Internal ophthalmoplegia is a rare complication that has been described with a variety of inferior oblique weakening procedures. Most reported instances of internal ophthalmoplegia were temporary, with a near-complete recovery of the pupillary function within 3–6 months after surgery.[2] In severe cases though, permanent tonic pupil after inferior oblique weakening has been described, including after recession.[1,3,5] The proposed mechanism is traction on the neurovascular bundle of the inferior oblique and subsequent stretching of the ciliary ganglion, which provides parasympathetic innervation for pupillary constriction and accommodation. It is plausible that procedures that place greater traction to the neurovascular bundle, such as myectomy or denervation and extirpation, may be at higher risks of this complication.

There is no clear treatment to improve the long-term recovery of the condition. Some authors have advocated for the use of 1% pilocarpine to reduce anisocoria and blurred vision while awaiting resolution.[2] Treatment with oral steroids had been described in one previous case of internal ophthalmoplegia after sinus surgery.[4] Although our patient received a 3-week tapering course of oral prednisolone, we lack sufficient data to determine if steroids contributed to her recovery. Our patient's accommodation recovered sooner than her pupillary function.

In summary, our case demonstrates that internal ophthalmoplegia is a possible complication of inferior oblique recession. We recommend minimizing traction on the inferior oblique as much as possible during surgery. If a muscle clamp or hemostat is used on the inferior oblique, care should be given to reduce traction and release the clamp as soon as feasible.

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Footnotes and Disclosure

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

Case of Coats disease associated with neurofibromatosis type 1

A 24-month-old boy with neurofibromatosis type 1 (NF1) was found to have Coats disease during clinical follow-up examination. The relationship between the 2 conditions is discussed.

A newborn was diagnosed with NF1 after birth based on clinical criteria[1]: his mother was diagnosed with NF1, and he had more than 6 *café au lait* spots. The patient's mother did not have any ocular history. When the boy was 3 months old, we performed a complete ophthalmic examination for optic nerve glioma screening. Findings of the anterior segment examination and funduscopic examination were unremarkable, especially optic nerve head edema was not detected, and he was asked to come back at age of 9 months to conduct the refractive screening (Supplementary Fig. 1, available online). The patient was lost to follow-up. He was referred to our department at the age of 17 months for leukocoria in his right eye. Findings of his neurologic examination were normal. He was uncomfortable when his left eye was covered. Cycloplegic refraction performed under cyclopentolate 1% was within normal range. Anterior segment examination showed clear cornea and normal crystalline lens, and no Lisch nodules were seen.

Funduscopic examination revealed peripheral retinal telangiectasia associated with diffuse intraretinal exudation, which was consistent with stage 2b Coats disease according to Shields et al. Classification[2] (Fig. 1A). He subsequently underwent peripheral laser photocoagulation under general anaesthesia. Intraocular pressure was normal.

Intraoperative spectral-domain optical coherence tomography images were obtained using the Retcan 700 (Carl Zeiss Lumera 700 with integrative HD OCT, Carl Zeiss Meditec AG) and demonstrated marked intraretinal and subretinal exudation in the macular area (Fig. 1B). After 1 month, a second peripheral laser photocoagulation session was required for persistent active telangiectasia. Follow-up examination at 1 month disclosed regression of the peripheral telangiectasia and development of foveolar nodule (Supplementary Fig. 2, available online). The child did not require further laser photocoagulation during the 7 months' follow-up.

