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Surgical Management of Retinal Detachment Secondary to Acute Retinal Necrosis: Clinical Features, Surgical Techniques, and Long-term Results

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Purpose: To describe the clinical features of complicated retinal detachment secondary to acute retinal necrosis (ARN) and to present the long-term results of vitreous surgery in these cases.

Methods: A retrospective study was conducted on 16 immunocompetent patients (18 eyes). The average follow-up period was 60 months.

Results: Proliferative vitreoretinopathy (PVR) grade C, with the predominance of anterior PVR, and characteristic changes in the vitreous base area were present in all cases before surgery. All eyes underwent vitrectomy, membrane peeling, endolaser photocoagulation, and intraocular tamponade without scleral buckling. Additional procedures were performed in 13 eyes. Retinal reattachment was achieved in the 18 eyes (100%) in the short term. Variable degrees of reproliferation occurred in all cases after surgery. Other delayed complications included ocular hypotony, macular pucker, peripheral retinal neovascularization, and severe preretinal fibrosis. Improvement of visual acuity occurred in 13 eyes (72.2%). Eleven eyes (61.1%) achieved final ambulatory visual acuity of 5/200 or better.

Conclusions: Rhegmatogenous retinal detachment secondary to ARN has characteristic clinical features. Severe proliferative vitreoretinopathy with the predominance of anterior PVR develops rapidly. Reproliferation is the most important late postvitrectomy complication necessitating multiple surgical procedures in these cases. The visual results remain unfavorable due to the destructive nature of ARN. **Jpn J Ophthalmol 2003;47:484–491**

Key Words: Acute retinal necrosis, complex retinal detachment, ocular hypotony, reproliferation.

Introduction

Rhegmatogenous retinal detachment is the serious late complication of acute retinal necrosis (ARN).[1] The incidence of this complication has been reported to be 50–85%.[2,3] Severe retinal ischemia and vitreous organization that accompanies widespread zones of necrotizing retinitis have been determined to have roles in the high incidence of retinal detachment in these cases. Retinal detachment secondary to ARN has a complex nature and usually results in severe visual loss. The presence of multiple large posterior retinal breaks located at the junction of involved and uninvolved retina as well as the large holes over the areas of necrotic retina and the predisposition to proliferative vitreoretinopathy (PVR) are the most important causes of the devastating course of retinal detachment in this disease.[1–4] Early attempts to repair retinal detachment in these cases have shown disappointing results.[1] Nevertheless, with the refinement of vitreoretinal surgical techniques, there has been an increased rate of retinal reattachment.[2–6]

During the past several years, patients with retinal detachment secondary to ARN have been managed in our department as a tertiary referral center. We have noticed unique clinical findings in these cases and have also found their postoperative course to be different from other types of rhegmatogenous retinal detachment.

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In this report, we retrospectively present the clinical features of retinal detachment secondary to ARN and describe the surgical techniques that were employed. The long- term anatomic and functional results with emphasis on late complications are also reported.

Materials and Methods

The records of 16 patients (18 eyes) undergoing vitrectomy for repair of retinal detachment secondary to acute retinal necrosis were reviewed. The diagnosis of ARN had been made according to the clinical findings. The patients had been treated medically in different centers. They were then referred to the vitreoretinal service of the Labbafinejad Medical Center for management of retinal detachment secondary to ARN. The operations were performed consecutively from February 1992 to June 1998. In 2 patients, both eyes underwent surgery due to bilateral involvement. The following data were extracted from the charts: the status of the patient's immune system, age, sex, duration of follow-up, the time interval between the onset of ARN symptoms and retinal detachment, preoperative visual acuity (VA), final VA, causes of decreased vision after surgery, the status of retina before and after surgery, presence of optic atrophy, grading of PVR, the status of retinal vessels, history of prophylactic laser therapy, preoperative and postoperative measured intraocular pressure (IOP), presence of relative afferent pupillary defect (RAPD), the initial and additional surgical procedures, and early and late postoperative complications. The updated classification of the Retina Society was used for PVR grading.

