LETTERS TO THE EDITOR

Retina to retina adhesions following suprachoroidal haemorrhage

EDITOR,—Retina to retina adhesions occur as a rare complication of suprachoroidal haemorrhage. Previous reports indicate a poor prognosis. We observed a patient who developed suprachoroidal haemorrhage and retina to retina adhesions following a glaucoma shunt procedure. Modern vitreoretinal surgical techniques were used to lyse the adhesions.

CASE REPORT

An 80-year-old white woman with a history of aphakia and recent penetrating keratoplasty developed glaucoma requiring the placement of a Krupin valve with disc. Three days later the patient noted a severe, sharp pain in her right eye.

Visual acuity was light perception. The intraocular pressure was 14 mm Hg. The corneal graft had 2+ deep striae. The anterior chamber was deep with a small hyphaema present. The tube shunt was properly positioned in a deep anterior chamber. A strand of vitreous was incarcerated within the proximal tube. Large, dark, 'kissing' choroidal detachments were present. Ultrasound examination showed massive suprachoroidal haemorrhage.

Three days later, the choroidals were somewhat smaller, with the nasal and temporal components slightly separated. Two regions of the nasal and temporal retina remained adherent to one another, with resulting traction retinal detachments (Fig 1).

It was feared that further spontaneous recession of the choroidals would cause tearing of the retina. A surgical procedure was performed using techniques similar to those described for retinal adhesions to anterior segment structures.¹ Saline was infused via a 20 gauge anterior chamber maintainer in the inferotemporal limbus. Very low infusion pressure was used to avoid forceful separation of the choroidal detachments. Two additional limbal 20 gauge incisions were created. Using microscope illumination and a vitrectomy probe, vitreous was removed from the anterior segment and tube shunt. Using endoillumination, the vitrectomy was extended posteriorly until the retinal adhesions were reached.

The retina to retina adhesions were gently separated with a spatula-shaped retinal pick. The suprachoroidal haemorrhage was drained via two equatorial, radial sclerotomies. The vitrectomy was continued through the limbal approach, removing as much vitreous as safely

possible. No tears or persistent traction were found. To avoid postoperative hypotony, the shunt tube was ligated with a 5-0 polyglactin suture.

The patient has been followed for 9 months. Vision recovered to 20/100. The intraocular pressure has been adequately controlled. The retina has remained attached.

COMMENT

Large suprachoroidal haemorrhages may bring the retina into contact with other retinal surfaces, ciliary body, anterior segment structures, or surgical wounds. If firm adhesions form, traction retinal detachment, retinal tears, and rhegmatogenous retinal detachment are likely to occur as the choroidals recede.

We found two references describing retina to retina adhesions complicating suprachoroidal haemorrhage. Berrocal described three cases due to appositional choroidal detachments following scleral buckling surgery.² Two of the cases were haemorrhagic choroidals. In one eye the condition was successfully treated by draining the choroidals, and reforming the globe with saline and air. The others were judged to be inoperable. Lakhanpal described one case each of 'interretinal' and 'intraretinal' adhesions, neither of which could be separated surgically.³

Retina to retina adhesions may occur more frequently than the literature suggests. Media opacities associated with suprachoroidal haemorrhage may preclude recognition of adhesions. Rhegmatogenous detachment may supervene, without recognition of preceding adhesions. Some may lyse spontaneously, without sequelae. We intervened because we felt the risk of rhegmatogenous retinal detachment was high, and the prognosis for repair of such a detachment would be poor.

We believe this is the first case of retina to retina adhesions documented photographically, and the first description of successful management using vitreoretinal surgical techniques.

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DAVID V WEINBERG

LISA F ROSENBERG

Department of Ophthalmology, Northwestern University Medical School, Chicago, USA

Correspondence to: Dr DV Weinberg, 645 N Michigan Avenue, Suite 440, Chicago, IL 60611, USA.

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Solitary astrocytoma of the retina in a child

EDITOR,—Astrocytic proliferations in the eye are rare lesions usually associated with tuberous sclerosis or neurofibromatosis. Solitary

astrocytomas occurring outside this association are exceptional, fewer than 20 cases having been reported. We present an additional case which occurred in a unique anatomical location and discuss the clinical features and treatment of this rare condition.

