channels; and (*iii*) direct extension along or in peripheral nerves.[14] We found no evidence of these processes histologically, aided with immunohistochemical markers for vascular and lymphatic vessels. The inability to identify a structural conduit could reflect the relatively sparse amount of tissue that was available to study surrounding the skin and conjunctival tumours.

This case also brings up a relevant issue concerning the risk for regional spread from incisional biopsy of CM. Several studies have addressed this question, but the most definitive answer comes from analysis of the Sunbelt Melanoma Trial. This large, multicentre, randomized, clinical trial involved 2164 patients with CM and found that incomplete excision of primary melanoma (incisional and shave biopsies) did not adversely affect locoregional or distant recurrence as long as appropriate treatment followed diagnostic biopsy.[15]

In summary, we describe a patient with CM of temporal skin that caused an ipsilateral conjunctiva melanoma with unusual compartmentalization of malignant melanocytes within the epithelium and substantia propria. This pattern of tissue involvement is similar to that described for epidermotropic metastatic melanoma of skin. Although definitive evidence of lymphatic, perivascular, or perineural spread was lacking, the proximity of the CM suggests regional rather than blood-borne spread.

Andrew R. Carey, Edgar M. Espana, Curtis E. Margo
Morsani College of Medicine, University of South Florida, Tampa, Fla.

Correspondence to:
Curtis E. Margo, MD: cmargo@health.usf.edu

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Pseudo-Foster Kennedy syndrome due to idiopathic intracranial hypertension

Foster Kennedy syndrome is characterized by optic atrophy in one eye and papilledema in the other caused by a frontal lobe mass ipsilateral to the atrophic optic nerve. It was first described by Foster Kennedy in 1911 in a case series of 6 patients with expanding frontal lobe lesions.[1] Foster Kennedy syndrome is thought to be present in 1% to 2.5% of intracranial masses.[2]

Optic nerve atrophy with contralateral optic disc edema in the absence of an intracranial mass has been coined pseudo-Foster Kennedy syndrome and is seen most often with nonsimultaneous bilateral nonarteritic anterior ischemic optic neuropathy (NAION).[3] Other causes of pseudo-Foster Kennedy syndrome that have been reported include optic neuritis, trauma, and syphilis.[4] Pseudo-Foster Kennedy syndrome due to idiopathic intracranial hypertension (IIH) is very rare and has been reported only once in the literature.[5] Here, we report an additional case of pseudo-Foster Kennedy syndrome due to IIH.

Case Report

A 36-year-old overweight female patient (BMI 26.6 m/kg) was referred to the neuro-ophthalmology clinic by an optometrist who incidentally noticed optic disc edema in the right eye and a pale optic disc in the left eye during a routine eye examination. The patient denied blurry vision, transient visual obscurations, diplopia, headache, and tinnitus. She had no notable medical history and denied

