Neovascularization of the pars plana vitrectomy sclerotomy site

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Introduction

Neovascularization of the pars plana vitrectomy site (NVPVS) is a rare complication. The normal fibrovascular response expressed in the healing response to close the vitrectomy wound site is severely exaggerated and episcleral vessels prolif-erate through the lips of the sclerotomy wound site into the intracoarlar pars plana region. Correct diagnosis and effective treatment of both the fibrotic and vascular components of this destructive process are necessary to repair complications which include recurrent vitreous hemorrhages, retinal detachment and hypotony.

Case review and observations

In a review of 1000 consecutive vitrectomy cases performed in my surgical practice at the Jules Stein Eye Institute, I can recall only 4 cases of neovascularization of the pars plana vitrectomy site(NVPVS), yielding an incidence of 0.4%. The actual incidence is probably higher because it is often unrecognized especially when spontaneous regression of the neovascularization occurs without clinical sequelae. The histopathology of NVPVS has been reported previously by our group and the Bonn Eye Institute at the University of Bonn. NVPVS can be clinical appreciated when symptoms including vitreous hemorrhage, retinal detachment and hypotony are recognized.

Recurrent vitreous hemorrhages occurs from the bleeding neovascularization in the region of the pars plana. Scleral depression of the extreme peripheral retina and pars plana in the region of the vitrectomy wound sites reveals the ingrowth of blood vessels originating from the wound site and extending circumferentially along the extent of the pars plana (Fig. 1–3). Neovascularization originating in one sclerotomy wound site joins the neovascular growth originating from the other pars plana sclerotomy sites and completely encircles the pars plana region for 360 degrees.

The massive fibrosis fueled by the neovascularization causes contraction of the vitreous base and tractional retinal detachment in the region of the pars plana and anterior retina. The tractional forces may cause retinal tears transforming a purely tractional retinal detachment into a combined tractional and rhegmatogenous retinal detachment.

Effective surgical correction of this type of retinal detachment requires recognizing the syndrome of NVPVS and performing the appropriate surgical procedures to address both its fibrotic and vascular components. Lensectomy, vitreous base dissection and lysis of the anterior hyaloidal membrane with application of intraocular diathermy to the pars plana neovascularization at its origin in the sclerotomy wound sites are important surgical steps.

A lensectomy at the beginning of the surgical repair is recommended in order to create the

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room for the surgical maneuvers necessary to treat the anterior hyaloidal fibrosis and neovascularization. The vitreous base must be meticulously dissected and shaved down close to the retinal surface. The anterior loop traction that remains can be relieved by inserting a Machemer MPC intraocular scissors into the potential space between the pars plana and the overlying fibrotic anterior hyaloidal membrane. Special attention must be directed to the internal aspect of the sclerotomy wound sites. Fibrosis is extraordinarily thick at the internal wound site and after cutting back vitreous strands to the wound, the fibrosis must be circumcised to relieve any surrounding traction upon the peripheral nonpigmented pars plana epithelium and anterior retina. The remaining nubbin of tissue emanating from the sclerotomy wound site contains well established vascular elements which are at the root of the neovascular process extending circumferentially along the entire pars plana. These vascular elements must be carefully identified and treated with ablative intraocular diathermy in order to prevent recurrent fibrovascular proliferation and vitreous hemorrhages.

Certain conditions which predispose to the development of NVPVS can be avoided at the time of the initial pars plana vitrectomy procedure. Incarceration of vitreous strands from the vitreous base in the sclerotomy at the end of a pars plana vitrectomy can be avoided. When incarceration of the vitreous is recognized, the extruding vitreous can be cut away with a Weckcel sponge and scissors or the vitrectomy instrument may be used on the external scleral surface and the extruding vitreous cut away using gentle aspiration until the wound is free of vitreous strands. The incarceration of vitreous strands in the pars plana wound site form “wick” along which the fibrovascular ingrowth may proliferate
into the intraocular locus. Tight and meticulous closure of the sclerotomy wound site after the pars plana vitrectomy operation is an important mechanical barrier to the development of fibrovascular ingrowth from the episcleral vessels. In histopathological studies of the pars plana incisions poorly closed sclerotomy wounds were found in some cases of NVPVS (Figure 3).

