



Brief Report

Microsurgical observation of the posterior vitreous in patients with vitreous hemorrhage caused by Terson syndrome

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ABSTRACT

Purpose: To describe features characteristic of vitreous hemorrhage in patients with Terson syndrome observed through a microsurgical scope.

Methods: Between May 2015 and February 2019, 12 eyes of 10 patients with vitreous hemorrhage occurring after subarachnoid hemorrhage (SAH) underwent pars plana vitrectomy.

Results: During vitreous surgery, we found 10 of 12 eyes did not have posterior vitreous detachment (PVD). Furthermore, we observed in 9 of the 10 eyes without PVD (90.0%) that there was no hemorrhage in the posterior vitreous cavity at the posterior pole while we removed vitreous hemorrhage. We confirmed that this clean space could be the posterior precortical vitreous pocket (PPVP).

Conclusion and importance: Terson syndrome may have no hemorrhage in the PPVP regardless of the presence of severe vitreous hemorrhage. The cases presented in our study may suggest one of the mechanisms of Terson syndrome.

1. Introduction

Terson syndrome is recognized as intraocular hemorrhage associated with subarachnoid hemorrhage (SAH) or, intracerebral hemorrhage.¹ Hemorrhage may be present in the vitreous, sub-hyaloid, or intraretinal/sub-internal limiting membrane (ILM). Although there have been many case reports of Terson syndrome^{2–7} since Litten⁸ first described the occurrence of vitreous hemorrhage associated with SAH in 1881, the characteristics of vitreous hemorrhage in Terson syndrome remains unclear.

Recently, swept source optical coherence tomography (SS-OCT) has enabled visualization of the morphologic change of the posterior vitreous on the macula such as premacular membrane or vitreomacular traction.^{9–11} However, even with OCT, it remains very difficult to observe precisely the morphologic features of the posterior vitreous in a patient with severe vitreous hemorrhage.

The purpose of the present study was to evaluate the characteristic features of the posterior vitreous in patients with vitreous hemorrhage caused by Terson syndrome through a microsurgical scope.

2. Methods

This study followed the tenets of the Declaration of Helsinki and

abided by the regulation of the local ethics community (Ethics committee at Nakamura Memorial Hospital). From November 2015 to February 2019, 222 patients with SAH were admitted to our emergency institute at Nakamura Memorial Hospital. We conducted bedside ophthalmoscopic exams of all the patients within 14 days after onset of SAH. Among the patients, 25 (11.3%) showed vitreous, sub-ILM, or subretinal hemorrhage and 11 (4.9%) showed intraretinal hemorrhage without vitreous hemorrhage.

10 patients (12 eyes) with Terson syndrome underwent a 25-gauge pars plana vitrectomy (PPV), which was followed by a phacoemulsification procedure and intraocular lens (IOL) implantation for 8 of the eyes. We retrospectively analyzed the video records of vitrectomy for these patients.

3. Results

The baseline characteristics of the 10 patients (12 eyes) with Terson syndrome who underwent PPV are designated in [Table 1](#). Their mean age was 53.3 ± 8.0 years. The ratio of men and women was 1 to 1. The origins of SAH included ruptured anterior cerebral artery aneurysms (ACA AN) (n = 3), anterior communicating artery aneurysms (Acom AN) (n = 3), middle cerebral artery aneurysms (MCA AN) (n = 1), posterior inferior cerebellar artery aneurysms (PICA AN) (n = 1), and

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Table 1
Clinical characteristics of patients with Terson syndrome who underwent PPV.

Case No.	Age (yrs), Sex	Origin of Hemorrhage	Eye	Preoperative		Surgery	PVD	PPVP	Hemorrhage in PPVP	Postoperative BCVA	Other clinical features
				BCVA	HM						
1	54, M	MCA AN	OD	HM	PEA + IOL + PPV	-	+	-	20/20		
2	64, M	Acom AN	OS	HM	PEA + IOL + PPV	-	+	-	20/20		
3	55, M	ACA AN	OS	20/200	PEA + IOL + PPV	-	+	-	20/20		
4	56, F	ACA AN	OD	HM	PEA + IOL + PPV	+	unknown	unknown	20/28	macular hole	
5	67, F	Acom AN	OS	20/2000	PEA + IOL + PPV	+	unknown	unknown	20/20		
6	43, F	ACA AN	OD	20/20	PPV	-	unknown	unknown	20/16		
7	54, F	PICA AN	OS	HM	PEA + IOL + PPV	-	+	-	20/25		
8	50, M	VA dissection	OD	HM	PEA + IOL + PPV	-	+	-	20/50	corneal opacity	
			OS	HM	PEA + IOL + PPV	-	+	-	20/16		
9	44, M	VA dissection	OD	CF	PPV	-	+	-	20/25		
			OS	20/200	PPV	-	+	-	20/20		
10	46, F	Acom AN	OS	LP	PPV	-	+	-	20/20		

