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CLINICAL INVESTIGATION

Prophylactic Vitrectomy for Acute Retinal Necrosis

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Abstract

Purpose: To evaluate the efficacy of prophylactic vitrectomy for acute retinal necrosis.

Methods: The clinical charts of 17 patients (18 eyes) with acute retinal necrosis and no retinal break or rhegmatogenous retinal detachment (RRD) were retrospectively analyzed for the efficacy of prophylactic vitrectomy. The retinal necrotic lesions at the initial presentation were classified into three groups according to the lesion site as described by Holland: zone 1 (posterior pole; $n = 3$), zone 2 (midperiphery; $n = 12$), and zone 3 (periphery; $n = 3$). All patients were treated with intravenous antiviral therapy. Three zone 1 eyes and eight zone 2 eyes underwent prophylactic vitrectomy. Four zone 2 eyes and three zone 3 eyes did not receive prophylactic vitrectomy.

Results: All zone 1 eyes developed RRD despite prophylactic vitrectomy. Among the 12 zone 2 eyes, eight of the eyes that underwent prophylactic vitrectomy did not develop RRD, whereas three of the four zone 2 eyes without prophylactic vitrectomy developed RRD. All zone 3 eyes were cured with only antiviral medication.

Conclusions: Prophylactic vitrectomy is effective in preventing the development of RRD in eyes where necrotic lesions do not extend beyond the midperiphery (zone 2). **Jpn J Ophthalmol** 2009;53:486–489

Keywords: acute retinal necrosis, prophylactic vitrectomy, rhegmatogenous retinal detachment

Introduction

Acute retinal necrosis (ARN) is necrotizing vaso-occlusive retinitis caused by the human herpes viruses.[1–12] Although antiviral medication is effective against necrotizing retinitis, ARN may be complicated by the development of rhegmatogenous retinal detachment (RRD), which is a major contributing factor to a poor visual prognosis.[1,13–19]

Treatment with prophylactic photocoagulation in combination with antiviral medical therapy for ARN has decreased the incidence of RRD to 0%,[20] 17%,[21] and 35%,[22] considerably lower incidences than those in untreated patients (50%–75%).[1] Recently, a case in which prophylactic vitrectomy during ARN effectively prevented the development of RRD was reported.[23]

Here, we report the results of prophylactic vitrectomy performed during the inflammatory phase of ARN in conjunction with antiviral treatment compared with antiviral treatment alone.

Subjects and Methods

The records of patients with ARN at Tokyo Medical and Dental University Hospital between January 1998 and September 2006 were studied. Eyes with RRD due to ARN at the initial presentation were excluded from the study. A total of 18 eyes of 17 patients (11 men and 6 women)

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between the ages of 17 and 81 years (mean age, 49 years) was studied. The follow-up period ranged from 3 to 80 months (mean follow-up, 30 months).

All patients were diagnosed with ARN on the basis of following ocular findings: moderate anterior uveitis with small or medium size mutton-fat keratic precipitates, mild to severe vitreous opacities, optic disc hyperemia, and multiple necrotizing areas of the retina.

An aliquot of aqueous humor from each patient was collected for the diagnosis of pathogenic viruses, conducted using qualitative multiplex polymerase chain reaction (PCR). In those patients who had had vitrectomy, virus loads were examined by real-time PCR. The details of our PCR methods are described elsewhere.[24]

Immediately after the diagnosis of ARN, our standard antiviral medical treatment, that is, intravenous acyclovir (15 mg/kg, three times daily), oral prednisolone (40 mg daily), and oral aspirin (100 mg daily), was administered. Medical treatment continued for at least 2 months. Betamethasone sodium phosphate, levofloxacin, tropicamide, and atropine sulfate were also applied topically to the affected eyes.

The eyes were classified according to Holland's classification for cytomegalovirus retinopathy[25] according to the site of the retinal necrosis as follows: zone 1, extending to the posterior pole area from the ora serrata, the posterior pole within 3000 μm (about 2 disc diameters) of the fovea (approximately the area enclosed by the major temporal vascular arcades) or 1500 μm from the margins of the optic nerve head ($n = 3$); zone 2, extending anteriorly from zone 1 to the clinical equator of the eye, identified by the anterior borders of the ampullae of the vortex veins ($n = 12$); and zone 3, extending anteriorly from zone 2 to the ora serrata ($n = 3$).

