repair. Pressure on the wound produced a small amount of discharge, which later grew methicillin-sensitive *Staphylococcus aureus* in culture. The wound was irrigated with bacitracin solution. The silicone sling ends were tucked into a preperiosteal pocket, without any trimming. Direct closure in 2 layers was performed with additional wound support provided by adhesive strips fixed with tincture of benzoin. Ophthalmic antibiotic ointment was applied after shedding of the adhesive strips. There was subsequent dehiscence over a couple weeks. The silicone sling was removed 6 months after insertion without difficulty. Exploration of the wound revealed pyogenic granuloma, which was excised. The wound was left to heal by secondary intention and closed by the first postoperative visit at 2 weeks. After the explant, the lid height and contour were unchanged after 1 year.

Silicone is a safe and effective material for the frontalis sling procedure.[1,2] It is inert and easy to work with and has become a popular choice.[3] Adjustability and elasticity are among the touted advantages. Removal is usually simple and, as demonstrated in our case, the fibrous connections between the eyelid and frontalis can be sufficient to maintain lid height and contour after explant. Complications are uncommon and limited to only the periocular area, because a remote harvest site is not required as in the traditional tensor fascia lata frontalis sling procedure popularized at our institution by Jack Crawford.[4] Extrusion of the silicone sling is an established infrequent complication. In the case described here, the unusual feature is that exposure did not occur at a surgical wound. The precise mechanism in our case cannot be established with certainty, but presumably the silicone tips prevented wound healing with secondary pyogenic granuloma formation and *S. aureus* colonization/infection. It is possible that the tissue overlying the silicone ends was more susceptible to laceration with trauma as a result of local inflammation or infection as a result of the foreign material. Despite mechanistic uncertainty, the case demonstrates an unusual event after frontalis silicone sling ptosis repair.

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Michel J. Belliveau, Dan D. DeAngelis
University of Toronto and The Hospital for Sick Children, Toronto, Ont.

Correspondence to:
Michel J. Belliveau, MD, FRCSC; mbelliveau42@gmail.com

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Multiple parallel-line endotheliitis: case report and literature review of an emerging entity

Although rare in the nontransplant setting, several forms of corneal endotheliitis have been described.[1] Viral replication (Herpes Simplex Virus, Varicella Zoster Virus, Cytomegalovirus and mumps) within the endothelium is a proposed etiology although response to antivirals and topical corticosteroids has been variable.[2–8] Multiple parallel-line endotheliitis (MPLE) is a rare form of endothelial inflammation characterized by horizontal lines of endothelial dots, stromal edema, and minimal anterior chamber reaction.[9–11] We present a case of MPLE and discuss the current literature on this emerging clinical entity.

A 56-year-old female presented with acute painless vision loss of the right eye noted when waking from sleep. She had no history of trauma, flashes, floaters, curtain defects, redness, epiphora, or discharge. The left eye was unaffected. She had a 3-day history of oral vesicular lesions and a recent upper respiratory tract infection. There was no ocular history and no ocular medication use. Medical history was significant for recurrent herpes simplex of the mouth, diabetes mellitus, hypercholesterolemia, gastroesophageal reflux, obstructive sleep apnea, overactive bladder, and post-traumatic stress disorder. Systemic medication had not changed within the past 12 months and included metformin, rosuvastatin, esomeprazole, tramadol, quetiapine, duloxetine, pregabalin, mirabegron, and lorazepam. The patient used a CPAP machine intermittently but not on the night preceding presentation. She did not recall sleeping with her hand against her face.

Best corrected visual acuity was 20/60 in the right eye and 20/30-1 in the left eye. Intraocular pressures were 18 and 19 mm Hg, respectively. Pupils, visual fields, and ocular motility were unremarkable. Ishihara colour testing was symmetric (10/14). Floppy eyelids were noted.

