Is BAK the Culprit?: A Glaucoma Case Report Featuring Anterior Uveitis and Uncontrolled IOP

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PURPOSE

Congenital glaucoma is an aggressive form of glaucoma. It can cause large variations of intraocular pressure (IOP) meriting difficult control. Multiple IOP lowering agents are often required to achieve acceptable IOP. Benzalkonium chloride (BAK), a common preservative, has been well-documented to cause or exacerbate ocular surface disease (OSD).[1] We present a case of a patient with congenital glaucoma who developed uncontrolled IOP and chronic anterior uveitis (AU) following an uncomplicated YAG capsulotomy. Herpes simplex virus-1 (HSV-1) was initially thought to be the underlying cause. After treatment with oral antiviral medication, the AU and IOP improved significantly, but did not resolve. Only when the IOP lowering medications were changed to BAK-free medications did the AU completely resolve and IOP normalize. The purpose of this case report is to consider BAK as a confounding factor influencing IOP control and AU resolution.

CASE REPORT

A 42-year-old Caucasian female with congenital glaucoma was using BAK-free travoprost 0.004% QHS, brinzolamide-brimonidine 1%-0.2% BID and timolol 0.5% BID. An uncomplicated YAG capsulotomy was performed OS. One week later, IOP OS was 27mmHg with 0.5+ anterior chamber cells. Prednisolone acetate 1% QID was added and BAK- free travoprost was switched to latanoprostene bunod 0.024% QHS. Brinzolamide-brimonidine and timolol were continued BID. IOP fluctuated (18-32 mmHg OS) for 6 months with persistent low-grade AU. The AU resolved after addition of valacyclovir 500mg BID, but IOP remained above target even after discontinuing prednisolone acetate 1%. Micro pulse (MP3) laser was performed to further lower IOP. Thirteen months after MP3, IOP was 43mmHg OS with low grade AU. At this time latanoprostene bunod, brinzolamide-brimondine and timolol were discontinued. The patient was switched to BAK-free medications including BAK-free travoprost 0.004% QHS, brimonidine 0.1% with Purite BID, and preservative-free dorzolamide-timolol 2%-0.5% BID. One-week later IOP was 28mmHg and after four weeks IOP was 13mmHg with resolution of the AU. IOP has been consistently 11-17mmHg for 2 years since switching to BAK-free glaucoma medications (see Table 1).

TABLE 1

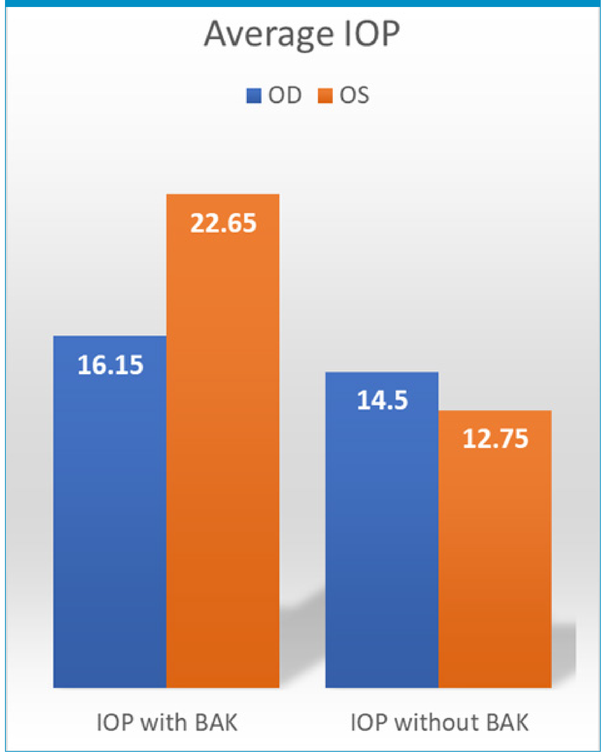


FIGURE 1
Pre-YAG capsulotomy of the visually significant posterior capsule opacification in the left eye.

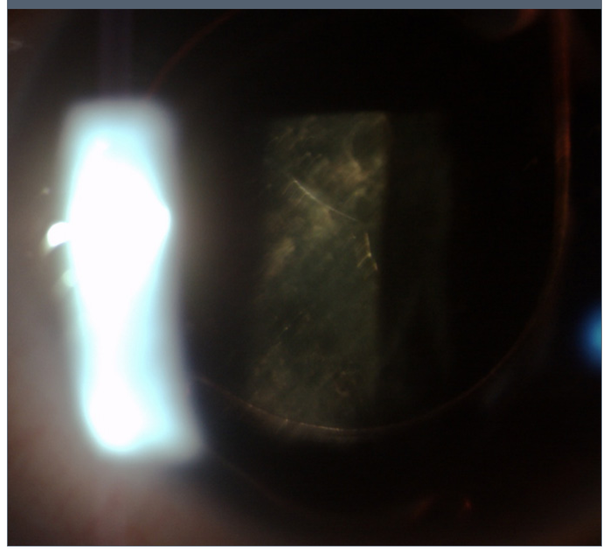
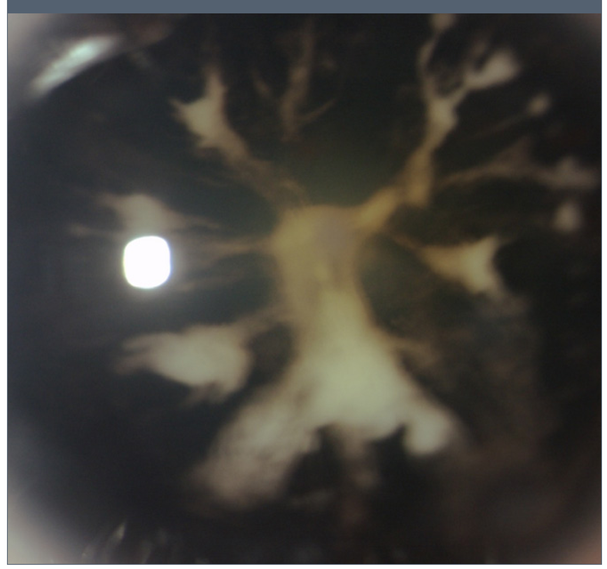


FIGURE 2
Congenital cataract of the right eye.



CONCLUSION

BAK causes ocular surface inflammation and may contribute to an already low-grade anterior uveitis and IOP elevation.[2-5] We hypothesize that switching to BAK-free medications, in this case, may have reduced inflammation not only to the ocular surface but also to intraocular structures. Reducing intraocular inflammation, especially if the trabecular meshwork is involved, may help lower IOP. Use of BAK-free glaucoma medication should be considered in susceptible individuals on multiple medications. Although HSV-1 was initially suspected as the underlying etiology, this cannot be excluded as an additional comorbid condition since it is well known that inflammation can reactivate dormant HSV-1.

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