ophthalmoscopy with scleral indentation. He suggested that the genetic basis for simultaneous bilateral inferotemporal retinal dialysis is poorly supported.

Interestingly, none of the cases of simultaneous bilateral rhegmatogenous retinal detachment due to retinal dialysis were associated with other multiple retinal breaks. Our patient has a combination of retinal breaks in the form of horseshoe retinal tears and atrophic holes in the absence of any generalized progressive vitreoretinal disorders. Trauma seems to be the most likely a etiology in our case.

We believe that this is the first case ever reported with simultaneous bilateral inferotemporal rhegmatogenous retinal detachment secondary to dialysis with multiple retinal breaks. Our case emphasizes the importance of binocular indirect ophthalmoscopy with scleral indentation in all cases of bilateral inferotemporal retinal dialysis, despite the absence of history or evidence of ocular trauma.

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Sir,

Acute bilateral blindness in meningeal carcinomatosis
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Diffuse or multifocal seeding of the leptomeninges by carcinoma, so-called meningeal carcinomatosis, often presents as simultaneous or rapidly sequential cranial neuropathy, with or without headache, altered mental status, or signs of meningeal irritation. Visual loss may occur in up to 30% of these patients, usually rapid, painless, and unilateral, and often progressing to the other eye.[1] Only a few cases of meningeal carcinomatosis owing to ovarian carcinoma have been reported. To the best of our knowledge there are no reports in the literature on acute bilateral blindness occurring during hours owing to meningeal carcinomatosis or ovarian malignancies. We present a case of acute bilateral blindness secondary to ovarian adenocarcinoma with meningeal infiltration.

Case report

A 50-year-old woman presented in May 2000 with abdominal distention and ill-defined abdominal pain. Examination revealed a firm pelvic mass. Ultrasound showed a mass compatible with ovarian cancer. Surgical exploration and pathologic examination assessed an ovarian serous cystoadenocarcinoma stage IIIc (with abdominal implants over 2 cm in diameter and positive retroperitoneal and inguinal nodes). Total hysterectomy with bilateral salpingo-oophorectomy was performed, and three cycles of systemic chemotherapy of cisplatine and cyclophosphamide were administered. During the follow-up period, no visual complaints were reported. After 2 years, the patient presented with altered mental status and headaches, without visual complaints, or pupil abnormalities. Lumbar puncture revealed elevated cerebrospinal fluid (CSF) protein, and cytology of the centrifugated CSF sediment demonstrated numerous cells consistent with adenocarcinoma. Magnetic resonance imaging (MRI) Tl-weighted scan of the orbit showed thickening of both apical intraorbital optic nerves with slight enhancement postgadolinium (Figures 1 and 2). MRI of the brain demonstrated minimal meningeal enhancement and normal postchiasmatic visual pathways.

During admission, the patient complained of acute painless bilateral loss of vision, and mental status deteriorated. The patient reported that the loss of vision occurred within less than 12 h. At examination, visual acuity was no light perception in both eyes. Anterior segment and intraocular pressure were normal in both eyes. Pupils were amaurotic and nonreactive to light. Optic disc and fundus examination were normal in both

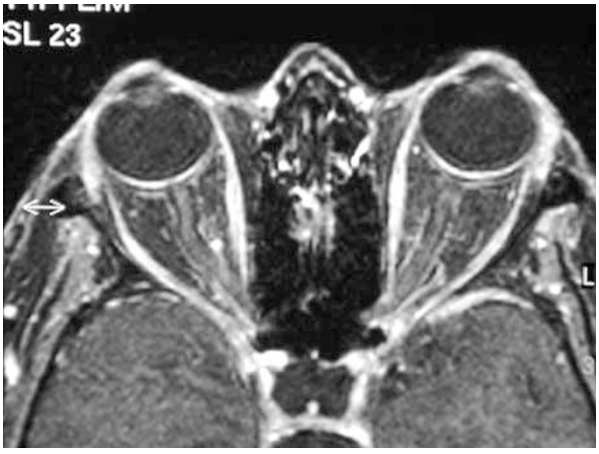

Figure 1 Gadolinium-enhanced axial MRI Tl-weighted SPIR demonstrates bilateral slight enhancement of the apical optic sheath.

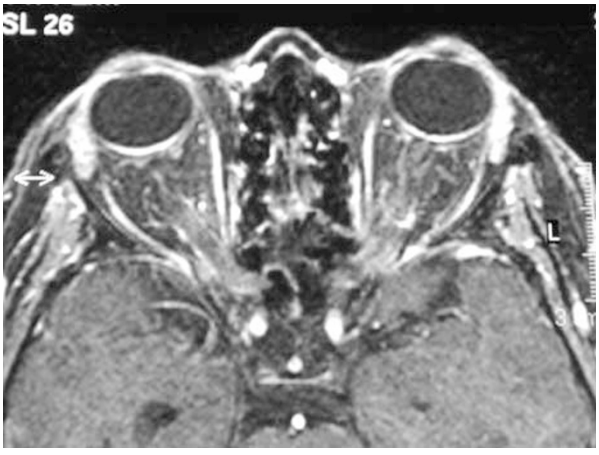

Figure 2 Gadolinium-enhanced axial MRI Tl-weighted SPIR demonstrates bilateral slight enhancement of the apical optic sheath.

eyes. Full ocular ductions were observed. Fluorescein angiography did not reveal any pathology. Despite radiation therapy, the patient died 1 week after the presentation of the visual loss. No autopsy was performed.

Comment

The most common tumours to metastasize to the meninges are breast carcinoma, lung carcinoma, and melanoma. Leptomeningeal involvement has been estimated to occur in 2.5–5% of all breast cancers, 9–25% of small cell lung cancers, and 23% of melanomas.[2] A history of known malignancy is absent in 6–38% of patients with meningeal carcinomatosis.

