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Case Report

Timing of Prophylactic and Early Vitrectomy for First-Presenting or Recurrent Acute Retinal Necrosis Syndrome

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Acute retinal necrosis syndrome (ARNS) is a herpetic infectious eye disease that presents clinicians with difficult decisions to make about the indication and timing of surgical intervention. Here I report 2 patients who underwent prophylactic and early vitrectomy with good visual outcomes. Case 1, a 72–year-old man, had a second recurrence of ARNS in the left eye in 2011 and underwent early vitrectomy in the acute inflammatory phase to remove previously formed vitreous opacity and vitreoretinal adhesions, in parallel with intravenous acyclovir and oral prednisolone administration. He had experienced ARNS in the right eye in 1983, in the left eye in 1986, and a recurrence in the left eye in 1999. Case 2, a 66–year-old woman, developed ARNS in the right eye. All of the circumferential retinal lesions became degenerative with intravenous acyclovir and prednisolone. She underwent a vitrectomy in the post-inflammatory phase, since epiretinal proliferation was noted through vitreous opacity with complete posterior vitreous detachment. These cases suggest that early vitrectomy in the acute inflammatory phase would be indicated for pre-existing vitreoretinal adhesions, while prophylactic vitrectomy in the post-inflammatory phase would be indicated for epiretinal proliferation.

Key words: acute retinal necrosis syndrome (ARNS), early vitrectomy, epiretinal proliferation, prophylactic vitrectomy, herpetic retinitis

Acute retinal necrosis syndrome is a clinically defined entity of herpetic retinitis, occurring in otherwise systemically healthy patients. The extent of retinal involvement with herpetic necrotic lesions differs from patient to patient: in some patients, entire circumferential lesions rapidly become confluent and extend to the posterior pole with sheathing of all retinal arteries, and these lesions are designated as the fulminant type; in other patients, self-limiting necrotic lesions scatter in the fundus, and these are designated the mild type [1, 2]. Vitreous opacity and subsequent formation of vitreoretinal adhesions, caused by vitreous inflammation in association with herpetic retinitis, are major factors leading to rhegmatogenous and traction retinal detachment in the late post-inflammatory phase of the disease.

Vitrectomy, entire circumferential scleral buckling (encircling), and silicone oil tamponade, which are often combined with cataract surgery, are mandatory to achieve retinal reattachment for rhegmatogenous and traction retinal detachment with multiple retinal tears after the fulminant type of acute retinal necrosis syndrome [3]. Early vitrectomy is defined as vitrectomy in the acute inflammatory phase, whereas prophylactic vitrectomy is defined as vitrectomy in the post-inflammatory phase to prevent the supposed development of retinal detachment. These 2 phrases are often used interchangeably. Several case reports

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[4–7] and case series [8, 9] have described successful early and prophylactic vitrectomy in acute retinal necrosis syndrome to prevent retinal detachment. However, the indication and the timing for early and prophylactic vitrectomy in acute retinal necrosis syndrome remain to be determined. In this report, I described early vitrectomy for recurrent mild-type acute retinal necrosis syndrome in the acute inflammatory phase and prophylactic vitrectomy for first-presenting fulminant-type acute retinal necrosis syndrome in the post-inflammatory phase, and discussed the indication and the timing of surgical intervention.

Patients and Methods

Medical records were retrospectively reviewed for 2 consecutive patients who had undergone prophylactic or early vitrectomy with a transconjunctival 25-gauge trocar system at Okayama University Hospital. Informed consent for vitrectomy was obtained from each patient after the risks and benefits of surgical intervention were explained.

Case Reports

Case 1. A 72-year-old man presented with a 2-week history of blurred vision in the left eye in February 2011. His best-corrected visual acuity was 0.5 in the right eye and 0.6 in the left. The intraocular pressure was 15 mmHg in the right eye and 26 mmHg in the left. Each eye had an intraocular lens implantation. The right eye had chorioretinal degeneration but no aqueous inflammation. The left eye had 2+ muttonfat keratic precipitates and several aqueous cells. A large retinal necrotic lesion (Fig. 1D) was observed temporal to the macular area through dense vitreous opacity (Fig. 1C) in the left eye.

He had experienced acute retinal necrosis syndrome in the right eye in July 1983 and in the left eye in May 1986, as described in detail in a previous report [10]. On those 2 occasions, he was given intravenous prednisolone alone but no acyclovir. In March 1999, acute retinal necrosis syndrome recurred in the left eye, and he underwent a 2-week course of intravenous acyclovir 1,500 mg daily, combined with oral prednisolone tapered from 20 mg daily. The superonasal midperipheral necrotic lesions became inactive chorioretinal scars, together with the initial chorioretinal scars mainly located on the temporal side, as described in a previous report [11]. He underwent cataract surgery in both eyes in 2004. In December 2006, he underwent vitrectomy in the right eye for traction retinal detachment caused by dense vitreous opacity and vitreoretinal adhesions with chorioretinal scars (Fig. 1A). Mild vitreous opacity was left behind in the left eye (Fig. 1B). His visual acuity in November 2010 was 0.7 in the right eye and 0.8 in the left.

