



Case Report of Terson's Syndrome Suggesting Pathway of Subarachnoid Hemorrhaged Blood into Eye

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Authors' contributions

This work was carried out in collaboration between all authors. Author YC treated the patient, collected the patient data and prepared the manuscript. Author MT treated the patient and managed the analyses of the study. Authors YK and HY collected the patient data and managed the literature searches. Author SO managed the analyses of the study. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

Aims: The pathway of subarachnoid hemorrhaged blood into the eye in Terson's syndrome has not been definitively determined. We present our findings in a patient with Terson's syndrome that provided clues on how the subarachnoid hemorrhaged blood entered the eye.

Presentation of Case: A 47-year-old man visited our department three months after a subarachnoid hemorrhage. His visual acuity was hand motion in the right eye and light perception in the left eye. B-scan ultrasonography showed dense vitreous opacities in both eyes and a total retinal detachment in the left eye. Vitrectomies were performed on both eyes. An old subretinal

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hemorrhage was found in the right eye. A circular opacity firmly attached to the posterior capsule of the lens was found during the vitrectomy on the left eye. His final visual acuity was 20/20 in the right eye and 20/400 in the left eye.

Discussion and Conclusions: The subretinal hemorrhage in the right eye suggests the blood spread along the choroid. The localized circular sublenticular opacity in the left eye suggests that the blood reached the posterior capsule of the lens through the hyaloid canal. A review of the development of the eye suggests that the blood can flow into the eye through the subarachnoid space in the choroid fissure and the choroid in eyes with Terson's syndrome.

Keywords: Terson's syndrome; development of eye; hyaloid canal; choroid fissure; choroid.

1. INTRODUCTION

Intraocular hemorrhage associated with subarachnoid hemorrhage (SAH) is called Terson's syndrome. Ko et al. [1] presented the ocular pathology of Terson's syndrome from their findings in 30 eyes: subarachnoid hemorrhage along the optic nerve was present in 20 eyes, vitreous hemorrhage in 11 eyes, diffuse subhyaloid hemorrhage in 10 eyes, intraretinal hemorrhage in all eyes, and subretinal hemorrhage in 14 eyes. Computed tomography [2] and magnetic resonance imaging [3] have shown that the hemorrhaged blood traverses to the optic nerve sheath in patients with Terson's syndrome. However, because the optic nerve sheath is not continuous with the intraocular space anatomically, the exact pathway of how blood enters the eye has not been definitively determined [1-7]. One commonly accepted explanation is that the increased intracranial pressure is transmitted through the optic nerve sheath to the retina, causing ruptures of thin retinal vessels [4,5]. However, there are other publications which indicated that the subarachnoid hemorrhaged blood flowed into the eye without ruptures of retinal vessels [3,6,7].

We present our findings in a patient with Terson's syndrome that provided clues on how the blood entered the eye.

2. PRESENTATION OF CASE

A 47-year-old healthy man with good vision had a sudden loss of consciousness on December 7, 2014, and immediately underwent surgery to clip a ruptured aneurysm of the left internal carotid artery at C2. He recovered from the surgery, but a severe visual impairment developed in both eyes. His mental condition became extremely unstable during the rehabilitation. He was forcibly admitted to the psychiatry department of Kohnodai Hospital on February 15, 2015, and

then referred to our department for ophthalmic examinations on February 17. His visual acuity was hand motion in the right eye and light perception in the left eye. Ophthalmoscopy showed dense vitreous opacities in both eyes, and the fundi were not visible. B-scan ultrasonography showed dense vitreous opacity with a posterior vitreous detachment in the right eye, and dense vitreous opacity with a total retinal detachment in the left eye (Fig. 1).

