the right internal capsule and left frontal premotor area that were believed to be secondary to vasospasm. He was started on a 2-week prophylactic dose of intravenous vancomycin and meropenem. The psychiatric team evaluated him for psychosis and seizures and started him on valproate and olanzapine. The patient remained intubated in the intensive care unit for 1 month, after which he was transferred to the neurological ward for continuation of care. During a lengthy 6-month admission, the patient showed severe cognitive impairment and was discharged to a supervised nursing home for further treatment and care.

Discussion

Over 50 cases of unilateral or bilateral psychotic oedipism have been reported in the last 50 years.[9–13] Most cases were in association with psychotic episodes, mainly untreated schizophrenia.[14] In this article, we report the first case of cocaine-induced self-enucleation in a healthy male with no known history of psychiatric disease, as well as another case of simultaneous bilateral self-enucleation in a patient with untreated schizophrenia. Self-enucleation is a rare ophthalmic and psychiatric emergency requiring immediate attention and collaboration between the ophthalmologist and the psychiatrist because it results in severe ophthalmic and possibly lethal neurological complications. Patients should be admitted, started on prophylactic antibiotics, and assessed by psychiatry. Continuous nursing supervision is warranted, particularly for patients with unilateral ocular damage. These patients are at a higher risk for further self-mutilation. Long-term psychiatric hospital admission and therapy may be necessary because these patients are at risk of suicide.[15]

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References

1. Large M. Self-enucleation and the real meaning of Oedipus. *Aust N Z J Psychiatry*. 2015;49:846.
2. Sophoecles (trans. Paul Roche). *Oedipus Rex*. New York: Mentor; 1958.
3. Khatib T, Dua A, Singh A, Harminder SD. Oedipus: repenting eyes. *Br J Ophthalomol*. 2011;95:1371.
4. Large MM, Nielssen OB. Self-enucleation: forget Freud and Oedipus, it's all about untreated psychosis. *Br J Ophthalmol*. 2012;96:1056-7.
5. Eshraghy B, Abdi F, Faramarzi N, Esfahani M, Akbari BM. Auto-evisceration in an elderly schizophrenic female. *Int Ophthalmol*. 2013;33:717-20.
6. Schwerkoske JP, Caplan JP, Benford DM. Self-mutilation and biblical delusions: a review. *Psychosomatics*. 2012;53:327-33.
7. Sabahi AR, Amini-Ranjbar Z, Sharifi A, Kheradmand A. Enucleation of eye using finger following cannabis consumption: a case report. *Addict Health*. 2014;6:81-4.
8. Large M, Babidge N, Andrews D, Storey P, Nielssen O. Major self-mutilation in the first episode of psychosis. *Schizophr Bull*. 2009;35:1012-21.
9. Large M, Andrews D, Babidge N, Hume F, Nielssen O. Self-inflicted eye injuries in first-episode and previously treated psychosis. *Aust N Z J Psychiatry*. 2008;42:183-91.
10. Marrag I, Anes I, Hajji K, Essid N, Nasr M. Bilateral concomitant oedipism: two case reports. *Encephale*. 2017;43:195-6.
11. Bababeygy SR, Sadun AA. Bilateral oedipism. *Ophthal Plast Reconstr Surg*. 2013;29:e11-2.
12. Harish T, Chawan N, Rajkumar RP, Chaturvedi SK. Bilateral self-enucleation in acute transient psychotic disorder: the influence of sociocultural factors on psychopathology. *Comprehensive Psychiatry*. 2012;53:576-8.
13. Pérignon S, Cornut PL, Boyer F, Manificat HJ, Burillon C, Denis P. Bilateral oedipism: a case report. *J Fr Ophtalmol*. 2008;31:614-7.
14. Leibovitch I, Pietris G, Casson R, Selva D. Oedipism: bilateral self-enucleation. *Am J Emerg Med*. 2006;24:127-8.
15. Imes RK. Drug induced autoenucleation with resultant chiasmal damage. *Br J Ophthalmol*. 2005;89:783.

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Bilateral intratarsal dystrophic calcification and ossification in localized immunoglobulin light chain amyloid

Amyloidosis is a heterogeneous group of rare disorders wherein insoluble fibrillar proteins are deposited in extracellular spaces within the body. The disorders may be localized, systemic, hereditary, and nonhereditary. Various subtypes of amyloid proteins are involved, and are most accurately characterized by mass spectrometry. We report herein a unique case of unsuspected bilateral localized intratarsal amyloid within specimens derived from blepharoptosis surgery. Bilateral focal dystrophic calcification and bone formation were present within the amyloid deposits, a unique finding in the eyelids.

A healthy 75-year-old male presented with bilateral blepharoptosis (Fig. 1) and a mild "dry eye" condition. Visual acuity

Fig. 1—Preoperative view of blepharoptosis.

was 20/20 OU. Extraocular muscle function was normal. There was Meibomian gland dysfunction of the lower lids with mild corneal punctate staining. A well-healed bilateral LASIK flap from a several-year-old procedure was present. There was bilateral pseudophakia. Findings of each fundus examination, including optic nerves, were normal.

