Tube Erosion Following Insertion of a Glaucoma Drainage Device With a Pericardial Patch Graft

Glaucoma drainage devices are currently used to manage high-risk, complicated, adult and pediatric glaucoma when standard filtration surgery with an antimetabolite is unsuccessful. The drainage device consists of a plate and a tube. The tube is directly implanted either into the anterior chamber or through the pars plana in eyes undergoing vitrectomy. Tube coverage is imperative to prevent conjunctival erosion, which would lead to tube exposure and pose a risk for the development of endophthalmitis.

Sclera, dura, fascia lata, and pericardium have been employed to cover the tube and fistula sites. Raviv et al recently published safety data in a study of 44 patients (44 eyes) who had a pericardial patch graft placed to cover a glaucoma drainage device. In their retrospective study, they reported 5 cases of asymptomatic thinning of the pericardial patch graft without evidence of tube erosion. No cases of infection were reported. The mean ± SD follow-up was 10.2 ± 4.0 months. Herein, we report 2 cases of tube erosion through the conjunctiva following use of commercially prepared pericardial patch grafts occurring 7 and 8 months postoperatively.

Report of Cases. Case 1. A 74-year-old African American woman with a long-standing history of primary open-angle glaucoma in both eyes underwent an uncomplicated insertion of a 350-mm² Baerveldt glaucoma implant (Pharmacia Upjohn Co, Kalamazoo, Mich) in the right eye with a pericardial patch graft through a fornix-based conjunctival incision. One month later, because of conjunctival retraction with exposure of the tube and fistula site, she underwent exchange of the original pericardial patch graft for a larger pericardial patch graft as well as a conjunctival autograft. The original pericardial patch graft appeared intact but was replaced to provide better coverage of the insertion site.

Three months later the patient was found to have asymptomatic melting of the pericardial patch graft without conjunctival erosion. Four months later the patient returned complaining of foreign body sensation and mild ocular tenderness. On examination, her visual acuity was 20/40 + 1 OD. Slitlamp examination revealed trace conjunctival hyperemia with a 2-mm area of conjunctival erosion and tube exposure. There was no visible pericardial graft tissue in the subconjunctival space. The cornea was clear. The anterior chamber demonstrated 1+ cells and flare without hypopyon. There were no vitreous cells. Fundus examination findings were unremarkable.

The patient was immediately treated with a topical fluoroquinolone (ciprofloxacin) hourly for presumed endophthalmitis. The following day she underwent repositioning of the tube, which was covered with a 5 × 5-mm piece of full-thickness donor sclera. The conjunctiva was mobilized from the supronasal quadrant to cover the scleral patch graft.

The patient did well with complete resolution of the anterior chamber reaction. The topical ciprofloxacin antibiotics and prednisolone acetate were tapered. All cultures continued to yield no organisms. Fifteen months after surgery, her best corrected visual acuity was 20/25 - 1 OD, the overlying conjunctival tissue was intact, and the scleral patch graft had no observable thinning.

Case 2. An 89-year-old African American woman with a long-standing history of primary open-angle glaucoma in both eyes and multiple ocular surgeries underwent insertion of an Ahmed glaucoma valve (New World Medical Inc, Rancho Cucamonga, Calif) in the left eye with a 4 × 4-mm pericardial patch graft through a fornix-based conjunctival incision. Three months after surgery the pericardial patch graft was no longer visible. Five months later she came to the office complaining of a 4-day history of pain and blurred vision in the left eye. On examination, her visual acuity had decreased from 20/30 to 20/400 OS. Slitlamp examination revealed diffuse conjunctival hyperemia and a full-thickness conjunctival defect overlying the tube 3 mm posterior to the limbus. No pericardial tissue was visible. There were no sutures at the site of the defect. The anterior chamber demonstrated 3+ cells and flare with a 2-mm layering hypopyon. There were 1+ anterior vitreous cells. Fundus examination findings were unremarkable.