CASE REPORT

A 3-year-old girl presented with a 6 week history of left divergent squint and an abnormal left retinal reflex. Indirect ophthalmoscopy revealed a large inferior retinal mass and a 360° retinal detachment. A presumptive diagnosis of retinoblastoma was made. There was no significant medical or family history and developmental milestones were normal. She was admitted for examination under anaesthesia. Ultrasound, lumbar puncture, bone marrow aspiration, and bone scan were also performed. The presence of a retinal mass was confirmed and the affected eye was enucleated. The patient made a good recovery and a prosthesis was fitted. Examination of the right eye showed two small areas of anomalous retinal pigment epithelium change.

In view of the histological findings discussed below, the patient underwent further examination for signs of tuberous sclerosis and neurofibromatosis. Chromosomal analysis, computed tomography scan of the brain, and dermatological examination including Wood's light were all normal. After 4 years of follow up the appearances of the right retina were unchanged and there were no signs of systemic disease.

The surgical specimen was a 20 mm diameter globe with 11 mm of attached optic nerve. A white reflex was visible through the normal cornea; the iris and pupil were symmetrical. After fixation, sectioning of the globe showed complete retinal detachment. The intervening space contained opaque fluid. A firm, grey, spherical tumour was present. It arose at the junction of the iris and retina and pressed on the lens posteriorly (Fig 1). No other tumour nodules or other macroscopical abnormalities were present.

Histological examination showed that the tumour arose at the junction of the nervous and non-nervous parts of the retina. It was composed of spindle-shaped cells with moderate nuclear pleomorphism and abundant, fibrillary, eosinophilic cytoplasm. The cells were arranged in storiform patterns and also formed ill defined palisades, some around blood vessels (Fig 2). Several areas of microcystic degeneration were present (Fig 1)

Figure 1 One of two areas of retina to retina adhesion, with traction retinal detachments (small arrows) bridging large nasal and temporal choroidal elevations (large arrows).

Figure 1 A low power view of the sectioned eye showing the solitary tumour pressing on and deforming the posterior surface of the lens. Tumour diameter 7 mm.

and focal collections of macrophages were associated with these areas. No mitoses were noted. Small vessels were present throughout the tumour, but no vascular tufting was seen. Rosenthal fibres, granular bodies, and multinucleate change were not noted. Scanty reticulin fibres were present within the tumour, but no collagen was seen. With immunohistochemistry the tumour cells stained positively for S100 protein, glial fibrillary acidic protein, neuron specific enolase, and vimentin. They were negative for desmin, the proliferation marker Ki-67, and the oncogene p53. The choroid, sclera, and ciliary body were not involved and no evidence of tumour was seen in the optic nerve. A diagnosis of solitary retinal astrocytoma was made.

COMMENT

A hamartoma is defined as a nodular or tumour-like mass arising from faulty embryonal development of cells and tissues natural to the place where it arises as seen, for example, in vascular birthmarks. Astrocytic proliferations arising from the retina are uncommon and usually represent hamartomas occurring in association with tuberous sclerosis or neurofibromatosis.¹ Although their growth rate does not usually exceed that of the surrounding tissues, on occasion they may enlarge sufficiently to compress the optic disc and cause visual field defects. Very rarely they may bleed, causing vitreous haemorrhage.

Outside the syndromic settings astrocytic proliferations in the eye are extremely rare and usually single. Most of the reported cases were situated on the optic nerve head or were in continuity with it.²⁻⁴ The features of the present case, consisting of an astrocytic proliferation arising in the peripheral retina in a patient without evidence of genetic disease on extensive examination and follow up, are thus exceptional. To our knowledge there have only been four previous reports of solitary astrocytoma not involving the optic disc.^{5,6}

Previously described lesions were well circumscribed and composed of well differentiated astrocytes. Some were described as low grade astrocytic neoplasms ('astrocytoma'), while others were called astrocytic hamartomas. In a paediatric setting, differentiation between these two designations may not be possible. In view of the cytological features, absence of systemic findings, and unusual peripheral location, we feel that the present case represents a solitary astrocytoma.

