CNV can occur and is well documented, we hypothesize that almost total regression of the long-standing CNV in the patient described in this article was a consequence of the systemic chemotherapeutic agents targeting the proliferating endothelial cells within the CNV lesion.

This theory is not without precedent. Although systemic therapy for cancer has been dominated by the use of cytotoxic chemotherapeutics administered in single doses or short courses at the highest doses possible without causing life-threatening levels of toxicity (referred to as the maximum tolerated dose [MTD]), this approach has been under recent reappraisal. More frequent administration of chemotherapeutics at doses significantly below the MTD, with no prolonged drug-free breaks, known as metronomic chemotherapy, may more effectively target the vascular endothelial cells that play an essential role in supplying oxygen and nourishment to the tumour.[9,10] Although too low to kill the cancer cells directly, the doses are high enough to arrest capillary growth that would have supplied nutrients to the cancer cells.

Our patient did not get typical metronomic therapy because he had 1-week breaks. Nonetheless, the concept of low-dose chemotherapy that targets the endothelial cells rather than the tumour cells (a fundamental component of metronomic chemotherapy) is consistent with the effects we saw with our patient and suggests that such an approach may be effective for patients with CNV. We propose that intrachoroidal concentrations of the chemotherapeutic agents used systemically in our patient may have approached metronomic levels, thereby targeting the proliferating endothelial cells in the small vessels within the choroidal neovascular lesion. We further hypothesize that a local (rather than systemic) approach, using more frequent but lower concentrations (metronomic) of intraocular antiproliferative (chemotherapeutic) agents, may possibly target capillary growth in CNV by similar mechanisms. Administration of antiproliferative molecules intravitreally (or by other local means), rather than systemically, may provide higher local concentrations with minimal systemic side effects. Further studies examining the safety of intravitreal (or other local) injection of these and other antiproliferative therapeutic agents would be necessary before using these drugs in patients with CNV. More extensive studies of systemic administration of such drugs are also indicated.

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Rapid growth of an epibulbar complex choristoma in organoid nevus syndrome

A 6-year-old male presented with a large, fleshy mass protruding from the left eye that has been rapidly enlarging for 4 weeks before presentation (Fig. 1A). Since infancy, he manifested with mental retardation, right hemiparesis, motor seizures, and he underwent surgical correction of an undescended testicle. His photograph at the age of 10 months revealed left eye esotropia, hazy cornea, and a flat red lesion on the temporal conjunctiva (Fig. 1B). On examination, the patient exhibited frontoparietal alopecia, faint scalp nevus, multiple eyelid and periocular skin tags, and dental anomalies. A smooth subcutaneous fullness at the left temple was previously biopsied and diagnosed as lipoma. Previous three-dimensional computed tomographic scanning images showed multiple skull defects (Fig. 1C), and magnetic resonance images showed atrophy of the left cerebral hemisphere, with widening of the left lateral ventricle (Fig. 1D). Ophthalmic examination under anaesthesia disclosed a pedunculated epibulbar mass of

