examination showed mild venous dilation and normal optic nerves (Fig. 1). Five months later, there was bilateral optic atrophy temporally (Fig. 2). The cup-to-disc ratio was 0.1 OU.

Humphrey visual field testing showed a dense inferior altitudinal field defect and a central scotoma OD (Fig. 3) and a central scotoma with nasal step defect superimposed on a central scotoma OS. Optical coherence tomography performed 4 months after acute vision loss confirmed retinal nerve fiber layer loss consistent with optic atrophy OU (Fig. 4).

Testing for anti-aquaporin 4 neuromyelitis optica antibodies, rapid plasma reagin, fluorescent treponemal antibody, vitamin B12, folate, quantiferon, and paraneoplastic panel including antibodies against collapsin response–mediated protein 5 were all negative.

Several cases of NA-AION have been reported in users of PDE5i, which occur typically between 30 minutes to 36 hours after ingestion of the sildenafil.[3] Sildenafil, vardenafil, and avanafil have half-lives of approximately 4–5 hours, compared with tadalafil, with a half-life of 17.5 hours. A recent multicenter observational case–crossover design investigated the daily relative risk for acute NA-AION on days within 5 half-lives of PDE5i use versus other days by estimating an odds ratio (OR) from conditional logistic regression. The potential cases of NA-AION were classified as definite, possible, or not NA-AION. Among 43 definite NA-AION cases with PDE5i exposure in the prior 30 days, the OR was 2.15 (95% confidence interval [CI]: 1.06–4.34), implying that there is a 2-fold increased risk of acute NA-AION within 5 half-lives of PDE5i use compared with use in a prior time period.[5]

We believe, based on exclusion of other etiologies for her retrobulbar optic neuropathy, that our patient suffered simultaneous bilateral PION.

Su et al.[4] published a case of bilateral sequential presumed PION within 36 hours after a sildenafil overdose with 3 pills at once in a patient with NA-AION risk factors, including systemic hypertension, hyperlipidemia, and stroke. Initially, the patient had no light perception OU, but after 6 weeks, the visual acuity improved to count fingers OD and hand movements OS with both optic discs developing pallor. In our patient, after PDE5i was discontinued, the vision remained stable over the next 5 months.

We believe that the bilateral, acute, simultaneous retrobulbar optic neuropathy with subsequent optic atrophy OU in our patient was attributable to PION, and we hypothesize a possible relationship to sildenafil use. Further study will be necessary to determine whether the OR seen for NA-AION and PDE5i use holds true for PION as well.

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Sebaceous gland carcinoma of tarsus can be misdiagnosed as intratarsal keratinous cyst

Chalazia, sebaceous gland carcinomas (SGCs), and intratarsal keratinous cysts (IKCs) are the 3 main mass lesions of the tarsus that have received clinical attention. SGC is an aggressive, malignant neoplasm that is especially notorious for masquerading as other benign and less malignant lesions, such as chalazion, chronic keratoconjunctivitis, basal cell carcinoma, or squamous cell carcinoma.[1]

IKC is a relatively new, benign entity first described by Jakobiec et al., who reported 6 cases in 2010.[2] Clinical presentations of usual IKC include a yellowish-white,

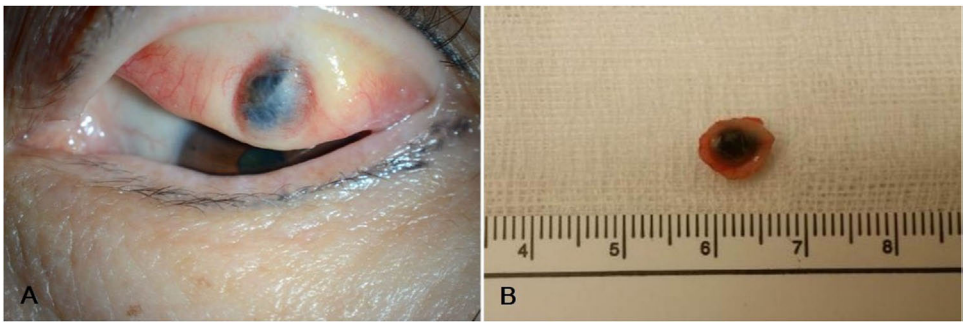

Fig. 1—Photographs showing clinical features of the tarsal mass in this case. (A) A blue-greyish firm nodule elevated on the middle portion of the left upper tarsus. (B) A 6-mm-sized mass, with a well-developed wall, which was excised by partial full-thickness tarsectomy.