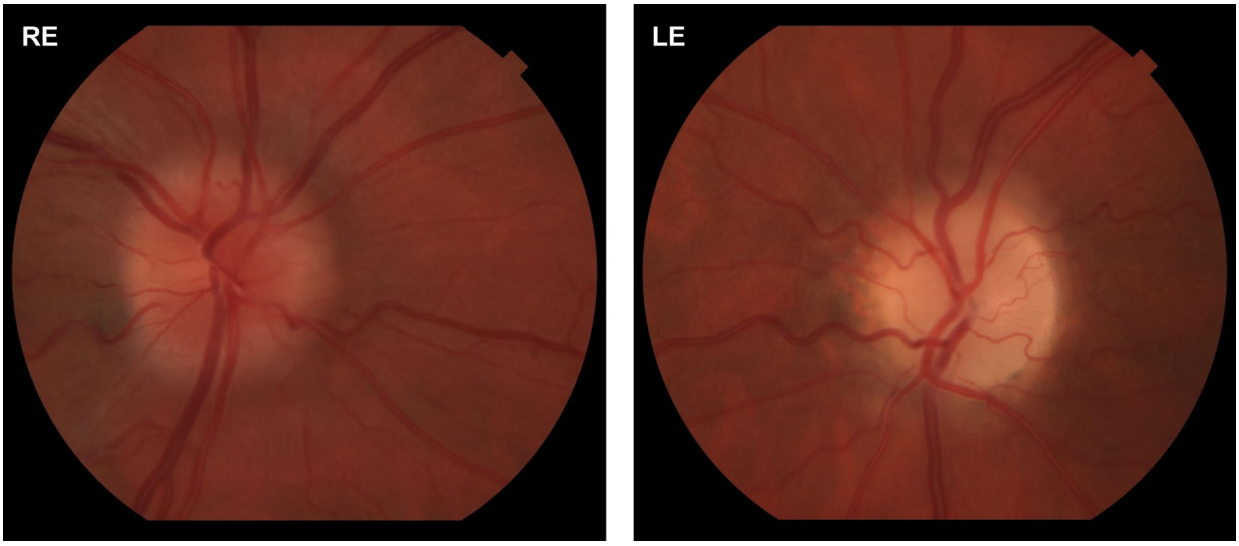

Fig. 1—Optic disc photographs show optic disc edema in the right eye (RE) and optic atrophy in the left eye (LE).

any previous trauma and the use of any medications including oral contraceptives, vitamin A derivatives, or any recent antibiotics.

During our initial assessment, her best corrected visual acuity was 20/20 OD and 20/30 OS. She had a relative afferent pupillary defect in the left eye and intraocular pressures of 10 mm Hg in each eye. She correctly identified 12 of 12 Ishihara colour plates in the right eye and saw the control plate, and 0 of 12 plates in the left eye. Her extraocular movements were full and there was no proptosis. Her anterior segment examination was normal. Dilated fundus examination revealed grade II optic disc edema in her right eye and a pale atrophic left optic disc (Fig. 1). The macula and peripheral retina appeared normal in each eye. Humphrey 24-2 visual field testing revealed a constricted visual field in the left eye with a mean deviation of –27.51 dB and a normal visual field in the right eye with a mean deviation of –1.96 dB (Fig. 2). The neurologic examination was unremarkable. Cardiovascular, respiratory, and head and neck clinical examinations were also within normal limits.

A magnetic resonance (MR) image of the brain revealed tortuosity of the optic nerves and flattening of the posterior globe. There were no space-occupying lesions. An MR angiogram was normal. MR venogram revealed narrowing of bilateral transverse sinuses, but no evidence

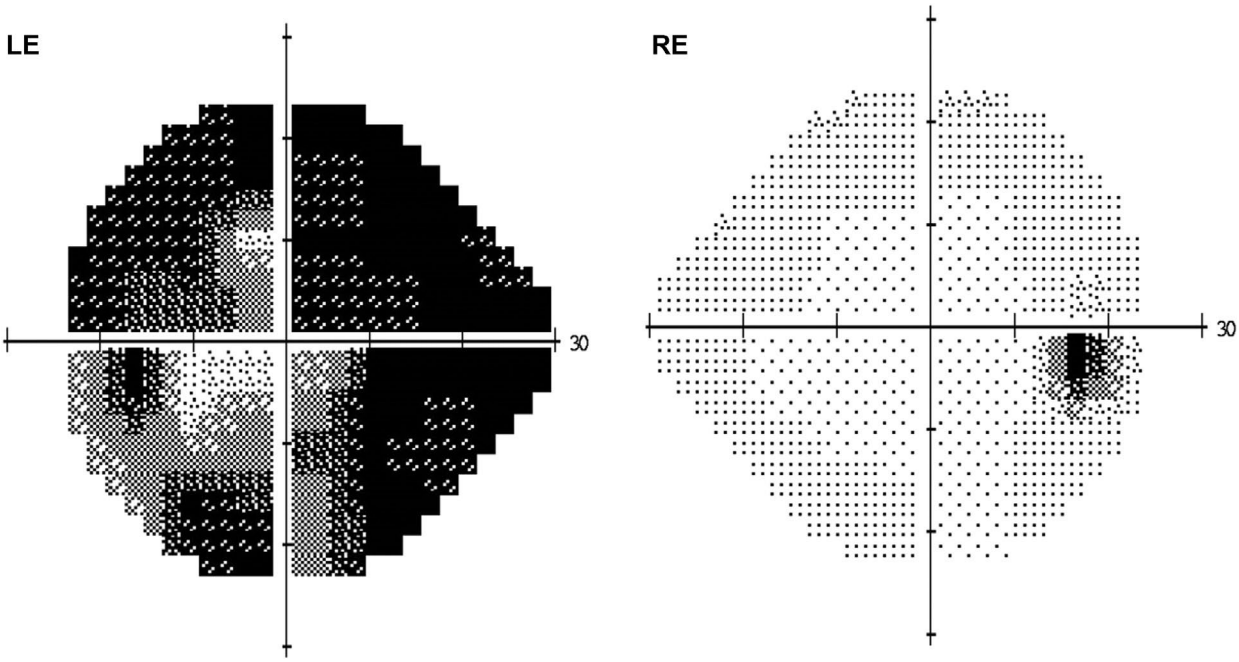

Fig. 2—24-2 Humphrey visual field shows visual field loss in the left eye (LE) and a normal visual field in the right eye (RE).