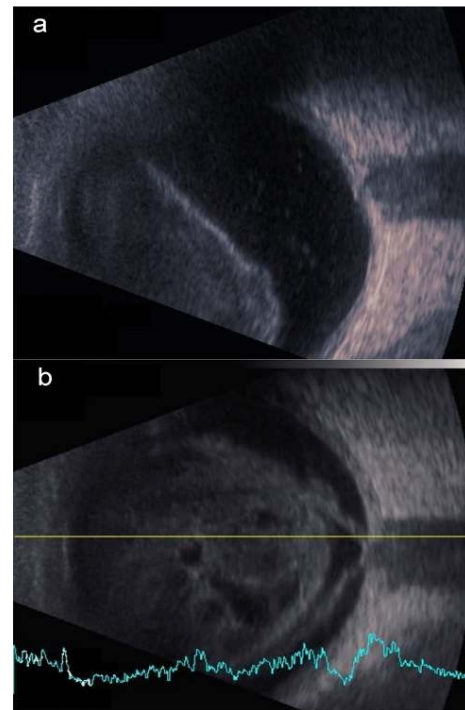


Fig. 1. B-scan ultrasonographic images showing dense vitreous opacity with a posterior vitreous detachment in the right eye (a), and dense vitreous opacity with a retinal detachment in the left eye (b)

Pars plana vitrectomy of the right eye was performed under general anesthesia on

February 25, 2015. After removing the dense vitreous opacity, an old subretinal hemorrhage was found in the superior-temporal arcade area. The fovea and the optic disc were intact. His best-corrected visual acuity (BCVA) improved to 20/20 postoperatively. His mental condition also became stable. Postoperative fundus photograph and optical coherence tomography showed that the hemorrhage was in the subretinal space (Fig. 2a, 2b). The subretinal hemorrhage disappeared six months after the surgery.

Pars plana vitrectomy of the left eye was performed under general anesthesia on March 5th. Following combined phacoemulsification and aspiration, the intraocular space could still not be seen clearly because of a circular hard opacity firmly attached to the posterior capsule of the lens. We used the vitreous cutter to remove the opacity until the intraocular space became

visible. The vitreous cavity was filled with old hemorrhage (Fig. 3). After cutting the core vitreous, we noted that the posterior vitreous was still attached to the optic disc, and created a posterior vitreous detachment. A total retinal detachment was found due to a hole in the superior-temporal arcade with proliferative vitreoretinopathy. We removed the hemorrhage as much as possible and then peeled off the epiretinal membrane. Finally, we injected silicone oil to tamponade the retina. A retinal reattachment was obtained after two additional vitrectomies because of the recurrences of the retinal detachment. His BCVA in the left eye was 20/400 at the last examination on August 23, 2016. Postoperative fundus photograph and optical coherence tomography showed that the retina was reattached with degeneration and an epiretinal membrane under the silicone oil (Fig. 2c, 2d).