Surgical Techniques

Three-port pars plana vitrectomy was carried out in all cases. The lens was removed either through pars plana lensectomy, phacoemulsification, or extracapsular lens extraction. Extensive peeling of the epiretinal membranes was done. Perfluorocarbon liquid (PFCL) was used and was removed at the end of the surgical procedure. Vitreous base dissection and/or debulking was carried out. Relaxing retinotomies/retinectomies were performed if indicated, and the peripheral retinal flap anterior to retinotomy was removed. After flattening of retina, barrier endolaser photocoagulation with argon green laser was applied around the retinal breaks and to the posterior edge of retinotomies/retinectomies. Removal of PFCL was followed by either fluid/gas exchange or silicone oil injection. Silicone oil with viscosity of 1000 cs or 5000 cs was used and inferior peripheral iridectomy (PI) was performed.

Results

Of 16 patients, 9 were male. All patients were immunocompetent. The age range was 22–56 years with a mean age of 38.5 years. The average follow-up period was 5 years. All of the patients had been treated by intravenous acyclovir (30 mg/kg per day in three divided doses) for 10 days followed by oral acyclovir (800 mg 5 times daily) for 6 to 14 weeks. Oral prednisolone (1 mg/kg per day) was started 48 hours after the prescription of intravenous acyclovir and was tapered and discontinued within 3 weeks. Retinal detachment (RD) was diagnosed 4–24 weeks after the onset of ARN symptoms (except in case 16, retinal detachment occurred 2 years later, following vitrectomy for management of macular pucker). The average time interval was 10.5 weeks. In 2 patients, both eyes were operated on. Prophylactic barrier laser photocoagulation was carried out in 5 eyes but retinal detachment occurred despite previous laser therapy in these cases. The patient data have been summarized in Table 1.

The extent of retinal detachment was total in all but 2 cases (Table 1). Partial posterior vitreous detachment was present in all eyes. PVR grade C was observed in all cases before surgery. Anterior PVR was predominant and was present in all eyes. Anterior PVR involved 12 clock hours in 15 eyes. Posterior PVR was present in 12 eyes involving 3–12 clock hours. The retinal tears were large, multiple, and posteriorly located. The coalescence of retinal tears led to giant retinal tear formation in some cases. Retina was thin, avascular, and necrotic anterior to retinal breaks. Very severe condensation of vitreous base was apparent in all cases. There were avascular membranes in vitreous base area, vertically located and firmly attached to the thin, ischemic peripheral retina. These changes induced a characteristic appearance (Figure 1A).

Vitrectomy associated with peeling of epiretinal membranes and endolaser photocoagulation was carried out in all cases. PFCL was used temporarily in most of the cases. The lens was removed in 15 eyes during initial surgery. Extraction of the lens was performed during the reoperation in the right eye of case 7. The lens remained intact in case 10. However, cataract developed as a late complication. Extracapsular cataract extraction and posterior chamber intraocular lens implantation were carried out 2 years later and a final visual acuity of 20/30 was achieved. Case 16 was pseudophakic. Vitreous base debulking was carried out in 16 eyes. Relaxing retinotomies/retinectomies were performed in 8 eyes extending from 90 to 360º (Figure 1B) during the initial surgeries and in 1 eye during the reoperation. Injection of silicone oil with viscosity of 1000 or 5000 cs was performed in 17 cases. Long-acting gas was used in only 1 case with less severe PVR changes (case 10, Table 1). Scleral buckling procedure was not performed in any of the cases. Retinal

Table 1. Patient Characteristics

No./Sex/ Age (y)*	Eye	Time Interval (wks)	Follow-up (mos)	Extent of RD†	PVR‡	Pre-op IOP§ (mm Hg)	Post-op IOP (mm Hg)	Pre-op VA‖	Post-op VA (Final)	Causes of Decreased Post-op VA	Initial Procedures¶	Subsequent Procedures#
1/F/22	OD	13	20	Total	CA12,P12	10	8	HM	HM	Severe optic neuropathy (4+ pale disc)	Lensx, Vitx, MP, ELP, VBD, SO	
1/F/22	OS	11	20	Total	CA12,P12	11	9	HM	HM	Severe retinal ischemia (4+ pale disc)	Lensx, Vitx, MP, ELP, VBD, SO	
2/M/32	OD	16	84	Total	CA12,P6	4	6	CF	20/120	Partial optic atrophy (2+ pale disc) macular pucker	Lensx, Vitx, PFCL, MP, ELP, VBD, SO	Argon laser photocoagulation, reopening of inferior PI with YAG
3/M/30	OS	12	72	Total	CA12,P12	10	8	HM	HM	Severe optic neuropathy (4+ pale disc) complete occlusion of retinal vessels	Lensx, Vitx, MP, PFCL, ELP, retinotomy 360º, SO	
4/M/48	OD	4	72	Total	CA12	8	12	CF	HM	Severe optic neuropathy (4+ pale disc) Severe retinal ischemia Severe preretinal fibrosis	ECCE, Vitx, MP, PFCL, retinotomy 180º, ELP, SO	
5/F/35	OS	8	96	Total	CA12	6	2	HM	HM	Sever optic neuropathy ($3–4^{+}$ pale disc) Severe retinal ischemia, macular pucker and macular cystoid changes	Lensx, Vitx, PFCL, MP, ELP, VBD, SO	AC refromation
6/M/44	OD	6	78	Total	CA12	10	5	CF	20/200	Partial optic atrophy (2+ pale disc) macular pucker	Lensx, Vitx, PFCL, MP, retinotomy & reinectomy, VBD, SO	Argon laser photocoagulation
7/F/56	OD	8	92	Total	CA12,P3	16	3	HM	20/160	Partial optic atrophy, macular pucker	Vitx, ELP, retinotomy	ECCE, VBD, PFCL, extension of retinotomy, ELP, SO. Reopening of PI with YAG. Removal of SO and MP. Reinjection of SO and endolaser