In 1962, this patient had experienced right upper lung lobectomy for pulmonary tuberculosis and blood transfusion. In December 2003, transfusion hepatitis-screening programs detected that he was positive for hepatitis C virus (HCV) antibody with HCV-RNA 400 IU/mL and genotype 1b. Observation was chosen because there was neither a rise in serum liver enzymes nor abnormalities on ultrasonographic liver examinations. However, when his serum liver enzyme levels rose in October 2008, he was treated with peginterferon-α2a and ribavirin from then until November 2009, successfully eradicating the HCV.

In February 2011, he underwent vitrectomy in the left eye to remove vitreous opacity and to release old vitreoretinal adhesions, together with endo-laser photocoagulation applied to the previously degenerative retina and around the new lesions (Fig. 1E). At the same time, he received a 9-day course of acyclovir 1,500 mg daily, combined with oral prednisolone tapered from 20 mg daily, which was followed by a 2-week course of oral valacyclovir 3,000 mg daily. The necrotic lesions and laser spots became degenerative (Fig. 1F). His visual acuity was 0.5 in the right eye and 1.0 in the left. The aqueous inflammation subsided, and the intraocular pressure returned to a normal level in the left eye, with 0.1% betamethasone, latanoprost, and dorzolamide-timolol eye drops.

Case 2. In March 2011, a 66-year-old woman developed acute retinal necrosis syndrome in the right eye, involving the entire retinal quadrants with total arterial and venous sheathing, together with dense vitreous opacity (Fig. 2A). Her best-corrected visual acuity was 0.05 in the right eye and 1.2 in the left. Intraocular pressure was 28 mmHg in the right eye and 17 mmHg in the left. Her right eye had 2+ muttonfat keratic precipitates but no aqueous cells. She was taking toremifene citrate 40 mg daily after a right-side breast cancer resection combined with sentinel lymph

