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ORIGINAL RESEARCH

Optic Nerve Head Structural Changes After Intravitreal Injection

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Purpose: To evaluate the acute anatomical changes of Bruch's membrane opening (BMO) and optic nerve head (ONH) pit depth in patients receiving 0.05cc of anti-VEGF intravitreal injections (IVIs).

Methods: We prospectively enrolled patients receiving IVIs and collected data including age, sex, race, phakic status, presence or absence of glaucoma, injection agent utilized, axial length, and cup-to-disc ratio (C/D). High-resolution spectral domain OCT imaging and measurements were made pre- and within five minutes post-IVI.

Results: Fifty-one eyes of 51 patients were included in the study. 35.29% of patients were male. The mean age at the time of IVI was 78.27 ± 11.17 years. The average change in the pit depth after IVI was 31.57 ± 24.36 μm ($p < 0.001$). The number of IVIs previously received significantly correlated with a decreased change in pit depth post-injection ($r = -0.369$, $p = 0.004$). No significant relationship was identified between categorical or continuous variables and the change in pit depth or BMO after IVI. No significant associations were identified between the magnitude of IOP elevation and the change in pit depth post-IVI ($r = 0.03$, $p = 0.834$) or the number of previous IVIs ($r = 0.005$, $p = 0.973$).

Conclusion: IVI was associated with a modest increase in ONH pit depth. The number of prior injections inversely correlated with this change, suggesting a potential cumulative effect on structural response. Given the link between ONH biomechanics and retinal nerve fiber layer (RNFL) thinning, our findings raise the possibility that repeated IVIs may contribute to structural changes relevant to glaucomatous progression.

Keywords: optic nerve, injections, intravitreal, pit, depth

Introduction

Intravitreal injections (IVIs) are one of the most common procedures worldwide and the leading ophthalmic procedure performed globally.[1,2] Anti-vascular endothelial growth factor (VEGF) IVIs are indicated for the treatment of several retinal conditions, including wet age-related macular degeneration (ARMD), diabetic macular edema (DME),[3] retinal vein occlusion (RVO),[4] and choroidal neovascular membrane (CNVM).[5] Repeated injections are integral to the long-term management of these chronic conditions.[6] Although IVIs are well tolerated over years of treatment, there are risks involved with each injection.[7]

It has been established that IVIs may result in a transient spike in intraocular pressure (IOP) due to the added volume to the globe.[6] The rise in IOP typically returns to baseline within 30 min,[8] though a minor subset of patients may have persistently elevated IOP, suggesting elevated risk for adverse effects.[9] Though largely temporary, the increased IOP raises concern for damage to the optic nerve head (ONH), central retinal artery blood flow, and axonal transport.[8]

Documented changes to the ONH immediately post-injection include enlargement of Bruch's membrane opening (BMO), deepening and enlargement of the ONH cup, and prelaminar tissue thinning.[10] Current studies suggest that IVIs have a minimal association with retinal nerve fiber layer (RNFL) thinning in non-glaucomatous eyes, although this relationship is complex with findings varying between studies.[11]

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The current literature on the ONH morphological changes caused by IVIs is limited by small patient numbers and does not address the impact of ocular and patient characteristics on the ONH morphologic changes induced by the acute rise in IOP. This is particularly relevant given evidence suggesting that patients with a history of glaucoma have a higher rate of prolonged IOP elevation post-IVI.[12] Risk factors for glaucoma include advanced age, African American or Asian race, family history of glaucoma, hypertension, and myopia.[13,14] Females are also at a higher risk for closed-angle glaucoma.[15] To better understand risks associated with IVIs, we aim to assess the acute impact of anti-VEGF IVIs on ONH pit depth and BMO and compare these structural changes across demographic and ocular characteristics.

Materials and Methods

This was a prospective study conducted at the University of Arkansas for Medical Sciences (UAMS) Harvey & Bernice Jones Eye Institute. The study protocol was approved by the UAMS Institutional Review Board and was carried out according to its research standards. The study adhered to the principles of the Declaration of Helsinki (2013 version).