NF1 is an inherited autosomal dominant disease caused by mutation of the *NF1* gene on chromosome 17, which encodes the protein neurofibromin. However, the diagnosis is based on clinical criteria, and mutational screening is not

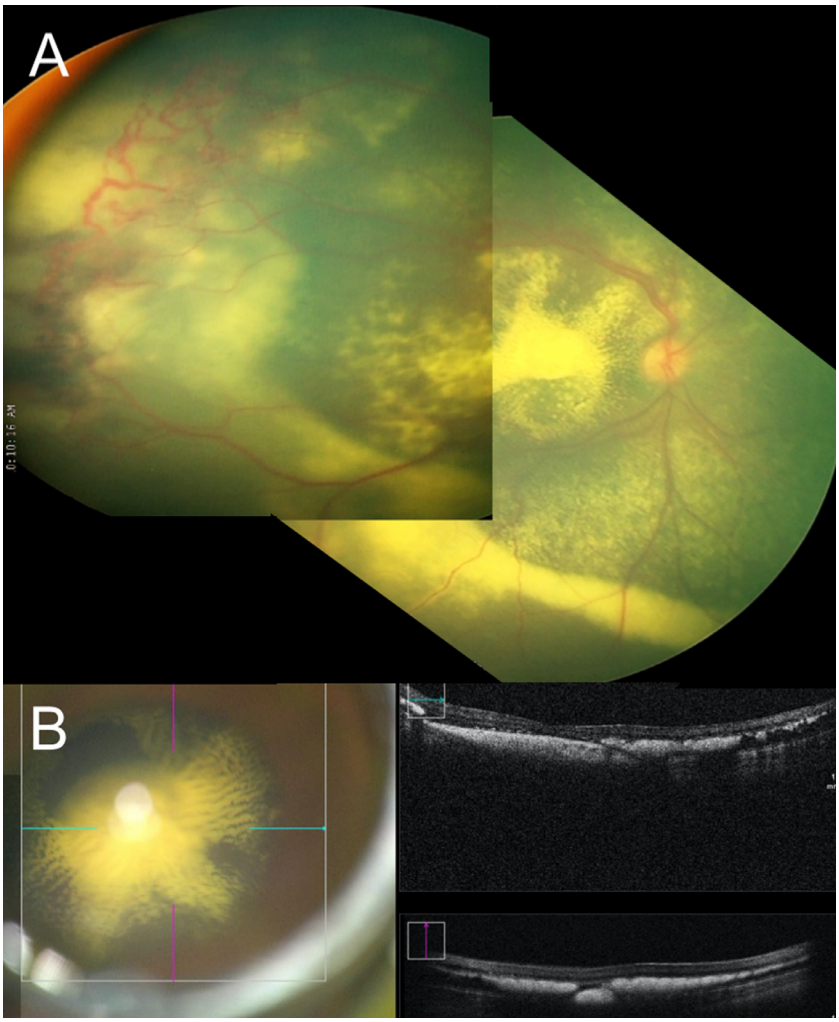

Fig. 1—Funduscopic examination at 17 months of age showed peripheral telangiectasia with diffuse intraretinal exudation (A), which was treated by laser photocoagulation. Intraoperative optical coherence tomography demonstrated intra- and subretinal exudates (B).

required for the diagnosis. The main objective of the ophthalmic examination in children with NF1 is early detection of optic nerve glioma. Other common ophthalmic signs include Lisch nodule and choroidal schwannoma, which are asymptomatic. Parrozzani et al. found that 6.1% of patients[3] with NF1 had retinal vascular abnormalities called corkscrew vessels defined as well-defined, small, tortuous retinal vessels with a spiral aspect, originating from small tributaries of retinal veins, localized by optical coherence tomography angiography in the superficial vascular plexus. Shields et al. reported 6 patients with NF1 and retinal vasoproliferative tumours[4] and hypothesized that there is retinal ischemia, retinal vascular proliferation, and glial proliferation leading to retinal vasoproliferative tumours. It has been shown experimentally that neurofibromin deficiency induced endothelial cell proliferation and retinal neovascularization.[5] However, we found only one previous report on the association between NF1 and Coats disease. We hypothesize that the loss of neurofibromin function leads to retinal vascular abnormalities associated with NF1 that result in localized retinal ischemia with vascular proliferation and exudation leading to Coats disease.

Coats disease is much rarer than NF1, with an estimated prevalence of 0.09/100 000 children. It is a congenital disease but not hereditary; the ratio of boys to girls is 3:1. Characteristic features include peripheral telangiectasia, intraretinal exudation, and retinal detachment. The Norrie disease gene has been suggested as the causal gene in Coats disease.