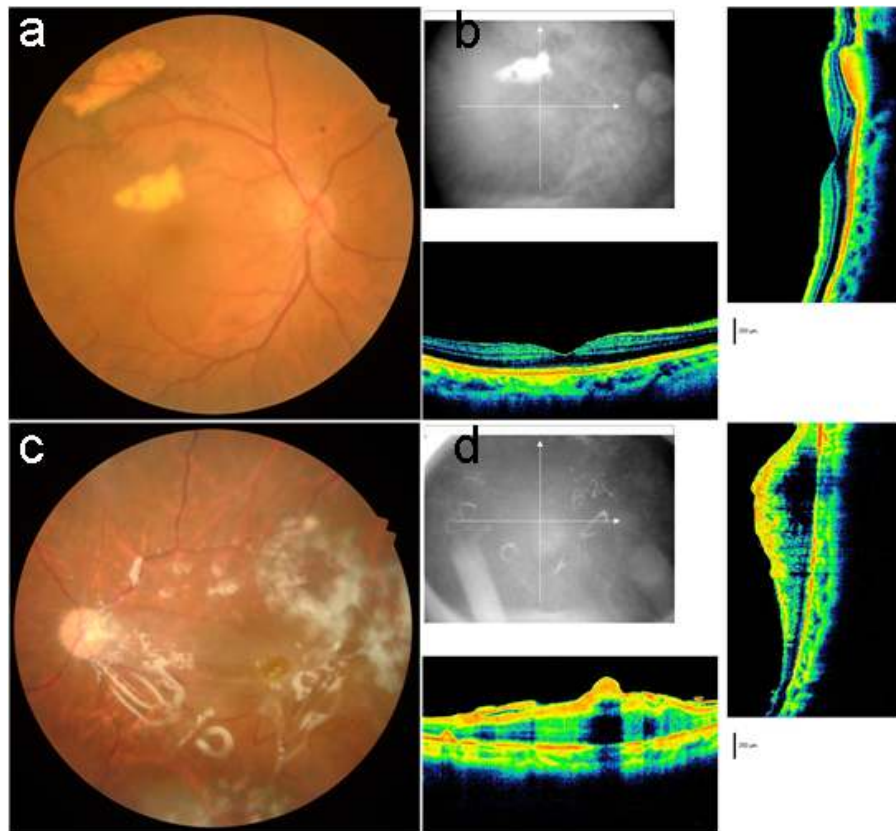


Fig. 2. Postoperative fundus photographs and optical coherence tomographic images showing an old subretinal hemorrhage in the superior-temporal arcade area in the right eye (a, b), and reattached retina with degeneration and an epiretinal membrane under the silicone oil in the left eye (c, d)

3. DISCUSSION

The pathway of subarachnoid hemorrhaged blood into the eye in Terson's syndrome has not been definitively determined. Doubler et al. [6] suggested that the blood entered the vitreous space directly by passing through the lamina cribrosa in 1917. Because there is little evidence for a connection between the optic nerve sheath and the vitreous, this suggestion has not been well accepted. Manschot reported that an injection of a mixture of gelatin and India ink into the suboccipital subarachnoid space of human cadavers spread to the subarachnoid space in the optic canal, and he suggested that the hypertension in the optic nerve sheath could lead to closure of the central retinal vein resulting in ruptures of the retinal veins [4]. Medele et al. [5] believed that an acute increase of intracranial pressure could be transmitted through the optic nerve sheath to the retina, causing ruptures of thin retinal vessels. Sakamoto et al. [3] made a new suggestion that the blood may enter beneath the internal limiting membrane through the perivascular space surrounding the central retinal vessels and the massive sub-internal limiting membrane hemorrhage may rupture the internal limiting membrane, resulting in the vitreous hemorrhage. The vitreous hemorrhage in our case might be explained by the ruptures of the intraretinal hemorrhage, however when a posterior vitreous detachment is not present, the hemorrhage is supposed to be in the subhyaloid space and it would be difficult to spread through the vitreous to the sublenticular area. In addition, the subretinal hemorrhage in the right eye was also difficult to be explained by the ruptures of the retinal vessels. So, we believe that there must be other pathways for the subarachnoid hemorrhaged blood to enter the eye.

According to Langman's Medical Embryology [8], the eye primordium is completely surrounded by loose mesenchyme at the end of the 5th week of pregnancy. At the end of the 6th week, the optic cup and optic stalk invaginate inferiorly, forming the choroid fissure. The mesenchyme not only surrounds the eye from the outside but also invades the inside of the optic cup by way of the choroid fissure. Outside the eye, the mesenchyme soon differentiates into an inner layer comparable with the pia mater of the brain and outer layer comparable with the dura mater. The inner layer later forms a highly vascularized pigmented layer known as the choroid; outer layer develops into the sclera and is continuous with the dura mater around the optic nerve.