Eligibility included patients undergoing IVIs of anti-VEGF medications (including bevacizumab, aflibercept, and faricimab-svoa) with prior diagnoses of wet ARMD, DME, RVO, or CNVM. Exclusion criteria included patients with prior history of vitrectomy, low-quality OCT images, and retinal pathology preventing clear ONH visualization. In patients requiring same day bilateral injections, only one eye was arbitrarily enrolled in the study.

We collected baseline demographic data, such as age, race, sex, indication for IVIs, anti-VEGF medication administered, lens status (phakic vs pseudophakic), and glaucoma history. Ocular data collected included pre-injection best corrected visual acuity, C/D, and axial length measured using the Zeiss IOLMaster 500 (Carl Zeiss Meditec USA, Inc, Dublin, USA). IOP pre- and within 5 min post-injection was measured using the Tono-Pen XL (Reichert, Inc, New York, USA).

Imaging

OCT imaging was performed using the OCT Spectralis system (Heidelberg Engineering, Inc., Heidelberg, Germany). Pre-injection images were captured prior to prepping patients for injection. A horizontal raster scan consisting of 131 scans (8 mm length) was done, with one side transversing the ONH and the other side covering the fovea. Post-injection images were taken within 5 min using the same acquisition protocol and the tracking function activated.

Procedure

Consenting patients were recruited from the clinic at UAMS. After identifying the need for an injection, pre-injection IOP was measured, and an OCT was acquired. Patients subsequently underwent IVI. All injections were performed by the same surgeon (S.H.U). A proparacaine drop was initially applied, followed by one drop of 5% betadine. Five minutes later, one drop of lidocaine was placed. The process was repeated after 5 minutes. A lid speculum was inserted, and the medication was injected 3–4 mm from the limbus using a 30-gauge needle. The volume of all injections was 0.05 mL (Eylea HD, Syfovre, and Izervay were not available at our institution at the time of the study). A cotton swab was gently applied to the injection site to prevent any reflux. The lid speculum was immediately removed, and the patient was escorted to the imaging suite, where repeat imaging was performed. The IOP was measured with the Tonopen as soon as the imaging was completed. The patient was escorted back to the examination room, where the eye was rinsed with BSS.

Analysis of the Optic Nerve Structures

We used the Heidelberg Eye Explorer software to analyze the OCT images. Frames displaying the maximum ONH pit depth and complete visualization of BMO were used as a baseline scan. These measurements were chosen as primary morphological parameters due to their consistent visibility and reproducibility on standard OCT imaging. BMO was measured as the linear distance from tip to tip of the retinal pigmented epithelium (Figure 1A). ONH pit depth was measured by using a reference line parallel to BMO, with a perpendicular line drawn to the deepest part of the ONH pit (Figure 1B). Measurements were averaged from individual data points taken by two masked interpreters. Measurements discordant by more than 10% were reviewed by a third interpreter. In this case, the measurement closest to the third interpreter's value was used for analysis to resolve discrepancies and exclude outliers.

