rare but devastating ocular complications that can occur.

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Glucose transporter isoform-1 receptor–positive infantile capillary hemangiomas: case report and literature review

A previously healthy 4-month-old child presented with a right-sided medial canthal subcutaneous mass that was present for 2 weeks (Fig. 1). Parents stated it had been growing in size since it appeared, and there was tearing from this eye. Clinically, location and discolouration gave the appearance of a dacryocystocele; however, the lesion was firm and appeared adherent to the bone. The ocular examination was otherwise normal. Probing of the nasolacrimal duct showed free flow into the nose and did not decompress the lesion. A magnetic resonance imaging scan was obtained and demonstrated a soft-tissue medial canthal mass, with long T1-T2 characteristics, and a homogeneous and lobulated contrast enhancement. The differential diagnosis included hemangioma, lymphangioma, histiocytosis, or an atypical rhabdomyosarcoma. The mass was operatively excised, and pathology demonstrated hemangioma. Immunohistochemistry staining was highly glucose transporter isoform-1 (GLUT-1)–positive (Fig. 2), which is consistent with an infantile hemangioma.

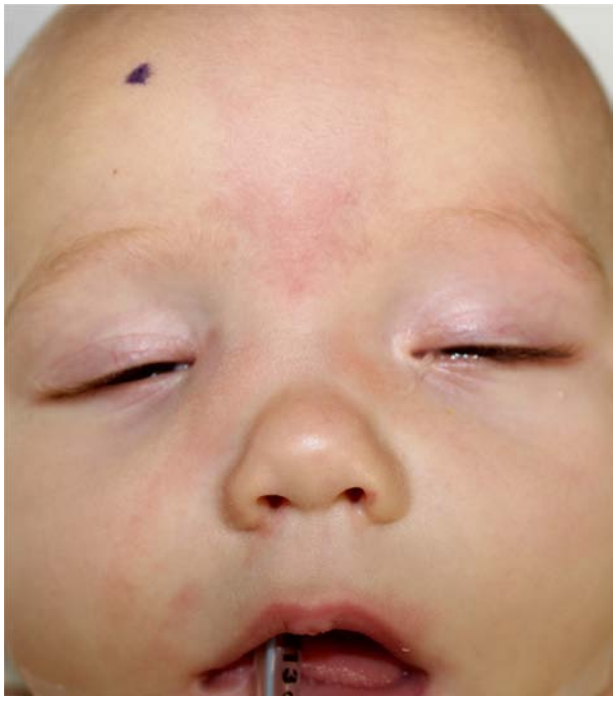

Fig. 1—Right eye medial canthal mass.

Hemangiomas are benign vascular tumours. They represent the most common benign tumour of infancy, affecting up to 5% of all infants in the United States.[1] Hemangiomas occur more commonly in products of multiple gestations, older maternal age, and placental complications such as placenta previa and pre-eclampsia.[2] They are hypothesized to originate from a subset of specific endothelial progenitor cells that are driven to proliferate.[1] Hemangiomas have recently been classified as infantile or congenital, each with vastly different clinical courses. Infantile hemangiomas are characterized by 2 phases: the rapid proliferation phase (occurs over 3–6 months of age) and the involution phase (starts at 1 year but can continue for many).[3] The involution phase occurs as vascular components are replaced by fibrofatty

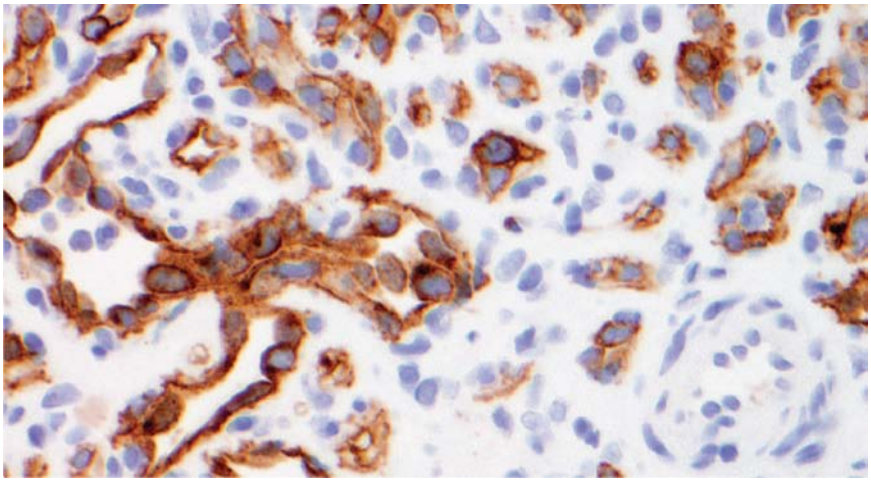

Fig. 2—Glucose transporter isoform-1 receptor–positive immunohistochemical staining.

tissue.[3] They possess a specific histochemical marker named GLUT-1, which is not found on other vascular tumours.[1] In comparison, congenital hemangiomas are present at birth, are stationary, and, unlike infantile hemangiomas, they have no involution phase.[4] Furthermore, hemangiomas can be classified as superficial, subcutaneous (as in our case), or orbital extension, and occur either locally or segmentally.[4] Because the management and prognosis of infantile versus congenital hemangiomas differ greatly, accurate diagnosis of these lesions carries significant implications for the patient.[5]