(*continued*)

Table 1. *Continued*

No./Sex/ Age (y)*	Eye	Time Interval (wks)	Follow-up (mos)	Extent of RD†	PVR‡	Pre-op IOP§ (mm Hg)	Post-op IOP (mm Hg)	Pre-op VA‖	Post-op VA (Final)	Causes of Decreased Post-op VA	Initial Procedures¶	Subsequent Procedures#
7/F/56	OS	14	92	Total	CA12,P3	14	3	CF	20/200	Partial optic atrophy, cystoid changes of macula and lamellar hole	ECCE, Vitx, MP, PFCL, VBD, ELP, retinotomy	Reopening of PI with YAG laser and additional argon laser photocoagulation
8/M/34	OS	12	72	Total	CA12	8	16	HM	18/200	Optic atrophy (3+ pale), retinal ischemia, macular pucker	Lensx, Vitx, MP, PFCL, ELP, SO	Barrier laser, SO removal
9/M/40	OD	20	42	Total	CA12	18	12	HM	20/60		Lensx, Vitx, MP, PFCL, ELP, SO	
10/M/44	OD	8	75	Partial	CA6,CP3	8	12	CF	20/30		Vitx, MP, retinotomy, ELP, GFX	ECCE, PC-IOL
11/M/36	OS	4	42	Total	CA12	4	10	HM	20/80	Partial optic atrophy and retinal ischemia	ECCE, Vitx, MP, PFCL, ELP, SO	Barrier laser
12/F/47	OS	4	48	Total	CA12,P6	10	4	LP	CF	Optic neuropathy, severe retinal ischemia	Lensx, Vitx, MP, VBD, retinotomy/retinectomy 360º, ELP, PFCL, SO	Reopening of PI with YAG Removal of emulsified SO and reinjection of SO (two times)
13/F/25	OD	6	44	Total	CA12,P8	9	12	LP	20/200	Partial optic atrophy, retinal ischemia	Lensx, vitx, MP, VBD, PFCL, ELP, SO	So removal, repeat vitx, MP, PFCL, ELP, reinjection of SO, barrier laser, SO removal
14/M/41	OD	10	42	Total	CA8,P6	5	0	CF	20/200	Partial optic atrophy, retinal ischemia	Lensx, Vitx, MP, VBD, PFCL, ELP, SO	SO removal
15/F/40	OD	24	32	Total	CA12,P12	4	0	HM	CF	Optic neuropathy, severe retinal ischemia, macular dragging	Lensx, Vitx, MP, retinotomy/retinectomy 300º, VBD, ELP, removal of subretinal fibrosis, SO	Barrier laser, SO removal
16/F/44	OS	2 yrs	54	Posterior pole	CA12,P3	2	6	CF	10/200	Macular hole	Repeat vitx, PFCL, MP, SO	Translimbal removal of SO bubble from AC

*F: female, M: male.
†RD: retinal detachment.
‡PVR: proliferative vitreoretinopathy.
§IOP: intraocular pressure.
‖VA: visual acuity, HM: hand movement, CF: counting fingers.
¶Lensx: lensectomy, Vitx: vitrectomy, MP: membrane peeling, ELP: endolaser photocoagulation, VBD: vitreous base dissection, SO: silicone oil, PFCL: perfluorocarbon liquid, ECCE: extracapsular cataract extraction, GFX: gas/fluid exchange.
#PI: peripheral iridectomy, AC: anterior chamber, PC-IOL: posterior chamber intraocular lens.