The patient underwent a bilateral Fasanella-Servat tarsoconjunctival resection for correction of blepharoptosis. During surgery, a white, "spongy" material was noted near the upper border of each tarsus, presumably reflecting some sort of scarring. Each tarsus measured 16 mm in height, approximately double the average measurement. Pathologic examination of both tarsoconjunctival fragments showed intratarsal deposits of Congo-red–positive material that exhibited apple-green birefringence under polarized light. Some areas showed a foreign body granulomatous reaction to the amyloid (Fig. 2A). Each tarsus contained focal basophilic calcification of the amyloid deposits, some of which had undergone bone formation (Fig. 2C, D). Some nonossified amyloid also was present in Mueller's muscle. Immunohistochemistry of a scant subconjunctival lymphoplasmacytic infiltrate disclosed polyclonality with respect to lymphocytes (an admixture of CD3-positive T cells and CD20-positive B cells) and plasma cells (an admixture of kappa and lambda light chains). These configurations denoted a garden-variety reactive pattern (chronic conjunctivitis) rather than a localized plasma cell proliferative disorder. Mass spectrometry performed at the Mayo Clinic disclosed the amyloid to be of the AL kappa type. Healing was unremarkable, and postoperative examination with lid-eversion showed no residual white deposits. Subsequent findings of an extensive systemic work-up, including complete blood count, basic metabolic panel, serum electrophoresis, serum calcium, and phosphorous, were all normal without evidence of systemic amyloidosis.

Localized AL amyloidosis represents a rare occurrence within the spectrum of amyloid disorders and is most accurately diagnosed by mass spectrometry protein sequencing. A recent report of 2 cases of localized conjunctival AL light chain amyloid without systemic disease used mass spectrometry analysis at the Mayo Clinic.[1] Our case illustrates the third such case. Because the disorder has been theorized to result from site-specific plasma cell clones producing fragmented monoclonal light chains,[2] we investigated the plasma cells and lymphocytes at the specimen borders and found no evidence of a monoclonal proliferation. A characteristic finding in the current case, unusual in systemic amyloidosis, was the presence of giant cells adjacent to both noncalcified and calcified amyloid deposits. This pattern may reflect a granulomatous foreign body reaction[3] or, in noncalcified amyloid, an immunohistochemical reaction to light chains.[1] Amyloidal calcification, with and without ossification, has been described in various systemic sites, such as lung,[4] subcutaneous tissues,[5] and orbit,[6] but this is—to our knowledge—the only case of such occurrence in the tarsus. The pathogenesis of the

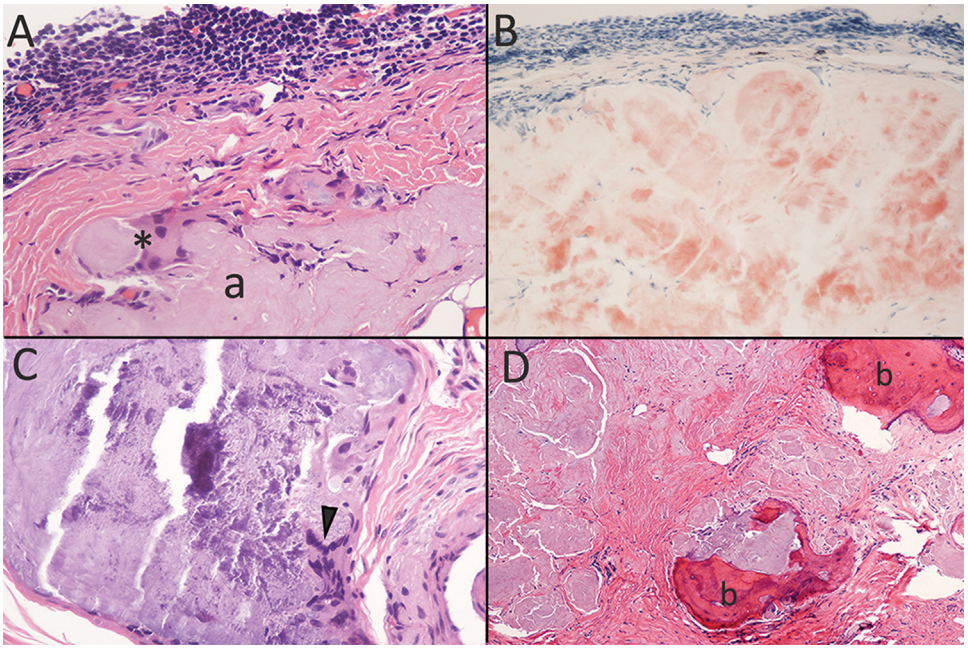

Fig. 2—(A) Tarsoconjunctival fragment shows conjunctivitis and contains amyloid deposition (a) with foreign body reaction (*) (hematoxylin–eosin, ×200). (B) Amyloid highlighted by Congo-red stain (×200). (C) Purplish calcification with granulomatous inflammation (arrowhead) (hematoxylin–eosin, ×200). (D) Bone formation (b) within amyloid (hematoxylin–eosin, ×100).

exceptional dystrophic calcification with bone formation in the current case and its absence in previously reported cases of conjunctival/eyelid amyloid is unclear.

