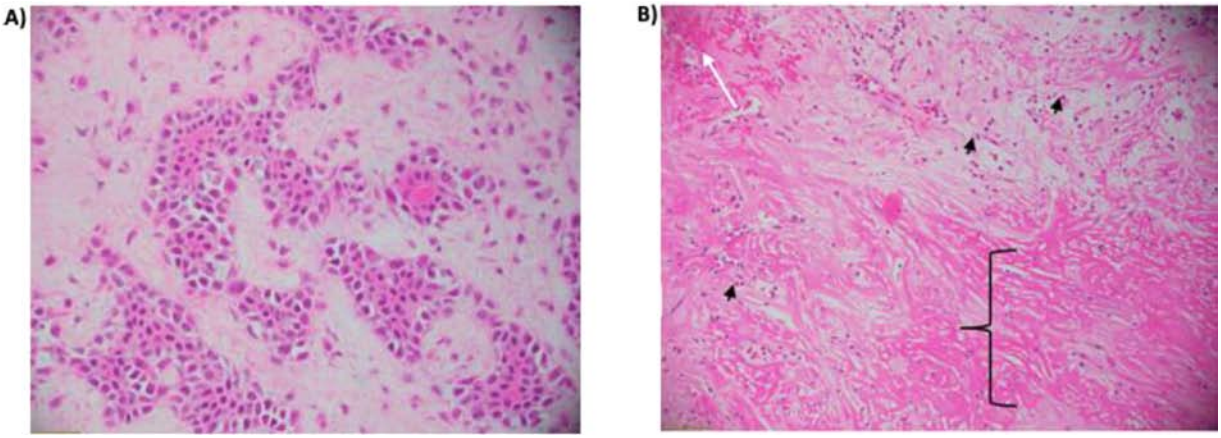


**Fig. 2—Histologic sections of the LGPA. (A) Section of pleomorphic adenoma (high-power field, × 40 magnification) displaying epithelial and myoepithelial populations suspended in a myxoid stromal matrix giving the appearance of a biphasic tumour. (B) Section of coagulative necrosis (high-power field, × 20 magnification) showing hyalinization area (black bracket) and areas of hemorrhage (white arrow) and monocytes (black arrows).**

diagnosis of carcinoma ex pleomorphic adenoma. The incidental presentation of our case is in keeping with the benign diagnosis.

In summary, we present a patient with cystic LGPA showing coagulative necrosis, further adding to the growing evidence that coagulative necrosis and cystic changes are not always associated with malignancy and that careful correlation of radiohistopathologic features is crucial.

## Supplementary Materials

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.jcjo.2021.09.020.

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Originally received Jul. 31, 2021. Final revision Sep. 21, 2021. Accepted Sep. 27, 2021.

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## Footnotes and Disclosure

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

# Treatment of an orbital pseudomeningocele through an eyelid incision

Pseudomeningoceles (PMs) occur from extravasation of cerebrospinal fluid (CSF) into soft tissue secondary to a dural tear.[1–3] Unlike a true meningocele, which is lined by arachnoid tissue, PMs are associated with the formation of a fibrous capsule.[2] Although PMs can form due to surgical insults to the dural covering of the brain or spine, they can also occur secondary to trauma or congenital abnormalities.[1,2] Clinical symptoms usually arise when there is mass effect on the

