of corticosteroids[2-4]. Many complications have been reported after IVTA which include elevation of intraocular pressure, progression of cataract, Acute Retinal Necrosis (ARN), retinal hemorrhage, retinal detachment, and endophthalmitis etc. Many case reports have shown the local immunomodulatory effects after IVTA may cause viral retinitis, therefore close observation is obligatory[5-8]. Various studies have shown that intraocular corticosteroids might reduce the ocular immune response resulting in either a primary infection or a latent viral reactivation. On a

whole intraocular steroids will cause localised immunosuppression. In immunocompromised patients, necrotizing retinitis may be usually caused by varicella zoster virus (VZV) or herpes simplex virus (HSV) and may lead to loss of vision. Hence, timely diagnosis and treatment is mandatory to prevent vision loss. But still there is a lack of definitive proof of periocular or intraocular corticosteroids causing viral retinitis[9-11]. Here we report a case of Acute Retinal Necrosis (ARN) following IVTA and review the relevant literature.

Case Report

A 65 years old female reported to retina clinic with sudden painless loss of vision since 2 days. She underwent cataract surgery 3 months back. Intraocular lens was not implanted due to posterior capsule rent. As she was not improving even with spectacle correction, she was referred to retina clinic by general ophthalmologist. On examination right eye showed immature senile cataract and left eye showed aphakia, her 'Best Corrected Visual Acuity' (BCVA) in right eye was 6/18. Right eye fundus was normal, but left eye fundus showed macular edema. Fundus fluorescein angiography revealed flower petal pattern, Optical coherence tomography (OCT) showed intra retinal cystic spaces with diffuse retinal thickening. A diagnosis of aphakic cystoid macular edema was given and IVTA was given 0.1 ml /4 mg. 4 weeks after injection patient vision improved to 6/24 with + 10 Dsp, secondary Scleral fixated intraocular lens

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(SFIOL) was planned. Before secondary Intraocular lens (IOL) implantation patient came with loss of vision, on examination her left eye showed anterior chamber flare, fundus was hazy due to mild vitritis and wide confluent areas of yellow white necrotic lesions with superficial haemorrhages in the periphery was noted (Fig 1 to 5).

Uveitic and systemic workup was normal. HIV 1 and 2 results were non reactive. Anterior chamber paracentesis for viral PCR was performed. The patient was clinically diagnosed as ARN based on the typical clinical presentation, but did not have confirmatory PCR. Although the causative virus was not detected, patient was advised intravitreal anti virals with systemic steroids after physician clearance.

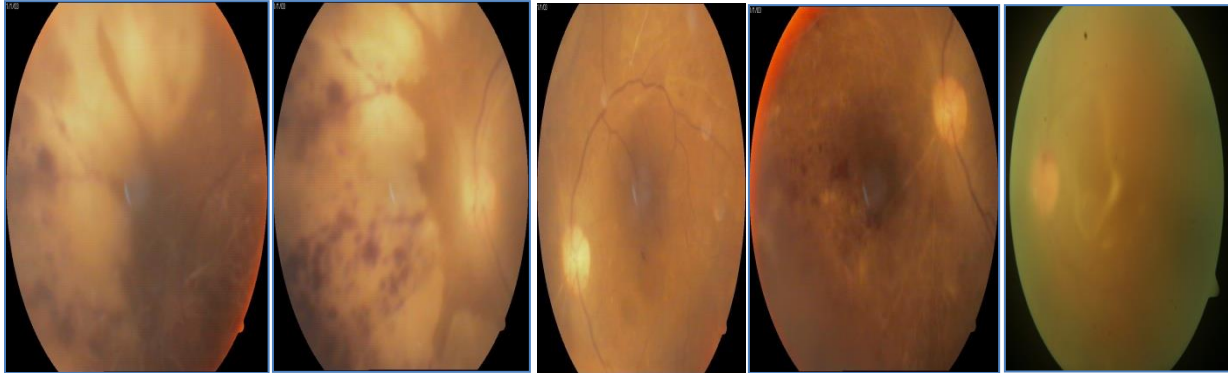


Fig 1: Fundus image showing vitritis with confluent areas of yellow white necrotic retina

Fig 2: Fundus image showing haemorrhagic necrosis of retina

Fig 3: Fundus image after treatment showing resolving vitritis with occlusive vasculopathy

Fig 4: Fundus image showing resolving haemorrhagic necrotic retina.