GLUT-1 has emerged as a valuable immunohistochemical marker to distinguish between infantile hemangiomas and other vascular lesions.[6] A study of 50 specimens from patients with vascular anomalies found that GLUT-1 was positive in 18 of 19 cases of infantile hemangiomas. The GLUT-1 marker was negative in all other cases (n = 31, 2 noninvoluting congenital hemangiomas and 29 vascular malformations).[6] GLUT-1 accurately distinguishes infantile hemangiomas from other vascular malformations and may be used to histopathologically differentiate between the 2.[6] In fact, this marker is not expressed in normal dermal or subcutaneous capillaries, nor is it expressed in other types of vascular tumours.[7,8] The only other tissue known to share this constellation of markers is that of placental chorionic villi.[8]

Infantile hemangiomas of the periorbital area may threaten or permanently compromise vision by occluding the visual axis, compressing the globe, or expanding into the retrobulbar space.[9] Up to 80% of patients with untreated periorbital infantile hemangiomas will experience complications such as refractive error, amblyopia, or strabismus.[9] Although infantile hemangiomas are often monitored or treated with pharmacologic agents, some congenital hemangiomas may require more aggressive management such as laser therapy, sclerotherapy, or surgical approaches.[10]

Topical, systemic, or intralesional corticosteroid therapies have traditionally been viewed as the first-line treatment for periorbital infantile hemangiomas.[5] Recently, systemic propranolol has been found to have a favourable effect on infantile hemangiomas.[5] Its action was first noted after administration of high-dose corticosteroids caused cardiomyopathy, which was then treated with propranolol. Within days the hemangiomas changed colour and decreased in size.[11] Furthermore, propranolol may work synergistically with corticosteroids and represent an effective adjunct therapy.[12] Léauté-Labrèze et al.[11] first proposed that propranolol inhibits growth and promotes involution of infantile hemangiomas through vasoconstriction and downregulation of angiogenic factors. The specific site of action has not yet been established; however, links between propranolol and the GLUT-1 receptor have been proposed.[13] Research into preeclampsia has demonstrated the effects of propranolol on placental tissue, which is also highly GLUT-1–positive. It has been suggested that beta-blockers induce apoptosis via the GLUT-1 receptor.[14] The histochemical similarities between infantile hemangiomas and the placenta would explain their natural history: rapidly growing, then involuting.[15] Placental tissue and infantile hemangioma tissues share multiple other markers aside from GLUT-1.[8] Other connections exist, as infantile hemangiomas exclusively occur perinatal and are associated with placental injury.[2] Proposed pathology includes embolization of placental cells in utero or at birth with clonal expansion, or somatic mutation and local inductive influences.[8] Furthermore, animal studies into retinopathy of prematurity show topical beta-blockers downregulate vascular endothelial growth factor, and this could be another site of action.[16] Infantile hemangiomas are highly GLUT-1–positive, and this is a likely source of action of beta-blockers, possibly in combination with decreased expression of vascular endothelial growth factor.[3,13]

Systemic beta-blockers should be avoided in the first week of life, and use of these agents in infants should be closely monitored.[17] A variety of treatment protocols exist, and monitoring of infants may vary from institution to institution. The following treatment protocol is one suggested in the literature: baseline echocardiography and 48-hour hospitalization to monitor vital signs and blood glucose levels. The medication should be initially dosed at 0.16 mg/kg and administered q8h. If vitals and blood glucose levels are in the normal range, the dose is incrementally doubled to a maximum of 0.67 mg/kg (with a daily maximum of 2 mg/kg). Propranolol should gradually be tapered over a 2-week period.[17]

In summary, GLUT-1 has emerged as a vital diagnostic tool to differentiate infantile hemangiomas from other vascular lesions.[1] Propranolol represents an effective treatment for infantile hemangiomas, with a proposed site of action on the GLUT-1 receptor, and should be used as a first-line modality in cases where treatment is required.[12] We recommend considering the usefulness of the GLUT-1 receptor in biopsy of undiagnosed vascular tumours, and the possible eventuality of immunohistochemical markers guiding treatment decisions.

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Isolated choroidal macrovessel: a tracklike choroidal lesion

Enhanced depth imaging optical coherence tomography (EDI-OCT) has provided significant new insights into the choroidal involvement in numerous vitreoretinal diseases, including age-related choroidal atrophy and central serous.[1,2] In this report, a patient presented with a lesion suggesting a variety of diagnoses including a potential intraocular parasite, but noninvasive visualization of ocular structures, particularly the choroid, established the diagnosis of a rare ocular lesion: a choroidal macrovessel possibly secondary to an aberrant long posterior ciliary artery.

A 76-year-old asymptomatic female was referred for evaluation of a possible "worm" in the right eye. Visual acuity was 20/30. Examination revealed macular drusen with a prominent temporal curvilinear red–orange lesion extending from the temporal fovea to the periphery. The lesion appeared deep, but caused elevation of the overlying retina. Associated pigment rarefaction and isolated pigmentary clumping with a bulbous termination in the macular region were noted. Fundus autofluorescence demonstrated variable hyperautofluorescence over the lesion (Fig. 1). Fluorescein angiography showed a subtle transmission defect. Indocyanine green angiography showed a large vascular structure with overlying blocking caused by the focal areas of hyperpigmentation. The vessel filled early, suggesting it to be arterial. The intensity of the fluorescence paralleled the fluorescence seen in the surrounding, presumably normal choroidal vasculature (Fig. 2). EDI-OCT demonstrated a large hyporeflective tubular structure in the choroidal space consistent with a choroidal macrovessel. Above the retinal pigment epithelium