American Journal of Ophthalmology Case Reports 36 (2024) 102131

Contents lists available at ScienceDirect

American Journal of Ophthalmology Case Reports

journal homepage: www.ajocasereports.com/

Secondary glaucoma after bevacizumab injection in Type-1 retinopathy of prematurity

Anthony A. Jones [a,b,*], Jacob S. Martin [a,b], Annette L. Giangiacomo [a,b], Deborah M. Costakos [a,b]

[a] Department of Ophthalmology, Medical College of Wisconsin, 8701 W Watertown Plank Rd, Milwaukee, WI 53226, USA
[b] Department of Pediatric Ophthalmology, Children's Hospital Milwaukee, 8915 W Connell Ct, Milwaukee, WI 53226, USA

ARTICLE INFO

Keywords:
Avastin
Bevacizumab
Secondary glaucoma
Retinopathy of prematurity

ABSTRACT

Purpose: The authors report three separate cases of type 1 retinopathy of prematurity (ROP) treated with intravitreal bevacizumab before, or at 34 weeks postmenstrual age (PMA), with subsequent development of secondary glaucoma.

Observations: All three cases involve patients born ≤24 weeks and meeting the American Academy of Pediatrics criteria for ROP screening. Prior to treatment, each patient was noted to have normal anterior chamber structures with no signs of glaucoma. Each patient developed type 1 ROP and was treated with intravitreal bevacizumab, which was administered at or before 34 weeks PMA. Following the administration of intravitreal anti-vascular endothelial growth factor (VEGF), each patient developed a suspected open-angle glaucoma (OAG) within an approximate 4-week time frame. In these cases, the presentation of glaucoma differed from those that have been previously reported in the literature.

Conclusion and importance: Based on similar timing of glaucoma development following intravitreal bevacizumab injections, we hypothesize that the administration of anti-VEGF agents to very premature infants (≤24 weeks) at or before 34 weeks PMA, may predispose them to the development of secondary glaucoma through an unknown and possibly novel pathway.

1. Introduction

Retinopathy of prematurity (ROP) is a retinal vascular disease that affects preterm infants and is the leading cause of preventable childhood blindness worldwide.[1] It is understood that the incomplete development of retinal vasculature leads to ischemic retina and the release of vascular endothelial growth factor (VEGF), which results in neovascularization.[1] The mainstay of treatment is aimed at reducing VEGF. Treatments include panretinal photocoagulation (PRP) and intravitreal anti-VEGF agents (e.g. bevacizumab), the latter being preferentially used in posterior ROP.[2]

Prematurity itself is an independent risk factor for the development of congenital glaucoma.[1,3–5] The pathogenesis is believed to be secondary to structural maldevelopment of the trabecular meshwork (TM), as it does not reach anatomical maturity until near 25 weeks' gestation.[6,7] Glaucoma in ROP, however, typically occurs in late stage, untreated ROP, and is mostly attributed to neovascular glaucoma or from an associated cicatricial retrolental membrane pushing the iris–lens diaphragm forward causing secondary angle closure glaucoma (ACG).[8–12] Glaucoma in ROP has also been reported as secondary to other modalities of treatment including PRP, cryotherapy, and vitreoretinal surgery. In these instances, the development of the glaucoma has been attributed to such things as recurrent hyphema, or angle closure from surgically induced mechanical changes to the anterior chamber (AC).[13–17]

In this case series, we present three patients with extreme prematurity and type 1 ROP who were treated with intravitreal bevacizumab and subsequently developed open angle glaucoma (OAG) within an approximate 4-week time frame.

