Table 1 Number of HIV, HCV, or HBV infections in patients intending to donate blood for use as autologous serum eye drops, compared with new blood donors in Germany in 2004[6]

	No of individuals tested	No of patients/donors with infection			Overall proportion of individuals with infection
		HIV	HCV	HBV	
Present study	88	0	1 (1.1%)	1 (1.1%)	2.3%
New blood donors[6]	per 100 000	6.3 (0.006%)	102.7 (0.1%)	136.2 (0.1%)	0.2%

HBV, hepatitis B virus; HCV, hepatitis C virus; HIV, human immunodeficiency virus.

patients with positive infection markers in the study.

Comment

In blood transfusion, the risk of receiving a blood component which was intended for a different patient is about 1:10 000.[8] This refers to a standardised procedure carried out by trained staff in a controlled hospital setting and led to the recommendation that all PABD patients in Germany should be tested for HIV, HCV, and HBV. In outpatient practice, discrepancies over the dosage of drugs, taking drugs that were not recorded, or not taking a recorded drug were reported in 76% of patients.[9] Serum eye drops are normally used in an ''uncontrolled'' outpatient setting. This suggests that the risk that autologous serum eye drops are not given to the intended patient or are unintentionally used by someone else at home (for example, children), and therefore give rise to the transmission of viral infection, might be considerably greater than 1:10 000. This strongly suggests that virology screening should be carried out in all patients undergoing donation for autologous serum eye drops.

In conclusion, significant proportions of patients intending to donate blood for use as autologous serum eye drops show infections with hepatitis B and C virus and should therefore generally be tested for HIV, HCV, and HBV infection. Patients with viral infections should not be allowed to donate autologous serum eye drops, to prevent the risk of viral transmission by unintended use of autologous serum eye drops by a third party. This principle should generally be applied in all cases where autologous blood is drawn and stored for therapeutic purposes.

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Irreversible aneurysm-like ectasia of the optic nerve sheaths in a patient with bilateral subdural hematomata

The differential diagnosis of retrobulbar processes as detected by computed tomography or magnetic resonance imaging (MRI) comprise a variety of pathological processes. While the most frequent diagnoses are tumours or inflammation, others are incidental findings that might point to an underlying cause. Among the latter is enlargement of the optic nerve sheath, an expansion of the cerebrospinal fluid space around the optic nerve that in some cases is linked to raised intracranial pressure.[1]

The so called ''optical nerve sheath ectasias'' or meningoceles are a rare finding, with only around 40 documented patients.[2 3] The appearance of these ectasias has been described as tube-like. Aneurysm-like ectasias of the optical nerve sheath have not been reported so far. We present a case of bilateral aneurysm-shaped ectasia of the optic nerve sheath associated with bilateral subdural haematomas and presumably with temporarily raised intracranial pressure.

Case report

A 36 year old female patient presenting with a history of focal Jackson seizures in her right arm two months ago, and being repeatedly beaten by her husband, complained of impaired vision and headaches for two days. Ophthalmoscopy revealed bilateral papilloedema, and MRI showed bilateral chronic subdural haematomas, empty sella, and bilateral, aneurysm-like ectasia of the optical nerve sheaths (fig 1). After neurosurgical evacuation of the subdural haematomas, the appearance of the optic nerve head normalised and clinical examination was unremarkable at three and six months after the intervention. On MRI, the ectasia of the optic nerve sheaths remained unchanged.

Comment

Ectasia of the optic nerve sheath is a rare finding in imaging studies, and various terms have been used to desribe this entity: arachnoid cyst,[4] optic hydrops,[5] patulous subarachnoid space,[6] and meningocele.[2]

Concerning pathogenesis and associated diseases, Shanmuganathan *et al*[7] reviewed the literature and identified a patient group with associated progressive hyperopia and choroidal folds.

Lövblad *et al* reported three patients with neurofibromatosis type 1 and tube-like ectasia of the optic nerve sheath.[8] In neurofibromatosis type 1, dural ectasias are a typical finding and are most often present in the spinal canal.[9 10] In contrast to an enlargement of the optic nerve sheath by optic gliomas, which are also common in neurofibromatosis type 1, the ectasias are isointense to cerebrospinal fluid.