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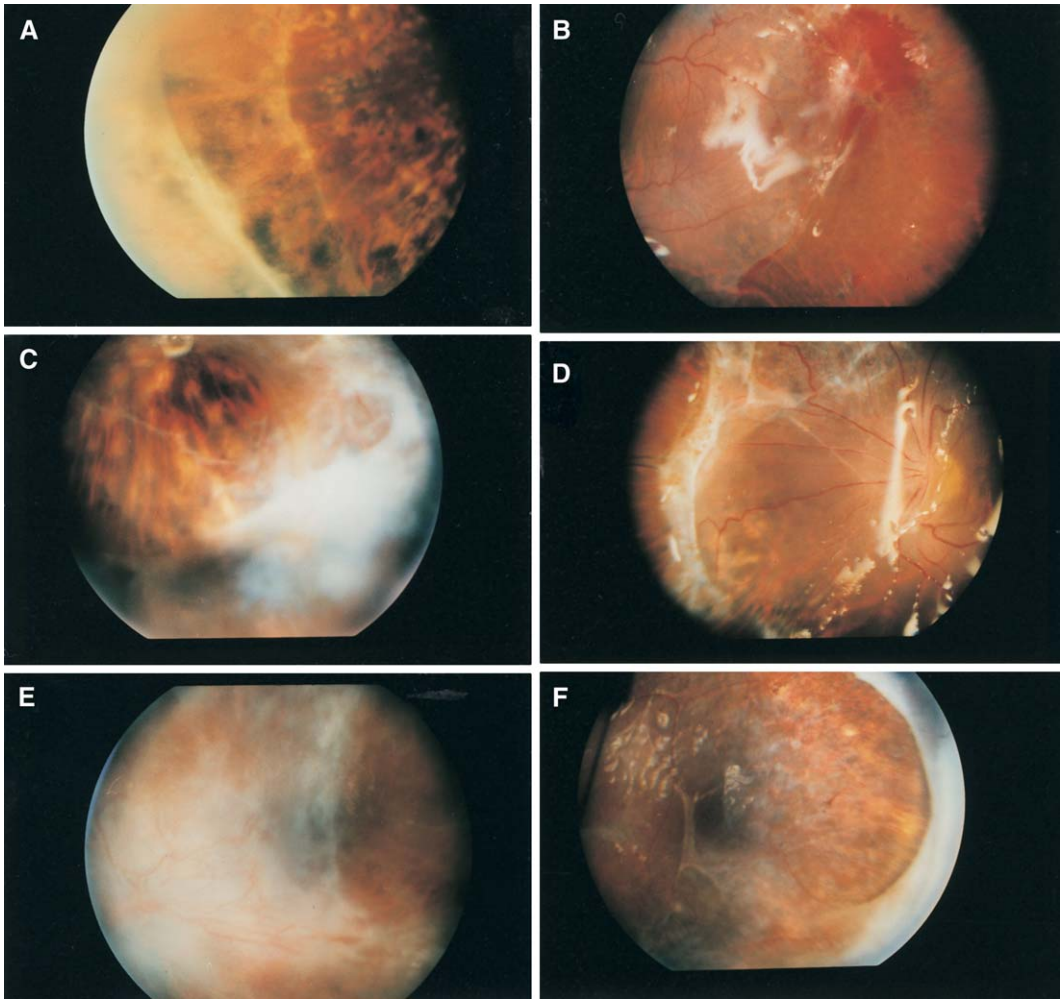

Figure 1. (**A**) Tenting of vitreous base. Characteristic changes called ''tenting of vitreous base.'' Vitreous base behaves as a scaffold for vertically located membranes with firm attachment to the peripheral avascular retina. (**B**) Retinal reattachment after a 360º retinotomy/retinectomy. Early postoperative days after a 360º retinotomy/retinectomy leading to retinal reattachment (case 12). Note the flat margin of the retinectomy with a clot of preretinal hemorrhage. (**C**) Localized retinal redetachment due to reproliferation. Reproliferation occurred with variable severity in all eyes as the most important late postvitrectomy complication leading to localized areas of retinal redetachment. (**D**) Elevation of retinectomy edges due to reproliferation. Reproliferation resulted in elevation of the edges of 360º retinotomy/retinectomy in case 12. (**E**) Preretinal fibrovascular tissue. Extensive preretinal fibrovascular tissue in case 4 with optic atrophy and occluded retinal vessels. (**F**) Epiciliary membranes. Peripheral reproliferation and involvement of ciliary body resulted in persistent ocular hypotony in most cases.

reattachment was achieved in all cases at the end of the surgical procedures.

