Case Report

Postoperative *Nocardia* Endophthalmitis and the Challenge of Managing with Intravitreal Amikacin

Sikander A. K. Lodhi,¹ G. Ashok Kumar Reddy,² and C. Aruna Sunder¹

¹Sarojini Devi Eye Hospital, Hyderabad 500028, India

²GHR Micro Diagnostics, Dwarakapuri Colony, Hyderabad 500082, India

Correspondence should be addressed to Sikander A. K. Lodhi; sikanderlodhi@gmail.com

Received 24 December 2015; Revised 15 February 2016; Accepted 16 February 2016

Academic Editor: Stephen G. Schwartz

Copyright © 2016 Sikander A. K. Lodhi et al. This is an open access article distributed under the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original work is properly cited.

Nocardia is a rare cause of delayed onset postoperative endophthalmitis after cataract surgery and it usually carries a guarded visual prognosis. **Purpose.** To highlight the clinical presentation, microbiological profile, and treatment outcome in a case of nocardial endophthalmitis after manual small incision cataract surgery. **Methods.** This case report highlights the typical features of *Nocardia* endophthalmitis, which presented six weeks after undergoing small incision cataract surgery. The case was managed by pars plana vitrectomy with intravitreal antibiotics. Intravitreal amikacin was used based on microbiologic work-up. **Results.** The endophthalmitis part was controlled but the case developed amikacin induced macular infarction which jeopardized a good visual outcome. **Conclusion.** *Nocardia* endophthalmitis manifests late after cataract surgery in an aggressive manner and carries a poor visual prognosis. An early diagnosis and the use of correct antibiotic regimen may salvage the vision. But the present case shows that one should always be wary of potential retinal toxicity with intravitreal amikacin.

1. Introduction

Nocardia are aerobic, Gram-positive, weakly acid fast filamentous bacteria and are a rare cause of endophthalmitis. Postoperative nocardial endophthalmitis is a delayed onset infection after cataract surgery and it usually carries a guarded visual prognosis. Nocardial endophthalmitis can occur in both immunocompromised and immunocompetent hosts. Most of these cases are reported from south India [1–5].

We report a case of postoperative *Nocardia* endophthalmitis in an immunocompetent individual. The infection was successfully managed by vitrectomy and intravitreal antibiotics but the case developed retinal toxicity due to intravitreal aminoglycoside antibiotic.

2. Case Report

A fifty-year-old female patient presented to us 45 days after an uneventful manual small incision cataract surgery. The

patient was from a farming background and otherwise she did not have any health issues and she was not a diabetic. She gave history of mild pain, redness, and blurred vision in the operated eye (left eye) for five days. On examination, visual acuity in the right eye was 6/6 and in the left eye hand movements were perceived close to face. On slit lamp examination, left eye had corneal epithelial edema and fluffy white cotton ball-like exudates in the anterior chamber and the intraocular lens was surrounded by the same exudates in the capsular bag (Figure 1). Intraocular pressure was normal. Ultrasound of the eye showed fine vitreous opacities of low reflectivity with flat retina (Figure 2).

Because of delayed onset and the type of exudates in the anterior chamber, fungal endophthalmitis was a strong suspicion. Pars plana vitrectomy with anterior chamber wash was done and intravitreal vancomycin, ceftazidime, and voriconazole were given. Direct microscopic examination of vitreous and anterior chamber fluid showed Gram-positive thin long beaded branching filaments and acid fast thin long filaments in modified acid fast staining (Kinyoun stain)

FIGURE 1: Fluffy white exudates in anterior chamber and surrounding the IOL in capsular bag.

FIGURE 2: Ultrasound showing fine vitreous opacities with flat retina.

(Figure 3). On blood agar dry, raised, chalky white colonies were seen after 2 days of incubation (Figure 4). The colonies were further identified as *Nocardia asteroides* based on colony morphology and biochemical reactions. The isolate was susceptible to amikacin, gentamicin, ciprofloxacin, ofloxacin, gatifloxacin, moxifloxacin, and co-trimoxazole.

Depending on the sensitivity report, intravitreal amikacin (200 $\mu\text{g}/0.1\text{ mL}$) was given. The dose of intravitreal amikacin was halved because it was a vitrectomised eye. Topical gatifloxacin and gentamicin eye drops were continued. Systemically, tablet co-trimoxazole was started and given for 6 weeks. Two weeks later, vision had improved to counting fingers at

one meter. Anterior segment was quiet, mildly hazy vitreous and the macula was pale and cloudy looking with multiple intraretinal hemorrhages (Figure 5). Amikacin induced macular infarction was suspected. Fluorescein angiography showed multiple capillary nonperfusion areas (Figure 6). The OCT showed increased macular thickness (central foveal thickness 342 μm), hyperreflectivity of inner retinal layers corresponding to areas of retinal edema (Figure 7).

To treat this ischemia and inflammation induced macular edema, intravitreal steroids could not be used in the backdrop of endophthalmitis. Intravitreal Bevacizumab was used for its anti-inflammatory properties. The vision improved

FIGURE 3: Gram-positive thin long beaded branching filaments of *Nocardia*.

FIGURE 5: Cloudy swelling, cotton wool spots, and scattered intraretinal haemorrhages.

FIGURE 4: Dry, raised, chalky white colonies of *Nocardia* on blood agar.

FIGURE 6: Fluorescein angiogram showing macular arteriolar occlusion with staining of the arteriolar walls along superior and inferior arcades.

to counting fingers at 2 meters and the macular edema subsided (Figure 8). On OCT, the macular thickness had decreased but the differentiation of different retinal layers was not evident and foveal centre showed hyper dense and adherent RPE and retinal layers (Figure 9). After follow-up of more than three months now, the condition of the eye is stable.