2. Cases

Case 1: An infant boy born at gestational age 23 weeks and 2 days with a birthweight of 550 g received his first ROP screening exam at 30 weeks postmenstrual age postmenstrual age (PMA) showing persistent tunica vasculosa and no other obvious AC abnormalities by handheld portable slit lamp exam. Posteriorly, he had stage 2 ROP in zone I without plus disease in both eyes (OU). At 31 weeks PMA, there was

* Corresponding author. Medical College of Wisconsin 8701 W Watertown Plank Rd. Milwaukee, WI, 53226, USA.
E-mail address: anjones@mcw.edu (A.A. Jones).

https://doi.org/10.1016/j.ajoc.2024.102131
Received 16 November 2023; Received in revised form 19 June 2024; Accepted 12 July 2024
Available online 25 July 2024

notable progression to stage 3 ROP in zone I without plus disease OU. Meeting Early Treatment of ROP (ETROP) criteria for Type 1 ROP, bilateral intravitreal bevacizumab was administered (0.625 mg/0.025ml bevacizumab injected 1.5 mm behind the limbus temporally on a 32-gauge needle) without procedural complications. On post op day (POD) #1 there was a slight haziness to the corneas OU which prompted an intraocular pressure (IOP) check. The IOP was found to be elevated at 28 mmHg in the right eye (OD) and 35 mmHg in the left eye (OS); and dorzolamide was started two times a day (BID) OU. The IOP was checked weekly during ROP screening exams and remained moderately controlled (IOP ranging from 22 to 25 mmHg OD and 21–24 mmHg OS) on topical medical therapy alone. During this time, the ROP was noted to have regressed to stage 1 zone II without plus, though there remained temporal notch of avascular retina extending into zone I OU. At 35 weeks PMA (Post op week 4) the patient developed bilateral persistent diffuse corneal haze and elevated IOP of 30 mmHg OD and 40 mmHg OS. At this time, our institution's glaucoma specialist examined the patient and confirmed the diagnosis of secondary glaucoma which has been managed with escalated topical therapy alone, including dorzolamide BID OU, timolol BID OU, and latanoprost nightly OU. At 36 weeks PMA, there was significant improvement of the IOP and clearing of the corneal haze OU. On exam, the temporal notches of avascular retina persisted in zone I OU. At the vascular/avascular junction, there were terminal buds with arborization of vessels OU, which were highly concerning for stage 3 ROP. Given these findings, PRP was promptly administered OU for treatment of recurrent type 1 ROP. Currently, at 56 weeks PMA, the patient has had no recurrence of ROP and has had no further treatments. The infant remains on dorzolamide BID OU and timolol BID OU (parents self-discontinued latanoprost OU) and is scheduled to see our pediatric glaucoma specialist in the coming months, who will decide on definitive management of the glaucoma at that time as the IOP OS remains poorly controlled (high teens), while OD remains at a goal IOP ranging from the high single digits to the low teens.

