Late Closure of Argon Laser Iridotomies Following Regrowth of Iris Pigment Epithelium

Closure of previously patent laser iridotomies has been described as occurring early or late after the procedure. Early closure, occurring from minutes to hours after completion of the iridotomy, results from landsliding of iris pigment epithelium from above the site of the iridotomy or from large pigment particles floating in the posterior chamber, lodging in the iridotomy site, and occluding it. Late closure may occur due to occlusion by the settling of dispersed pigment and/or inflammatory debris into a small iridotomy and/or proliferation of pigment with bridging of the iridotomy and usually occurs after days to weeks. In most cases, this pigment appears to come from the stroma, and the pigment plugging of the iridotomy is anatomically at the level of the iris stroma.

Iris pigment epithelial proliferation has been considered extremely rare and to our knowledge has not been previously described after laser iridotomy. Closure of iridotomies by proliferation of iris pigment epithelium has been alluded to in various reports, but published photographs indicate pigment proliferation plugging the iridotomy at the level of the stroma, not at the level of the iris pigment epithelium. We present 2 cases in which true regrowth of the iris pigment epithelium solely at the appropriate anatomic level resulted in recurrence of pupillary block.

Report of Cases. Case 1. A 57-year-old man underwent argon laser iridotomy in his right eye for appositional angle closure. Postsurgery, the angles were grade 2 and grade 3 open. The iridotomy was noted to be closed by iris pigment epithelial regrowth 30 months later. There was no filling of the stromal defect, which remained fully patent (Figure 1, left). Gonioscopy revealed 180° of appositional closure, while the remainder of the angle was grade slit-I. This was confirmed by ultrasound biomicroscopy (UBM; Paradigm Medical Laboratories Inc, Salt Lake City, Utah) (Figure 1, right). The pigment was easily dispersed with a few bursts of low energy argon laser, and the iridotomy has remained patent for 5 years without iris pigment epithelial regrowth.

Case 2. A 71-year-old women with angle-closure glaucoma underwent argon laser iridotomy in her left eye. Postsurgery, the iridotomy was patent, and the angle was grade 3. Four months later, iris pigment epithelial regrowth was noted to occlude the iridotomy site and the angle was narrow (Figure 2). A touch-up laser procedure was performed, following which the iridotomy has remained patent.

Comment. Pigment proliferation has been implicated in closure of iridotomies created by both argon and Nd:YAG lasers but is more commonly seen with those made with the argon laser. What has been termed late closure classically occurs anatomically at the level of the iris stroma within a few weeks to months after the procedure. This type of closure rarely occurs after the iridotomy has been patent for longer. It is not really clear whether this proliferation is of pigment epithelium or stromal pigment or both. Thermal drainage to the iris pigment epithelium surrounding the iridotomy may be responsible.

Our cases differ from these findings. The first patient had an iridotomy that was functioning and patent until regrowth of iris pigment epithelium 21⁄2 years later. The second had closure of the iridotomy approximately 4 months postsurgery. In both cases, ultrasound biomicroscopy was able to demonstrate occlusion of the iridotomy solely at the iris pigment epithelial level with no filling of the stromal defect. In eyes with late closure of the iridotomy due to true iris pigment epithelial regrowth, the cells proliferate into the opening from the en-
Figure 2. Ultrasound biomicroscopy demonstrating closure of iridotomy by iris pigment epithelium pupillary block (arrow). AC indicates anterior chamber; C, cornea; I, iris; and CB, ciliary body.

Figure 3. Clinical photograph demonstrating partial late closure of argon laser iridotomy by iris pigment epithelium with a small central patency (arrow) 3 years after iridotomy.

Figure 3. Clinical photograph demonstrating partial late closure of argon laser iridotomy by iris pigment epithelium with a small central patency (arrow) 3 years after iridotomy.

Malignant Melanoma Arising From a Large Uveal Melanocytoma in a Patient With Oculodermal Melanocytosis

A 51-year-old man with ocular melanocytosis was seen with a pigmented uveal mass that extended from the iris to the macular region in the ipsilateral left eye. Diffuse uveal melanoma was suspected, and multiple foci of extrascleral pigment were discovered at enucleation. Histopathologically, the lesion was a diffuse uveal tumor composed mostly of cells identical to a melanocytoma. In the center of the mass was an island of amelanotic melanocytoma. The extracocular component was consistent with benign melanocytoma. The patient is alive and well after 20 years. Based on observations in this case, we discuss the relationship between ocular melanocytosis and melanocytoma and emphasize that they may represent different clinical expressions of the same congenital pigmentary abnormality.

Ocular melanocytosis is a congenital condition characterized by hyperpigmentation of the uvea, tract, sclera, and episclera. When the periocular skin is involved, it is termed ocular melanocytosis or nevus of Ota. Melanocytoma is a deeply pigmented variant of uveal nevus that was originally described in the optic disc but is now known to occur in all parts of the uveal tract. Although both ocular melanocytosis and melanocytoma can give rise to uveal melanoma, there has been a tendency among ophthalmologists to consider them as different entities. We describe a patient with ocular melanocytosis and a large diffuse uveal melanocytoma that gave rise to melanoma.

