ophthalmology with symptoms and signs of direct compression (Horner syndrome) or indirect distal thromboembolic/hypoperfusion events (transient ischemic attacks/TMVL). Although the combination of Horner syndrome and TMVL is a well-known constellation for ipsilateral ICAD, the combination of TMVL and dysgeusia is also a highly suggestive symptom complex for ICAD of the petrous ICA even without the Horner syndrome.

Supplementary Materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.jcjo.2020.05.004.

Preston Baker,* Shruthi Harish Bindiganavile,† Ross Taylor,† Nita Bhat,† Andrew G. Lee†
*McGovern Medical School, Houston, TX; †Houston Methodist Hospital, Houston, TX.

Originally received Apr. 5, 2020. Final revision Apr. 23, 2020. Accepted May. 17, 2020.

Correspondence to:
Andrew G. Lee, MD; aglee@houstonmethodist.org.

References

1. Debette S. Pathophysiology and risk factors of cervical artery dissection: what have we learnt from large hospital-based cohorts? Curr Opin Neurol 2014;27:20–8.
2. Downer J, Nadarajah M, Briggs E, Wrigley P, McAuliffe W. The location of origin of spontaneous extracranial internal carotid artery dissection is adjacent to the skull base. J Med Imaging Radiat Oncol 2014;58:408–14.
3. Sturzenegger M, Huber P. Cranial nerve palsies in spontaneous carotid artery dissection. J Neurol Neurosurg Psychiatry 1993;56:1191–9.
4. Biousse V, Touboul P-J, D'Anglejan-Chatillon J, Lévy C, Schaison M, Bousser M-G. Ophthalmologic manifestions of internal carotid artery dissection. Am J Ophthalmol 1998;126:565–77.
5. Givre S., Van Stavern G.P.Amaurosis fugax (transient monocular or binocular visual loss). www.uptodate.com/contents/amaurosis-fugax-transient-monocular-or-binocular-visual-loss. Accessed
6. Debette S, Grond-Ginsbach C, Bodenant M, et al. Differential features of carotid and vertebral artery dissections: the CADISP Study. Neurology 2011;77:1174–81.

Footnotes and Disclosure

The authors have no proprietary or commercial interest in any materials discussed in this article.

Severe optic neuropathy as the presenting sign of leptomeningeal carcinomatosis from pancreatic cancer

Pancreatic cancer is one of the deadliest cancers in North America with an overall 5-year survival rate of less than 10%.[1] The majority of pancreatic solid tumours metastasize to other organs, mainly to the liver and other peritoneal organs.[1] Leptomeningeal metastasis (LM) or carcinomatous meningitis is infiltration of cancer cells to the leptomeninges and cerebrospinal fluid and is extremely rare in the setting of pancreatic adenocarcinoma with only 19 English cases reported (Supplementary Table 1, available online). There are no previous reports of an optic neuropathy from metastatic pancreatic cancer in the context of LM. Here, we report a case of pancreatic adenocarcinoma leading to LM and a severe optic neuropathy.

A 69-year-old woman with a history of pancreatic cancer presented with progressive, painless vision loss in her left eye. She was diagnosed with T2N1 well-differentiated pancreatic adenocarcinoma 9 years ago, for which she underwent a Whipple procedure followed by adjuvant gemcitabine for 5 months. Her first recurrence at 3.5 years from surgery consisted of low-volume lung nodules that were observed and, when proven indolent, resected and confirmed to be the same adenocarcinoma as the pancreas. Two years later she had radiological evidence of peritoneal metastatic disease and more lung metastasis. She commenced on standard-of-care first-line FOLFIRINOX chemotherapy. Over the next 4 years, she was treated with multiple FOLFIRINOX cycles, demonstrating radiological and biochemical partial responses followed by surveillance periods off chemotherapy and then rechallenge of FOLFIRINOX with progression. She had a repeat lung resection for dominant disease. More recently, she developed radiological signs of bone metastasis, enlarged mediastinal lymph nodes, and bilateral lung nodules consistent with progressive disease.

She developed blurry vision in her left eye 10 days before presentation, and the blurry vision gradually worsened. She had a visual acuity of 20/20 in the right eye and no light perception in the left eye. There was a left relative afferent pupillary defect, and dilated fundus examination was normal in both eyes. Humphrey 24-2 SITA-Fast visual field testing was normal in the right eye. Due to the concern for metastatic disease to the left retrobulbar optic nerve, she underwent urgent magnetic resonance imaging of the brain and orbits with contrast, which showed enhancement of both optic nerves and leptomeningeal enhancement of the corpus callosum, cingulate gyrus, and medial aspect of the temporal lobe (Fig. 1). This was consistent with metastatic disease. A