Case 2: An infant boy born at gestational age 21 weeks and 6 days with a birthweight of 480 g received his first ROP screening exam at 31 weeks PMA, which showed no obvious AC abnormalities by handheld portable slit lamp exam, and the dilated fundus exam revealed stage 0 ROP in zone I without plus disease OU. At 32 weeks PMA, there was notable ROP progression to stage 3 ROP in zone I without plus disease OU. Meeting ETROP criteria for Type 1 ROP, bilateral intravitreal bevacizumab was administered (0.625 mg/0.025ml bevacizumab injected 1.5 mm behind the limbus temporally on a 32-gauge needle) without procedural complications. One week after intravitreal bevacizumab injections, ROP screening exams revealed regression of ROP to stage 1 zone II without plus, with a temporal notch of avascular retina extending into zone I OU. The level of ROP remained stable, and there were no signs of glaucoma on screening exam until 36 weeks PMA (Post op week 4) when the corneas began to show slight haziness. The IOP was checked via Tonopen and was found to be 21 mmHg OU. At this time, the ROP had progressed to stage 2 zone II without plus, with persistence of the temporal notch into zone I, without clear evidence of ROP at the site of the temporal notch OU. At 37 weeks PMA, the corneal haze was so severe that views posteriorly were limited OU, and the IOP was found to be 45 mmHg OD and 46 mmHg OS. A diagnosis of secondary glaucoma OU was made and the patient was started on dorzolamide three times a day OU. An exam under anesthesia (EUA) was performed by the pediatric glaucoma specialist who noted IOP of 33 mmHg OU (Tonopen), no notably abnormal AC structures by handheld portable slit lamp, no clear view to the fundus OU, and axial length of 18.18 mm OD/17.74 mm OS. Gonioscopy showed hazy views OU with high and flat iris insertions OS, and minimal appreciable details of the angle structures OD due to the severity of the corneal haze. Following this EUA the diagnosis of secondary glaucoma was confirmed, with OD being more severe than OS. Therefore, surgical intervention was planned initially for OD, followed by OS 1–2 weeks later. At 40 weeks PMA, a 360 trabeculotomy OD was performed which had a complicated post op course involving a larger than expected hyphema requiring an AC washout OD at 41 weeks PMA. Following this washout there was a re-bleed (thought to be from neovascularization secondary to reactivated ROP) and the trabeculotomy was deemed to have failed. Throughout this time, the views posteriorly remained poor with minimal appreciable details, however, it was noted that there was no definite plus disease OU. An ultrasound (B-scan) was performed OU which confirmed no retinal detachments in either eye. Given the re-bleed OD from suspected neovascularization and recurrent ROP, it was deemed necessary to treat with repeat intravitreal anti-VEGF. Given the known symmetric nature of ROP, and to prevent similar complications at the time of the glaucoma surgery for OS, it was deemed necessary to also treat with intravitreal anti-VEGF. Therefore, at 42 weeks PMA bilateral intravitreal bevacizumab was administered (0.625 mg/0.025ml bevacizumab injected 1.5 mm behind the limbus temporally on a 32-gauge needle) without procedural complications. Subsequently, an Ahmed implant was placed OU at 43 weeks PMA. Several days later the corneal haze began to clear and ROP screening revealed stage 1 ROP, but very poorly vascularized retina barely extending into zone II without plus disease OU, furthermore, OD displayed nasal and temporal choroidal folds. Subsequently, near 44 weeks PMA, the retina service was consulted. Based on their exam there was choroidal engorgement causing folds, and they deemed laser was not ideal given the current level of inflammation following glaucoma surgery, and not technically possible given the amount of corneal haze. At 45 and 46 weeks PMA, ROP screening showed resolution of the choroidal folds OD but minimal improvement in avascular retina OU. Both eyes remained at stage 1 ROP with minimal extension past zone II without plus disease. The most peripheral nasal and temporal vessels were beginning to anastomose near the zone I and II border OU. Given this large degree of avascular retina OU as well as the anastomosis of the large vessels, PRP was planned OU under the assumption that no further normal retinal vascularization was likely to occur further into the periphery. An Ahmed revision OD was performed at 47 weeks PMA due to exposure of the tube and PRP was performed OU at the same time to minimize anesthesia. Following PRP, at 48 weeks PMA, OD was noted to develop a temporal band of traction that did not involve the fovea and was classified as stage 4A ROP, and OS showed complete regression of ROP. There were no skip areas noted on exam and there was no indication for fill-in laser. With regards to the glaucoma, OD has since been stable (ranging from IOP of 10–16 mmHg with most recent IOP of 13 mmHg) on a drop regimen of dorzolamide BID, and OS has been stable (ranging from IOP of 12–20 mmHg with a most recent IOP of 15) with an Ahmed implant alone and has not required further drops. At the most recent follow up, 52 weeks PMA, the ROP in OD has remained stable at stage 4A disease without plus, and the retinal fold appears to be flattening, whereas OS has remained with complete regression of ROP.