Of the 18 eyes, 13 (72.2%) showed partial improvement in visual acuity after surgery. Eleven eyes (61.1%) gained postoperative vision of 5/200 or better but only 2 eyes (11.1%) achieved the visual acuity of 20/60 or better. In 4 eyes, the preoperative vision of light perception or hand movement remained stable and in 1 case (case 4, Table 1), deterioration of visual acuity occurred after surgery. The severity of optic atrophy and retinal ischemia were marked in these 5 eyes. Extensive preretinal fibrosis and formation of fibrovascular tissue was observed in case 4 after surgery. The principal causes of decreased postoperative vision in our series were optic atrophy, prior severe retinal damage due to virus activity, retinal ischemia secondary to previous occlusive vasculitis, and pathologic changes of macula including macular pucker, dragging of macula, and macular hole formation.

Different degrees of optic atrophy were present in all cases. This was evaluated to be moderate (1–2+ pallor

of disc) in 12 eyes and severe (3–4+ pallor of disc) in 6 eyes. Before surgery, marked relative afferent pupillary defect (at least 3+) was detected in 12 eyes. It was undetectable in 4 eyes. In case 7 with bilateral acute retinal necrosis, RAPD was found to be 1–2+ in the right eye while retina was still attached in the left eye. After retinal reattachment was achieved in the right eye, retinal detachment occurred in the left eye and 1–2+ RAPD was detected in the left eye. After surgery, RAPD was undetectable in 6 eyes, remained at the same level as before surgery in 5 eyes, and decreased in 7 eyes.

Mild or moderate fibrin response occurred in all cases as an early postoperative complication. The fibrin was formed on the surface of silicone oil at the pupillary margin and occluded the inferior PI temporarily. Despite initiation of topical steroid, reopening of inferior PI with YAG laser was indicated in 4 eyes (Table 1).

The most important late postoperative complication was reproliferation (Figure 1C). Different degrees of reproliferation occurred in all eyes with variable severity and led to redetachment of retina in some cases. In 8 eyes, the reproliferation led to foci of localized traction RD posterior to the equator. However, the macula remained attached during the follow-up period in all of these cases. Redetachment of peripheral, thin, and avascular retina occurred in both eyes of case 7. Fibrous tissue was formed in the superior and temporal periphery of the right eye of this patient. The circumferential extent and also the thickness of this proliferative tissue gradually increased during the follow-up period. Peripheral fibrous proliferation also occurred in the left eye of the same patient, but to a lesser extent. In case 12 with 360º retinectomy, the edges of the retinectomy became elevated due to the late reproliferation (Figure 1D). Extensive preretinal fibrosis was observed in case 4 (Figure 1E).

The occurrence of peripheral reproliferation and involvement of the ciliary body (Figure 1F) by the proliferative tissue (epiciliary membrane) resulted in ocular hypotony in 9 eyes (Table 1). In case 5, the dysfunction of ciliary body was so severe that it led to the flattening of the anterior chamber in a silicone-oil–filled eye. Attempts for reformation of the anterior chamber were unsuccessful due to marked hyposecretion of the aqueous.

Pathologic changes of the macula were frequently observed after surgery. Macular pucker was common and affected the final vision in 5 eyes. Macular holes were observed in 2 eyes and dragging of macula due to the presence of fibrous tissue temporal to macula occurred in 1 eye.

Marked retinal ischemia secondary to previous occlusive vasculitis was present in all cases. This led to retinal neovascularization in 2 eyes. In the right eye of case 7, a tuft of fibrovascular tissue appeared in superior temporal periphery of retina after surgery and was complicated by hemorrhage into the silicone-oil–filled vitreous cavity. Absorption of hemorrhage occurred after a few weeks and additional scatter photocoagulation was carried out followed by regression of the neovascularization. However, there was an increase in the thickness and extent of the peripheral fibrous tissue involving all quadrants, with the most severity in the superior temporal quadrant. In case 4, the neovascularization was very extensive, involving almost the whole retina and was associated with severe preretinal fibrosis (Figure 1E).