The combination of headache, rapidly progressive visual loss, sluggish pupil reactions, and normal optic discs should bring to mind the diagnosis of meningeal carcinomatosis.[3] The pathophysiologic mechanisms of visual loss include migration of tumour cells along pial septae with invasion of the optic nerve mesenchyma, 'tumour cuffing' with compression of the nerve, and invasion of the arachnoid surrounding the intracranial optic nerves and chiasm with secondary interruption of the neural microvasculature.[4] The diagnosis of meningeal carcinomatosis is based on cytological confirmation, by either CSF cytology or leptomeningeal biopsy, and can also be supported by MRI findings. Median survival in untreated patients is 4–8 weeks, which is extended to 6 months by aggressive treatment in selected patients.[5] The differential diagnosis of visual loss in a patient with a known malignancy besides meningeal carcinomatosis includes cancer-associated retinopathy (CAR). But in CAR patients, visual loss is usually slowly progressive, ophthalmoscopy reveals attenuated retinal arterioles and changes in retinal pigment epithelium and vitreal cells; electroretinogram (ERG) is almost always abnormal, and in many patients autoantibodies to a 23 kDa retinal antigen may be detected by Western blot immunoelectrophoresis.

In our patient, ERG and CAR antibody testing were considered unnecessary because of the CSF confirmation of the tumour, and the MRI findings. Visual loss as a result of CAR caused by ovarian carcinoma has been described, but to the best of our knowledge there are no reports in the literature on meningeal carcinomatosis with acute bilateral blindness owing to ovarian adenocarcinoma.

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Sir,

Use of Pilocarpine following Hyphaema-Related Ocular Hypertension

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With the wide range of antiglaucoma drugs available to us now, the main use of pilocarpine is in the management of primary angle closure glaucoma. It is also useful in paediatric patients with pseudophakic glaucoma or in the short term following goniotomy. In the current literature, pilocarpine is not recommended for the treatment of hyphaema-related ocular hypertension. We have found it to be useful in a number of cases of raised intraocular pressure secondary to hyphaema which were refractory to other forms of treatment.

Case reports

Case 1 A 37-year-old man was admitted with an extensive left hyphaema following trauma to the eye with an exploding cartridge. The intraocular pressure (IOP) was 34 mmHg. He was commenced on oral acetazolamide, apraclonidine, and dorzolamide. He rebled 3 days later and the IOP rose again. It remained high at 30–40 mmHg for 5 days. Latanoprost was added with no effect. On day 6 following the rebleed the IOP was 52 mmHg, so a washout was performed under general anaesthesia (GA). Postoperatively, the IOP was 42 mmHg. Aqueous was released from the paracentesis on four occasions but the pressure rose within hours on each occasion. He remained on maximum medical treatment. After 3 days, pilocarpine 1% q.i.d. was commenced and the IOP fell to 22 mmHg on day one and 10 mmHg on day 2. All medications apart from pilocarpine were discontinued after 1 week. The pilocarpine was discontinued after a further 2 weeks and the IOP remained low. On gonioscopy he was noted to have angle recession with some areas of peripheral anterior syncechiae.

Case 2 A 35-year-old man was admitted with hyphaema and secondary ocular hypertension (OHT) of 52 mmHg following a blow to the eye. He was treated with topical steroid, cycloplegics, apraclonidine, betaxalol, and oral acetazolamide. The IOP returned to normal and the hypotensive treatment was reduced to betaxalol. Following a rebleed on day three, the IOP remained at 30–40 mmHg for 7 days in spite of recommencement of apraclonidine and maximum dose oral acetazolamide. The addition of latanoprost after 2 days had no effect and mannitol on day 6 reduced the IOP for less than 24 h. On day 8 after the rebleed he had a washout under GA. In spite of an initial drop to 10 mmHg the IOP remained high for 3 days (28–40 mmHg). No reduction was made in his IOP-lowering medications apart from discontinuing latanoprost. Pilocarpine 4% qid was added and the IOP fell over the next 3 days to 12 mmHg. All medications apart from pilocarpine were discontinued. The pilocarpine was discontinued 3 weeks later and the pressure remained at 15 mmHg. He was noted to have angle recession when the view of the angle improved sufficiently to allow gonioscopy.

Case 3 A 19-year-old man was admitted with a right hyphaema and IOP of 38 mmHg following a blow to the right eye. He was commenced on topical steroid, cyclopentolate, acetazolamide, and betaxalol which lowered the IOP. He rebled 2 days later and the IOP remained high at 30–40 mmHg for 5 days, in spite of the addition of apraclonidine and latanoprost. We commenced pilocarpine 2% q.i.d. on day 6 and stopped cyclopentolate and latanoprost. The IOP dropped from 38 to 23 mmHg after 2 days on pilocarpine. The other ocular hypotensive medications were discontinued 3 days after starting pilocarpine. The IOP remained low on pilocarpine alone at 12 mmHg after the other medications were discontinued. There was no evidence of angle recession on gonioscopy.

Comment

Current mangement of hyphaema emphasises the importance of cycloplegia, because of the concurrent anterior chamber activity, to break any pupillary block[1] and to allow posterior segment examination as soon as there is a clear view. In the current literature, pilocarpine is not recommended for ocular pressure lowering.[1,2] However, some cases of secondary glaucoma can be refractory to all treatment, including AC washout. In this small series, pilocarpine appeared to lower the IOP where all other forms of treatment failed. Prior to the development of the wide range of antiglaucoma medications that are available today, pilocarpine and acetazolamide were the only agents for treating hyphaema-related OHT.[3] Pilocarpine and homatropine were tried in combination to speed up resorption of the