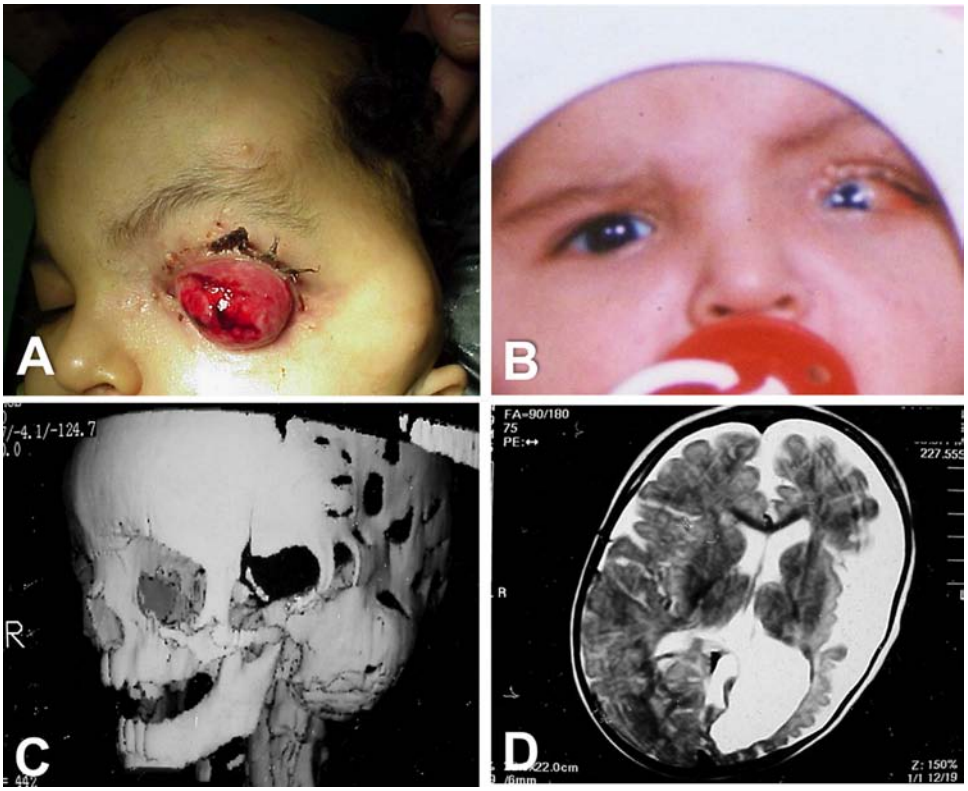

Fig. 1—A, External photo shows large, fleshy mass protruding from the left eye in a patient exhibiting left frontoparietal alopecia, faint scalp nevus, multiple eyelid and periocular skin tags, and subcutaneous temporal lipoma. B, Photograph of patient at age 10 months showing left eye esotropia, hazy cornea, and a flat red lesion on the temporal conjunctiva. C, Three-dimensional computed tomographic scan shows multiple skull defects. D, Magnetic resonance imaging shows atrophy of the left cerebral hemisphere and widening of the left lateral ventricle.

approximately 2 × 3 cm originating from the superior temporal conjunctiva overhanging the eye and lower eyelid. The mass was soft with cystlike consistency, irreducible, and nonpulsatile. The findings suggested manifestations of organoid nevus syndrome, which is typically associated with epibulbar choristoma. However, the nature of the bizarre rapid growth of this epibulbar mass was elusive. An incisional biopsy at the mass apex revealed conjunctival subepithelial nonspecific inflammation without evidence of malignancy. Oral amoxicillin clavulanate 250 mg, twice daily, for a week was prescribed. Two weeks later, the epibulbar mass showed further significant enlargement with spontaneous bleeding, which prompted excision biopsy (Fig. 2A). Surgically, the mass was dissected at its base that was attached to the limbus and the upper and lateral conjunctival fornices by using alternate applications of prophylactic bipolar diathermy and small-snips scissors dissection (Fig. 2B). The mass was not adhering to sclera, and the conjunctival surgical defect could be reconstructed with a local flap from the residual redundant conjunctiva. Left eye examination demonstrated translucent microcornea with a thick white plaque at the limbus laterally. The anterior chamber appeared shallow with pupillary dysgenesis that precluded fundus viewing (Fig. 2C). Intraoperative B-scan ultrasonography revealed a normal-sized eye with an echogenic lesion involving posterior sclera (Fig. 2D). Histopathologic analysis of the mass demonstrated conjunctival stratified squamous epithelium at the surface, with subepithelial inflammatory cellular infiltrates, mainly lymphocytes, and dilated vascular channels (Fig. 3A). The main mass volume was predominantly myxomatous with dispersed collagen fibres and blood vessels (Fig. 3B). Focal areas of serous glandular ducts and acini, presumably lacrimal, were detected (Fig. 3C). Adipose tissue was present near the base of the mass (Fig. 3D). These findings were consistent with complex choristoma with subepithelial inflammation and myxomatous tissue, with no evidence of malignancy. No subsequent regrowth occurred in the following 3 years.

Organoid nevus syndrome, first described by Jadassohn,[1] is a congenital, nonhereditary phacomatosis that encompasses numerous cutaneous, neurologic, and ocular manifestations.[1–4] This syndrome presumably results from a lateralized disorder in the proliferation, differentiation, and migration of neuroectoderm in early embryogenesis.[3] Our patient displayed the typical manifestations of organoid nevus syndrome, but he developed an unusual, rapidly enlarging, pedunculated, epibulbar mass that raised the suspicion of a malignant lesion. Several histopathologic types of epibulbar lesions have been described in organoid nevus