Report of a Case. In April 1979, a 51-year-old white man was referred to one of us (J.A.S.) because of a pigmented lesion in his left eye. His visual acuity was 20/15 OS and intraocular pressure was 8 mm Hg OU. The right eye was normal. The patient had a congenital cutaneous blue nevus, 2 cm in diameter, near the lateral aspect of the left eyebrow but not periocular cutaneous pigmentation. There were several well-defined patches of gray pigmentation on the sclera inferiorly (Figure 1). A dark brown lesion was present in the iris inferotemporally (Figure 2). Gonioscopy revealed that the inferior aspect of the trabecular meshwork was deeply pigmented.

Findings from fundus examination disclosed a pigmented, sectorial, inferotemporal ciliochoroidal mass that measured 15 mm in diameter and 8 mm in thickness. It was contiguous with the pigmented lesion of the iris and trabecular meshwork. The remainder of the choroid inferiorly was deeply pigmented with extension of the flat pigmentation into the central macular region (Figure 3). Fluorescein angiography of the mass demonstrated relative hypofluorescence in the venous phase and mild late hyperfluorescence. A-scan ultrasonography showed a solid mass with
gradually diminishing internal reflectivity, and B-scan ultrasonography showed a peripheral choroidal mass with low internal reflectivity.

Based on clinical findings and ancillary studies, our diagnosis was ocular melanocytosis with sector uveal melanocytosis, giving rise to diffuse choroidal melanoma, and enucleation was advised. Inspection of the globe immediately after enucleation disclosed multiple, deeply pigmented nodules on the posterior aspect of the globe inferotemporally and around the optic nerve (Figure 4).

Pathologic Findings. The sectioned globe showed a diffuse, elevated, deeply pigmented uveal mass, measuring $20 \times 20 \times 8$ mm in the inferotemporal quadrant with patches of black pigmentation in the corresponding episcleral tissue (Figure 5).
Low magnification microscopy showed cystic degeneration in the peripheral part of the pigmented choroidal mass and a large focus of depigmentation in the postequatorial region (Figure 6). Review of bleached sections showed that the deeply pigmented portions of tumor and the extrascleral nodules were composed of bland, ovoid cells with uniform small nuclei, compatible with the diagnosis of ocular melanocytosis or melanocytoma (Figure 7). In contrast, the less pigmented foci were composed of spindle cells and a few epithelioid cells, compatible with a mixed-cell type melanoma (Figure 8). No mitotic activity was identified in the numerous sections studied.

Based on the clinical and histopathologic findings, the diagnosis was ocular melanocytosis with segmental uveal melanocytosis forming a melanocytoma with foci of amelanotic choroidal melanoma. The patient is alive and well after 20 years with no local recurrence or metastasis.

**Comment.** Although it was originally described as a lesion of the optic disc, melanocytoma subsequently has been recognized to occur in the uveal tract, including the iris, ciliary body, and choroid. Patients with ocular melanocytosis have an increased risk of developing uveal melanoma. Zimmerman has stressed the microscopic similarities shared by ocular melanocytosis and melanocytoma of the optic disc. He coined the term melanocytoma for the pigmented lesions of the optic disc because he observed that...
the cells comprising such lesions were nearly identical to the cells that diffusely involve the uveal tract in ocular melanocytosis.2

Based on our experience, we believe that most ophthalmologists consider melanocytoma and ocular melanocytosis to be 2 distinctly different pigmentary conditions. However, in our practice of ocular oncology, we have seen patients who had a melanocytoma of the optic disc that was contiguous with a widespread area of choroidal pigmentation identical to the choroidal changes of ocular melanocytosis.

Our belief that melanocytosis and melanocytoma represent different expressions of the same congenital pigmenitary process is also supported by the case reported here. Our patient, who had ocular melanocytosis, also had marked uveal thickening, which was composed of cells identical to those seen in melanocytoma. In 1986, Haas et al14 reported a similar case in a 10-year-old boy. They also observed that the cells that comprised the choroidal thickening were identical to those seen in a melanocytoma of the optic disc. In contrast to our case, the tumor in their patient did not have histopathologic evidence of a malignant component.

We believe that there is a variant of ocular melanocytosis characterized by marked thickening of the uveal tract by cells that have features typical of those seen with melanocytoma of the optic disc. This large uveal melanocytoma can undergo malignant transformation into melanoma, as shown in our case. A curious feature of this malignant transformation is that the malignant portion is only minimally pigmented whereas the primary benign lesion is deeply pigmented. We have documented 2 other cases in which a clone of relatively amelanotic melanoma cells was present in a deeply pigmented melanocytoma of the ciliary body.

In summary, our patient with ocular melanocytosis was atypical in that the uveal tract was markedly thickened and assumed tumorous proportions. The deeply pigmented uveal mass was composed of cells identical to those found in melanocytoma of the optic nerve. The melanocytosis contained an area of malignant transformation into amelanotic melanoma cells. The patient is alive and well after 20 years, suggesting that he may have a favorable prognosis. This unusual case underscores the similarity between ocular melanocytosis and melanocytoma and suggests that they may represent variations of the same entity.