Fig 5: Fundus image 1 week after surgery, Silicone oil filled globe with attached retina

After 3 weeks, patient presented with tractional retinal detachment involving posterior pole one month after intra vitreal gancyclovir. Patient was explained about the prognosis regarding further management, pars plana vitrectomy with silicone oil tamponade was done. Patient was advised to continue anti virals for 4 weeks with tapering dose of steroids.

Discussion

Very few reports have described the occurrence of ARN after injection of corticosteroids. **Toh and Borthwick** reported that corticosteroids reduce the eye's immune response thereby increasing the risk of infection or reactivation of dormant organisms. Urayama et al (1971) first reported ARN and is most commonly seen in young immunocompetent patients, but in our case the patient was 65 years old [10-12]. Various clinical manifestations of ARN include anterior uveitis, vitritis, necrotizing retinitis, retinal vasculitis and optic disc involvement with mild to moderate hypermia. Usually the delay in diagnosis of ARN is due to the condition being overlooked as another uveitic entity and also the lack of use of molecular diagnosis. This delay may lead to complications resulting in blindness. Poor vision usually is due to rhegmatogenous retinal detachment or

optic neuritis [11-13]. Risk factors for ARN include young age, history of neurosurgery, periocular trauma, compromised immune status, Administration of local immunosuppressives in the vitreous, and a history of viral retinitis [12-14]. In our case patient was administered IVTA. **Ankur** et al found a half life of 113 days of 20 mg of IVTA in the vitrectomized eye.¹⁵ Whereas Beer et al found a half-life of 18.6 days and 3.2 days in non vitrectomized and vitrectomized patients respectively after administration of standard 4 mg dose of IVTA. They suggested that IVTA may cause the risk of long-lasting local immunosuppression and vulnerability to primary infection or reactivation of endogenous latent viral retinitis. In our case TA dose was only 4 mg and we found that lower dose can also cause immunosuppression and induce viral retinitis [14,15].

Bacterial endophthalmitis, is also a serious complication of IVTA. In our case typical exudates in vitreous as well as in anterior chamber and RCS thickening were not noted. CMV retinitis also excluded by aqueous chamber PCR sample, in CMV retinitis lesions were located along the retinal blood vessels and sometimes on fovea and disc[14-16].

Few reports have shown that complications like immune choroiditis developed as late as 34 years after first diagnosis of eye involvement in ARN. Hence it is vital to distinguish a purely inflammatory, reactive choroiditis and an infective etiology. The choroidal involvement might be due to the granulomatous infiltration of the choriocapillaris and choroid in ARN as also seen in sympathetic ophthalmia and Vogt Koyanagi Harada syndrome. Hence, all case of unilateral ARN have a risk of developing ARN in other eye and also choroiditis[16,17].

Management of ARN should include a combination of IVTA and anti virals. Few authors however prefer oral corticosteroids. In our case systemic steroids were given to reduce the inflammation, not IVTA, thinking that IVTA was the cause of the ARN[15-17].

Hence IVTA must be judiciously used, as it may cause few complications. We recommend judicious use of IVTA and monitoring of these injected patients as there is a possibility of ARN.

Conclusion

We report a case of ARN after IVTA in a 65 year old female patient. We recommend that physicians should be aware of the possibility of necrotizing herpetic retinopathy after IVTA, which may lead to blindness. The combined findings of severe posterior segment inflammation along with peripheral retinal whitening in a patient of unknown immune status must alert ophthalmologists a likelihood of underlying viral infection. Early diagnosis and timely treatment using antiviral agents may save these patients from loss of visual acuity.