slow-growing, well-circumscribed, and round mass that usually fixed to upper tarsus, although rare bluish or translucent lesions have been reported.[2] Herein we introduce the first case of tarsal mass that was clinically diagnosed as IKC, but finally revealed as SGC.

A 55-year-old Korean woman presented with a 4-year history of a palpable nodule in her left upper eyelid. Her medical and ocular history were otherwise unremarkable. The nodule demonstrated slow growth over the preceding few months. Incision and curettage was repeated 3 times over the preceding 4 years, and the lesion recurred after incision and curettage.

Physical examination showed a nontender, nonerythematous, and blue-greyish firm nodule that was fixed to the underlying tarsus with mobile overlying skin (Fig. 1A).

As the mass was originated from tarsus and shared many morphologic characteristics with IKC, we provisionally diagnosed the nodule as IKC. Under local anaesthesia, the patient underwent partial full-thickness tarsectomy on her left upper eyelid. The lesion was about 6 mm and embedded in tarsus with a well-developed wall, representing defined circular demarcation (Fig. 1B). However, the pathologic examination demonstrated atypical dark, pleomorphic sebaceous cells with foamy, vacuolated cytoplasm

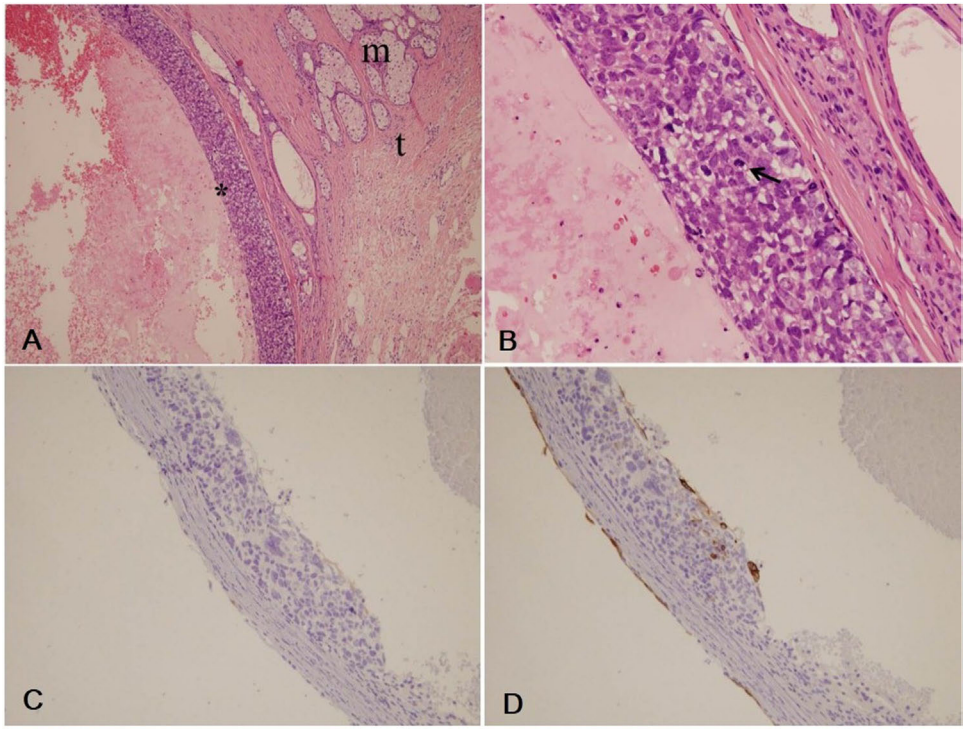

Fig. 2—Photographs showing pathological features and immunohistochemical stainings in this case. (A) Pathologic examination revealed sebaceous cell carcinoma with atypical dark, pleomorphic sebaceous cells contrast to the normal foamy sebaceous glands. t, tarsus; m, meibomian gland; asterisk, tumour with cystic change (hematoxylin and eosin, ×100). (B) Tumour cells with foamy, vacuolated cytoplasm and mitotic figures (arrow) (hematoxylin and eosin, ×400). (C) Negative for carcinoembryonic antigen. (D) Positive for epithelial membrane antigen (×200).

