Society for Clinical Electrophysiology of Vision (ISCEV) standards showed moderately reduced rod and scotopic cone amplitudes OD and mild photopic cone dysfunction OU, manifest primarily as reduced 30 Hz flicker responses OD and delayed cone implicit times OU. The widespread, asymmetric photoreceptor dysfunction is consistent with those seen in outer retinopathies such as AZOOR. No retinal autoantibodies were detected by western blot. HLA testing was not performed.

Six months later, the visual fields were unchanged and acuity remained unaffected. No ocular or systemic therapy has been initiated.

COMMENT

In this patient with intermediate uveitis (pars planitis), the clinical and electrophysiological findings also suggest the additional diagnosis of AZOOR, which presents with bilateral asymmetric progressive field loss, photopsia with scotoma, occasional retinal pigment epithelial changes and global outer photoreceptor/retinal pigment epithelium abnormalities on ERG.[1 2] Other causes of retinopathy, both genetic and acquired (infectious, autoimmune and inflammatory) were not supported by our evaluation.

To our knowledge, AZOOR has not been reported in association with pars planitis. AZOOR frequently presents in isolation, but it is well documented that ocular idiopathic inflammatory conditions may precede the clinical and electrophysiological features that characterise the disorder.[2] The pathogenesis of AZOOR remains obscure; circumstantial evidence supports an immune-related cause.[3] However, auto-antibodies are not consistently identified and the condition shows no tendency to improve with immunomodulatory therapy. There is also no clear evidence for genetic association between the clinical entities described as the AZOOR complex.

Pars planitis is an intraocular inflammatory disorder also of unknown aetiology that shares features with other uveitides previously documented to precede AZOOR. Additionally, pars planitis is associated with other diseases in which autoimmunity is recognised, for example multiple sclerosis.[4] These observations suggest a common disease process that leads to the subsequent development of outer retinopathy.

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Short-term intraocular pressure trends following intravitreal injections of ranibizumab (Lucentis) for the treatment of wet age-related macular degeneration

Rosenfeld and colleagues reported that ranibizumab (Lucentis) had no long-term effect on intraocular pressure (IOP) as determined by monthly measurements during the 2-year MARINA study.[1] We are unaware of previous studies examining the short-term effect on IOP by intravitreal ranibizumab in patients with wet age-related macular degeneration.

METHODS

We conducted a retrospective chart review of 50 ranibizumab injections from 23 patients. IOP was recorded using Goldmann applanation tonometry. All patients included in this series received ranibizumab injection as directed by the instructions provided in the package insert (volume 0.05 mL). Prior to injection, the mean baseline IOP was 14.04 (SD 3.16) mm Hg with a range of 9–22 mm Hg (n = 50). Thirty minutes after injection, the mean IOP was 21.10 (4.74) mm Hg with a range of 10–31 mm Hg (n = 50). At 1-week follow-up, the average IOP was 13.94 mm Hg (n = 19). The 2-week follow-up IOP averaged 12.5 mm Hg (n = 6). At the 4-week follow-up visit, the average IOP was 13.75 mm Hg (n = 37). The difference between the preinjection IOP and the IOP measured at all follow-up visits was not statistically significant (p = 0.297).

None of the 50 eyes experienced an IOP rise to greater than 31 mm Hg as measured 30 min after ranibizumab injection. Following the injection, none of the 50 eyes required IOP-lowering drops or anterior chamber paracentesis to lower IOP.

COMMENT

After intravitreal injections of ranibizumab, patients typically wait in the clinic another 30 min to have the IOP checked. Our series reveals that intravitreal injection of ranibizumab results in an expected mild transient elevation of IOP immediately after injection. However, the IOP normalises to baseline soon after without employing therapy for IOP control. We therefore challenge the need for patients to have IOP checked after ranibizumab injection and leave this decision to physician discretion. Shorter clinic visits translate into improved allocation of resources for both patients and physicians. It is reassuring that IOP returns to baseline at the 1-week, 2-week and 4-week follow-up visits. In such cases where a patient is unable to return to their follow-up visit, our data reassure that IOP theoretically should not remain elevated.

In summary, our limited series reveals that ranibizumab injection is safe in terms of elevation of IOP in a short-term setting. Anterior chamber paracentesis immediately after injection is not recommended, as this procedure has a risk of lens damage and a theoretical risk of increasing the chance of infection. The MARINA study also shows that monthly ranibizumab injections are safe in terms of its effect on long-term IOP.[1]

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CORRECTION

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