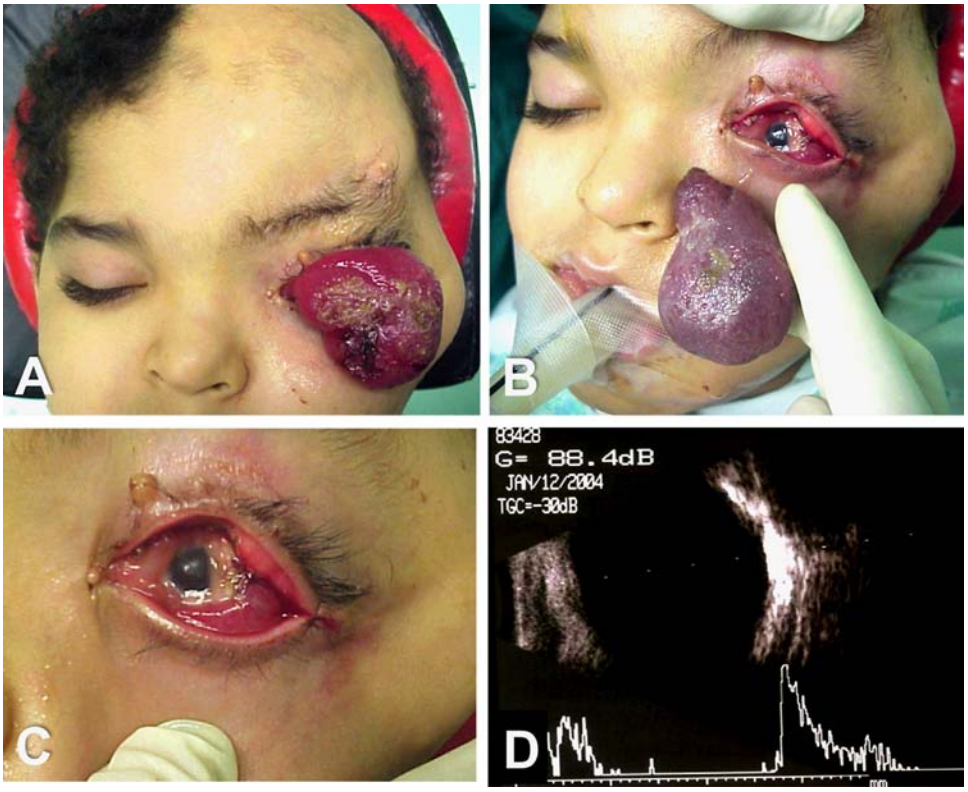

Fig. 2—A, Significant enlargement of the epibulbar mass after 2 weeks. B, Epibulbar mass after excision at the base, and reconstruction of the conjunctiva with a local flap, exposing the left eye underneath. C, The left eye shows translucent microcornea with a thick white plaque at the limbus laterally. D, B-scan ultrasonography shows a hyperreflective lesion involving posterior sclera.