Recurrent episodes of anterior uveitis occurred during the follow-up in 3 cases and were treated with topical steroids.

Silicone oil removal was carried out in 6 eyes. Removal of silicone oil was followed by reinjection in 2 eyes. In the right eye of case 7, silicone oil became emulsified and signs of silicone keratopathy appeared 2.5 years after the initial surgery. There was a rise in the IOP. The patient underwent silicone-oil removal associated with peeling of the epiretinal membrane. This resulted in the resolution of macular pucker. Retinal redetachment, however, occurred 1 week later due to reopening of a retinal break at the posterior margin of the peripheral fibrous tissue. Injection of SF_6 was followed by severe fibrin response leading to formation of sheets and membranes in the vitreous cavity. Reoperation was carried out including reinjection of silicone oil with viscosity of 5000 cs. The visual acuity and status of the retina remained stable and the silicone-oil surface was behind the pupillary plane thereafter. Ocular hypotony, however, developed 7 years after the initial surgery.

In case 12 with a 360º posterior retinectomy, band-shaped silicone keratopathy appeared as a late complication. Due to the high risk of retinal redetachment, removal of the emulsified silicone oil was followed by reinjection of silicone oil 5000 cs. The same procedure was repeated after 4 years. At the last follow-up examination, the edges of 360º retinectomies were more elevated due to the exaggerated reproliferation. The cornea was clear and ocular hypotony persisted.

Discussion

Acute retinal necrosis usually occurs in otherwise healthy patients, although it has also been reported in immunosuppressed cases.[7,8] All patients in our series were immunocompetent. Although retinal detachment may occur within the first month after the onset of symptoms, it usually develops 2–3 months later.[1,2] In our series, this time interval ranged from 4 to 24 weeks with an average of 10.5 weeks.

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In the acute phase of ARN, there is an increase in vitreous turbidity due to the massive breakdown of the blood—ocular barrier, which is associated with proliferative and chemotactic effects on pigment epithelium and fibroblasts.[9] In the cicatricial phase, formation of membranes and contraction of vitreous leads to the development of severe PVR. In one series,[3] 8 of 11 eyes (72.7%), and in another report,[5] 4 of 6 eyes, with ARN complicated by retinal detachment demonstrated PVR grade C or greater. We used the updated classification of the Retina Society[10] for PVR grading in our series and found PVR grade C in all of the cases. The anterior PVR was more pronounced, involving 12 clock hours in 83.3% of the cases. The predominant process of anterior PVR was the result of the primary pathologic changes in ARN, including involvement of peripheral retina as the initial and main site of necrotizing retinitis and involvement of the vitreous base during the disease process. We observed the condensation of the vitreous base in our cases and found the condensed vitreous base to be a scaffold for proliferation of membranes with firm adherence to the peripheral, avascular, thin, and necrotic retina. These pathologic changes resulted in a characteristic clinical picture, which we called ''tenting of the vitreous base.'' Vitreous base dissection and debulking as well as relaxing retinotomies and retinectomies were essential in mobilizing the retina in most of the cases in our series. Posterior PVR was also present in 66.6% of the eyes extending from 3 to 12 clock hours.

Repair of retinal detachment following ARN is very complex. Before 1982, of 41 eyes with retinal detachment, surgical repair with scleral buckling was attempted in 18 eyes and anatomic success was achieved in only 4 (22%) of these eyes, with only 2 achieving a final visual acuity of 20/400 or better.[1] With a combination of vitrectomy and scleral buckling, the success rate improved. In a report by Fisher et al[1], of the 7 eyes that developed retinal detachment, surgery was carried out in 4. The retina was reattached in 3 cases. Nevertheless, the visual acuity improved to 20/50 in only 1 eye and the rest had a visual acuity of less than 20/400.[1] In a report by Clarkson et al[2], surgical repair was attempted in 13 eyes. Using a combination of pars plana vitrectomy and scleral buckling, over 70% of eyes operated on were successfully reattached. Blumenkranz et al[3], reported that they could reattach 10 of 11 (91%) cases of retinal detachment secondary to ARN with modern microsurgical methods including the use of silicone oil. However, multiple operations were required (average: 2 per patient). Their results compared favorably with success rates of 22–77% previously reported. This was the first report of using silicone oil as an internal tamponade during the surgical repair of retinal detachment secondary to ARN.[3]