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An Infratemporal Fossa Foreign Body Presents as an Infraorbital Mass

We share an interesting case of a child whom we saw with a low-grade inflammatory infraorbital mass.

Figure 1. Facial asymmetry as noted by narrowed left palpebral fissure, erythematous infraorbital skin, and fullness of the inferolateral left orbital rim.
secondary to a foreign body that localized to the infratemporal fossa. There are several unanswered questions about the ultimate source of the foreign body. However, this case stresses the importance of a broad differential diagnosis and the choice of imaging modality in children.

Report of a Case. A 7-year-old girl was seen with a 2-month history of increased prominence and erythema of her left zygomatic arch and surrounding tissues. The family recalled minor, blunt trauma that occurred several weeks prior to the change in the child’s appearance. The patient was in good health and medical history was otherwise unremarkable.

On examination, the patient was afebrile and in no distress. Visual acuity was 20/20 OU without correction. There was no globe malposition subjectively or by exophthalmetry. There was a firm, minimally tender immobile mass that was continuous with the left zygoma by palpation. The skin medial to the lesion had a violet discoloration (Figure 1). The subacute presentation without constitutional signs increased our suspicion of a primary malignant neoplasm of either the bone or soft tissue, as opposed to a noninfectious inflammatory process.

A computed tomographic scan of the head and orbit was obtained. It revealed a soft tissue lesion surrounding the left lateral orbit with extension into the infratemporal fossa. Additionally, there was hyperostosis of the left frontozygomatic arch (Figure 2, top). Within the infratemporal fossa extending to the superior alveolar ridge, there was a high-density foreign body that was contiguous with the soft tissue density (Figure 2, bottom). Subsequent examination of the oral cavity demonstrated a pyogenic granuloma in the buccal sulcus at the left level of the first upper molar (Figure 3).

The patient underwent a left anterior orbitotomy to explore the region of the inferolateral orbital rim corresponding to the region of clinical involvement and to further evaluate the abnormality seen on the computed tomographic scan. A fibrous soft tissue mass overlying the orbital rim was removed. Incision into the mass produced a scant exudate. The underlying periosteum of the zygoma was thickened. Through introral blunt dissection of the pyogenic granuloma and reflection of the periosteum of the maxilla, a 24-mm, metallic, cylindrical for-
eign body was located on the an- 
terolateral face of the maxilla and re- 
moved (Figure 4). The cavity was 
copiously irrigated and a Penrose 
 drain was temporarily left in place 
exiting into the mouth. Intraopera-
tively, cefazolin was administered in-
travenously and followed with a 
postoperative course of oral amoxicillin. Review of the frozen and per-
manent pathological specimens re-
vealed fibrotic tissue with chronic 
inflammation and reactive woven bone formation.

The postoperative course was 
uneventful. At 5 months, there was 
no suggestion of recurrence and the 
mouth was well healed. Further 
questioning of the child and family 
did not glean insight into the source 
of the foreign body. The case social 
worker and pediatrician found no 
evidence of child abuse.

Comment. To our knowl-
edge, this case represents the first re-
port of an infratemporal foreign body 
presenting as an inferior orbital 
mass. There are several cases of for-
eign bodies in this region present-
ing with trismus and acute inflam-
mation.1 However, these cases have 
been associated with a known di-
rect injury (ie, broken pen tip from 
stabbing). We speculate that entry 
to the infratemporal fossa was via the 
buccal sulcus.

Despite a thorough multidisci-
plinary inquiry, the exact mecha-
nism of injury could not be ascer-
tained. Without invoking alien 
intervention, our best working hy-
pothesis is that this pinlike foreign 
body may have become imbedded in 
the child’s face during a lacerating 
sledding accident 2 years prior to 
presentation. The foreign body may 
have been lodged within the epicen-
ter of the perizygomatic mass seen 
on computed tomography. The rela-
tively minor trauma sustained 2 
months prior to presentation may 
have dislodged the foreign body. Dis-
rup tion of the reactive fibrous cap-
sule may have permitted leakage of 
its contents, resulting in the low-
grade inflammatory picture seen on 
presentation.

This case is instructive on sev-
eral levels. Foreign bodies should be 
considered in the differential of all 
orbital and periorbital mass les-
ions, especially in children, who are 
notoriously poor historians. Accord-
ingly, the choice of initial radiologi-
cal imaging should attempt to rule 
out the existence of such mate-
rial.2,3 Also, in very rare cases, oral 
examination may provide insight 
to the origin of lesions present-
ing in the periorbital region.

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1. Shemen LJ, Schechter LS, Godfrey N. Needle-
wire localization of an infratemporal fossa for-
eign body using computer tomography. Arch Oto-
2. Kelly WM, Paglen PG, Pearson JA, San Diego AG, 
Soloman MA. Ferromagnetism of intraocular for-
eign body causes unilateral blindness after NM 
3. Murphy KJ, Brunberg JA. Orbital plain films pre-
requisite for MR imaging: is a known history of 
injury a sufficient screening criterion? AJR Am J 