Optical coherence tomography and fundus fluorescein angiography of a case of macular toxicity from intravitreal amikacin

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DESCRIPTION

A 78-year-old man presented with a painful red right eye 9 days after uncomplicated phacoemulsification with intraocular lens implant. He had no relevant ocular or systemic history. Postoperative

subconjunctival cefuroxime had been given at the end of the procedure.

On current presentation, he had a best corrected vision of 6/12. He had a hypopyon with a dense fibrinous membrane. He was treated as having

Figure 1 Series of optical coherence tomography (OCT) in chronological order showing the macular infarct and corresponding OCTs.

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Figure 2 Fundus fluorescein angiography images showing the hypofluorescent areas of macular infarction and capillary occlusion. There is a large hypofluorescent masking defect corresponding to the infarct, with scattered specks of dense hypofluorescence. The macular capillary arterioles are occluded with some staining of the walls giving a 'pruned off' appearance together with non-filling of some adjacent venules.

postoperative bacterial endophthalmitis and an anterior chamber and vitreous tap were performed. He was given 0.4 mg/0.1 mL of amikacin and 1 mg/0.1 mL of vancomycin as per departmental protocol. Microbiology results did not reveal any causative organism.

On day 1 after the intravitreal injection, his vision dropped to counting fingers.

Fundus images and optical coherence tomography (OCT) (figure 1) showed an area of white macular infarct with perifoveal swelling. Over the next 5 days, this swelling resolved. However, the retinal whitening increased in intensity with no recovery of vision. The whitening slowly reduced in size and intensity over time, although at day 75, the patient was noted to

have cystic intra-retinal pockets—but this resolved within 2 weeks. At 5 months, the fluid had mostly settled, with a normal macular colour, and the patient's vision was 6/60.

The fluorescein angiogram (figure 2), performed at 19 days post injection, confirmed the diagnosis and showed compromised vasculature.

Aminoglycosides have been known to be linked with retinal toxicity.^{1–3} Although this is a rare event, certain departments have switched to using ceftazidime instead of amikacin, to avoid any risk of retinal toxicity.¹ Some theories regarding the mechanism suggest dilution errors, raised intraocular pressure postinjection, variations in vitreous concentration, or the supine position of the patient following an injection.^{1 2}

Although similar cases have been reported, this is a well-documented case showing the evolution of the infarct via OCT and fundus fluorescein angiography images in the first 3 months.

Learning points

- ▶ Amikacin, although known to cause retinal toxicity even in small doses, is still one of the drugs of choice in patients with exogenous endophthalmitis in some units. We recommend the use of ceftazidime, as it has a better safety profile.
- ▶ Although the exact mechanism is unclear, the images here capture the natural evolution of the retinal pathophysiology following amikacin toxicity.
- ▶ We wish to share these images to be used as references for future cases of patients suspected of amikacin toxicity.

Competing interests None declared.

Patient consent Obtained.

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