Intratarsal amyloid is much rarer than conjunctival or orbital amyloid and is nearly always unilateral.[7,8] We could find only one report of bilateral tarsal involvement, and it did not show the calcification and ossification found in the current case.[9]

A single report of a unilateral, papillomatous, calcified nonamyloid tarsal mass was termed, oddly, "calcinosis cutis" by its authors[10] and may represent calcification of an underlying neoplasm. Calcification of ocular tissues may occur in hypercalcemic states such as hyperparathyroidism, but these conditions were excluded in our patient. It is probable that the intratarsal deposits of calcified and ossified amyloid—along with nonossified Mueller's muscle deposits—contributed to the blepharoptosis.

This case, like its recently reported analogues,[1] illustrates that AL amyloidosis may be localized and characterized by a benign course. (Additional systemic follow-up is planned.) The case also underscores the importance of submitting tarsoconjunctival tissue for histologic examination rather than discarding the excised material.

Although some institutions use immunohistochemistry for traditional amyloid typing, the results can be inaccurate. Contamination or formalin-induced epitope loss may produce high background staining. Furthermore, c-terminus light chain variability can result in lack of staining.[11,12] We strongly endorse interinstitutional referral for protein sequencing by liquid chromatography and mass spectrometry, which is nearly 100% accurate.

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References

1. Hamill EB, Thyparampil PJ, Yen MT. Localized immunoglobulin light chain amyloid of the conjunctiva confirmed by mass spectrometry without evidence of systemic disease. *Ophthal Plast Reconstr Surg* 2016 [Epub ahead of print].
2. Westermark P. Localized AL amyloidosis: a suicidal neoplasm? *Ups J Med Sci*. 2012;117:244-50.
3. Mukhopadhyay S, Damron TA, Valenti AL. Recurrent amyloidoma of soft tissue with exuberant giant cell reaction *Arch Pathol Lab Med*. 2003;127:1609-11.
4. Xiang H, Wu Z, Wang Z, Yao H. Nodular pulmonary amyloidosis and obvious ossification due to primary pulmonary MALT lymphoma with extensive plasmacytic differentiation: report of a rare case and review of the literature. *Int J Clin Exp Pathol*. 2015;8:7482-7.
5. Bandyopadhyay A, Bhattacharya S, Maiti B, Bose K. Calcified amyloid tumor of neck with exuberant giant cell reaction. *J Lab Physicians*. 2015;7:61-3.
6. Gonçalves ACP, Moritz RB, Monteiro ML. Primary localized amyloidosis presenting as diffuse amorphous calcified mass in both orbits: case report. *Arq Bras Oftalmol*. 2011;74:374-6.
7. Hubbard AD, Brown A, Bonshek RE, et al. Surgical management of primary localised conjunctival amyloidosis causing ptosis. *Br J Ophthalmol*. 1995;79:707.
8. Hill VE, Brownstein S, Jordan DR. Ptosis secondary to amyloidosis of the tarsal conjunctiva and tarsus. *Am J Ophthalmol*. 1997;123:852-4.
9. Kamal S, Goel R, Bodh SA, Madhu S. Primary localized amyloidosis presenting as a tarsal mass: report of two cases. *Middle East Afr J Ophthalmol*. 2012;19:426-8.
10. Jun I, Kim SE, Lee SY, et al. Calcinosis cutis at the tarsus of the upper eyelid. *Korean J Ophthalmol*. 2011;25:440-2.
11. Vrana JA, Gamez JD, Madden BJ, et al. Classification of amyloidosisby laser microdissection and mass spectrometry-based proteomic analysis in clinical biopsy specimens. *Blood*. 2009 4957-9.
12. Leung N, Nasr SH, Sethi S. How I treat amyloidosis: the importance of accurate diagnosis and amyloid typing. *Blood*. 2012 3206-13.

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Bilateral neuroretinitis due to *Bartonella henselae* in a child

A 9-year-old male presented to the emergency department of a pediatric hospital with a 4–5-week history of lethargy, blurred vision, and intermittent temporal headaches. He was taking ibuprofen as needed for the headaches but no other medications. He had no other significant ocular or medical history except for a traumatic distal right radius fracture 3 months before presentation. The patient was afebrile with a blood pressure of 115/74.

Urgent ophthalmology consultation was requested. On examination, visual acuity was 20/60 OD and 20/200 OS. There was a moderate colour vision deficit in the right eye and a severe colour vision deficit in the left eye. On dilated fundus examination, there was 4+ bilateral optic disc edema with hemorrhages, cotton wool spots, and macular exudates (Fig. 1). There was no evidence of anterior uveitis or posterior vitreous cells. Extraocular motility was full with no evidence of a cranial nerve VI palsy in either eye. Goldmann visual field testing revealed bilateral blind spot enlargement.