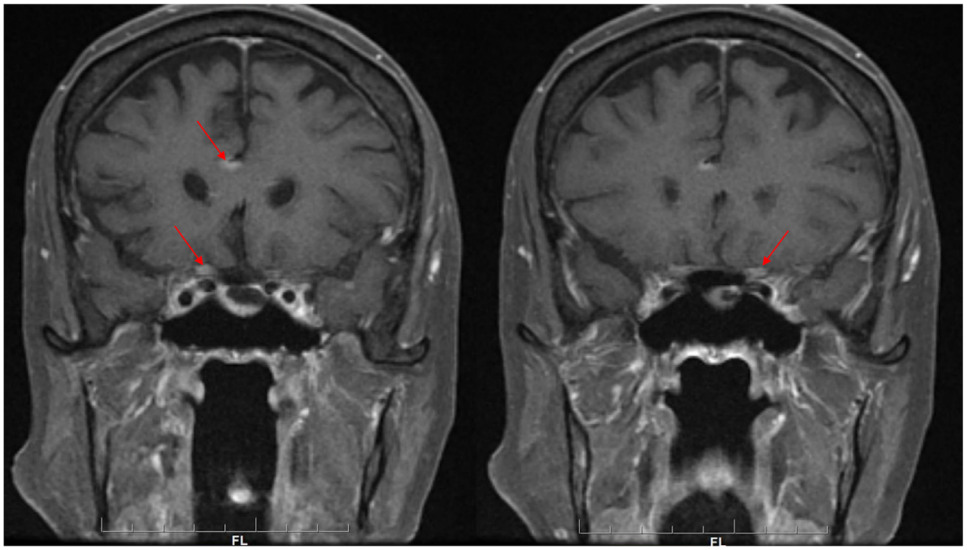

Fig. 1—Magnetic resonance imaging of the brain and orbits with contrast after vision loss demonstrating areas of leptomeningeal enhancement (red arrows) including both optic nerves.

lumbar puncture was performed with cytology, which confirmed the presence of adenocarcinoma.

She underwent whole-brain radiotherapy (20 Gy in 5 fractions) and concomitant treatment with dexamethasone. She subsequently developed hyponatremia, loss of rectal tone, and bladder retention owing to widespread leptomeningeal deposits in the spine. She underwent urgent radiotherapy to this area and was assessed by the palliative care team. The patient deteriorated further and died 3 months after developing vision loss. She had no light perception vision in her left eye at her final 1-month follow-up and maintained good visual function in her right eye until death.

Overall, LM develops in 5%–8% of metastatic cancer cases.[1] However, the rate of LM from pancreatic cancer is very low, with only 19 English cases reported (Supplementary Table 1). Here we present the first case of optic nerve dysfunction from LM in a woman with a 9-year history of pancreatic cancer. We confirmed a diagnosis of LM infiltrating the left optic nerve by both magnetic resonance imaging and cerebrospinal fluid cytology.

Grira et al. reported the only case of LM from pancreatic cancer with vision manifestations.[2] The report mainly focused on the neurological symptoms of the patient and the rare occurrence of LM carcinomatosis as the presenting sign of pancreatic cancer in general. There was only a brief mention of decreased visual acuity and papilledema, but no formal description of visual symptoms or ophthalmological assessment. Like most other reports, this patient had a poor outcome and died just 7 weeks after presentation. The remaining 18 cases of LM originating from pancreatic tumours did not have ophthalmological manifestations (Supplementary Table 1).

In contrast to the infrequency of reported cases of metastasis to the optic nerve, there have been more reports of vision loss owing to pancreatic cancer metastasis to the choroid and other structures of the eye.[3] In most of these cases, numerous yellow-tinged nodules are seen on the retina in fundoscopy examination.[3–5] Two such cases also had optic nerve involvement, but no investigations regarding a possible diagnosis of LM were conducted.[4,5] The choroidal lesions were treated with photocoagulation in one study,[4] and only palliative care was offered in the other report.[5] Overall, when approaching a patient with a history of pancreatic cancer presenting with vision loss, it is important to consider LM in the differential diagnosis and conduct the necessary tests if suspected. This will ensure that treatment with radiation, chemotherapy, or corticosteroids can be considered to help maintain quality of life in these patients and allow for palliative care services to be offered given the poor prognosis.

We believe that the unique presentation of this case is attributed to the unusually long survival of the patient after the diagnosis of metastatic pancreatic cancer. Her indolent disease course and metastatic distribution was atypical, despite typical pathology. She was certainly an exceptional responder to standard chemotherapy, where the typical control period is less than 1 year. On tumour sequencing, her genomic profile described a classical pancreas adenocarcinoma with no exceptional features to explain responsiveness or atypical progression patterns. Living for 9 years with pancreatic cancer certainly increased the chances of metastasis to spread to atypical parts of the body, including the central nervous system and, in this case, the optic nerve. With more effective systemic therapies that prolong the natural history, these rare cases may become a more common presentation.

Overall, we report the case of a 69-year-old woman with a history of pancreatic adenocarcinoma metastasising to the leptomeninges, presenting with complete vision loss of the left eye. This is the first case of an optic neuropathy in this setting.

Supplementary Materials

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.jcjo.2020.04.023.