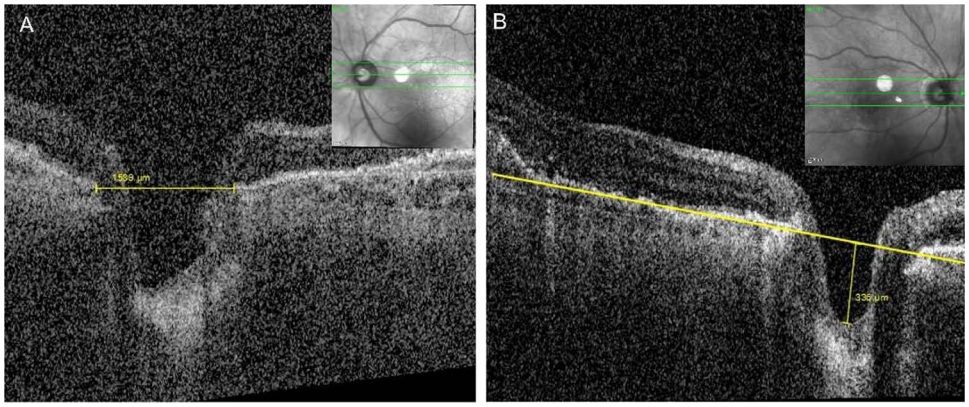

Figure 1 Measurement of Bruch's membrane opening (BMO) and optic nerve head (ONH) pit depth. The outer green lines represent the boundaries of the scan, while the middle line shows the exact location of the scan in view. (**A**) BMO was measured as the linear distance from tip to tip of the retinal pigment epithelium (yellow line). (**B**) ONH pit depth was measured using a reference line parallel to the BMO (horizontal yellow line); a perpendicular line was drawn from this reference to the deepest point of the ONH pit (vertical yellow line).

Statistical Analysis

Data was compiled on Microsoft Excel (Microsoft Corporation, Redmond, WA, USA, Version 2412) and exported to JASP (JASP Team, Amsterdam, Netherlands, Version 0.19.2) for analysis. A two-tailed *t*-test or ANOVA analysis was performed for categorical variables (sex, glaucoma status, agent utilized, and race). A Pearson correlation coefficient test was performed to evaluate the relationship between continuous variables (C/D ratio, axial length, age) and pit depth. Statistical significance was defined as p-value < 0.05.

Results

Fifty-six eyes were enrolled. Five were excluded due to poor imaging, leaving 51 eyes for final analysis. The majority of patients were White (76.47%), female (64.71%), pseudophakic (78.43%), and did not have a diagnosis of glaucoma (80.39%). The average age of patients was 78.27 ± 11.17 years. The average estimated C/D was 0.41 ± 0.18 with an average axial length of 23.99 ± 1.06 mm. A summary of patient demographics and continuous variables is shown in Table 1. The mean pre-injection IOP was 15.67 ± 3.83 mmHg, increasing to 32.54 ± 9.29 mmHg post-injection ($p < 0.001$).

Table 1 Summary of Demographics and Continuous Variables

Variable	Number of Eyes	% of Total
Total	51	100
Race[A]		
White	39	76.47
African American	10	23.53
Sex		
Male	18	35.29
Female	33	64.71
Lens Status		
Phakic	11	21.57
Pseudophakic	40	78.43
Glaucoma Status		
No	41	80.39
Yes	10	19.61
Diagnosis		
Age-related Macular Degeneration	29	56.86
Diabetic Macular Edema	7	13.73

(*Continued*)

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Table 1 (Continued).

Variable	Number of Eyes	% of Total
Retinal Vein Occlusion	13	25.49
Choroidal Neovascular Membrane	2	3.92
	Mean SD	**Range**
Age	78.27 11.17	38.00–97.00
C/D	0.41 0.18	0.10–0.85
Axial Length	23.99 1.06	20.39–27.53

Notes: [A]Due to small sample size, patient data for Hispanic and Asian individuals were not included in this portion of the analysis (N=49).

On average, patients received 19.65 ± 17.12 injections prior to the study, with a range of 1–70 injections. A significant negative correlation was found between the change in pit depth post-IVI and the number of injections a patient previously received ($r = -0.396$, $p = 0.004$).