Prophylactic vitrectomy during the active inflammatory phase, based on the progression of the whitish yellow necrotic lesion, was performed in all zone 1 eyes and in eight of the zone 2 eyes in which the necrotic lesions were located very close to zone 1 either at the initial presentation or when the necrotic lesions extended to zone 1 during antiviral medical therapy. The other four zone 2 eyes that had necrotic lesions located farther from zone 1 and all zone 3 eyes were treated with antiviral medication only. In all zone 2 eyes that underwent prophylactic vitrectomy, the lesions remained in zone 2, and in all zone 3 eyes, the lesions remained in zone 3.

Prophylactic vitrectomy was performed with a 20-gauge three-port system, and retrobulbar anesthesia was administered with 2% lidocaine and 0.5% bupivacaine. The perfusate was sterile intraocular irrigating solution (BSS plus, Alcon, Fort Worth, TX, USA) with acyclovir 40 μg/ml and dexamethasone 6 μg/ml. Whenever posterior vitreous detachment (PVD) was not present, it was artificially induced by suction with a vitreous cutter. Triamcinolone acetonide was used to visualize the posterior vitreous membrane, and the vitreous base was carefully shaved. After cutting of the vitreous, endolaser photocoagulation was performed 360° around the anterior edge of the healthy retina before performing the fluid–air exchange. Silicon oil was injected into the vitreous cavity in all cases. An encircling scleral buckling procedure was performed in one eye because a retinal tear was discovered during surgery. Efforts were made to keep the crystalline lenses intact, even if there were mild cataracts. Whenever possible, the silicon oil was removed several months after surgery.

Results

The efficacy of the ARN treatment was evaluated based on the incidence of RRD according to the extent of the necrotizing area at the initial presentation.

Zone 1

Varicella zoster virus was detected in all three eyes of the three patients with necrotic lesions in zone 1. The patients were referred to our clinic within 13 to 16 days after disease onset, and the best-corrected visual acuities (BCVAs) of the three eyes were 0.1, 0.5, and 0.9 (Table 1). Immediately after a clinical diagnosis of ARN was made, antiviral medical therapy was initiated and a prophylactic vitrectomy was performed in all three eyes within a few days. PVD was not present in two eyes before the prophylactic vitrectomy, and an artificial PVD was induced during the surgery. Iatrogenic retinal breaks occurred in one eye during the surgery. RRD developed in all three zone 1 eyes within 2 months after the vitrectomy. Retinal breaks or holes were present at the margin between the healthy retina and necrotic retina in all cases. Final visual acuity ranged from hand motion to 0.04, despite the fact that the RRD was repaired by other vitreoretinal surgery. Silicon oil could not be removed from any of the eyes.

Zone 2

Among the 12 eyes of 12 patients with necrotic lesions in zone 2, ten were considered to have surgical indications for prophylactic vitrectomy because the necrotizing retinitis was close to zone 1 or the eyes responded poorly to antiviral therapy. Two of these patients refused prophylactic vitrectomy and the remaining eight underwent the procedure. In the other two eyes surgery was not indicated because the necrotic lesions were located away from zone 1 and were not progressive. These eyes were treated with antiviral therapy alone.

Among the eight eyes that underwent prophylactic vitrectomy, PVD was not present in four eyes before the prophylactic vitrectomy and an artificial PVD was induced during surgery. Iatrogenic retinal breaks occurred in two eyes during surgery.

None of the eight eyes in zone 2 that underwent prophylactic vitrectomy developed RRD. In three of these, however, the silicon oil was not removed because the

Table 1. Profile of patients with acute retinal necrosis

Case	Sex	Age	Lesions at initial presentation	Initial BCVA	Virus	Interval between onset and medication (days)	PV	Interval between medication and PV (days)	SO removal	RRD	Interval from onset to RRD (months)	Final BCVA	Follow-up (months)
1	M	40	1	0.1	VZV	14	Yes	0	No	Yes	1	HM	14
2	M	44	1	0.5	VZV	16	Yes	1	No	Yes	1	HM	70
3	F	60	1	0.9	VZV	13	Yes	0	No	Yes	2	0.04	7
4	M	46	2	1	VZV	9	Yes	5	Yes	No		0.5	12
5	F	27	2	0.9	HSV-2	10	Yes	6	Yes	No		0.9	80
6	F	70	2	0.2	VZV	5	Yes	8	Yes	No		0.8	11
7	M	39	2	0.05	HSV-1	3	Yes	9	Yes	No		0.4	20
8	M	16	2	0.3	HSV-2	6	Yes	7	Yes	No		0.3	30
9	M	31	2	0.3	VZV	17	Yes	2	No	No		CF	26
10	M	61	2	0.1	VZV	8	Yes	0	No	No		HM	21
11	F	54	2	HM	VZV	12	Yes	2	No	No		HM	45
12	F	53	2	1.2	VZV	7	No	N/A	N/A	No		0.5	22
13	M	55	2	0.6	VZV	11	No	N/A	N/A	Yes	1	0.08	52
14	M	47	2	0.3	VZV	9	No	N/A	N/A	Yes	4	0.1	67
15	F	81	2	CF	VZV	18	No	N/A	N/A	Yes	4	CF	5
16	M	46	3	1.2	VZV	0	No	N/A	N/A	No		1.5	12
17	M	31	3	0.08	VZV	8	No	N/A	N/A	No		1.2	9
18	M	76	3	0.04	VZV	44	No	N/A	N/A	No		0.4	3