She underwent a vitreous tap and intravitreal injection of vancomycin hydrochloride and ceftazidime as well as topical and intravenous administration of vancomycin and ceftazidime for presumed endophthalmitis. Five days later she underwent surgical repair of the conjunctival defect. Intraoperatively, no remnants of the pericardial patch graft were visible. A 5 × 5-mm scleral patch graft was placed over the tube and covered with a conjunctival flap that was mobilized anteriorly and secured to the limbus.

Eight months postoperatively, her visual acuity improved to a baseline acuity of 20/30 OS. The conjunctiva was completely intact, and there was no evidence of scleral patch graft thinning.

Comment. The use of cadaveric allografts has been gaining wider acceptance for use in ophthalmic sur-
surgery, especially for coverage of glaucoma drainage devices. Pericardial, dura mater, and fascia lata patch grafts have been used. The reported advantages with pericardial patch grafts include uniform size and quality, commercial availability without dependence on an eye bank, potentially lower costs, and a processing method that leads to enhanced immunologic safety and reduced risk of viral transmission.

The dehydration process leaves the graft cell free and without antigenic stimuli. Tissue sterilization with organic solvents as well as low-dose irradiation leads to the inactivation of potential infectious pathogens, including human immunodeficiency virus and Creutzfeld-Jakob virus.

Despite the favorable result reported by Raviv et al., these 2 cases demonstrate that progressive thinning of the pericardial patch graft may occur in patients without predisposing ocular and systemic factors, such as uveitis, or other systemic immunologic disorders. Furthermore, specific antigen-mediated thinning is unlikely to be a major cause of progressive graft resorption owing to the manner in which the pericardial tissue is processed, leaving the tissue virtually antigen free. Surgical factors, such as exposed sutures, tight conjunctiva, or tube malposition, were not contributory in either case. Both cases of erosion occurred at a site over the tube previously covered with a pericardial patch graft. Moreover, in both cases a fornix-based conjunctival flap with superior and temporal relaxing incisions was used at the time of initial surgery to allow tension-free conjunctival apposition to the limbus. Although a conjunctival autograft was used in the first patient owing to retraction, the original pericardial patch graft was still intact. Additionally, the new pericardial patch graft and conjunctival autograft were placed over the tube without tension. Thus, it is unlikely that graft melting and conjunctival erosion, which occurred 7 months later, were related to excessive tension overlying the tube. Asymptomatic thinning of the graft may eventually lead to tube erosion with subsequent development of intraocular infection. Pericardial patch graft thinning was observed in both cases many months before the acute onset of presumed endophthalmitis.

In addition to these 2 cases, we have observed 7 other eyes with asymptomatic thinning of the pericardial patch graft. Currently, the appropriate mode of action in such cases is unknown, and judgment is reserved to the treating physician on a case-by-case basis.

The long-term safety of pericardial patch grafts for tube coverage is currently unknown. Without a prospective randomized study, the relative safety of pericardial vs other patch graft materials cannot be definitively determined. However, careful clinical observation may help identify potential problems with newer materials.

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**Permanent Ligation of Double-Plate Molteno Implant Distal Tube to Control Late Hypotony**

Hypotony is an uncommon complication to develop late after placement of a double-plate Molteno implant. Treatment to date for this has been limited to 2 options: permanent reocclusion of the proximal tube or removal of the tube from the anterior chamber. These methods have the disadvantage of eliminating the entire effect of the implant. The double-plate Molteno implant, however, has the potential advantage of reducing, not eliminating, drainage by stopping drainage to the distal plate. In the following case, we used a new technique, permanent ligation of the distal tube, which allowed treatment of late postoperative hypotony with long-term pressure control and minimal surgical morbidity.

To permanently ligate the distal tube, we incised the conjunctiva and Tenon layer in the space between the distal plate and the overlying rectus muscle. We then bluntly dissected under the muscle and located the tube, taking care to spare the fibrous capsule around the distal plate. Next, we tied a clove-hitch knot around the tube with 6-0 polypropylene suture (Figure). The conjunctiva and Tenon layer were sutured with a 9-0 braided polyglactin suture.