Armin Farahvash, Jennifer J. Knox, Jonathan A. Micieli
University of Toronto, Toronto, Ont.

Correspondence to: jonathanmicieli@gmail.com.

References

1. Hidalgo M. Pancreatic cancer. N Engl J Med 2010;362:1605–17.
2. Grira MT, Ben Jemaa HM, Lammouchi TM, Benammou SA. Meningitis revealing pancreatic carcinoma. Neurosciences (Riyadh) 2007;12:256–8.
3. Shields CL, Welch RJ, Malik K, et al. Uveal metastasis: clinical features and survival outcome of 2214 tumors in 1111 patients based on primary tumor origin. Middle East Afr J Ophthalmol 2018;25:81–90.
4. Ring HG. Pancreatic carcinoma with metastasis to the optic nerve. Arch Ophthalmol 1967;77:798–800.
5. Lin CJ, Yang CM, Chen MS. Intraocular metastasis of pancreatic cancer: report of two cases. Retina 2001;21:666–9.

Footnotes and Disclosure

The authors have no proprietary or commercial interest in any materials discussed in this article.

Exercise in isolating during novel coronavirus 19: a case report of bilateral ocular trauma from elastic resistant bands

The novel coronavirus disease 2019 (COVID-19) has become a worldwide pandemic.[1] Social distancing measures implemented to prevent the spread of disease have included the closure of gymnasiums worldwide. As a result, the public has turned to home exercise equipment. We have found a corresponding increase in patients attending a central London ophthalmic accident and emergency (A&E) with bilateral ocular trauma from elastic resistance bands. In this case report we describe 2 cases presenting within 2 weeks during the U.K. lockdown.

A 41-year-old woman presented to the A&E after a resistance band had slipped and hit both eyes. She had no ophthalmic or medical history. Her unaided Snellen visual acuity was 6/12 in both eyes; this improved to 6/9 with pinhole in both eyes. Her intraocular pressures were within normal limits at 10 mm Hg bilaterally. On examination of the anterior segment, the right eye had a 0.8 mm hyphema. The left eye had a 1 mm hyphema with 2 clock hours of iridodialysis from 4 to 6 o'clock. Dilated fundoscopy of both eyes revealed peripheral commotio retinae with no retinal tears found on indented examination. She was treated with bed rest and a tapering course of G. dexamethasone 0.1% beginning at 6 times a day and G. cyclopentolate 1% 3 times a day. She will require review within 2 weeks to monitor her progress and annual lifelong monitoring of the left eye for the development of angle recession glaucoma.

A 19-year-old male former personal trainer sustained a resistance band injury across both eyes. His unaided visual acuity on presentation was 6/6 on the right and 2/60 on the left with no improvement with pinhole. His intraocular pressure was 15 and 24 mm Hg in the right and left eye, respectively. Examination of the anterior segment of the right eye showed red blood cell deposits on the inferior corneal endothelium, and the left eye had a 1 mm hyphema, which had settled nasally owingto the patient laying on his right-hand side (Fig. 1). Dilated fundoscopy showed inferior peripheral commotio retinae on the right eye. The left eye had a vitreous hemorrhage with peripheral commotio retinae and intraretinal hemorrhages and an inferior retinal tear (Fig. 2). The patient was treated with bed rest and a tapering course of G. dexamethasone 0.1% starting 6 times a day and G. cyclopentolate 1% 3 times a day. He was referred to the on-call vitreoretinal team for laser retinopexy of the retinal tear. Forty-eight hours later, he reported that the vision in the left eye had decreased and he had developed an aching pain over the left eye. The visual acuity was recorded as 6/4.5 on the right and hand movements on the left. The intraocular pressure was 12 mm Hg on the right but had increased to 50 mm Hg on the left. This was treated with 500 mg oral acetazolamide, G. dorzolamide 20 mg/mL + timolol 5 mg/mL, and apraclonidine 1%, to which his pressure responded well. Spectral-domain optical coherence tomography of the macular showed a traumatic lamellar macular hole on the left with foveal disruption of the outer segment (Fig. 3). He remains under weekly review for monitoring of the intraocular pressure, traumatic uveitis, and retinal tear.

This case report describes the complex bilateral ocular injuries from elastic home exercise bands, requiring both short- and long-term management. There are sparse case reports of ocular injuries from elastic exercise bands causing unilateral retinal detachment,[2] hyphema,[3] and lens dislocation.[4] Our case report is the first where both cases have bilateral injury requiring treatment. The bilateral involvement is explained through the mechanism of injury where the exercise band slips, often from under the foot, and strikes both eyes. Both cases had asymmetrical injuries, with the left eye being more affected than the right. This highlights the need for clinicians to ensure that both eyes are adequately assessed because the apparently unaffected eye may still have injuries. This case report is also unusual in that all cases presented within quick succession due to increased home exercise during COVID-19 isolation. We wish to raise public awareness of the potential ocular risks arising from home exercise and the need for adequate eye protection.