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 onco- gene 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. Although their growth rate exceeds 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. 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.

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. 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.

We thank Professor A Garner of the Institute of Ophthalmology for his opinion on the histological sections.

J A LEE
Department of Pathology, Royal Hallamshire Hospital, Sheffield S10 2RX

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

R D FINLAY
Royal United Hospital, Bath BA1 3NG

PI BERRY
Directorate of Pathology, St Michael's Hospital, Southwell Road, Sheffield S10 2RX

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

Accepted for publication 8 March 1996


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, glucoma, rhegmatogenous and non-rhegmatogenous retinal detachment, retinoschisis, vitreous haemorrhage, and subretinal, retinal, and optic disc neovascularisation.

Coats' disease is a primarily unilateral disease of young males marked by vascular abnormalities, classically telangiectatic retinal vessels, with massive intraretinal and subretinal exudates. 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 funnel dilation of retinal arteries and/or veins. Other retinal findings include vascular loops and beading, neovascularisation, and focally or segmentally dilated capillaries. 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 pigmen-

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

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.
mentosa, chronic ischaemic branch retinal vein obstruction, and retinopathy of prematurity, as well as systemic diseases including muscular dystrophy, Senior-Loken syndrome (renal retinal dystrophy), and Turner’s syndrome. To our knowledge, this is the first report of a Coats’-like response in pars planitis.

The aetiology of retinal telangiectasia, either congenital or secondary, remains unclear. Histopathological specimens show functional or structural loss of the blood–retinal barrier, and disruption of retinal architecture. Reese conjectured that endothelial cell dysfunction causes localised microvascular atresia and occlusion, which leads to vascular ectasia and collateral vessels. A vessel-occlusive event may precede the development of retinal telangiectasia. In Coats’-type retinitis pigmentosa, additional possible aetiologies include autoimmune vasculopathy, and chronic subretinal uveitis from subretinal leakage of inflammatory and immunogenic serum constituents. The Coats’-like response in our patient occurred in the eye that had historically been more severely affected by pars planitis; progression of the Coats’-like response suggests ongoing damage to the retinal vasculature secondary to the chronic inflammation of pars planitis.

PHILIP P CHEN
LAWRENCE P CHONG
Doheny Eye Institute, University of Southern California School of Medicine, 1450 South Pacific Street, Los Angeles, CA 90033, USA

Correspondence to: Dr Chen.

Accepted for publication 11 March 1996


Management of a patient with pseudophakic malignant glaucoma: role of ultrasound biomicroscopy

Editor,—Malignant glaucoma is characterised by increased intracocular pressure (IOP) accompanied by shallowing or flattening of the anterior chamber, despite treatment with patent iridectomy and the presence of a normal posterior segment. We describe a patient with pseudophakic malignant glaucoma, a serious disorder, who was treated successfully with pars plana vitrectomy. High frequency ultrasound biomicroscopy, a new technique for examining the ocular tissue even behind the iris in vivo, clarified the mechanism of vitreoretinal block and the misdirection of aqueous flow following cataract surgery and implantation of a posterior chamber lens.

CASE REPORT

A 65-year-old Japanese woman with a history of bilateral primary angle closure glaucoma had undergone successful bilateral argon laser iridiotomies by a local physician in mid May 1994. However, she experienced a gradual increase in IOP in the left eye and shallowing of the anterior chamber in early March 1995. Despite medication, an increase in peripheral anterior synchiae (PAS) was observed in the left eye, and the IOP remained elevated (>21 mm Hg). On 23 March 1995, a slight posterior subcapsular cataract was removed by planned extracapsular extraction, with implantation of a posterior chamber intraocular lens (three piece IOL, optic diameter 6.5 mm) into the capsular bag, and a peripheral iridectomy performed at the 12 o’clock position. About 20 days postoperatively, the depth of the anterior chamber in the left eye gradually decreased, and the IOP again rose. A second peripheral iridectomy was performed at the 2 o’clock position on 3 April, but the IOP remained high despite full medication.

The patient was then referred to our department on 8 June 1995. The anterior chamber was slit-like and the IOP in the left eye was 33 mm Hg with marked corneal oedema. Despite the flatness of the anterior chamber with the patency of iridectomy, there were no signs of forward bowing of the iris, choroidal haemorrhage, or choroidal effusion in either eye. Ultrasound biomicroscopy (UBM-840, Humphrey Instruments Inc, USA) revealed a marked forward displacement of the lens haptic and a dense zonular capsular barrier, with the apposition to the iris root (Fig 1).

Nd-YAG hyaloidecctomy was performed on 8 June 1995. Photodisruption of the anterior hyaloid surface immediately deepened the anterior chamber and created a pathway for the anterior flow of aqueous humour. However, such laser treatment did not reverse the glaucoma, because of the broad peripheral anterior synchiae (PAS) due to long standing peripheral iridocorneal apposition. Ultrasound biomicroscopy showed that, despite deepening of the anterior chamber, the anatomical relative positions around the ciliary body were unchanged, and the apposition of the lens haptic to the zonular capsular barrier persisted (Fig 2). Two days later, trabeculectomy was performed on the temporal lower quadrant after application of mitomycin C. On the first postoperative day after trabeculectomy, a massive amount of fibrin and severe hyphaema were detected in the anterior chamber, and IOP was 55 mm Hg without a filtering bleb. Two days later, a pars plana vitrectomy was performed through the scleral flap created by the last trabeculectomy. Anterior cortical vitrectomy, which involved

Figure 1 Ultrasound biomicroscopy before Nd-YAG hyaloidecctomy. From the temporal angle, the anterior chamber (A) is slit-like with peripheral iridocorneal apposition. The zonular–capsular inflammatory barrier (B), including a shadow, suspected to be haptic of IOL (C), is attached behind the iris.

Figure 2 Ultrasound biomicroscopy after Nd-YAG hyaloidecctomy. The anterior chamber was deepened after performance of the Nd-YAG laser. In the temporal angle, the peripheral portion of the haptic (A) of the posterior chamber lens is still attached to the cilio–zonular barrier (B). Anterior rotation of the ciliary body (C) is observed near the haptic.