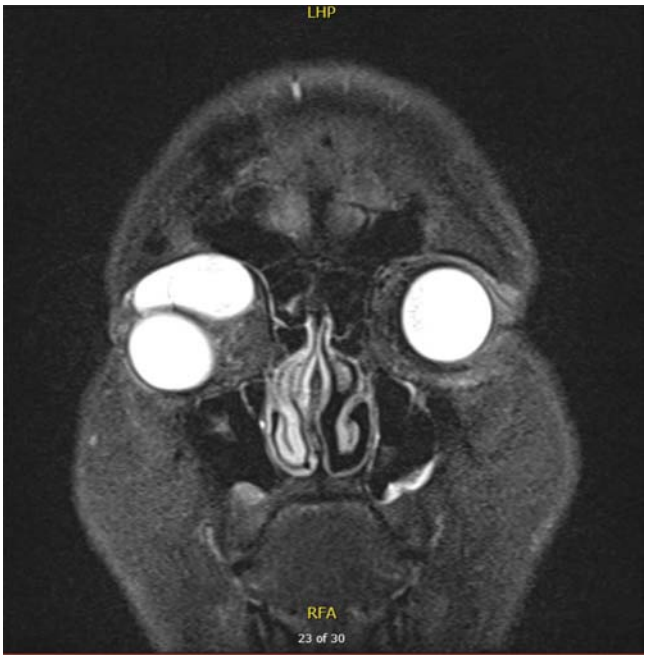


**Fig. 1—T2-weighted, coronal-cut magnetic resonance imaging demonstrating a 5.1 × 3.3 × 2.4 cm cystic mass affecting the optic canal continuous with the right superior orbit and right frontal extra-axial space, an effaced right superior muscle complex, mass effect and proptosis of the right globe, and crowding of the right orbital apex.**

surrounding structures.[3] There are very few reports of orbital PMs in the current literature.[1,4] Here, we report a case of a 17-year-old male status-post gross tumor resection of a pilocytic astrocytoma, who presented with a PM involving the right optic canal and superior orbit and was treated via an anterior orbitotomy.

## Case Report

A 17-year-old male with a history of type 2 diabetes mellitus, obesity, and cystic pilocytic astrocytoma of the right optic nerve up to the optic chiasm presented 3 months status-post gross tumor resection via right frontotemporal craniotomy with pulsatile proptosis and hypoglobus, blepharoptosis, and a supraduction deficit of his right eye. Seven years prior to the current presentation, the patient had decreased visual acuity OD to 20/25 and optic disc edema of the right optic nerve. There was no other evidence suggesting neurofibromatosis type 1. Neuroimaging demonstrated a fusiform mass affecting the right optic nerve. The patient underwent multiple rounds of chemotherapy, including carboplatin, Avastin, and vinblastine. Despite treatment, there was progression and growth of the tumor with extension to the chiasm. The patient's visual acuity in his right eye had progressed to no light perception, and the tumor threatened the contralateral optic pathways. Multidisciplinary optic nerve tumor resection was indicated. The procedure was uneventful, although the patient had a transient third, fourth, fifth, and sixth cranial neuropathy and developed a small left temporal visual defect secondary to tumor resection.

On follow-up two month post-operatively, the patient was no light perception OD and 20/20 OS. Intraocular pressure was 18 mm Hg OD and 12 mm Hg OS. There was an amaurotic pupil OD. Extraocular motility testing demonstrated a complete supraduction and adduction deficit OD and no motility deficits OS. External examination was significant for pulsatile exophthalmos, hypoglobus, and proptosis OD. Slit-lamp examination was significant for complete ptosis OD but was otherwise within normal limits OU. Dilated fundus examination demonstrated 4+ optic nerve pallor, an ischemic appearing macula, sclerotic arterial and venous vessels, and scattered pigmentary changes in the peripheral retina OD and was normal OS. Magnetic resonance imaging (MRI) of the brain and orbits with and without contrast demonstrated a 5.1 × 3.3 × 2.4 cm cystic mass affecting the optic canal continuous with the right superior orbit and right frontal extra-axial space, an effaced right superior muscle complex, mass effect and proptosis of the right globe, and crowding of the right orbital apex (Fig. 1). The findings were consistent with a PM.

Owing to the compressive effects of the PM causing blepharoptosis and motility deficits, intervention was indicated. The neurosurgery and oculoplastic services proceeded with an image-guided right transorbital approach for repair of the right pseudomeningocele. This was achieved via a supraorbital incision through the upper eyelid crease. The PM was drained, and an autologous fat and muscle graft from the right lateral thigh was grafted into the posterior superior orbit and optic canal to occlude the cranio-orbital communication. The patient also had placement of a lumbar drain to decrease cerebrospinal pressure and forestall PM reformation in the early postoperative period.

Postoperatively, right hypoglobus as well as the supraduction and adduction deficits improved. Repeat MRI orbits, one day postoperatively, demonstrated significant reduction in the PM, now measuring 3.2 × 1.6 × 0.9 cm with improved mass effect and decreased proptosis (Fig. 2). Six weeks after surgery, the patient was noted to have continued improvement in motility of the right eye and stable blepharoptosis OD with a margin reflex distance 1 of 0.5 mm. Repeat MRI orbits 3 months postoperatively demonstrated resolution of the PM.