Report of a Case. We implanted a Molteno double-plate drainage device with adjunctive intraoperative mitomycin C in the left eye of a 17-year-old woman who had a history of congenital ocular tubella syndrome (congenital glaucoma, aphakia, and high hyperopia). A previous trabeculectomy with mitomycin C in that eye had failed. To prevent early postoperative hypotony, we placed a 6-0 polypropylene suture in the proximal tube and ligated the tube with a 6-0 polypropylene suture. After
8 weeks, we removed the intraluminal suture, and her intraocular pressure (IOP) decreased from 28 to 7 mm Hg without medication. For the first 4 months after removing the intraluminal suture, her IOP ranged from 14 to 16 mm Hg and her visual acuity remained at 20/50 OS.

Six months after removing the intraluminal suture, the patient complained of fluctuating, decreased visual acuity. Her visual acuity was 20/200 OS. Her IOP was 7 mm Hg. Fundus examination revealed horizontal macular folds. We diagnosed hypotony with maculopathy and ligated the distal tube of her Molteno implant. In the next several months her visual acuity improved to 20/70 OS, her IOP ranged from 18 to 19 mm Hg with treatment of 1 drop (approximately 20 μL) per day of timolol maleate, and her macular folds substantially decreased.

Comment. To halt and reverse the progression of complications associated with hypotony requires elevation of IOP. Comparison of glaucoma implants in rabbits has shown that the amount of filtration relates to the surface area available for filtration. Consistent with this observation, it has been observed that single-plate Molteno implants result in higher IOP and fewer complications related to hypotony than double-plate implants. Ligation of the distal tube of a double-plate Molteno implant, therefore, raises IOP and treats hypotony because it halves the surface area available for filtration, and yet it still allows filtration through the proximal plate in those situations where some drainage is required.

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Incomplete Spontaneous Regression of Choroidal Melanoma Associated With Inflammation

Spontaneous regression of cancer is a rare occurrence that has been documented with several cancers, including cutaneous melanoma. In cutaneous melanoma the regression is typically partial, occasionally displaying transient dermal inflammation with subsequent pink-grey atrophic changes. Rarely does cutaneous melanoma spontaneously disappear completely, and relapse is common.

Observations of spontaneous regression of choroidal melanoma are exceedingly rare. Of over 8000 patients with choroidal melanoma cared for at the Ocular Oncology Service at Wills Eye Hospital, Philadelphia, Pa, we have personally witnessed and photographically documented spontaneous regression in only 3 patients. We suspect that there may be additional cases of tumor regression that remain subclinical with partial necrosis, not appreciated on ophthalmoscopy or ultrasonography.

Report of Cases. Case 1. A 60-year-old man noted pain and photopsia in his left eye for 3 days in September 1995. Supronasal conjunctival chemosis and episcleral hyperemia were noted. Funduscropy revealed a minimally pigmented choroidal mass measuring 6.0 mm at the base and 2.2 mm in thickness, with an overlying serous retinal detachment. On ultrasonography, the mass showed acoustic solidity, choroidal excavation, and echolucency in the overlying episcleral tissue. The patient returned 2 months later without pain or chemosis; the choroidal mass had flattened to 1.2 mm in thickness, and the serous retinal detachment had resolved.

In April 1996, the choroidal mass had enlarged to 2.8 mm in thickness, and the patient was referred to us in September 1996. On examination, the left fundus demonstrated a lightly pigmented choroidal mass measuring 11.0 mm at the base with prominent subretinal fluid. A necrotic central area within the tumor surrounded by a viable peripheral rim was noted. Ultrasonography revealed an excavated, acoustically hollow mass measuring 4.0 mm in thickness. The diagnosis of spontaneously regressed choroidal melanoma with recurrence was made. The tumor was treated with plaque radiotherapy.

Case 2. A 45-year-old man noted redness, irritation, and pain in his left eye of 1 day’s duration in March 1998 (Figure). Funduscropy revealed a supronasal, juxtapapillary pigmented choroidal mass measuring 14.5 mm at the base and 7.6 mm in thickness. Ocular B-scan ultrasonography revealed an acoustically hollow choroidal mass with overlying episcleral echolucency. A periocular corticosteroid injection was administered for relief of inflammatory pain.