BCVA, best-corrected visual acuity; HM, hand motion; CF, counting fingers; PV, prophylactic vitrectomy; SO, silicone oil; RRD, rhegmatogenous retinal detachment; HSV, herpes simplex virus; VZV, varicella zoster virus; N/A, not applicable because the patients did not undergo vitrectomy.

patients refused further surgery due to very poor vision. At the same time, three of the four eyes in zone 2 treated with antiviral medication alone developed RRD.

The eight eyes in zone 2 treated with prophylactic vitrectomy had BCVA ranging from counting fingers or worse (three eyes) to 0.3 or better (five eyes) (Table 1); only one of four eyes treated with antiviral medication alone had a BCVA of 0.4, the other three having a poor visual prognosis.

Zone 3

Varicella zoster virus was detected in all three eyes of the three patients with necrotic lesions in zone 3. All three were treated only with antiviral medical therapy. The necrotic retinal lesions did not extend into zone 2, and the eyes did not develop RRD and had good visual prognosis (Table 1).

Discussion

ARN is a sight-threatening disease with necrotizing retinitis caused by the human herpes virus. Although the first line of treatment is antiviral medication, RRD in an atrophic retina following necrotizing retinitis is the major cause of visual loss in this disease, and this complication occurs frequently. Therefore, the treatment strategy for ARN is aimed at preventing RRD during the clinical course of the disease. Some studies report that either laser photocoagulation of the normal retina at the margin of necrotizing retinitis[18,20–22] or prophylactic vitrectomy[23] is effective for preventing RRD, but the efficacy of these strategies remains controversial.

In the present study, prophylactic vitrectomy performed during the active phase of necrotizing retinitis was effective for preventing RRD in some eyes with ARN. Furthermore, the data indicate that the extent of necrotizing retinitis at the initial presentation can be used as a parameter to predict the development of RRD following prophylactic vitrectomy. Although the patients with zone 1 ARN underwent prophylactic vitrectomy in addition to treatment with antiviral medication, all developed RRD within a couple of months following vitrectomy. On the other hand, none of the eight eyes with ARN in zone 2 that underwent prophylactic vitrectomy developed RRD, whereas three of four eyes with zone 2 ARN treated only with antiviral medication without vitrectomy developed RRD within 2 months following initiation of medical therapy. None of the eyes with zone 3 ARN, all of which were treated with antiviral medication alone, developed RRD. These zone 3 lesions did not extend to zone 2. These findings suggest that prophylactic vitrectomy is effective in preventing the development of RRD in eyes in which necrotizing retinitis is limited to zone 2 at the initial presentation, and ARN limited to zone 3 can be successfully treated with antiviral medication alone. Eyes with ARN extending into zone 1, however, are difficult to treat, and RRD may not be preventable, even with prophylactic vitrectomy.

Although we carefully shaved the peripheral vitreous by pressing on the sclera from the outside with triamcinolone, RRD still occurred in all zone 1 cases within several months following prophylactic vitrectomy, with new breaks at the margin between the necrotic and healthy areas of the retina. We consider that the zone 1 RRD was due to traction

caused by the remaining peripheral vitreous. Whether or not the lens should be removed to adequately withdraw the peripheral vitreous is a matter of controversy. We did not remove the lens to avoid extensive invasion caused by cataract surgery together with vitrectomy during the active inflammatory stage of the disease. When peripheral vitreous remains, another option to avoid the development of RRD is encircling buckle surgery. The zone 1 cases in the present study were not treated by encircling buckle surgery, and all these patients subsequently developed RRD. The present findings suggest that encircling buckle surgery might be a beneficial treatment for zone 1 eyes. More studies are required to determine the best treatment strategy for advanced ARN with necrotizing retinitis extending into zone 1.

Interpretation of the results of the present study is limited by the small number of patients and the fact that this was a retrospective study. The disease is not common, and few patients with new cases with ARN present annually at this clinic; therefore, a multicenter clinical study to evaluate the therapeutic strategy of this sight-threatening disease is warranted.

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