BCVA, best corrected visual acuity. HM, hand motion. CF, count of fingers. LP, light perception. PEA, phacoemulsification and aspiration.

vertebral artery (VA) dissection (n = 2). All patients underwent PPV safely without any complications and improved visual acuity. In case 4, the macular hole was found after removing dense vitreous hemorrhage, so the patient was additionally treated with ILM peeling and 20% SF₆ gas tamponade. During vitreous surgery, we found 10 (83.3%) of 12 eyes did not have posterior vitreous detachment (PVD). A PVD was surgically induced in patients with incomplete PVD or normal posterior vitreoretinal interface. We observed the posterior vitreous space without hemorrhage in front of the posterior pole regardless of severe vitreous hemorrhage in 9/10 (90.0%) without PVD. In case 6, we could not find the presence of this clear space because there was only mild vitreous hemorrhage.

4. Discussion

In a 1990 study, Kishi et al.¹² examined the vitreous of 84 human autopsy eyes by scanning electron microscopy with a fluorescein staining technique and found the presence of bursa premacularis, which they termed posterior precortical vitreous pocket (PPVP). It is now understood that the PPVP is located almost immediately anterior to the posterior fundus surrounded by the temporal vascular arcades and can sometimes be visualized through the translucent formed vitreous during TA-assisted vitrectomy.^{13–15} In our study, we confirmed that the clean posterior vitreous space through a microscope in almost all the patients without PVD could be the PPVP because we could observe bag-shaped space and posterior vitreous cortex dyeing by injection of TA (Video 1).

Supplementary video related to this article can be found at <https://doi.org/10.1016/j.ajoc.2020.100613>.

There are several possible pathophysiologic mechanisms for Terson syndrome. One possible mechanism is that the arterial blood from the SAH enters the vitreous space directly through the intervaginal space around the optic nerve by penetrating the lamina cribrosa of the sclera.^{16–18} Sakamoto¹⁸ indicated that SAH within the optic nerve sheath may enter under the ILM through the perivascular space surrounding retinal vessels from magnetic resonance imaging (MRI) findings in a patient with Terson syndrome. We tried to analyze the MRI findings in several of our patients utilizing the same gradient echo sequence as Sakamoto. However, we could not detect the features suggesting the perivascular space such as Virchow-Robin space, which Sakamoto described, along the central retinal vessels within the retrobulbar optic nerve because it was too small to be demonstrated precisely on 3-T MRI of the orbit even using thin-slice high-resolution fast gradient-echo technique such as 3D turbo flip low angle shot (FLASH) sequence which reveals clearly arterial blood vessel. Another possible mechanism is that a sudden increase in intracranial pressure leads to rapid effusion of cerebrospinal flow into the optic nerve sheath.

Dilation of the retrobulbar optic nerve then mechanically compresses the central retinal vein and venous hypertension results in a rupture of thin retinal vessels.^{19–25} Naseri et al.²³ reported the patient with Terson syndrome following epidural saline injection and they imply that in Terson syndrome, the source of vitreous hemorrhage is not intracranial, but ocular. Ogawa et al.²⁴ reported that fluorescein angiography demonstrated a leakage site at the disc margin in a patient with Terson syndrome with vitreous hemorrhage. This suggests potential damage to the peripapillary retinal vasculature is induced by increased intracranial pressure transmitted through the optic nerve sheath, which would be expected as a potential source of hemorrhage given the above proposed mechanism. In our study, the clean PPVP was observed regardless of severe vitreous hemorrhage. Itakura et al.⁹ demonstrated that there is a connecting channel between PPVP and Cloquet's canal in 93.1% by SS-OCT. Additionally, the apex of Cloquet's canal, known as Martegiani's funnel, opens on the optic disc. Schaal et al.²⁶ demonstrated in 101/102 eyes that there was a connection between the pre-macular bursa and preoptic area of Martegiani using SS-OCT. Therefore, the presence of clean PPVP in our cases indicates that the arterial blood flow from the SAH may not enter the vitreous space directly through the optic disc. Our results may support the mechanism of Terson syndrome that dilation of the retrobulbar optic nerve due to rapid intracranial pressure mechanically compresses the central retinal vein and that venous hypertension results in a rupture of thin retinal vessels.

In conclusion, Terson syndrome without PVD may have no hemorrhage in the PPVP regardless of severe vitreous hemorrhage. The cases presented in our study may suggest one of the mechanisms of Terson syndrome.

Patient consent

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

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Authorship

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

Declaration of competing interest

The authors have no financial disclosures.

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