Blumenkranz et al[6] stated that placement of a broad encircling buckle in cases of retinal detachment secondary to ARN might be associated with the risk of significant complications. They attempted to reattach the retina without scleral buckling in 6 eyes. They removed all vitreous cortex, particularly that in the region of the vitreous base where tenacious adhesions were often adjacent to areas of necrosis or tears, and they were able to reattach retina in all cases. Primary lens removal was required in 4 eyes.[5]

We used the techniques of vitrectomy, temporary injection of PFCL, membrane peeling, vitreous base debulking, endolaser photocoagulation, and internal tamponade in our series. Retinal reattachment was achieved in all of the operated eyes with either one or multiple extensive vitreoretinal surgical procedures. The clear lens was removed in 16 of 17 eyes (94.1%). In cases of retinal detachment complicating ARN, removal of the lens facilitates the approach to the vitreous base area and improves the management of anterior PVR in such cases. Using PFCL, no drainage retinotomy was necessary in our cases. In addition, the presence of PFCL facilitated better management of the anterior component of PVR. We used silicone oil as an internal tamponade in 94.4% of the eyes. Long-acting gas was used in only 1 eye with less severe PVR. Silicone oil has also been used as an internal tamponade in retinal detachment following viral necrotizing retinitis associated with AIDS.[11–14]

Although prophylactic laser photocoagulation has been suggested by some of the authors,[15,16] retinal detachment may occur despite this treatment. In our series, retinal detachment developed in 5 eyes in spite of previous prophylactic laser photocoagulation.

Mild to moderate fibrin response occurred in all eyes in our series. Fibrin response is a common early postoperative complication in eyes undergoing surgery for management of retinal detachment secondary to ARN. Fibrin produces mesenchymal transformation of pigment epithelial cells[17] and contributes to the development of PVR. Aggravation of the PVR process, manifested as reproliferation of epiretinal membranes, was observed in all our cases after surgery. Reproliferation is the most important late complication in these cases and results in redetachment of retina, necessitating multiple reoperations. The predominance and severity of anterior PVR in ARN results in formation of epiciliary membranes followed by persistent ocular hypotony. The high rate of retinal detachment after silicone-oil removal in these cases is also the consequence of PVR reactivation and reproliferation.

Because of the avascularity of torn retina, significant vitreous hemorrhage is not common in ARN. Vitreous hemorrhage can occur, however, secondary to neovascularization created by the inflammatory and ischemic processes. Occlusive arteriopathy associated with vascular

fibrinoid necrosis are major clinical and histopathologic features of acute retinal necrosis.[18] Despite extensive areas of nonperfusion, neovascularization of iris and retina is uncommon in ARN. This may be due to less angiogenic stimulus as a result of necrosis of the retina and retinal pigment epithelium.[13] Panretinal photocoagulation to areas of necrotic and nonperfused retina has been shown to cause regression of optic nerve neovascular proliferation in these cases.[19] We observed the occurrence of retinal neovascularization in 3 eyes of 2 patients. These patients were the oldest in our series. Scatter photocoagulation resulted in regression of the peripheral retinal neovascularization in 1 of the cases. The fibrous tissue, however, showed progression and involved the retinal periphery. In the second case, retinal neovascularization was associated with very severe and extensive preretinal fibrosis. There was a history of varicella-zoster dermatitis in these 2 patients. Varicella zoster virus is a cause of ARN in older patients.[20]

Arteritic ischemic optic neuropathy is one of the most dramatic clinical manifestations of acute retinal necrosis and results in optic atrophy and contributes to severe visual loss in these patients. Different degrees of optic atrophy were present in all of our cases and the presence of relative afferent pupillary defect correlated with extensive retinal damage and optic nerve dysfunction.

In summary, in cases of retinal detachment and PVR secondary to ARN, vitrectomy with membrane peeling and vitreous base dissection, relaxing retinotomies and retinectomies in selected cases, endolaser photocoagulation and internal tamponade with silicone oil usually result in anatomic success in the short term. Nevertheless, due to the progression of the PVR process and occurrence of reproliferation, redetachment of retina may occur and multiple surgical procedures may be required. Ocular hypotony is also a common late complication. Removal of silicone oil is associated with a high risk of retinal redetachment and reinjection may be necessary in some cases. Despite the high anatomic success rate, the functional results remain unfavorable due to extensive retinal damage secondary to infection and ischemia, and the presence of severe optic neuropathy.

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