The mean pre-injection ONH pit depth was 239.46 ± 148.04 μm, increasing to a mean of 266.65 ± 155.62 μm following IVI ($p < 0.001$). The average change in ONH pit depth following IVI was 31.57 ± 24.26 μm, correlating with an increase of 13.18% in pre-IVI ONH pit depth. No significant associations between the change in pit depth post-IVI and categorical variables were identified, including race (White versus African American, $p = 0.713$), sex (male versus female, $p = 0.087$), phakia (phakic versus pseudophakic, $p = 0.501$), and glaucoma status (no versus yes, $p = 0.924$). Additionally, a change in ONH pit depth was not found to differ between bevacizumab, aflibercept and faricimab ($p = 0.245$). Table 2 summarizes the change in pit depth by categorical variable. There was no significant relationship between the change in IOP and the change in the pit depth post-IVI ($r = 0.03$, $p = 0.834$). Similarly, no significant relationship was found between the change in IOP post-IVI and the number of previous injections ($r = 0.005$, 0.973). Two cases exhibited more than 10% discrepancy between the two primary evaluators in pit depth measurement, requiring review by a third interpreter.

BMO was visualized in 50 patients. There was no significant change in BMO after IVI, with an average change of 75.45 ± 79.14 μm ($p = 0.785$). No significant relationship was identified between the change in BMO and any of the assessed categorical variables, including race ($p = 0.151$), sex ($p = 0.496$), phakia ($p = 0.752$), glaucoma status ($p = 0.831$), and agent utilized ($p = 0.424$). Table 2 additionally summarizes the change in BMO by categorical variable.

Of the assessed continuous variables, none were found to be significantly correlated with changes in the pit depth or BMO. Age had weak correlations with pit depth ($r = -0.005$, $p = 0.972$) and BMO ($r = -0.114$, $p = 0.432$). C/D ratio showed no significant association with pit depth ($r = 0.152$, $p = 0.287$) or BMO ($r = -0.055$, $p = 0.704$). Axial length demonstrated weak correlations with both pit depth ($r = 0.033$, $p = 0.820$) and BMO ($r = -0.141$, $p = 0.333$). Table 3 summarizes the change in BMO by categorical variable.

Discussion

The immediate rise in IOP following IVI caused an average increase in the ONH pit depth of 31.57 μm, or 13.18%, independent of assessed variables. This change was statistically significant ($p < 0.001$). This magnitude of displacement is well above the axial resolution of high-resolution spectral-domain OCT (<10μm).[16] Though clinical significance remains unclear, our findings suggest that IVIs can cause subtle but measurable structural changes at the ONH. Glaucoma risk factors (age, race) were not found to be significant predictors of increased change in pit depth post-IVI.[13,14] The change in pit depth post-IVI between males and females trended toward significance ($p = 0.087$) suggesting that females may be more susceptible to adverse effects. Continuous variables were not found to correlate with the change in pit depth post-IVI.

A study conducted by Gómez-Mariscal et al assessed twenty-nine eyes receiving IVIs for the first time.[10] In patients receiving a three-month course of monthly injections, BMO significantly enlarged after each injection. Patients receiving

Table 2 Change in ONH Pit Depth and BMO by Categorical Variable

ONH Pit Depth						BMO				
Variable	**Number of Eyes**	**Mean (Micron)**	**SD (Micron)**	**Range (Micron)**	**P-Value**	**Number of Eyes**	**Mean (Micron)**	**SD (Micron)**	**Range (Micron)**	**P-Value**
Total:	51	31.57	24.36	1.00–85.00	-	50	75.45	79.14	2.00–372.00	-
Race[a]:					0.713					0.151
White	39	32.68	24.57	1.00–85.00		38	86.46	87.04	2.00–372.00	
African American	10	29.40	26.52	1.00–67.00		10	45.55	25.44	7.00–78.00	
Sex:					0.087					0.496
Male	18	23.67	20.91	2.00–82.50		17	64.71	58.80	10.50–244.00	
Female	33	35.88	25.31	1.00–85.00		33	80.99	88.13	2.00–372.00	
Lens Status:					0.501					0.752
Phakic	11	36.00	21.68	9.00–71.50		11	68.68	104.47	2.00–372.00	
Pseudo-phakic	40	30.35	25.16	1.00–85.00		39	77.36	72.02	4.50–331.00	
Glaucoma Status:					0.924					0.831
No	41	31.73	24.92	1.00–82.50		40	76.66	75.60	2.00–372.00	
Yes	10	30.90	23.11	1.00–85.00		10	70.60	96.43	7.00–331.00	
Agent[b]:					0.245					0.424
Bevacizumab	9	32.06	27.11	1.00–71.5		9	50.61	41.44	2.00–104.00	
Aflibercept	28	34.93	25.85	1.00–85.00		27	73.22	72.55	4.50–331.00	
Faricimab	13	21.34	15.50	3.00–61.50		13	96.12	109.86	12.00–372.00	