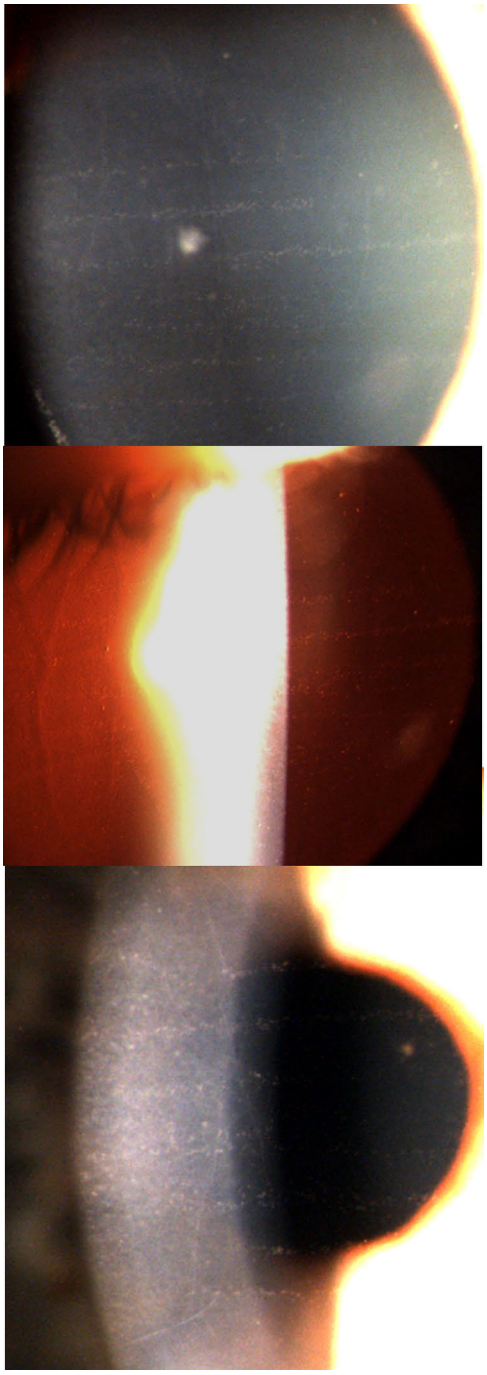

Fig. 1—Corneal changes seen on the right eye of a 54-year-old female presenting with acute painless decrease in vision demonstrating multiple dots arranged in horizontal parallel lines (A). The extent of these lines, from limbus to limbus, is highlighted on retroillumination (B), while a slit-beam localizes the changes to the endothelium (C).

Slit-lamp examination demonstrated mild inferior superficial punctate keratopathy. Evenly spaced horizontal dotted lines along the corneal endothelium were seen on the right in addition to mild stromal edema (Fig. 1). These lines were unchanged on indentation. The anterior chamber was deep and quiet bilaterally on presentation. The left cornea was clear and the remainder of the dilated ocular examination was unremarkable.

Central corneal thickness of 622 μm on the right and 572 μm on the left was measured. There was a decreased endothelial cell count on the right (2545 cell/mm^2) compared to the left eye (2941 cells/mm^2). Endothelial pleomorphism, spot-like holes, and polygonal deposits arranged in linear configurations were seen on confocal microscopy (Fig. 2). Corneal topography demonstrated no evidence of astigmatism or keratoconus, OCT of the anterior segment and macula were noncontributory (central macular thickness of 247 μm right and 248 μm left).

An aqueous sample from an anterior chamber paracentesis 4 days after presentation was submitted for polymerase chain reaction (PCR) testing before therapy. Findings of the PCR analysis for HSV 1 and 2, VZV, and CMV were negative.

Over the initial 4-day period without treatment, improvement in stromal edema and a decrease in endothelial dots were noted. Treatment with prednisolone acetate 1% and gatifloxacin 0.3% drops QID was initiated after the paracentesis. Within 4 days the endothelial lines had disappeared. Treatment with prednisolone was tapered, and 15 days after presentation, the findings had resolved.

Endotheliitis has been classified into 4 forms on the basis of keratic precipitate and stromal edema distribution: linear, sectoral, disciform, and diffuse.[2] Linear endotheliitis, which has been linked to HSV type 1 and CMV, is located peripherally with a single line of keratic precipitates demarcating the boundary of the process. Unlike linear endotheliitis, MPLE demonstrates multiple parallel lines arranged horizontally across the endothelium. To our knowledge, this case represents the 10th in the published English literature. There is no association with the direction of aqueous flow, corneal innervation, or other intraocular structures, and this patient had no identified history of mechanical trauma or pressure to the eye. As with linear endotheliitis, viral replication (particularly HSV) within endothelial cells has been implicated on the basis of positive blood serology in MPLE.[9] On the contrary, Le Piane et al. observed an absence of HSV-related confocal microscopy findings in a series of MPLE cases.[11] In another series,[10] a recurrent MPLE affecting both eyes was documented.