In April 1998, a strikingly smaller choroidal mass measuring 5.3 mm in thickness was noted. In May 1998, the mass continued to decrease to 3.5 mm in thickness and on referral to us in June 1998, it was 3.2 mm thick. Evidence of prior subretinal fluid with retinal pigment epithelium mottling of the inferonasal fundus was found. Our diagnosis was choroidal melanoma with partial spontaneous regression. Options for treatment included continued observation, radiotherapy, or enucleation. The tumor was treated with a notched radioactive plaque and supplemental thermotherapy.

Case 3. A 78-year-old woman with Alzheimer disease developed ocular redness, swelling, and extreme pain in her right eye in June 1998. Funduscropy revealed a total bullous serosanguineous retinal detachment with shifting subretinal fluid. A large pigmented choroidal mass measuring 20.0 mm at the base and 11.7 mm in thickness was seen. Ocular ultrasonography confirmed a mushroom-shaped chilochoroidal mass with intrinsic vascular pulsations, consistent with melanoma. Enucleation was advised. At the time
A 45-year-old man noted redness, irritation, and pain in his left eye of 1 day’s duration. A, March 1998. A juxtapapillary choroidal mass with overlying serous retinal detachment obscuring the optic disc is found. The ultrasonographic thickness was 7.6 mm. B, April 1998. After resolution of symptoms, the variably pigmented choroidal mass has spontaneously regressed, exposing the optic disc. The ultrasonographic thickness was 5.3 mm. C, June 1998. The choroidal mass continues to spontaneously regress with overlying retinal pigment epithelium alterations. The ultrasonographic thickness was 3.2 mm. D, June 1998. B-scan ultrasonogram demonstrates the untreated, regressed choroidal melanoma.

of enucleation, 8 days later, the inflammatory symptoms had completely resolved leaving the patient comfortable, without eyelid or conjunctival edema. On ophthalmoscopy, the tumor was dramatically smaller, measuring 8.5 mm in thickness. We suspected that the melanoma had undergone partial spontaneous regression.

Histopathologically, the enucleated globe demonstrated a highly necrotic ciliochoroidal malignant melanoma. The necrotic cells were rimmed by macrophages, and the viable cells demonstrated features of spindle and epithelioid malignant melanoma cells.

Comment. Spontaneous regression of cancer is a remarkable but rarely observed phenomenon, estimated to occur in 1 in 80,000 to 100,000 cases of cancer. It is defined as the complete or partial disappearance of a neoplasm in the absence of treatment. Spontaneous regression can occur with many systemic cancers, but it has not been correlated with complete tumor cure as most will ultimately recur. In a major review of all of the literature on 504 cases of spontaneously regressed cancer from 1966 to 1987, Challis and Stam found the primary cancer sites to be cutaneous malignant melanoma in 14%, renal cell carcinoma in 13%, lymphoma in 13%, leukemia in 11%, neuroblastoma in 8%, retinoblastoma in 6%, and breast cancer in 4%.

From the standpoint of cutaneous melanoma, it is believed that some degree of spontaneous regression is detected in 10% to 30% of cases. The clinical features suggesting regression are the development of depigmented areas and inflammation. The histopathologic appearance of cutaneous melanoma regression includes degeneration of tumor cells, lymphohistiocytic infiltrate, pigmented macrophages, dermal fibrosis, and epidermal atrophy. The prognostic significance of spontaneous regression with cutaneous melanoma is unclear, but some investigators feel it is associated with a worse prognosis. Sondergaard and Hou-Jensen found that stage I cutaneous melanoma has a 95% 10-year survival rate in those without regression and a 79% 10-year survival rate in those with spontaneous regression. In unusual circumstances, the primary site of cutaneous melanoma completely regresses leaving no clinical trace of tumor, but these patients are still at risk for metastatic melanoma.