Case 3: An infant girl with chromosome 1 partial deletion and microcephaly was born at gestational age 24 weeks and 5 days with a birthweight of 570 g. The infant's care was initially performed at an outside facility, and per chart review, her first ROP screening exam was at 31 weeks PMA, she was found to have stage 2 ROP in zone I without plus disease OU. On handheld portable slit lamp exam, there were no obvious AC abnormalities noted, however, she was found to have an anomalous optic nerve OD, and a small arteriovenous malformation (AVM) along the supero-temporal arcade OS. At 34 weeks PMA, screening exam revealed stage 3 ROP in zone I without plus disease OU and an increased size of left AVM. Meeting ETROP criteria for Type 1 ROP, intravitreal bevacizumab was administered (0.625 mg/0.025ml bevacizumab injected 1.5 mm behind the limbus temporally on a 32-gauge needle) OD, and to OS one day later (unknown why the outside provider chose to stagger injections). Screening exams were continued, and they showed regression of ROP to stage 2 in zone II without plus, but with significant avascular retina into posterior zone II OU. While ROP screening exams remained stable, at 39 weeks PMA (Post op week 5), the corneas were noted to have severe clouding OU. At 40 weeks PMA, the

child was transferred for specialized ROP care and was found to have mildly elevated IOP (25 mmHg OD/27 mmHg OS) and it was monitored at this time. At 41 weeks PMA, the ROP had further regressed to stage 1 zone II without plus OU, but the IOP had increased to 35 OD and 34 OS. At this time the infant was examined by our institution's pediatric glaucoma specialist who noted normal AC structures by handheld portable slit lamp, C/D ~0.4 OU, corneal diameters were 12 mm OD/11 mm OS and a diagnosis of secondary glaucoma OU was confirmed. The glaucoma was managed medically with dorzolamide BID and latanoprost QHS OU with the plan to perform surgery as a more definite measure once she was medically stable. At 42 weeks PMA the ROP exam was stable and the IOP had improved to 9 mmHg OD and 19 mmHg OS. Unfortunately, the patient did not survive her multitude of medical comorbidities and she passed away at 43 weeks PMA, prior to any further exams or intervention.

3. DISCUSSION/CONCLUSION

The goal of this report is to highlight the use of bevacizumab in a small population of extremely premature infants with type 1 ROP and to bring attention to the possibility that intravitreal anti-VEGF therapy in these eyes may contribute to the pathogenesis of glaucoma. Each patient in this series was born at or before 24 weeks PMA and developed type 1 ROP, receiving anti-VEGF agents before or at the age of 34 weeks PMA. Each patient subsequently developed OAG during the postoperative week 4 period. Given the timing of development of secondary glaucoma following anti-VEGF injection, we propose that anti-VEGF agents in these very premature eyes may alter aqueous outflow in mechanisms not yet elucidated.

In the setting of prematurity, the risk of developing glaucoma increases due to microscopic variations in the level of development of the TM, Schlemm's canal, and the uveal tract.[3–6,18] These changes cause obstruction of aqueous humor outflow and lead to OAG. Furthermore, several other relevant anterior segment anatomic abnormalities (steep corneal curvature, decreased AC depth, anteriorly displaced iris planes, and increased lens thickness) have classically been reported in premature infant eyes.[3–7,18,19] Each of the presented cases involves extremely preterm infants who developed an OAG with no other grossly observable anatomic abnormalities. While it is feasible that our patients may have developed OAG regardless of treatment, the timing of development of glaucoma, occurring shortly after intravitreal anti-VEGF injections, makes this seem less likely. Furthermore, this connection is strengthened by the fact that the majority of extremely premature infants, whether in the setting of ROP or not, do not go on to develop OAG. This then begs the question, does the introduction of anti-VEGF into the underdeveloped eye of these extremely premature infants cause disruption in the maturation of the angle structures?

With regards to the embryonic and fetal development of the TM, Schlemm's canal, and the uveal tract, it is known that VEGF plays a crucial role in the homeostasis of the hyaloid and primitive choroidal vasculature.[20,21] These vascular systems supply the angle structures with the necessary molecules and solutes to develop into properly functioning anatomical zones.[20,21] One can then infer that the introduction of anti-VEGF into such a system could cause disruption of the normal development. Similarly, the remainder of the AC structures, which when abnormal are known risk factors for the development of glaucoma in premature infants (such as the cornea, iris, and lens), are also reliant on adequate blood supply for normal development. Introducing anti-VEGF into the premature eye may stunt such development and lead to glaucoma via other avenues as well.