Owing to its extreme rarity and overlapping clinical appearances, solitary retinal astrocytoma is essentially indistinguishable at presentation from retinoblastoma. All reported cases have been surprise findings in enucleated eyes. In several instances tumour necrosis, sometimes associated with retinal detachment, has

been associated with the onset of symptoms.^{2,4} Necrosis was not seen in the present case, but total retinal detachment with extensive subretinal exudate was present and was presumably responsible for bringing this case to clinical attention.

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JA LEE

Department of Pathology, Royal Hallamshire Hospital, Sheffield S10 2RX

P HARVEY

Department of Ophthalmology, Royal Hallamshire Hospital, Sheffield S10 2RX

RD FINLAY

Royal United Hospital, Bath BA1 3NG

PJ BERRY

Directorate of Pathology, St Michael's Hospital, Southwell Street, Bristol BS2 8EG

Correspondence to: Dr JA Lee, University Department of Pathology, The Medical School, Beech Hill Road, Sheffield S10 2RX.

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Coats'-like response in a patient with pars planitis

EDITOR.—Pars planitis is a form of intermediate uveitis characterised by chronic, bilateral vitritis, vitreous cellular aggregates ('snowballs'), periphlebitis, and peripheral fibrovascular ('snowbank') accumulation over the inferior pars plana and vitreous base. Reported complications include cystoid macular oedema (CMO), cataract, band keratopathy, glaucoma, rhegmatogenous and non-rhegmatogenous retinal detachment, retinosis, vitreous haemorrhage, and subretinal, retinal, and optic disc neovascularisation.¹⁻³

Coats' disease is a primarily unilateral disease of young males marked by vascular abnormalities, classically telangiectatic retinal vessels, with massive intraretinal and subretinal exudates.^{4,5} The term 'Coats'-like response' refers to a fundus with the clinical appearance of Coats' disease in the setting of other ocular or systemic disease. We report a case of Coats'-like response in a patient with pars planitis.

CASE REPORT

A 31-year-old woman had been diagnosed in 1977 with pars planitis. She had initially presented with mild vitreous haemorrhage in the right eye without obvious neovascularisation, and had developed bilateral posterior subcapsular cataracts. Several times during the course of her disease she had exacerbations of CMO that necessitated sub-Tenon's steroid injections. Her medical history included hypothyroidism, and hospitalisation for a 'viral-inflammatory illness' in 1990.

In 1989, an area of subretinal blood and exudate was noted in the inferotemporal periphery of the right eye, without signs of active ocular inflammation or evidence of previous branch vein occlusion. This area appeared stable over the next several years. In 1991 the patient presented with keratic precipitates on the corneal endothelium of the right eye, with moderate anterior chamber and vitreous inflammation; the subretinal exudate remained unchanged. Ultrasonography revealed a mildly (maximum 1.9 mm) elevated mass with an irregular surface and medium to high reflectivity, without internal vascularity or associated retinal detachment. Observation was recommended because the visual acuity was 20/40 in the right eye.

In 1993, visual acuity was 20/40 in both eyes, with unchanged posterior subcapsular cataracts, quiet anterior segments, and no vitreous inflammation. Ophthalmoscopy in the right eye revealed that the subretinal exudate had extended posterior to the equator, within the inferotemporal arcade (Fig 1). Fluorescein angiography revealed telangiectasia of the retinal vasculature in this same area, with leakage later in the course of the angiogram (Fig 2).

COMMENT

Coats' original description of exudative retinal vascular disease has since been refined to describe a specific clinical picture of abnormal congenital telangiectatic retinal vessels accompanied by yellow subretinal exudates, with or without retinal detachment. The telangiectasia is a localised fusiform dilatation of retinal arteries and/or veins. Other retinal findings include vascular loops and beading, neovascularisation, and focally or segmentally dilated capillaries.^{4,5} A fundus appearance similar to that of Coats' disease, called a Coats'-like response, has been reported in the setting of ocular diseases such as retinitis pig-

Figure 1 Subretinal exudate within the inferotemporal arcade in the right eye. The arrowhead indicates the area seen on the fluorescein angiogram in Figure 2.

Figure 2 Fluorescein angiogram (corresponding to the inferotemporal area of Fig 1) demonstrating telangiectatic retinal vessels at the site of the subretinal exudate.

Figure 2 The microscopic appearance of the tumour. This typical field shows fibrillary astrocytic cells arranged in swirling patterns and forming ill defined palisades. A small area of microcystic degeneration is also present.