Spontaneous regression of choroidal melanoma has been recognized, often masquerading as scleritis. Unfortunately, the regressed tumor often relapses. In this report we document photographically the clinical features of 3 patients, all of whom had ocular inflammation. The rate of tumor regression varied from...
Intravascular Papillary Endothelial Hyperplasia With Presumed Bilateral Orbital Varices

Intravascular papillary endothelial hyperplasia (IPEH) is an unusual condition characterized by a benign proliferation of vascular endothelial cells that form papillary projections in the lumen of a blood vessel. It is not a specific neoplasm, but is generally believed to be a reactive response that develops secondary to a thrombus in vascular lesions such as varices or hemangiomas.1-3 It most often occurs as a painless, reddish purple lesion in the dermis or subcutis of the head and neck region or extremities.4-6 Intravascular papillary endothelial hyperplasia has been recognized to occur rarely in the ocular area, usually in the eyelid7-10 and less often in the orbit.11 In reported ocular cases, IPEH has occurred in association with a unilateral, solitary vascular lesion.7-10 We report a case of IPEH that developed in association with multiple bilateral orbital vascular tumors, thus expanding the known ocular spectrum of this condition.

Report of a Case. In August 1991, an 80-year-old woman was seen with a 6-month history of slowly progressive, painless proptosis of the right eye. She had a history of successfully treated uterine cervical cancer many years earlier and a cataract extraction in the right eye 6 years earlier, but had no other systemic or ocular problems. Specifically, she had no history of trauma, ocular inflammation, cutaneous hemangioma, or prior episodes of proptosis.

Her visual acuity was 20/20 OD and 20/30 OS and intraocular pressures were normal. There was bilateral fullness of the upper and lower eyelids and 5 mm of proptosis of the right eye (Figure 1). The proptosis was not exacerbated by head position or Valsalva maneuver. Most of the fullness seemed to be caused by anterior displacement of orbital fat, as there was no distinctly palpable mass. Ocular motility was normal. The remainder of her ocular examination revealed normal findings.

Orbital computed tomography disclosed bilateral orbital masses. In the right orbit there was an intracranial mass that measured 17 × 17 × 15 mm (Figure 2) and displaced the optic nerve superiorly. Lesions in the left orbit were not clearly delineated. However, on subsequent computed tomographic examination, a second separate tumor, measuring 12 × 12 × 12 mm, was noted in the region of the superior orbital fissure of the right orbit with posterior extension into the middle cranial fossa (Figure 3). In the left orbit was an irregular mass nasal to the globe with ill-defined extension toward the orbital apex (Figure 3). Our differential diagnosis included metastatic carcinoma, lymphoma, multiple orbital cavernous hemangiomas, and orbital varices.

We elected to perform an excisional biopsy of the larger, symptomatic, intracranal mass in the right orbit by a superolateral orbitotomy through a cutaneous incision with an extraperiosteal approach and lateral osteotomy (Kronlein approach).12 A reddish blue mass was identified and removed intact. The patient had an uneventful postoperative course.

Pathologic Findings. Grossly, the slightly deflated, irregular, dark-red mass measured 17 × 15 × 10 mm. Microscopically, the lesion was a vascular mass composed of large, thin-walled, blood-filled channels lined by benign endothelial cells. There were localized areas of chronic inflammation. Approximately 70% of the mass was composed of slender fibrous trabeculae lined by benign endothelial cells that projected into the lumen of large vascular channels (Figure 4). The final diagnosis was IPEH, arising in a thrombosed varix.

Three years after surgery, the patient's visual acuity was 20/40 OD and 20/30 OS. There was 2 mm of right proptosis and slight pallor of the right optic disc. Orbital computed tomography disclosed that all of the remaining lesions were stable.

Comment. Intravascular papillary endothelial hyperplasia usually occurs in the head and neck region and the extremities.14 Ocular involve-
ment is exceptionally rare. In the series of 44 cases reported by Clearkin and Enzinger, 4 17 cases reported by Kuo et al, 5 and the 91 cases reported by Hashimoto et al, 6 there was no mention of specific lesions in the eyelids or orbit.

To our knowledge, the first reported bona fide case of eyelid IPEH was by Wolter and Lewis 7 in 1974. Although these authors cited 3 prior eyelid cases, these subsequently were not accepted by Font et al, 9 who considered the patient of Wolter and Lewis 7 to be the first acceptable case. Additional eyelid cases were recorded by Sorenson et al 8 and Font et al 9 in 1983. In 1997, Werner et al 10 compiled the experience of several surgeons and added 4 eyelid cases, one of which extended to the lateral orbital rim.