3. Discussion

There are two large case series of postoperative *Nocardia* endophthalmitis reported from South India [1, 4]. Decroos et al. [1] from Hyderabad, India, presented 16 cases of culture proven postoperative *Nocardia* endophthalmitis. In this series, 75% of patients presented with distinctive anterior chamber findings, such as the exudates on corneal endothelium or nodule-like deposits at the pupillary border. Final visual outcome was poor (final mean BCVA 2.1 ± 0.89 logMAR units.). Haripriya et al. [4], from Madurai, India, reported 24 culture proven cases of exogenous nocardial endophthalmitis following cataract surgery. Cotton ball

exudates, fluffy exudates on corneal endothelium, and nodular exudates on iris and hypopyon were the anterior segment findings. Visual outcome was poor in most patients with 79% (19/24) of patients obtaining a final visual acuity of hand motions or worse. In both these studies, the intense anterior chamber involvement was in sharp contrast to minimal vitritis seen. Both these studies reported highest antibiotic sensitivity for amikacin (90% and 87.5%). In our case, the presentation of endophthalmitis was six weeks after cataract surgery. The anterior segment involvement was intense and comparatively vitreous exudates were minimal. Following vitrectomy and intravitreal amikacin (with a reduced dosage of $200 \mu\text{g}/0.1 \text{ mL}$), the infection part was controlled but the visual outcome was not satisfactory due to development of macular infarction. The rationale for amikacin use was based on the sensitivity report and the results of the previous case series [1, 4]. Previous reports have shown that amikacin is a frequently chosen option for *Nocardia* infections [5–7].

Though macular ischemia is a rare complication following intravitreal amikacin injection, it is reported both following vitrectomy with intravitreal injection and following intravitreal injection alone [8–10]. Only one case of macular toxicity, possibly due to amikacin, was reported out of 420

FIGURE 7: OCT showing increased macular thickness.

FIGURE 8: Macular edema subsided.

FIGURE 9: Hyperdense and adherent RPE and retinal layers.

cases in Endophthalmitis Vitrectomy Study [11]. Several explanations have been put forth to explain the amikacin induced retinal toxicity: dilution errors, increased intraocular pressure following intravitreal injection, and variation in the liquefaction status of vitreous humor. The reason for macular predilection could be the contact of the drug with macula in supine position. Individual susceptibility may also be a factor. It is recommended that amikacin be better avoided when other antibiotics are sensitive to the organism. Amikacin may be used with a calculated risk when other antibiotics are resistant.

Competing Interests

The authors declare that they have no competing interests.

References

[1] F. C. Decroos, P. Garg, A. K. Reddy et al., “Optimizing diagnosis and management of *Nocardia* keratitis, scleritis, and endophthalmitis: 11-year microbial and clinical overview,” *Ophthalmology*, vol. 118, no. 6, pp. 1193–1200, 2011.

[2] R. Ramakrishnan, M. J. Bharathi, C. Shivkumar et al., “Microbiological profile of culture-proven cases of exogenous and endogenous endophthalmitis: a 10-year retrospective study,” *Eye*, vol. 23, no. 4, pp. 945–956, 2009.

[3] P. Lalitha, J. Rajagopalan, K. Prakash, K. Ramasamy, N. V. Prajna, and M. Srinivasan, “Postcataract endophthalmitis in South India incidence and outcome,” *Ophthalmology*, vol. 112, no. 11, pp. 1884–1889, 2005.

[4] A. Haripriya, P. Lalitha, M. Mathen et al., “*Nocardia* endophthalmitis after cataract surgery: clinicomicrobiological study,”

American Journal of Ophthalmology, vol. 139, no. 5, pp. 837–846, 2005.

- [5] B. A. Brown-Elliott, J. M. Brown, P. S. Conville, and R. J. Wallace Jr., “Clinical and laboratory features of the *Nocardia* spp. based on current molecular taxonomy,” *Clinical Microbiology Reviews*, vol. 19, no. 2, pp. 259–282, 2006.
- [6] A. K. Reddy, P. Garg, and I. Kaur, “Speciation and susceptibility of *Nocardia* isolated from ocular infections,” *Clinical Microbiology and Infection*, vol. 16, no. 8, pp. 1168–1171, 2010.
- [7] P. Lalitha, M. Tiwari, N. V. Prajna, C. Gilpin, K. Prakash, and M. Srinivasan, “*Nocardia* keratitis: species, drug sensitivities, and clinical correlation,” *Cornea*, vol. 26, no. 3, pp. 255–259, 2007.
- [8] P. A. Campochiaro and B. P. Conway, “Aminoglycoside toxicity—the survey of retinal specialists. Implications for ocular use,” *Archives of Ophthalmology*, vol. 109, no. 7, pp. 946–950, 1991.
- [9] P. A. Campochiaro and J. I. Lim, “Aminoglycoside toxicity in the treatment of endophthalmitis. The Aminoglycoside Toxicity Study Group,” *Archives of Ophthalmology*, vol. 112, no. 1, pp. 48–53, 1994.
- [10] G. Galloway, A. Ramsay, K. Jordan, and A. Vivian, “Macular infarction after intravitreal amikacin: mounting evidence against amikacin,” *British Journal of Ophthalmology*, vol. 86, no. 3, pp. 359–360, 2002.
- [11] Endophthalmitis Vitrectomy Study Group, “Results of the Endophthalmitis Vitrectomy Study. A randomized trial of immediate vitrectomy and of intravenous antibiotics for the treatment of postoperative bacterial endophthalmitis,” *Archives of Ophthalmology*, vol. 113, no. 12, pp. 1479–1496, 1995.