Observation of a pulsatile choroidal vascular lesion in a patient with polypoidal choroidal vasculopathy during vitrectomy

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ARTICLE INFO

Keywords:
Polypoidal choroidal vasculopathy
Pulsatile choroidal lesion

ABSTRACT

Purpose: To describe a patient in whom a pulsating choroidal vascular lesion was observed during vitrectomy for the treatment of vitreous hemorrhage secondary to polypoidal choroidal vasculopathy (PCV).

Observations: A 67-year-old Chinese male was referred to our department for the treatment of vitreous hemorrhage secondary to PCV in his right eye. PCV was confirmed during surgery, accompanied by extensive vitreous and subretinal hemorrhage, and exudation in the posterior pole and temporal to the fovea. A reddish-orange pulsatile lesion was observed at the inferotemporal fundus. The pulsatility of the lesion varied with changes in the intraocular infusion pressure. The pulsating lesion was not visible on fluorescein and indocyanine green angiography performed 1 month after surgery.

Conclusions and importance: A pulsatile choroidal vascular lesion was observed during vitrectomy. Future studies elucidating the relationships between pulsation, intraocular pressure, and blood flow would help our understanding of the hemodynamics of polypoidal choroidal vessels.

1. Introduction

Fundus fluorescein angiography (FFA) and indocyanine green angiography (ICGA) are the gold-standard imaging techniques for the diagnosis of polypoidal choroidal vasculopathy (PCV). Both imaging methods transiently show the choroidal circulation from the retinal and choroidal arteries to the veins during the dye-filling phase. This circulation was originally described as a branching vascular network that formed polypoidal lesions. Pulsatile choroidal polyps were observed in previous studies that used fundus angiography to assess the choroidal hemodynamics, and provided important clues to understand the pathogenesis of PCV.\textsuperscript{1–3} Here we report an interesting finding: a pulsating choroidal vascular lesion that was visible during vitrectomy in a patient undergoing treatment of vitreous hemorrhage secondary to PCV. We manipulated the intraocular infusion pressure (IOIP) to observe changes in the pulsatility of the lesion. The findings might aid our understanding of the hemodynamics of choroidal vessels.

2. Case report

A 67-year-old Chinese male was admitted with a 4-month history of impaired vision in his right eye. The affected eye’s visual acuity was hand movement and the intraocular pressure (IOP) was 14 mmHg. The patient was suspected of having extensive vitreous hemorrhage secondary to PCV, which had been diagnosed 3 years earlier.

Pars plana vitrectomy combined with phacoemulsification and intraocular lens implantation were performed using the NGENUITY\textsuperscript{®} three-dimension visualization system and Constellation\textsuperscript{®} operation system (Alcon Laboratories, Fort Worth, TX, USA). The surgical findings confirmed PCV with extensive vitreous and subretinal hemorrhage, and exudation in the posterior pole and temporal to the fovea (Video).

A reddish-orange pulsatile vascular lesion was noticed at the inferotemporal fundus. The pulsation rate was 72 beats per minute (bpm) at an IOIP of 25 mmHg (millimeters of mercury), and increased to 78 bpm when the IOIP was raised to 35 mmHg. For the sake of clinical safety, IOIP was not raised above 35 mmHg, since the central retinal artery pulsation was observed at the IOIP of 35 mmHg. The pulsatility slowed considerably, to 35–36 bpm, when the IOIP was lowered to 15–20 mmHg and was almost undetectable at an IOIP of approximately 8 mmHg (Fig. 1 and Video). Vitrectomy was performed with intraoperative photocoagulation around the pulsating lesion.

Supplementary video related to this article can be found at https://doi.org/10.1016/j.ajo.c.2022.101526

Received 14 September 2021; Received in revised form 24 March 2022; Accepted 2 April 2022
Available online 10 April 2022
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The patient’s blood pressure during the surgery was around 150/85 to 160/90 mmHg and his heart rate was 75–82 bpm, as recorded by an intraoperative electrocardiographic monitoring system.

FFA combined with ICGA were performed one month after vitrectomy. A choroidal lesion was confirmed at the inferotemporal fundus corresponding to the intraoperative pulsating reddish-orange lesion (Fig. 2). However, no pulsation was detected during angiography. The subretinal hemorrhage gradually resolved during 6 months of follow-up. The right eye’s best-corrected visual acuity was 0.2 (Snellen equivalent 20/100) and the IOP was 11.6 mmHg at the patient’s last visit.