Inside the eye, the mesenchyme forms the hyaloid vessels, which supply the lens and form the vascular layer on the inner surface of the retina. In addition, the mesenchyme forms a delicate network of fibers between the lens and retina known as the vitreous body. The hyaloid vessels disappear during fetal life, leaving a clear zone through the center of the vitreous called the hyaloid canal. The proximal portion of the hyaloid vessels remains as the central retinal vessels (Fig. 4). Sebag showed a photomicrograph of a human eye during early embryogenesis which demonstrated the existence of the choroid fissure [9]. These embryonic features suggest that the subarachnoid space around the optic nerve is continuous with the vitreous, the retina, and the choroid during fetal life. The subarachnoid space in the choroid fissure might be able to serve as a connection between the optic nerve sheath and the vitreous and retina. The subarachnoid space in the choroid might be able to serve as a connection between the optic nerve sheath and the choroid.

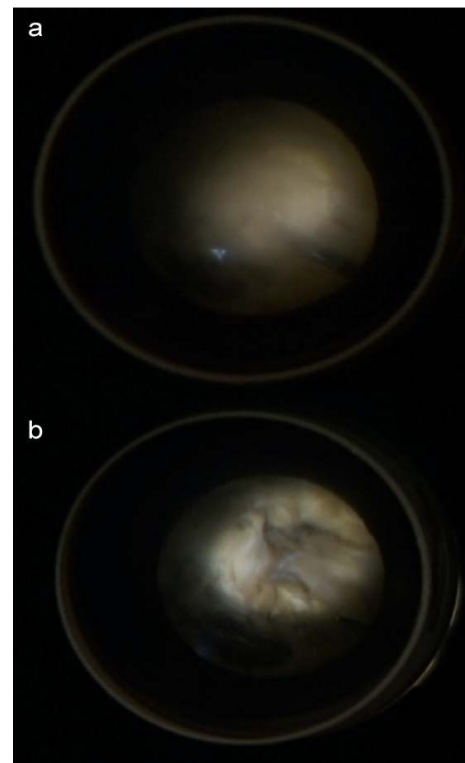


Fig. 3. Intraoperative views of the left eye showing a circular opacity firmly attached to the posterior capsule of the lens (a), and old vitreous hemorrhage after removing the opacity (b)

On the basis of the development of the eye, we present new possible pathways for the blood to enter the eye in Terson's syndrome to explain our intraoperative findings: The blood from the ruptured intracranial aneurysm spreads to the subarachnoid space around the optic nerve

resulting in hypertension in the optic nerve sheath. The acute hypertension might lead to a temporary reopening of the subarachnoid space in the choroid fissure and the choroid and allow the blood to enter the vitreous, retina and choroid (Fig. 5). In our case, the blood spread along the

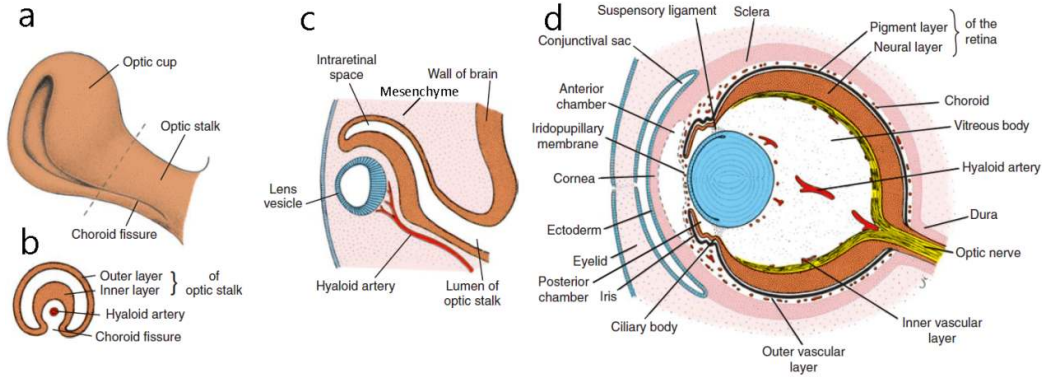


Fig. 4. Embryological development of the eye (permitted by Wolters Kluwer Health)