References

1. Machemer R, Sugita G, Tano Y. Treatment of intraocular proliferations with intravitreal steroids. *Trans Am Ophthalmol Soc.* 1979;7:171-8.
2. JM Han et al. Necrotizing Viral Retinitis after IVTA. *Korean J Ophthalmol* 2011;25(6):451-454.
3. Jonas JB, Kressig I, Kampeter B, Degenrin RF. Intravitreal triamcinolone acetonide for the treatment of intraocular oedematous and neovascular diseases. *Ophthalmology.* 2004;101:113-20.
4. Gillies MC, Larsson J. The effect of intravitreal triamcinolone on foveal edema in exudative macular degeneration. *Am J Ophthalmol.* 2007;144:134-5.
5. Sekiryu T, Iida T, Kaneko H, Saito M. Cytomegalovirus retinitis after intravitreal triamcinolone acetonide in an immunocompetent patient. *Jpn J Ophthalmol* 2008;52:414-6.
6. Park YS, Byeon SH. Cytomegalovirus retinitis after intravitreal triamcinolone injection in a patient with central retinal vein occlusion. *Korean J Ophthalmol* 2008;22:143-4.
7. Tugal-Tutkun I, Araz B, Cagatay A. CMV retinitis after intravitreal triamcinolone acetonide injection in a patient with Behcet's uveitis. *Int Ophthalmol* 2010;30:591-3.
8. Sonmez K, Ozturk F. Complications of intravitreal triamcinolone acetonide for macular edema and predictive factors for intraocular pressure elevation. *Int J Ophthalmol* 2012;5(6):719-725.
9. Shah AM, Oster SF, Freeman WR. Viral retinitis after intravitreal triamcinolone injection in patients with predisposing medical comorbidities. *Am J Ophthalmol* 2010;149:433-40.
10. Chang S, Weissgold DJ, Singer JA, Sobrin L. Acute Retinal Necrosis following Intraocular Triamcinolone Acetonide Injection. *Retin Cases Brief Rep.* 2010;4(4):306-308.
11. McGrath L, Woods M, Lee L, Conrad D. Acute retinal necrosis (ARN) in the context of neonatal HSV-2 exposure and subconjunctival dexamethasone: case report and literature review. *Digit J Ophthalmol.* 2013; 19(2):28-32.
12. Toh TY, Borthwick JH. Acute retinal necrosis post intravitreal injection of triamcinolone acetonide. *Clin Experiment Ophthalmol.* 2006;34 :380-382.
13. Takakura A et al. Viral Retinitis following Intraocular or Periocular Corticosteroid Administration: A Case Series and Comprehensive Review of the Literature. *Ocul Immunol Inflamm.* 2014;22(3):175-182.
14. Tsinopoulos I, Tranos P, Topouzis F, Hundal K, Brazitikos P. Atypical acute retinal necrosis (ARN) responding to late treatment. *Eye* 2004; 18: 203-204.
15. Ankur M. Shah, Stephen F. Oster, William R. Freeman. Viral Retinitis Following Intravitreal Triamcinolone Injection in Patients with Predisposing Medical Comorbidities. *Am J Ophthalmol.* 2010; 149(3):433-40.e1.

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16. Jeong Mo Han, Jeeyun Ahn, Kyu Hyung Park, Se Joon Woo. Presumed Necrotizing Viral Retinitis after Intravitreal Triamcinolone Injection: Case Report. *Korean J Ophthalmol* 2011;25(6):451-454.
17. Roger Wong, Elizabeth M. Graham, Moin Mohamed, R. Stanford. Immune Choroiditis Following Contralateral Acute Retinal Necrosis. *Arch Ophthalmol* 2010;128 (10): 1364 - 1366.

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