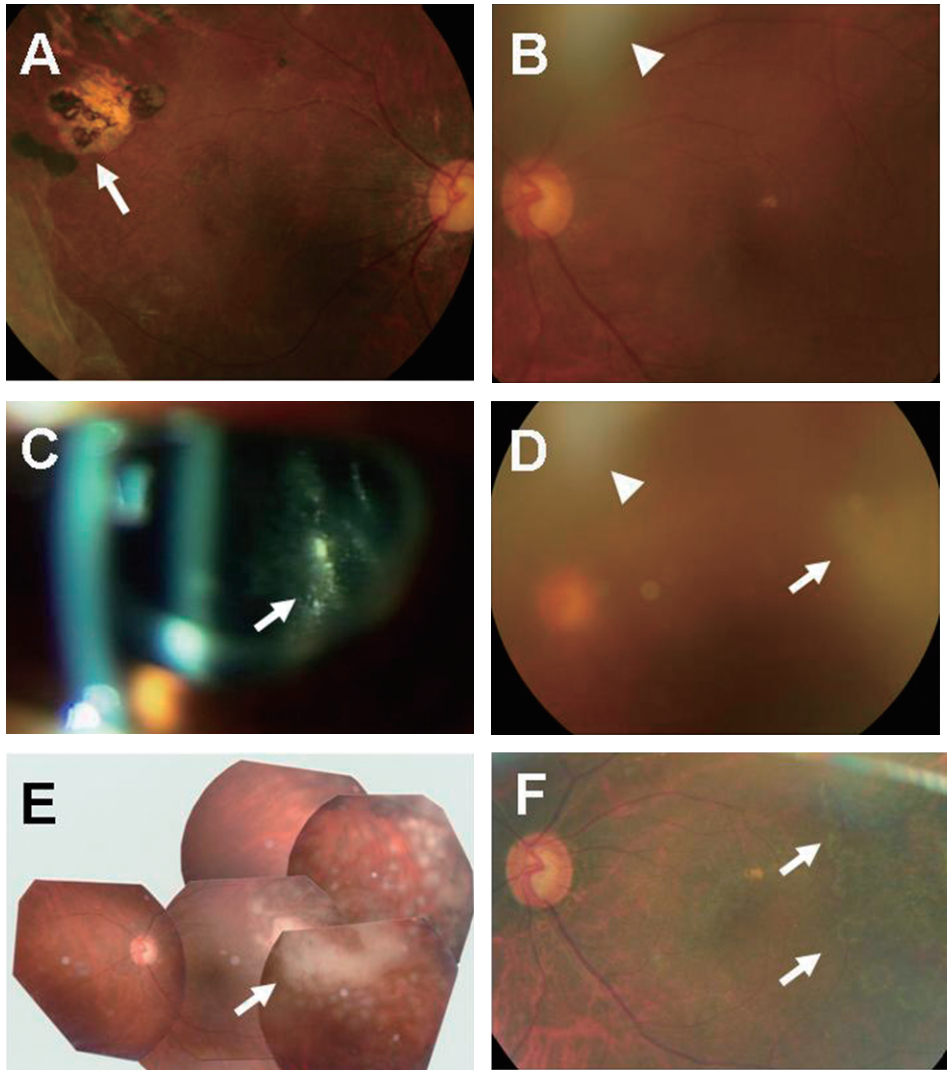

Fig. 1 Case 1. Fundus photographs (**A**, **B**) in July 2010, showing retinal degeneration (arrow in A) on the temporal side with peripheral residual vitreoretinal adhesion after vitrectomy in the right eye (A) and moderate vitreous opacity in the left eye (arrowhead in B) as sequelae to the 2 preceding episodes of acute retinal necrosis syndrome. Slit-lamp biomicroscopic photograph (**C**) and fundus photograph (**D**) in the left eye in February 2011 showing diffuse vitreous opacity (arrow in C) and new retinal necrotic lesions (arrow in D) temporal to the macula, with aggregated dense vitreous opacity superior to the optic disc (arrowhead in D). Merged fundus photographs (**E**) in the left eye, 2 days after vitrectomy, showing retinal necrotic lesions (arrow in E) with scattered laser photocoagulation to the atrophic retina and around the new lesions. Fundus photograph (**F**) in the left eye, 16 days after vitrectomy, showing that the retinal necrotic lesions and laser spots have become degenerative (arrows in F).

node dissection in 2009.

She was given 0.1% betamethasone and timolol eye drops in the right eye, and treated with a 2-week course of intravenous acyclovir 1,500 mg daily, combined with intravenous prednisolone tapered from 100 mg daily, followed by oral valacyclovir 3,000 mg daily for 2 weeks in combination with oral prednisolone tapering. The retinal necrotic lesions became degenerative, but her visual acuity in the right eye was 0.08, due mainly to the vitreous opacity. She was observed after developing complete posterior vitreous detachment with no vitreoretinal adhesion in the right eye (Fig. 2C). In June 2011, epiretinal proliferation with tangential retinal traction was noted in the super-