The current patient has a history of assumed recurrent oral HSV with an eruption 3 days before onset of visual symptoms. An aqueous humour PCR assay for HSV was negative. However, this assay is not validated for use on aqueous humour samples. Although high sensitivities are reported using this method in facial and genital specimens, a viral load of 200 copies/mL is required for positivity.[12] Lower level HSV activity as might be present within the anterior chamber may be undetected. Serological testing for HSV is of limited value in the setting of active facial

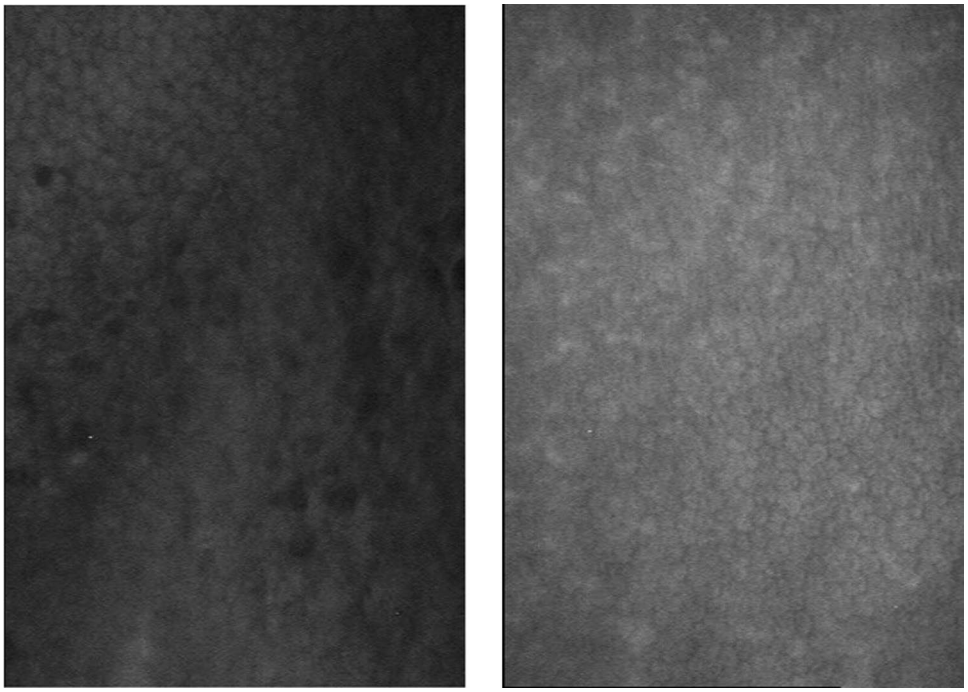

Fig. 2—Confocal microscopy images of corneal endothelium from the right eye (A) and the left eye (B) of the described patient. The parallel lines seen by slit-lamp biomicroscopy correspond to areas of pleomorphism, spot-like holes, and polygonal deposits arranged in linear configurations, shown in (A). The total endothelial cell count in the right eye was 2545/mm^2, and 2941/mm^2 in the left eye. A regular pattern of polygonal cells was seen on the left side (B).

vesicles as this would mask evidence of intraocular viral activation. Recurrences of MPLE in this patient will be scrutinized for association with other ocular and nonocular HSV activity. PCR testing for VZV and CMV was also negative.

Despite the temporal relationship between oral lesions and the ocular findings, HSV was not clearly causative in this patient, and a conservative approach with frequent reassessment was employed. Of note, she had a long-standing history of oral vesicular eruptions with no history of prior ocular symptoms. Spontaneous improvement in the absence of treatment was observed within days. In cases of high clinical suspicion, however, empiric treatment with systemic antiviral therapy may be warranted as treatment is well tolerated and may attenuate the course of this process.

Partial resolution of findings was observed before initiation of treatment. Initiation of topical steroid therapy caused no striking change in the clinical course. Given that complete resolution of MPLE has been observed in the absence of therapy,[11] the role played by topical steroids in this case may be limited.

MPLE is a rare form of endotheliitis resulting in transient, usually unilateral, painless decrease in vision with characteristic findings on examination. The differential diagnosis includes other patterns of endotheliitis, posterior polymorphous corneal dystrophy, mechanical trauma, and rupture of Descemet's membrane. Several questions remain regarding the cause and pathophysiology of MPLE, particularly with respect to the role of HSV. Complete resolution of manifestations has been well documented, even in the absence of therapy.

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Claire Hamilton, [*] **Christopher D. Seamone,** [*] **Mahshad Darvish,** [†] **R. Rishi Gupta**[*]

[*]Dalhousie University, Halifax, Nova Scotia; [†]McGill University, Montréal.