Glaucoma in ROP is labeled as a secondary glaucoma of childhood associated with ocular abnormalities and the reported clinical presentation is strikingly unique.[3,4,18] Changes observed in glaucoma secondary to ROP include cicatricial retrolental fibroplasia and anteriorization of the iris-lens diaphragm, which classically cause a secondary ACG.[9–12] None of these changes were noted in any of the presented cases. Of note, both OAG and neovascular glaucoma can occur in this setting but have been far less reported. With this regard, we highlight the fact that in glaucoma secondary to ROP, the age of presentation tends to be much later than that observed in our cases, with some reports describing the median age at presentation to be between 18 and 24 months (corrected age), and others describing an median age of 7.8 months (from birth).[5,8,22] In two of our cases, gonioscopy was unable to be performed due to corneal clouding, but the ACs were noted to be of normal depth. Although this does not preclude a diagnosis of secondary ACG, it does guide us away from this classification and more towards OAG.

The development of glaucoma secondary to treatments for ROP, such as vitreoretinal surgery, PRP, and cryotherapy, have all been reported. In these instances, the most common presentation involves a shallow or flat AC, with or without posterior synechiae or pupillary block.[13–17,23] Although there have been reported cases of glaucoma in ROP patients who have received intravitreal anti-VEGF treatment, there has been no mention of a clear association between the two, and to our knowledge, the possibility of this treatment causing iatrogenic glaucoma has not been proposed. In fact, many of the available studies using bevacizumab in ROP have shown it to be a safe, effective, and well tolerated treatment.[2,24–29] With that said, none of these studies have focused on the age of the patient at the time of the intravitreal injection, but rather on the stage and zone of ROP. Within these reports, the possible association between anti-VEGF and glaucoma is not discussed, as before now, it was presumably regarded as coincidence or more likely secondary to ROP. Each of the presented cases followed the same recommended guidelines for treatment, and when the extreme level of prematurity, as well as the PMA at which they received anti-VEGF injections are introduced as variables, we have noticed the possibility of this new association.

Lastly, there is a well-known transient rise in IOP following intravitreal injections of any substance. Several reports have postulated this to be one of the factors involved in the possible correlation between chronic intravitreal injections and the development of OAG in diseases such as exudative macular degeneration and diabetic retinopathy. In these cases, the likelihood of developing OAG does not arise until approximately 14 injections, and the risk increases thereafter.[30] In our cases, the patients only received one injection before showing signs of glaucoma. Furthermore, a small prospective case-series investigating the short-term changes in IOP following intravitreal injections of bevacizumab in ROP patients showed an average IOP spike of 12 mmHg at 1 minute following injection. Within three minutes following the injection the IOP had normalized to non-statistically significant levels when compared to pre-injection IOP.[31] Similarly, Kato et al. demonstrated an average increase in IOP of 12mmHG at 5 minutes following intravitreal injections for the treatment of ROP. In this study, the IOP also normalized within 15 minutes. They also reported that no clinical parameters, such as axial length, were correlated with high IOP following the injections.[32] When comparing intravitreal injections in neonates to adults, it is important to recognize that the volume of medicine injected into the vitreous cavity is half of that in neonates compared to adult dosing to compensate for the differences in ocular volume.

Again, we stress the fact that in each case these signs did not present themselves until 4 weeks after the intervention. An argument could be made that the injection of medication into the vitreous cavity caused a volume expansion and a subsequent anterior shift of the lens-iris diaphragm, this coupled with the possibility of unseen pathology such as neovascularization of the iris, angle, or persistent tunica vasculosa lentis – which are all inflammatory in nature – could explain a rise in IOP and the subsequent development of glaucoma. We believe that if this were truly the case, the glaucomatous changes seen on exam would have presented themselves sooner as this would have led to an ACG. We feel this further strengthens our hypothesis that within this subpopulation of ROP there is likely an unknown mechanism behind the introduction of anti-VEGF and the development of glaucoma at or near the 4 week post injection date. There are several possible limiting factors to our

hypothesis. We were limited in some cases by an inability to perform gonioscopy due to corneal haze and cannot classify the glaucoma with certainty. The infant in case #3 was noted to have a chromosomal abnormality, which may have predisposed her to glaucoma. Lastly, there was no other testing performed in these cases to reveal specific genetic abnormalities that could contribute to the development of glaucoma.