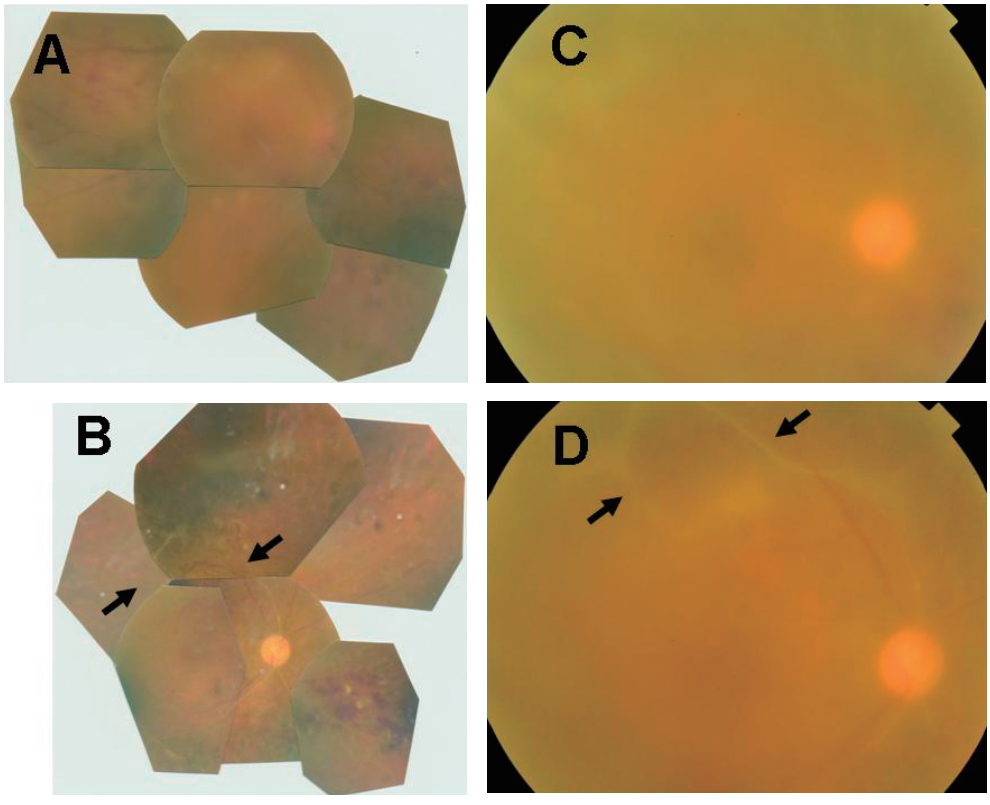

Fig. 2 Case 2. Merged fundus photographs (A) in the right eye taken on March 11, 2011, showing retinal necrotic lesions covering the entire retina with dense vitreous opacity. Fundus photographs in the right eye taken on April 27 (C) and June 8 (D), showing retinal degeneration with no proliferation under dense vitreous opacity (C), and epiretinal proliferation extending from the superotemporal midperiphery to the posterior pole (arrows in D), respectively. Merged fundus photographs (B) taken on June 29 after vitrectomy showing removal of epiretinal proliferation (arrows in B).

otemporal midperipheral retina to the posterior pole (Fig. 2D). Vitrectomy, endo-laser panretinal photocoagulation, and air tamponade, combined with cataract surgery, were performed to remove epiretinal proliferation, together with vitreous opacity (Fig. 2B). Her visual acuity in the right eye became 0.5.

Discussion

Acute retinal necrosis syndrome in the first patient (Case 1) was a re-recurrence and classified as the mild type. The vitreous opacity was a combination of sequelae to 2 episodes of previous inflammation and the immediate consequence of the present inflammation. The vitreous opacity prevented the new retinal necrotic lesions from being visualized clearly. Vitreoretinal adhesions, as sequelae to the previous inflammation, would be suspected to induce traction retinal detachment under the new inflammation. Vitrectomy was thus performed during the acute inflammatory phase and was called an early vitrectomy.

In contrast, acute retinal necrosis syndrome in the second patient (Case 2) was the fulminant type, involving the entire retina. Complete posterior vitreous detachment developed spontaneously in the acute inflammatory phase; thus, an early vitrectomy was not planned. The development of epiretinal proliferation with tangential retinal traction was noted in the post-inflammatory phase, and vitrectomy was done at this time as a prophylactic vitrectomy against subsequent retinal detachment.

Laser photocoagulation was also advocated as a prophylactic against retinal detachment in acute retinal necrosis syndrome [12, 13]. Based on this recommendation, laser photocoagulation was applied to

the first patient (Case 1) during vitrectomy around active retinal necrotic lesions and also on the atrophic retina as sequelae to the previous retinal inflammation. Panretinal photocoagulation was performed during vitrectomy in the second patient (Case 2) to cover the entire atrophic retina in the post-inflammatory phase.

The first patient (Case 1) develop acute retinal necrosis syndrome three times in the same (left) eye. The patient happened to have hepatitis C virus infection and underwent virus eradication therapy. Immunomodulation, caused possibly by HCV itself and also by interferon therapy, might underlie the repeated activation of herpetic viruses in the same eye.

The formation of vitreoretinal adhesions, followed by vitreous traction caused by inflammation-induced vitreous gel contraction in the post-inflammatory phase, leads to traction and rhegmatogenous retinal detachment with multiple retinal tears in acute retinal necrosis syndrome [1-3]. Spontaneous formation of complete posterior vitreous detachment in the acute inflammatory phase or in the late post-inflammatory phase is a good sign of the absence of vitreoretinal adhesions [1, 2]. Even under the circumstances described in Case 2, epiretinal proliferation would develop in the background of persistent weak inflammation and would cause tangential retinal traction, leading finally to traction and rhegmatogenous retinal detachment. Prophylactic vitrectomy to remove epiretinal proliferation under complete posterior vitreous detachment in the post-inflammatory phase has the rationale of preventing the subsequent development of traction and rhegmatogenous retinal detachment. In addition, pre-existing vitreous opacity with apparent vitreoretinal adhesions would be better removed by early vitrectomy even in the acute inflammatory phase to visualize the fundus and to avoid traction and rhegmatogenous retinal detachment under active inflammation, as shown in Case 1.

In conclusion, the timing of surgical intervention is crucial to good visual outcome in prophylactic and early vitrectomy for acute retinal necrosis syndrome. In the post-inflammatory phase, one indication for surgical intervention would be to remove epiretinal proliferation, which causes tangential retinal traction even in the absence of vitreoretinal adhesions, specifically under complete posterior vitreous detachment. Another indication would be to release apparent and marked vitreoretinal adhesions even in the acute inflammatory phase as well as in the late post-inflammatory phase of the disease.

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