To the best of our knowledge, the association between NF1 and Coats disease has been described only once in the literature.[6] There is no clue for a common genetic origin because the NF1 is much more prevalent than Coats disease.

There are several articles describing retinal vascular abnormalities associated with NF1. Our report suggests that NF1 may be a risk factor for Coats disease; however, we acknowledged that this association may also be fortuitous given the few reported cases.

In conclusion, this second case report of Coats disease associated with NF1 suggests that there may be an association between these entities. Further research into the pathogenesis of both conditions may elucidate the underlying mechanisms of this association.

Consent for Publication

The patient's parents gave verbal permission for the presentation of clinical details and images in this study. This report does not contain any personal information that could lead to the identification of the patient.

Supplementary Materials

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.jcjo.2020.09.005.

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

Late-onset spontaneous EX-PRESS shunt dislocation into anterior chamber

The EX-PRESS Glaucoma Filtration Device (Alcon Laboratories, Fort Worth, Tex) is a surgical option for management of uncontrolled open-angle glaucoma where medical and laser treatments have failed. It is a stainless steel, nonvalved device that is implanted under a scleral flap and secured by sutures. By using this surgical approach, the complications of erosion of overlying conjunctiva and shunt extrusion are reduced, and similar efficacy and safety, when compared with trabeculectomy, has been shown.[1] Even though it is influenced by magnetic field forces, magnetic resonance imaging (MRI) is considered safe in patients with EX-PRESS shunt.[2,3] We present a case of late-onset spontaneous EX-PRESS dislocation into the anterior chamber, coincidentally after a positron emission tomography–computed tomography (PET-CT) scan.

A 70-year-old Hispanic man being followed and treated for primary open-angle glaucoma presented with sudden pain and decreased vision in his right eye. Two years earlier he had underwent EX-PRESS P-50 model shunt insertion by our group in both eyes with a 2-month span, without any complications, due to severe visual field deterioration despite maximal topical hypotensive therapy. His medical history was relevant for prostate cancer in remission for 2 years. As part of follow-up, a PET-CT (Biograph mCT, Siemens, Siemens Health Care, Erlangen, Germany) scan was performed 2 weeks before the onset of ocular pain.

During consultation, examination revealed a best-corrected visual acuity of 20/40 in the right eye and 20/20 in the left eye with an intraocular pressure (IOP) of 12 and 14 mm Hg, respectively. The patient denied any history of trauma, rubbing, cough, or Valsalva manoeuvres. The biomicroscopy of the right eye showed mild conjunctival injection, a low diffuse bleb, and a localized corneal edema. The whole EX-PRESS shunt was noted to lie inferiorly in the anterior chamber angle (Fig. 1).

The patient was taken to the operating room, and the EX-PRESS shunt was removed via a 2.2 mm clear corneal incision assisted by ophthalmic viscosurgical devices and straight toothed 0.12 mm forceps, without complications or incidents (Fig. 2). On postoperative day 1, his vision was 20/30 and IOP 14 mm Hg without any topical medications, no corneal edema, and minimal anterior chamber reaction, which was controlled with topical steroids. One month after the surgery, his visual acuity was 20/20, IOP was 14 mm Hg, a mild iris atrophy in the sector of the wound was found, and the specular microscopy revealed a cell count of 1815 within normal morphology.

The EX-PRESS shunt was designed as an alternative to trabeculectomy with less complications for patients with open-angle glaucoma in which medical therapy has failed. It is made of stainless steel, which is an inert material and compatible with MRI. Implanting the device under a scleral flap has reduced the possibility of movement of the device and conjunctival exposure.

Coincidentally, our patient underwent PET-CT scan 2 weeks before the EX-PRESS dislocation. PET-CT is a nuclear imaging test in which a radiolabeled molecule enables functional imaging of a metabolic pathway. It has