**Discussion**

Pars plana vitrectomy has been performed for nearly 30 years. The complications of pars plana vitrectomy have been well documented. Acknowledged complications include cataract, vitreous hemorrhage, retinal tears and detachments, uveitis, sympathetic ophthalmia, hypotony, hyphema, choroidal effusion and suprachoroidal hemorrhage, glaucoma and endophthalmitis. Neovascularization of the pars plana vitrectomy site has been less well recognized. First described by Kreiger and Straatsma in 1977, it is an uncommon, but serious complication of pars plana vitrectomy. The fibrovascular response is unique and separate from anterior hyaloidal proliferation which can occur without antecedent vitrectomy operation and without extensive pars plana neovascularization which is typical of NVPVS.

Recognition of NVPVS and surgically addressing its essential components is crucial for repairing the associated retinal detachment. The incidence of NVPVS in my practice is 0.4%. Mild neovascularization of the pars plana vitrectomy wound site may occur frequently in the early wound healing stages in the first two or three weeks after the operation. In this early stage, fibrosis and vascularization are accepted as part of the normal healing process. The healing response requires vascularization in order to import monocytes and structural proteins such as fibrin and collagen to the wound site. After the scar of wound healing is complete, the local proliferative response normally involutes and the vascularization disappears leaving only a localized fibrous scar at the PPV sclerotomy wound site (Figure 2).
The abnormal response occurs when the normal healing fibrovascular response does not end with normal scar, but continues to proliferate into the PPV wound site. The process continues to spread over the entire pars plana forming dense fibrosis and an extensive network of neovascularization (Figure 1). The vascular network fuels the fibrotic response to develop extensive anterior loop tractional and retinal detachment. The condition is further complicated because the extensive neovascular component causes recurrent vitreous hemorrhages.

The stimulus to vascular proliferation is initially a part of the normal wound healing response, but the normal fibrovascular proliferation becomes exaggerated when accompanied by anterior retinal ischemia from underlying conditions such as diabetic retinopathy, carotid stenosis and arteriolosclerosis or even a tight encircling scleral buckle. The retinal ischemia elicits angiogenic factors and the episcleral vessels which supply the healing response at the pars plana vitrectomy wound site are transformed into aggressive neovascular networks which penetrates the sclerotomy wound site to proliferate in the region of the pars plana. This type of neovascularization, once established in the pars plana, further fuels the fibrotic response stimulating anterior hyaloidal proliferation and tractional retinal detachment. The anterior loop traction can also lead to hypotony caused by detachment of the ciliary body epithelium.

In addition to retinal ischemia, the detachment of the retina and nonpigmented neuroepithelium further stimulate the fibrotic response typical of proliferative vitreoretinopathy (PVR), but in the case of NVPVS, the proliferative response is more massive because it is fed by extensive neovascularization.

There are two surgical goals: 1) elimination of the fibrovascular response, and 2) reattachment of the retina. The anterior proliferative vitreor-
retinopathy must be carefully excised with meticulous anterior vitreous base dissection. The fibrosis of the anterior hyaloidal face results in anterior loop traction and tractional retinal detachment of the retina. The traction can only be relieved by cutting the contracted anterior hyaloidal membrane over the surface of the pars plana. A potential space exists between the pars plana epithelium and the overlying fibrotic and contracted anterior hyaloidal membrane. After making an incision into this space, the Machemer MPC instrument can be placed into the space and the anterior loop membrane cut. In areas where the adhesion of the anterior hyaloidal membrane is not firmly established to the ciliary body and pars plana, the vitrectomy instrument can be used to enter the potential space and cut the fibrotic anterior hyaloidal membrane bridging the pars plana. This surgical maneuver must be carried out for 360 degrees along the entire vitreous base.

All sclerotomy wound sites must be identified and the vascular elements which form the root of an extensive neovascular network must be destroyed with the application of intraocular diathermy or intraocular cautery. The internal wound site must be circumcised of all surrounding fibrosis attached to the surrounding pars plana and retina reducing the internal fibrovascular wound site to a small nubbin of atrophic, white scar tissue. A scleral buckle may be placed to relieve residual contraction of the vitreous base and to support retinal breaks in the retinal periphery in an effort to repair the retinal detachment.

Conclusion

NVPVS is a relatively rare and unrecognized complication of pars plana vitrectomy which can result in serious consequences including recurrent vitreous hemorrhages, tractional and rhegmatogenous retinal detachment and hypotony. Recognition of this unique process and the initiation of the appropriate surgical steps including vitrectomy, excision of the vitreous base fibrosis, lysis of anterior loop traction and application of intraocular diathermy or intraocular coagulation to the neovascular ingrowth at the sclerotomy wound sites are crucial steps in controlling the recurrent vitreous hemorrhages, repairing the retinal detachment and correcting hypotony.

References