Table 1—Non IIH causes of pseudo-Foster Kennedy syndrome reported in the literature in chronological order

Authors	Cases (n)	Cause	Journal
Semeraro F et al.[12]	1	Meningioma infiltrating superior sagittal sinus	*Clin Neurol Neurosurg.* 2012;114:272-4
Bansal S et al.[13]	1	Congenital optic nerve hypoplasia	*J Med Case Rep.* 2008;2:86
Sendrowski et al.[14]	1	AION and malignant hypertension	*Clin Refract Optom.* 2005;16:6-14
Tamai H et al.[15]	1	Pachymeningitis, p-ANCA positive	*Am J Ophthalmol.* 2000;130:535-7
Limaye SR et al.[16]	1	AION and nonbasal glioma	*J Clin Neuroophthalmol.* 1990;10:188-92
Lepore FE et al.[17]	1	Nonsimultaneous NAION	*Ann Ophthalmol.* 1985;17:411-2
Schatz NJ et al.[18]	2	Ischemic optic neuritis	*J Neurosurg.* 1967:27:37
	1	Congenital syphilis	*J Neurosurg.* 1967:27:37
	1	Optic neuritis	*J Neurosurg.* 1967:27:37
	2	Traumatic optic neuropathy	*J Neurosurg.* 1967:27:37

AION, anterior ischemic optic neuropathy; ANCA, anti-neutrophil cytoplasmic antibody; NAION, nonarteritic anterior ischemic optic neuropathy.

of venous thrombosis. A lumbar puncture was performed in the left lateral decubitus position with an opening pressure of 28 cm water. Cerebrospinal fluid analysis including cell counts, protein, and glucose were within normal limits.

In addition to recommending weight loss, the patient was started on oral acetazolamide 250 mg bid. She was seen in follow-up 3 times since the time of diagnosis. Although there was no reduction in her weight, she remained asymptomatic and was tolerating the medication well. Her visual acuity during the last visit at 9 months was unchanged in the right eye and had improved to 20/25 OS. The left optic disc remained atrophic and right optic disc edema evolved to grade I papilledema. As she still had papilledema, she was advised to continue acetazolamide and continue weight loss.

Discussion

Before the availability of neuroimaging, Foster Kennedy syndrome was thought to be pathognomonic for a space-occupying lesion in the basofrontal area on the side of optic atrophy, and exploratory craniotomy was performed to locate the mass.[4] Negative exploratory craniotomies subsequently led to description of other noncompressive causes, which are termed pseudo-Foster Kennedy syndrome (Table 1). The largest case series of pseudo-Foster Kennedy syndrome was published by Francois and Neetens in 1955, reporting that the top 3 causes were "arteriosclerosis and hypertensive cardiovascular disease, optochiasmatic arachnoiditis, and internal carotid aneurysms."[6] More recent literature clearly implicates bilateral (nonsimultaneous) NAION as the most common cause, by far, of pseudo-Foster Kennedy syndrome (which is almost certainly what is implied by "atherosclerosis and hypertensive cardiovascular disease" in historical reports).[3]

IIH encompasses patients with isolated increased intracranial pressure that is not related to intracranial compressive lesions, cerebral venous thrombosis, or a meningeal process. Approximately one fourth of patients, such as the one described in this report, are asymptomatic and diagnosed on routine eye examination.[7] Papilledema can result in slowly progressive visual loss, and up to 25% of patients with IIH develop secondary optic atrophy and permanent visual loss.[8] Previous studies have found that black patients, male patients, and those with the greatest degree of obesity are at highest risk for visual loss.[9]

Papilledema is swelling of the optic nerve head caused by increased intracranial pressure and is usually bilateral. However, asymmetric papilledema is not uncommon in IIH, and it is thought to be related to differences in impedance to cerebrospinal fluid flow from trabeculations within the subarachnoid space of the optic nerve sheath.[10] Unlike asymmetric papilledema in IIH (which has been described in the literature),[11] this patient had papilledema in one eye and optic atrophy in the other: pseudo-Foster Kennedy syndrome. In our case, it may be that the atrophic optic nerve had the most severe optic disc edema initially, leading to optic atrophy, with relative preservation of less affected optic nerve, which continues to manifest optic disc swelling.