Correspondence to:
Claire Hamilton, MD.; hamiltonc@cdha.nshealth.ca

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Lacrimal abscess mimicking a choroidal mass: an ultrawide field evaluation

A 13-year-old boy presented to us with mild pain, mild mucopurulent discharge, redness, upper eyelid swelling with an S-shaped upper lid, and dimness of vision in the right eye (RE) for 25 days. There was a history of fever at the onset of symptoms though the patient was afebrile at presentation. There was no history of trauma, spectacle use, or any systemic illness. Redness and subtle bulging were seen in the superotemporal forniceal conjunctiva in the RE with local tenderness. No limitation of ocular movement or proptosis was noted in either eye, though there was pain in the RE during dextroversion and dextroelevation. The RE had uncorrected visual acuity (UCVA) of 6/36 and best corrected visual acuity was 6/12 (+2.00 +1.00 × 90). The left eye (LE) had UCVA of 6/6 with normal fundus. There was no relative afferent papillary defect in the RE. No preauricular or submandibular lymphadenopathy was detected. There were no cells in the anterior chamber (AC) and vitreous. Intraocular pressure was 14 (RE) and 16 (LE) mm Hg. Fundus examination of the RE revealed an elevated choroidal mound (approx. 6 disc diameter) with a smooth surface and a well-defined nasal margin, located 4 disc diameters temporal to the fovea (Fig. 1a). Ultrawide field fundus fluorescein angiogram (UWFA) revealed mild crowding and tortuosity of retinal vessels overlying the mound. There was no leakage or abnormal fluorescence at the lesion (Fig. 1b). The ultrasonography of the RE showed an oval lesion in choroid with heterogeneous echotexture. The lesion showed areas of moderate reflectivity with pockets of reduced echo intensity. Posterior to the lesion there was an echolucent zone suggestive of fluid in the subtenon space (Fig. 1c). Our differential diagnoses included nodular posterior scleritis and an orbital mass (inflammatory or infective involvement of the superotemporal orbit or lacrimal gland region) indenting the ocular coats. The patient was advised hematological investigations (complete hemogram with ESR, rheumatoid factor, ANA, P-ANCA, C-ANCA), chest x-ray, Mantoux, contrast-enhanced computed tomography (CECT) orbit, and a conjunctival swab culture. The patient was started empirically on oral antibiotics (co-amoxiclav 375 mg with metronidazole 250 mg TDS). The patient showed a dramatic improvement of symptoms within 1 week. CECT orbit showed a bulky right lacrimal gland (21 mm × 8.5 mm) mildly indenting the globe with a nonenhancing hypodense area within it, suggestive of acute dacryoadenitis with lacrimal abscess formation (Fig. 1d). The CECT also showed an irregular thickening of the adjacent scleral envelope that may suggest scleritis. The choroidal thickening in our case might also be associated with this scleral thickening rather than simple indentation of the globe by the lacrimal gland mass. Subtenon fluid in the patient also may point towards a secondary inflammation of the sclera because of the lacrimal gland abscess.

There was no evidence of rhinosinusitis or bony erosion. Conjunctival swab revealed *Staphylococcus epidermidis*, which was sensitive to amoxicillin, ciprofloxacin, and chloramphenicol. Hemogram was within normal limits with the exception of a raised erythrocyte sedimentation rate of 45 mm in the first hour. Chest X-ray and peripheral blood smears were unremarkable. As the patient had started responding well to the empirical therapy, we did not go ahead with drainage/aspiration of the abscess. Oral antibiotics were continued for the next week, leading to resolution of the lacrimal abscess and the choroidal elevation. At 2 months' follow-up, the vision in the RE was 6/6 without glasses and the choroidal indentation disappeared with minimal pigmentary changes.

Acute suppurative bacterial dacryoadenitis (ASBD) is a rare condition with only 44 culture-positive cases reported to date.[1,2] Causative bacteria include *Staphylococcus aureus* (including methicillin-resistant strain, MRSA), *Streptococcus pyogenes, Haemophilus influenzae, Pneumococcus, Pseudomonus, Diphtheroids, Micrococcus*, and *Klebsiella pneumoniae*.[1,2] Here the authors describe a patient with presumed ASBD masquerading as a choroidal mass. Ultrawide field imaging (UWFI) by Optos™ (Optos Inc, Marlborough, Mass.) provides a panoramic 200° image of the fundus. Fundus imaging and ultrawide field fluorescein angiography (UWFA) using Optos helped us rule out a true choroidal mass. ASBD has been reported in all ages (1–72 years), with 53% cases occurring in the pediatric population.[1] Most cases present within 1 week with pain, redness, S-shaped upper lid, tenderness at