Given these noted complications, we recommend close monitoring of IOP in very premature infants receiving injections for ROP. We hope by bringing these associations to light, that other providers may notice similarities in their cases and report on such findings. This could help determine the need for more precise research investigations regarding the use of anti-VEGF injections in ROP, and specifically, the possible rate of glaucoma as a complication in extremely preterm infants.

Patient Consent

Consent to publish the case report was not obtained. This report does not contain any personal information that could lead to the identification of the patient.

IRB approval was not obtained as it did not meet criteria for necessity at our institution.

Acknowledgments and dislocsures

The following authors have no financial disclosures: AAJ, JSM, ALG, DMC.

The following authors have no conflicts or potential conflicts of interest: AAJ, JSM, ALG, DMC.

There was no funding or grant support.

Authorship

All authors attest that they meet the current ICMJE criteria for Authorship.

CRediT authorship contribution statement

Anthony A. Jones: Writing – review & editing, Writing – original draft, Validation, Formal analysis, Data curation, Conceptualization. **Jacob S. Martin:** Writing – review & editing, Supervision, Formal analysis, Data curation, Conceptualization. **Annette L. Giangiacomo:** Writing – review & editing, Investigation. **Deborah M. Costakos:** Writing – review & editing, Validation, Supervision, Project administration, Methodology, Investigation, Formal analysis, Data curation, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Acknowledgements

None.