## Discussion and Conclusion

Patients with PMs can have variable clinical presentations, including pulsatile or non-pulsatile proptosis, bruit, headache, seizures, epilepsy, decrease vision, diplopia, globe displacement, or defects in extraocular movements.[1] On clinical examination, palpation of the PMs demonstrates a soft, reducible mass that can enlarge with Valsalva

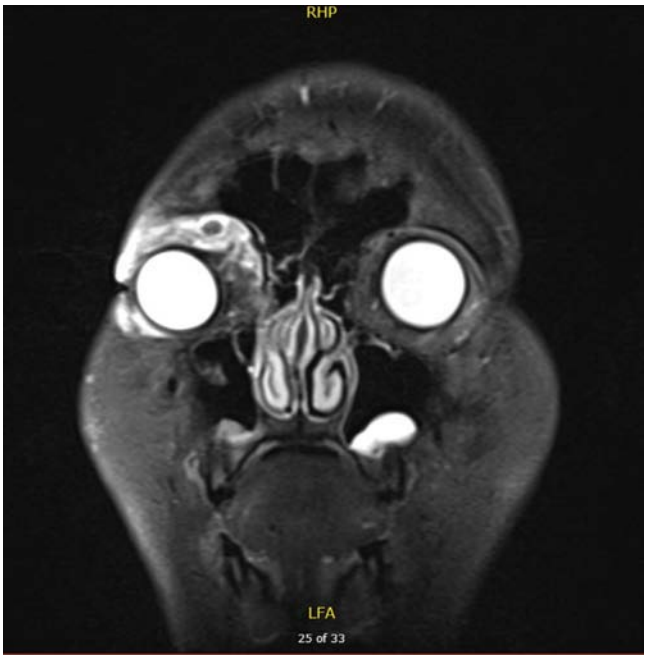


**Fig. 2—T2-weighted, coronal-cut magnetic resonance imaging demonstrating significant reduction in the pseudomeningocele, now measuring 3.2 × 1.6 × 0.9 cm, and decreased proptosis. This image was taken 1 day after surgery.**

maneuvers.[2] The development of a PM is variable and can occur anywhere from weeks to years after initial dural insult.[1,2,4] Interestingly, our patient presented with pulsatile proptosis and extraocular motility deficits that occurred within 3 months after his tumor resection.

Although it is understood that PMs develop from a CSF collection in the soft tissue after dural injury, there is debate regarding the risk factors that may hasten PM development. It is possible that elevated intracranial pressure (ICP) may be a modifiable risk factor in PM formation; prior studies have found a reduction in size of spinal PMs when using ventriculoperitoneal shunts or acetazolamide therapy to reduce ICP.[5] Other possible risk factors for PM formation include a prior diagnosis of syndromic craniosynostosis, a persistent CSF leak, the presence of a bony fracture or bony defect, or the presence of dead space.[3] Possible risk factors in our patient included the presence of a bony defect and the presence of dead space.

The treatment for orbital PMs is variable. Many asymptomatic PMs can be observed.[2] Symptomatic PM can be treated via nonsurgical or surgical means. Nonsurgical treatment options include conservative measures, such as head elevation and pressure dressings; medication management, such as acetazolamide and steroids to reduce CSF pressure; or repeated transcutaneous needle aspirations.[1–3] Surgery, such as ventriculoperitoneal shunts or cranioplasty with surgical watertight repair of the dural defect (with or without fibrin glue) remains as treatment options if nonsurgical management fails or if there is concern for progressive orbital signs or vision loss.[1–3] Small defects may be amenable to fibrin glue. In this instance, a fat and muscle graft was selected because of the size of the cranio-orbital defect.

MRI imaging of both the brain and orbits with and without contrast is recommended if an orbital PM is suspected. Intervention is indicated if there is evidence of progressive proptosis, vision loss, cranial neuropathy, or optic nerve compression.

## Declaration of Competing Interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Originally received Oct. 21, 2021. Final revision Oct. 31, 2021. Accepted Nov. 4, 2021.

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## Footnotes and Disclosure

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**Supported by:** Gavin Herbert Eye Institute is recipient of an institutional Research to Prevent Blindness grant.