Even fewer cases of deeper orbital involvement with IPEH have been recorded. The first case was reported by Weber and Babel 11 in 1981. In 1983, Font et al 9 added 3 orbital cases from the files of the Armed Forces Institute of Pathology. A similar histopathologic response was also observed in a patient with a variant of the Sturge-Weber syndrome. 13 Based on the aforementioned reports, it seems that there have been approximately 8 reported cases of eyelid IPEH and 5 reported cases of orbital IPEH in the English-language literature.

Our case of IPEH is unusual for 2 reasons. First, it occurred in bilateral orbital vascular lesions, presumably varices. Second, it was associated with another lesion that extended through the superior orbital fissure into the cranial cavity. Recently, a few cases of IPEH have been recognized to arise from intracranial vascular lesions, and these were summarized by Werner et al. 10 It has also been recognized in the region of the superior orbital fissure, 14 as occurred in our case.

Based on the histopathologic findings, the IPEH in our patient probably arose in a thrombosed varix. Orbital varices usually are primary lesions, but they occasionally can arise secondary to an intracranial arteriovenous communication that shunts arterial blood to the venous system, causing secondary dilation of orbital veins. 5 The fact that the vascular lesions in our patient were bilateral and multiple raised the possibility of secondary orbital varices. However, our patient had no history of trauma or clinical signs of carotid-cavernous fistula. Therefore, the pathogenesis of the underlying vascular lesion in our case remains obscure. It is possible that the presumed varices in our patient had been present for many years and that IPEH was a response to thrombosis in the larger lesion in the right or-

Figure 1. Facial appearance showing bilateral fullness of eyelids and proptosis of the right eye.

Figure 2. Axial computed tomographic scan of orbits showing large circumscribed right intracranial mass. The lesion in the right orbital apex and the lesions in the left orbit were not clearly delineated.

Figure 3. Axial computed tomographic scan of orbits done 5 months later, better delineating an irregular mass in right muscle cone with extension into the cranial cavity and a mass in the nasal aspect of the left orbit. The lateral bony wall of the right orbit is missing following the lateral orbitotomy.

Figure 4. Photomicrograph shows intravascular papillary endothelial hyperplasia filling lumen of an orbit varix. (Left, hematoxylin-eosin; original magnification ×10; right, hematoxylin-eosin; original magnification ×100.)
bit that contributed the progressive proptosis.

Intravascular papillary endothelial hyperplasia can be found histopathologically in vascular lesions of the eyelid and orbit, such as varic, cavernous hemangioma, and lymphangioma. Small intraluminal foci of IPEH are not uncommon, which attests to the reactive nature of the process. Many patients who have presumed benign vascular lesions of the orbit are diagnosed with computed tomography or magnetic resonance imaging and are followed up without treatment. If such a patient with a previously dormant lesion develops acute or progressive proptosis, however, the possibility of IPEH, rather than tumor growth or malignant transformation, should be considered. In IPEH, the proptosis can be gradual or rapid in onset, the latter possibly resulting from inflammation associated with an acute thrombus.

The histopathologic differential diagnosis includes angiosarcoma and angiolymphoid hyperplasia with eosinophilia (Kimura disease). Angiosarcoma is particularly difficult to differentiate from IPEH. However, it has more malignant cytologic features and invades tissue outside the vascular lumen.2-4 Kimura disease has a predominance of lymphocytes and eosinophils.2-4

In summary, this case demonstrates that orbital IPEH can occur in patients with multiple orbital vascular tumors, as well as solitary lesions. Intravascular papillary endothelial hyperplasia should be included in the differential diagnosis of acquired progressive proptosis in adults.

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From the Archives of the ARCHIVES

A look at the past . . .

The roots of the trouble go back very far. When aniseikonia was first presented to ophthalmologists, serious objections were raised by the leaders of the medical profession. These were never properly answered. Instead, exaggerated claims for aniseikonia were made which could not be substantiated. The cool reception by the members of the profession led, in turn, to an exaggerated sensitivity toward criticism, and an approach to less critical laymen was made in person or through the press which would not be approved by the members of the medical profession, either within or outside the Institute.