References

1. Brown, A.C. and K. Nwanyanwu, Retinopathy of prematurity, in *StatPearls*. 2023, StatPearls Publishing Copyright © 2023, StatPearls Publishing LLC.: Treasure Island (FL).
2. Sankar MJ, Sankar J, Chandra P. Anti-vascular endothelial growth factor (VEGF) drugs for treatment of retinopathy of prematurity. *Cochrane Database Syst Rev*. 2018; 1(1):Cd009734.
3. Zertsalova MA, et al. The features development and clinical course of primary congenital glacucoma in infants. *Pediatrician*. 2013;4(2):18–23.
4. Stamper R, Lieberman M, Drake M. *Becker-Shaffer's diagnosis and therapy of the glaucomas*. In: *Becker-Shaffer's Diagnosis and Therapy of the Glaucomas*. vol. 1. Mosby; 2009.
5. Consensus 9. In: Glaucoma Childhood, Weinreb AGRN, Papadopoulos M, Grigg J, Freedman S, eds. *9th Consensus Meeting*. Kugler Punblications: Vancouver, Canada. 2013:239.
6. Hosaka F, et al. Qualitative changes in fetal trabecular meshwork fibers at the human iridocorneal angle. *Anat Cell Biol*. 2013;46(1):49–56.
7. Anderson DR. The development of the trabecular meshwork and its abnormality in primary infantile glaucoma. *Trans Am Ophthalmol Soc*. 1981;79:458–485.
8. Senthil S, et al. Management outcomes of secondary glaucoma due to retinopathy of prematurity: a 19-year prospective study at a tertiary eye care Institute. The Indian Twin cities ROP Screening (ITCROPS) database report number 8. *PLoS One*. 2020;15 (9), e0238633.
9. Pollard ZF. Secondary angle-closure glaucoma in cicatricial retrolental fibroplasia. *Am J Ophthalmol*. 1980;89(5):651–653.
10. Ueda N, Ogino N. Angle-closure glaucoma with pupillary block mechanism in cicatricial retinopathy of prematurity. *Ophthalmologica*. 1988;196(1):15–18.
11. Smith J, Shivitz I. Angle-closure glaucoma in adults with cicatricial retinopathy of prematurity. *Arch Ophthalmol*. 1984;102(3):371–372.
12. Kushner BJ, Sondheimer S. Medical treatment of glaucoma associated with cicatricial retinopathy of prematurity. *Am J Ophthalmol*. 1982;94(3):313–317.
13. Bremer DL, et al. Glaucoma in the early treatment for retinopathy of prematurity (ETROP) study. *J aapos*. 2012;16(5):449–452.
14. Uehara A, et al. Angle closure glaucoma after laser photocoagulation for retinopathy of prematurity. *Br J Ophthalmol*. 2004;88(8):1099–1100.
15. Trigler L, et al. Case series of angle-closure glaucoma after laser treatment for retinopathy of prematurity. *J aapos*. 2005;9(1):17–21.
16. Lee GA, Lee LR, Gole GA. Angle-closure glaucoma after laser treatment for retinopathy of prematurity. *J Am Assoc Pediatr Ophthalmol Strabismus*. 1998;2(6): 383–384.
17. Lenis TL, et al. Case report: glaucoma in an infant with retinopathy of prematurity. *Front Pediatr*. 2021;9, 786327.
18. Ko F, Papadopoulos M, Khaw PT. Primary congenital glaucoma. *Prog Brain Res*. 2015;221:177–189.
19. Hartnett ME, et al. Anterior segment evaluation of infants with retinopathy of prematurity. *Ophthalmology*. 1990;97(1):122–130.
20. Ma IT, et al. VEGF mRNA and protein concentrations in the developing human eye. *Pediatr Res*. 2015;77(4):500–505.
21. Baba T, et al. VEGF 165 b in the developing vasculatures of the fetal human eye. *Dev Dynam*. 2012;241(3):595–607.
22. Hong EH, et al. Ophthalmic complications in retinopathy of prematurity in the first decade of life in Korea using the national health insurance database. *Sci Rep*. 2022; 12(1):911.
23. Mills MD. Evaluating the cryotherapy for retinopathy of prematurity study (CRYO-ROP). *Arch Ophthalmol*. 2007;125(9):1276–1281.
24. Taher NO, et al. Intravitreal anti-vascular endothelial growth factor injection for retinopathy of prematurity: a systematic review and meta-analysis. *Front Med*. 2022; 9, 884608.
25. Mintz-Hittner HA, Kennedy KA, Chuang AZ. Efficacy of intravitreal bevacizumab for stage 3+ retinopathy of prematurity. *N Engl J Med*. 2011;364(7):603–615.
26. Roohipoor R, et al. Comparison of intravitreal bevacizumab injection and laser photocoagulation for type 1 zone II retinopathy of prematurity. *J Curr Ophthalmol*. 2019;31(1):61–65.
27. Tsiropoulos GN, et al. Comparison of adverse events between intravitreal anti-VEGF and laser photocoagulation for treatment-requiring retinopathy of prematurity: a systematic review. *Int Ophthalmol*. 2023;43(3):1027–1062.
28. Karkhaneh R, et al. Efficacy of intravitreal bevacizumab for zone-II retinopathy of prematurity. *Acta Ophthalmol*. 2016;94(6):e417–e420.
29. Autrata R, et al. [Effects of intravitreal pegaptanib or bevacizumab and laser in treatment of threshold retinopathy of prematurity in zone I and posterior zone II–four years results]. *Ceska a Slov Oftalmol*. 2012;68(1):29–36.
30. Levin AM, et al. Intraocular pressure elevation following intravitreal anti-VEGF injections: short- and long-term considerations. *J Glaucoma*. 2021;30(12): 1019–1026.
31. Obata S, et al. Short-term changes in intraocular pressure after intravitreal injection of bevacizumab for the treatment of retinopathy of prematurity. *Clin Ophthalmol*. 2019;13:2445–2449.
32. Kato A, et al. Short-term intraocular pressure changes after intravitreal injection of bevacizumab for retinopathy of prematurity. *Jpn J Ophthalmol*. 2019;63(3):262–268.