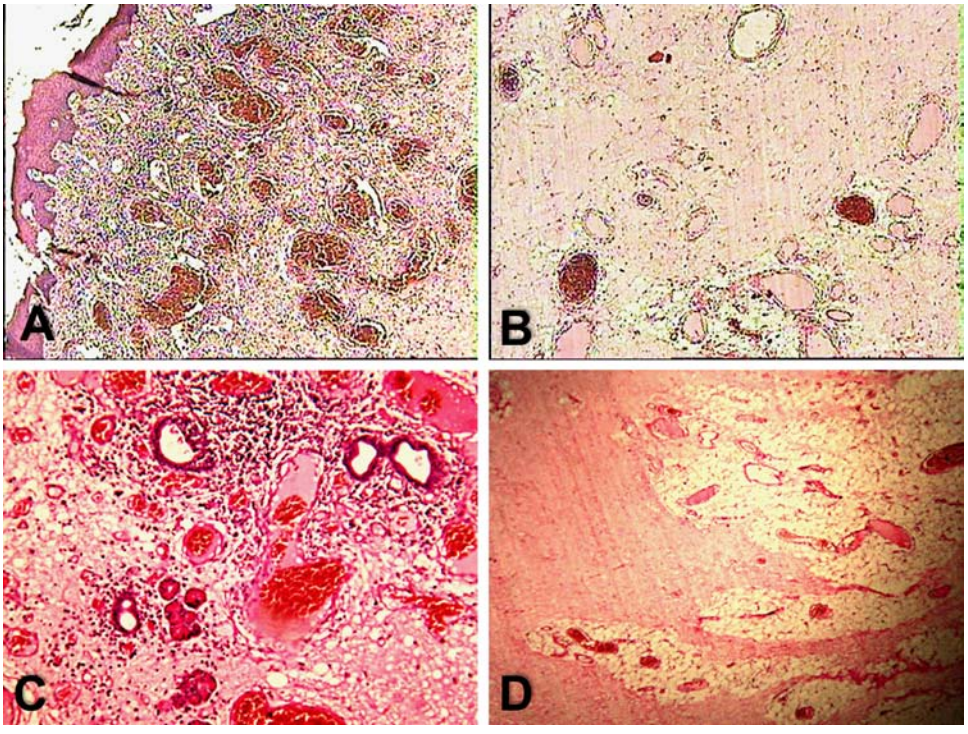

Fig. 3—Photomicrographs of the histopathology of the epibulbar mass showing multiple tissues in complex choristoma (hematoxylin and eosin staining; original magnification ×12.5). A, Conjunctival stratified squamous epithelium at the surface, with subepithelial inflammatory cellular infiltrates, mainly lymphocytes, and dilated vascular channels. B, The main mass volume is predominantly myxomatous with dispersed collagen fibres and blood vessels. C, Focal areas of serous glandular ducts and acini. D, Adipose tissue is present near the base of the mass.

syndrome, including dermoid, lipodermoid, presumed hemangioma, and simple and complex choristoma.[2,4,5] Contrary to the single-tissue structure of simple choristoma, complex choristoma contains ≥ 2 ectopic tissues. The epibulbar mass in our patient was a complex choristoma enclosing glandular, myxomatous, and adipose tissues. Although epibulbar complex choristoma is regarded as a congenital stationary lesion, few reports demonstrated slow progressive growth.[2,6,7] The mechanism of the rapid growth as manifested in our patient that was alarming for a malignant process is ambiguous. Roth and Keltner[8] reported a similar considerable growth of a complex choristoma over 4 weeks in a patient with organoid nevus syndrome. The authors attributed this high growth rate to an inflammatory reaction, as evidenced by increased vascularity, inflammatory cellular infiltration, and reactive epithelial hyperplasia, as demonstrated in their case.[8] Trubnik et al.,[7] in a report on 2 cases, concurred with the possibility of a reactive process secondary to inflammation, trauma, or hormonal factors. Our patient, however, did not display the systemic or local signs of inflammation. Conversely, he showed continued rapid enlargement of his choristoma despite broad-spectrum antibiotic therapy. Interestingly, Shields et al.[2] have observed that the choristomas that were reported to manifest progressive growth harboured myxomatous tissue. Presence of myxomatous tissue is associated with the deposition of glycosaminoglycans including hyaluronic acid, which has a strong water-binding capacity.[9] The resulting osmotic edema may have caused the rapid swelling of the choristoma. This could explain the soft cystlike consistency from the high water content as observed in the myxomatous choristoma of our patient. Moreover, the choristomas in those reports suggesting inflammatory response as a causative factor for rapid lesion expansion have also disclosed myxomatous stroma.[2,7,8] It is thus possible that tissue edema resulting from deposition of hydrophilic myxomatous stromal matrix could have contributed to the pathogenesis of this unusual rapid expansion of the choristoma in this patient.

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Visante OCT in the diagnosis of caterpillar-induced iritis

Lepidoptera are among the most common insects, with approximately 175,000 described species worldwide.[1] Larvae of the species *Thaumetopoea pityocampa*, the "pine processionary caterpillar," are found in late winter and early spring in southern Europe, the Near East, and North Africa; however, milder winters have resulted in them expanding farther north in Europe, and they have been observed in England, Holland, and Germany. These small insects, around 3 to 4 cm long, have the habit of moving over the ground in long head-to-tail processions. They are covered with approximately 63,000 pointed defensive hairs (setae), which contain an urticating toxin, break off readily, and easily become airborne. Contact with the caterpillar's hair can cause a severe itchy rash, respiratory problems, and/or ocular lesions. Caterpillar setae are a rare cause of ocular trauma. Following the entry of setae into the eye, several lesions may occur, including conjunctivitis, conjunctival nodules, keratoconjunctivitis, iridocyclitis, iris nodules, vitreitis, chorioretinopathy, and papillitis.[2,3] We describe in this article a case of caterpillar-induced iritis with acute ocular hypertension, diagnosed by Visante OCT (Carl Zeiss Meditec, Dublin, CA).

A 11-year-old female complained of sudden pain and visual loss in her left eye while she was playing in a pinewood. Physical examination revealed a maculopapular

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