Notes: [A]Due to small sample size, patient data for Hispanic and Asian individuals were not included in this portion of the analysis (N=49 for pit-depth, N=48 for BMO). [B]Due to small sample size, agent data for Ranibizumab was not included in this portion of the analysis (N=50 for pit depth, N=49 for BMO).

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Table 3 Correlation of Continuous Variables with Changes in Pit Depth and BMO

Variable	P-Value	Pearson's r
Age:		
Pit Depth	0.972	−0.005
Bruch's Membrane	0.432	−0.114
C/D:		
Pit Depth	0.287	0.152
Bruch's Membrane	0.704	−0.055
Axial Length:		
Pit Depth	0.820	0.033
Bruch's Membrane	0.333	−0.141

six monthly injections showed smaller, insignificant BMO changes, yet size was still significantly increased when compared to baseline.[10] Contrasting with previous studies, we did not find the change in BMO to be significant post-IVI.[10] We further determined no significant relationship between the assessed categorical and continuous variables and an acutely increased change in BMO.

Several studies have evaluated the impact of intravitreal injections (IVI) on intraocular pressure (IOP). Immediately after IVI, IOP can increase by 11.9 to 39.1 mmHg for an injected volume of 0.05 mL,[17] 34.49 to 52.43 mmHg for an injected volume of 0.07,[18] 14.6 to 47.1 mmHg with an injected volume of 0.09,[19] and 28.9 to 52.7 mmHg for an injected volume of 0.1.[20] There has been a reported exponential increase in the IOP values with reduced length of the globe, with more pronounced changes seen in eyes with shorter axial lengths. This may explain the less pronounced increase in BMO we observed in longer eyes, though the correlation was weak ($r = -0.141$).[20,21]

Prior investigations on the chronic effects of IVIs have shown significant long-term effects of regularly scheduled IVIs on Bruch's membrane expansion, prelaminar tissue thinning, and cup deepening one year following the initial IVI, although no significant long-term changes in IOP were observed at this time.[10] BMO was determined to significantly enlarge by 27 μm in eyes receiving more than six IVIs compared to the pre-injection baseline.[10] After six IVIs, progressive prelaminar tissue thinning and cup deepening in the inferior sector were observed, correlating with lower collagen density and greater susceptibility to strain.[10] Though an inverse correlation between prelaminar tissue thinning and cup deepening has been identified,[10] enlargement of the ONH may also result from direct laminar deformation.[22] It has been found that the ONH dynamically responds to changes in IOP. In the proposed mechanical behavior, increased IOP results in scleral expansion and a subsequent expansion of the scleral canal, pulling the laminar cribrosa taut into a maximally anterior position in conjunction with the retrolaminar tissue pressure.[23,24] The pressure applied to the sclera by the IOP redistributes as a circumferential "hoop stress" pushing the lamina outward. At some level of IOP, this outward force produces a net posterior displacement of the lamina.[23,24] Models further suggest that the compliance of the sclera determines the extent of laminar deformation.[23] It has been proposed that the aged ONH should demonstrate less deformation for a given IOP insult based on greater likelihood for stiffened peripapillary scleral and lamina cribrosa connective tissue and laminar beam stiffness.[22,23,25] Despite this, our study did not determine a significant correlation between age and the change in pit depth or BMO post-IVI. This may be due to a limited variance in age within the sample, as the majority (84.0%) of patients were aged 70 years or greater. Alternatively, the absence of this pattern may reflect a more complex relationship between age and ONH stiffening in the context of repeated IVIs, which may obscure a simple linear relationship.