Evolution of optic atrophy in papilledema depends on the severity and persistence of the elevated intracranial pressure. Postpapilledema optic atrophy can happen within weeks or days, even before the phase of chronic papilledema, if the intracranial pressure rises rapidly and remains constantly high. In other cases with chronic papilledema, the optic disc swelling can gradually melt away into optic atrophy over a period of months to years. After the development of optic atrophy, papilledema does not occur without the presence of sufficient viable optic nerve fibres. Another potential cause of optic atrophy in this setting is a superimposed NAION or some other cause of optic atrophy before the development of IIH. These alternative explanations cannot be excluded particularly because the patient was asymptomatic and she was found to have to this condition during a routine eye examination.

Jonathan A. Micieli, Murad Al-Obthani, Arun N.E. Sundaram
Department of Ophthalmology and Vision Sciences, Sunnybrook Health Sciences Centre, University of Toronto, Toronto, Ontario, Canada

Correspondence to:
Jonathan A. Micieli, MD: jonathan.micieli@utoronto.ca

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Sympathetic ophthalmia after diode laser cyclophotocoagulation: now an issue in informed consent

A 40-year-old male with a history of retinopathy of prematurity presented with chronic left eye pain. No surgery had been performed on either eye, nor had there been any previous episodes of uveitis. Best corrected Snellen visual acuity was 3/36 OD and no perception of light OS; intraocular pressure (IOP) was 17 mm Hg OD and 57 mm Hg OD. The left eye showed iris neovascularization, a dense cataract, and no view of the posterior segment. Ultrasound revealed a long-standing left total retinal detachment. The right eye had clear optical media and a dragged disc.

The patient underwent left diode laser transscleral cyclophotocoagulation (TSCPC). This treatment proved insufficient because of persistent IOP elevation and ongoing pain, and he required 2 further treatments, each separated by 1 month, to gain IOP control (i.e., 3 treatments in 2 months). Each of the 3 treatments involved 30 burns distributed over 3 quadrants; no quadrant was ultimately spared treatment. Settings were 2000 mW × 2000 msec and there was a total of 10 pops over the 3 episodes. Total energy delivered was thus 360 J. One month after the third and final TSCPC, the IOP was 10 mm Hg OS and the eye felt more comfortable.

After a further 2 months, there was recurrence of pain in the left eye, without IOP elevation, and concurrent painless reduction of vision in the right eye (the untreated, better seeing eye) to count fingers at 1 m. Bilateral granulomatous anterior uveitis was diagnosed and treated. The patient requested enucleation of the left eye because of ongoing pain, and this was performed approximately 4 weeks later.

Histopathology showed a small globe (axial length 18 mm). Typical changes of cyclodiode therapy were noted in the pars plicata, which also showed granulomatous inflammation.[1] Findings characteristic of sympathetic ophthalmia (SO) were observed, with prominent nonnecrotizing granulomatous inflammation in the choroid and ciliary body, focally involving the subretinal space and retinal pigment epithelial layer (Fig. 1).

An inferior serous retinal detachment was noted in the right eye, and the patient was admitted for pulse intravenous methylprednisolone treatment, with a subsequent weaning dose of oral prednisolone and commencement of oral cyclosporine (200 mg twice daily). The right visual acuity improved to its pre-SO level.

SO is a rare complication of diode laser TSCPC and tends not to be recorded in treatment series but as sporadic case reports. Three cases of diode laser TSCPC-induced SO, involving eyes that had not undergone prior surgery or trauma, have been documented in the literature.[2–4] A fourth case was reported, although 2 trabeculectomies, a vitrectomy, retinal cryotherapy, and buckle had previously been performed on this eye.[4] A fifth case in a 2-year-old with Coats' disease has been documented in a poster presentation.[5] The case reported here had onset of symptoms in the sympathizing eye 4 months after the first laser treatment, which was an interval similar to other reported cases,[2–4] and used representative laser settings.[6] An estimate of the incidence of SO after diode laser TSCPC has recently been calculated based on an extensive literature review.[7] The numerator used in the calculation was the number of published cases of the complication (4), whereas the denominator was the total number of eyes that had undergone diode laser TSCPC (5979) in peer-reviewed studies. The computed incidence rate of 0.07% (95% CI 0.03%–0.17%)[7] was admitted by those authors to be likely