and mitotic figures (Fig. 2A, B). Immunohistochemical stainings revealed positive carcinoembryonic antigen and negative epithelial membrane antigen stain (Fig. 2C, D). The findings were consistent with SGC containing cystic changes. Additional wide surgical excision was performed on her left upper eyelid.

Clinical differentiation of chalazion, SGC, and IKC is often challenging because they all arise from tarsus and can be similar in gross morphology. Histopathologically, however, each shows distinct features. Chalazion manifests as a chronic lipogranulomatous inflammation with a mixture of inflammatory cells, including neutrophils, lymphocytes, plasma cells, and eosinophils. IKC is lined with stratified squamous epithelium, abrupt keratinization (no granular layer), and an eosinophilic cuticle. The lumen contains abundant keratinous debris and is surrounded by thick fibrous wall. No adnexae, such as sebaceous lobules or follicles, are attached to the cyst.[3,4] By immunohistochemical staining, the inner lining of IKC reveals distinct expressions of carcinoembryonic antigen and epithelial membrane antigen such as cytokeratin 17. SGC is composed of irregular lobular masses with signs of invasion. The cytoplasm is pale, foamy, and vacuolated. This feature of foamy cytoplasm is seen only in SGC.[5] SGC can be composed of cells with varying degrees of sebaceous differentiation, and so it can exhibit various clinical and histopathological properties.

The treatment and prognosis of each entity are also different from each other. Chalazion may resolve with conservative managements like warm compression and lid hygiene. In select cases, incision and curettage may be beneficial. In case of IKC, incision and curettage is not an effective long-term treatment. Total removal of the lesion, including full-thickness excision of tarsus at the cyst's base, is suggested for definitive treatment. On the other hand, SGC requires a much more aggressive approach, such as surgical excision with wide margins or orbital exenteration.

As mentioned earlier, SGC is a great mimicker. This masquerading feature often leads to delayed management with increased morbidity and mortality rates. Shields et al. reported that the diagnosis of SGC was suspected initially in only 32% of patients at first examination and in only 50% at histopathologic examination elsewhere.[6] It is known that overall mortality rate of SGC is 5–10% because of frequent difficulties or mistakes in diagnosis and delay in treatment. The mortality from metastasis may increase to 25%.[5] Thus, it is essential to perform meticulous history-taking and to maintain a high clinical suspicion for SGC so as to accurately and promptly diagnosis the malignancy.

Referred from this case, IKC also should be included in the list of diseases under which SGC can masquerade mainly because both lesions are situated in the tarsus, and SGC could contain cystic portions as in IKC. Thus, greater awareness and clinical suspicion is crucial in diagnosing IKC in order to discriminate it from SGC.

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Granulomatosis with polyangiitis provoked by trauma

A 31-year-old Somali male bus driver presented with left periorbital swelling and diplopia after an alleged assault, having received punches to his face. Testing of ocular movements demonstrated restriction of left upgaze. Examination was consistent with an orbital floor fracture. Surgical exploration confirmed a fracture over the infraorbital nerve canal associated with soft tissue tethering and inferior rectus muscle entrapment. Surgical repair was successful.

At a postoperative follow-up appointment, he had persistent eyelid swelling and conjunctival injection. Despite partial response to a short course of oral steroids, he developed progressive swelling, pain, and diplopia. Hypoglobus and proptosis (Fig. 1A) suggested a superior orbital mass.

A further computed tomography (CT) of the orbits revealed a superiomedial orbital mass (Fig. 1B) causing inferior globe displacement. A retrospective review of CT images at the time of presentation (Fig. 1C) revealed the same mass to be present, albeit smaller. Biopsies were taken of a firm rubbery mass in the superior orbit. These