We found a significant relationship between the change in pit depth and the number of injections a patient previously received, with more injections corresponding to a smaller change in the pit depth post-IVI. While a long-term increase in IOP in patients undergoing repeated IVIs has been established, the chronic effects on the ONH or glaucoma progression remain undetermined.[26] Acute IOP spikes following IVI have been associated with thinning of the RNFL within 2 to

5 min specifically in the nasal and inferior RNFL. There is a lack of studies on chronic glaucomatous changes in the ONH in response to repeated IVIs.[27] A recent study found a significant negative correlation between RNFL thickness and lamina cribrosa strain, suggesting that a stiffer ONH may be more vulnerable to damage.[28] Reduced pit depth movement may reflect lamina cribrosa stiffening. Over time, such structural changes could predispose to RNFL thinning. This pattern suggests a potential association between repeated IVIs and glaucomatous changes although further studies are needed to clarify this relationship.

Given the chronic effects of IVIs on the RNFL, studies have been conducted to mitigate the effect of repeated increased IOP with IVIs on RNFL damage over time. One study compared RNFL thinning in patients receiving IVIs alone versus those also undergoing prophylactic anterior chamber paracentesis (ACP). Notably, the ACP group showed no significant RNFL thinning.[29] Acutely, IOP spikes cause posterior deflection of the lamina cribrosa predominantly near the periphery, potentially preserving structural stability in central regions critical for vision by minimizing strain in central vasculature and the nerve fibers.[30] In a theoretical model, larger scleral ONH canal diameter and thinner sclera increase IOP-related stress at a given pressure, increasing susceptibility to damage.[31]

Conclusion

Ultimately, we found that the number of injections received in an eye correlated to a lesser increase in pit depth following IVI, suggesting sustained structural changes of the ONH with repeated IVI. While no demographic category was found to be at significant risk for greater post-IVI changes in pit depth or BMO in the acute setting, the literature suggests that patients receiving long-term IVI therapy may be at a greater risk for complications.[21] Given the relationship between ONH stiffening and RNFL thinning, it is worth considering that repeated IVIs may contribute to glaucomatous changes.

This study had some limitations. Natural anatomical variation of the ONH between patients required careful evaluation to ensure data reliability. We chose the pit of the ONH as a reference point to measure the variation in the ON anatomy post IVI, as the quality of the OCT scans, mainly post IVI, did not allow a clear distinction between the lamina cribrosa and the prelaminar tissue. The betadine solution used to sterilize the eye for IVIs resulted in epithelial keratopathy, leading to reduced post IVI OCT image quality. Sample demographics limited the ability to draw generalized conclusions across all populations. An additional limitation of the study is the small sample size and the lack of longitudinal data. This study was not designed to evaluate the long-term effect of the acute rise in IOP following IVI. Rather, it aimed to address the gap in the literature regarding acute anatomic changes in the ONH structure post-IVI. Future studies with more stringent inclusion and exclusion criteria, a larger sample, and additional long-term follow-up may help reduce variability and increase statistical power. Despite these limitations, we believe these findings offer valuable preliminary insight into the acute ONH biomechanics following IVIs, which may have the potential to lead to sustained structural modification and subsequent glaucomatous changes.

Abbreviations

IVIs, intravitreal injections; ONH, optic nerve head; BMO, Bruch's membrane opening.

Data Sharing Statement

Data is available upon request from the corresponding author.

Ethics Approval and Informed Consent

This study was performed in line with the principles of the Declaration of Helsinki and approved by the UAMS Institutional Review Board. Informed consent was obtained from all individuals included in the study.

Consent for Publication

Consent for publication was obtained.

Author Contributions

All authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare they have no competing interests for this work.

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