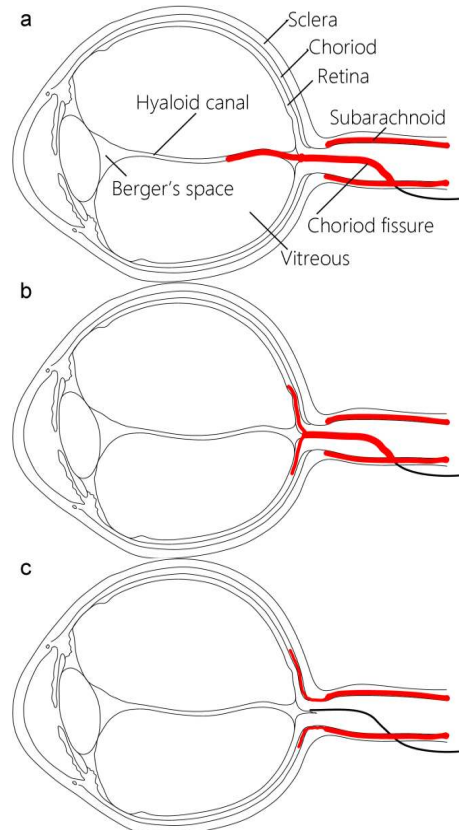


Fig. 5. Schema of the blood pathways into the eye in Terson's syndrome. a) Blood flows into the subarachnoid space in the choroid fissure and then spreads directly from the optic disc into the vitreous causing vitreous hemorrhage. b) Blood flows into the subarachnoid space in the choroid fissure and then spreads along the perivascular space surrounding the central retinal vessels causing intraretinal hemorrhage. c) Blood spreads along the choroid causing subretinal hemorrhage

choroid causing the subretinal hemorrhage and the blood also flowed into the vitreous causing the vitreous hemorrhage in the right eye. In the left eye, the blood spread from the optic disc through the hyaloid canal into the vitreous causing the vitreous hemorrhage. Because more blood flowed into the left eye than into the right eye, the blood even reached the posterior capsule of the lens and formed the circular sublenticular opacity. The retinal hole and the retinal detachment were caused by the traction of the epiretinal membrane induced by the old vitreous hemorrhage.

Our B-scan ultrasonography failed to detect the route of the hemorrhage through the hyaloid canal, because three months had passed since SAH. However, Doubler et al. [6] reported that a fresh hemorrhage located on the disc grew in size until the disc was completely obscured from repeated ophthalmoscopic examinations of a patient in the first hours after SAH, who died 6 hours after the admission. Autopsy showed a large, chocolate brown mass of clot completely overlying the disc, extending in an irregular manner into the vitreous. Michalewska et al. [7] investigated nine eyes by optical coherence tomography, scanning laser ophthalmoscopy and intraoperative images, and suggested that there were three possible pathways for blood to enter the eye: direct spread into the vitreous from the optic disc area, along the retinal vessels into the retinal layers, and the subretinal pathway. Sakamoto et al. [3] showed magnetic resonance images of a patient two days after SAH demonstrating an area with high signal intensity along the central portion of the optic nerve which indicated that the blood flowed into the center of the optic nerve. Although the blood pathways through the choroid fissure and the choroid were not discussed in these publications, these previously reported findings could support our hypothesis. Further histopathological studies will be required to test our hypothesis.

4. CONCLUSION

The subretinal hemorrhage in the right eye suggests the blood spread along the choroid. The localized circular sublenticular opacity suggests that the blood reached the posterior capsule of the lens through the hyaloid canal. A review of the development of the eye suggests that the blood can flow into the eye through the subarachnoid space in the choroid fissure and the choroid in eyes with Terson's syndrome.

CONSENT

All authors declare that 'written informed consent was obtained from the patient for publication of this case report and accompanying images'.

ETHICAL APPROVAL

It is not applicable.

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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