3. Discussion

The phenomenon of pulsatile or turbulent choroidal blood flow has been described in studies using ICGA.8–10 To our knowledge, the most interesting finding of the present case is that pulsatile movements of polypoidal vessels were visible ophthalmoscopically during vitrectomy. This pulsatility was visible for almost the entire surgical procedure. To our knowledge, this is the first time this has been reported.

PCV was first named by Yanuzzi in 1990 as choroid orange nodules.4 Recent studies using swept-source optical coherence tomographic angiography have revealed that the internal structures of polypoidal lesions have been suggested to present as aneurysmal dilations, tangled vascular networks, or even a mixture of both morphologies.5–7 Under normal circumstances, the pulse in the small branching vessels is too weak to be observed. The pulsating polypoidal lesions may indicate the sites of arteriovenous shunts or arteriovenous crossings in the choroidal vasculature, and the small arteriole supplying the pulsating lesion might be stenotic.1,3 According to Starling’s law of resistance, the inflow of blood causes a gradient increase in the local pressure within the vessel near the constriction, resulting in flow instability and vessel wall oscillation.5,9

Previous studies reported that following trabeculectomy (with IOP lowered), blood flow was increased and resistive index was decreased in the short posterior ciliary arteries, which supply the choroid.10,11 This might have resulted in the increased choroidal blood flow and more pulsation might be noticed if other mechanical factors were not altered. However, as IOP is lowered, the external pressure on the flexible blood vessel is reduced, and the constriction of blood vessels could become relaxed, making the blood flow less turbulent and the pulsation less obvious. Therefore, the change of pulsatility was the result of the balance among blood pressure, resistance of vessel wall, and extent of the vessel narrowing, all of which might be part of blood flow autorregulation.5,12 A mathematical model developed by Guidoboni did show that patients can regulate retinal blood flow as IOP varies within certain limits.8 In addition, the relationship of IOP and pulsatility might not be linear (Fig. 1), indicating vessel autorregulation during the change of hemodynamics.

In our observation, the pulsatile rate was decreased and blood flow was less obvious when the IOP was lowered from 35 mmHg to 8 mmHg, suggesting that relaxation of the constriction at the pulsating vessel (instead of blood flow increase) plays a key role in the change of pulsation. Therefore, it might be speculated that if the IOP exceeded a certain level, the constriction point of the vessel narrowed further, making the blood flow more turbulent and pulsation more prominent. We speculated that, if the IOIP was raised above 35 mmHg, the pulsatility would have reached a plateau, synchronizing with the patient’s cardiac rhythm, which was 75–82 bpm during the surgery. In 2008, Watanabe et al. used laser speckle flowgraphy and reported that the pulsatility of polypoidal lesions synchronized with the cardiac rhythm.2 Based on our findings, the pulsatility at an IOIP of 15–20 mmHg was approximately half of the heart rate, and was thus not synchronized with the patient’s heart rate. The different locations of the pulsating lesions might account for this discrepancy. Most of the previously reported pulsatile polyps were located in the posterior pole, a region with exceptionally high blood flow.1–3,13,14 Therefore, the pulsatility of these polyps showed near-perfect synchronization with the heart rate. However, in our patient, the pulsatile lesion was located in a peripheral region (inferotemporal fundus). The pulsatile movements might be less visible in peripheral vessels because the pulse pressure of the inflowing branching arterioles is much lower than that of the main arteries.9,12 An increase in the IOIP might cause narrowing of the vessel wall at the point of constriction, increasing the visibility of the pulsation, which might eventually reach a plateau equivalent to the heart rate.

4. Conclusions

We have reported an interesting finding of a pulsatile choroidal vascular lesion located in the inferotemporal fundus that was visible during vitrectomy. The pulsation was visualized during surgery and was more visible when the IOP was raised. Future studies elucidating the relationships between pulsation, IOP, and blood flow would help us to...
better understand the hemodynamics of polypoidal choroidal vessels.

**Patient consent**

The patient provided verbal consent for the publication of the case.

**Author contributions**

Gezhi Xu: conceptualization, supervision and manuscript review. Ting Zhang: formal analysis; investigation and writing the original draft. Chen Jiang and Fang Song: data curation.

**Funding**

This work was supported by the Clinical Research Plan of SHDC (Shanghai clinical three-year action plan-major clinical research project) (SHDC2020CR2041B).

**Authorship**

All authors that they meet the current ICMJE criteria for authorship.

**Declaration of competing interest**

The authors declare that they have no known competing interests.

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