Hansen and Helmke[1] investigated the optic nerve sheath response to pressure during CSF absorption studies in 12 patients undergoing neurological testing, and found that changes in the diameter of the sheath followed changes in the intracranial pressure.

In our case, neither progressive hyperopia, choroidal folds, nor features of neurofibromatosis were present. The intracranial pressure was at least temporarily raised, but the morphological changes remained after normalisation of the intracranial pressure.

The morphology of the optic nerve sheath ectasia in our case is different from what has been reported previously. Whereas previous reports describe a tube-like enlargement, in our case it was aneurysm-like.

Vascular aneurysms usually arise from weak parts of the vessels, from the vascular division sites. In our case the aneurysm-like ectasia of the optic nerve may have been pre-existing and

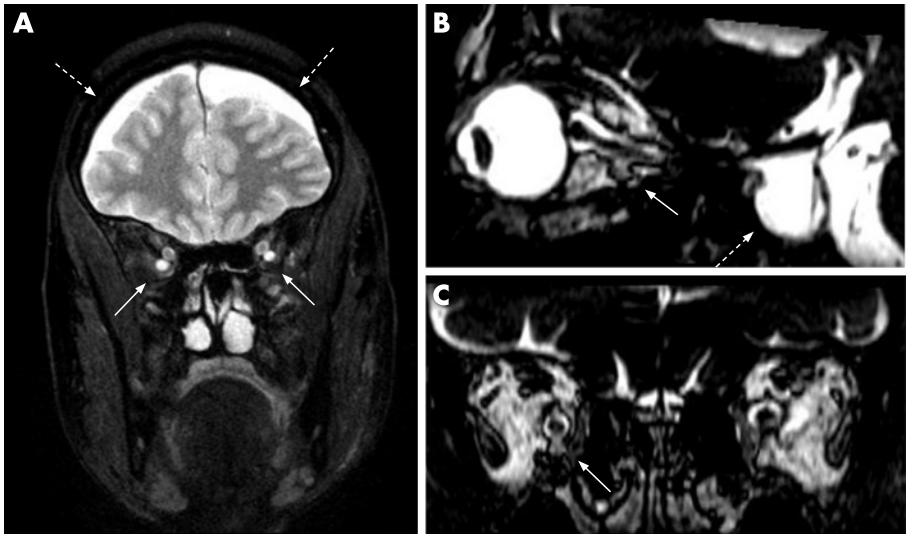

Figure 1 Magnetic resonance images showing aneurysm-like ectasias (solid arrows) of the optic nerve sheaths, subdural haematomas (dotted arrows in (A)), and empty sella (dotted arrow in (B)). (A) T2 weighted image with fat suppression. (B) (C) Multiplanar reconstruction of a three dimensional T2 weighted sequence in sagittal (B) and coronal projection (C).

only accidently detected. Alternatively, one may hypothesise that a focal weakness in the optic nerve meninges may have been induced by some indirect trauma to the orbit. The increased pressure within the optic nerve sheath may then have contributed to the formation of the optic nerve sheath aneurysm.

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Unilateral diffuse uveal melanocytic proliferation (DUMP)

Bilateral diffuse uveal melanocytic proliferation (BDUMP) is a rare paraneoplastic syndrome first described by Machemer 40 years ago in a patient with pancreatic carcinoma.[1] Gass and associates later expanded the syndrome to include round red patches in the fundus, multifocal areas of retinal pigment epithelial (RPE) atrophy, rapidly progressive cataracts, retinal detachments, and choroidal thickening.[2] There have been 34 reported cases since Machemer first described it, and all have had bilateral involvement.[3–8] After searching databases such as MEDLINE (1947 to the present), Exerpta Medica/EMBASE (1947 to the present), and Ophthalmic Literature (1947 to 1988) using the key words BDUMP, melanocytic proliferation, paraneoplastic, and uvea, we report the first case—to the best of our knowledge—of unilateral diffuse uveal melanocytic proliferation (DUMP) found in a patient with metastatic lung cancer.

Case report

A 55 year old woman with a two year history of metastatic small cell lung carcinoma undergoing chemotherapy was referred for a decrease in vision and anterior displacement of the temporal iris. Visual acuity was 20/20 in the right eye and 20/40 in the left. Examination revealed anterior bowing of her left iris, a nuclear cataract, and multiple grey oval patches separated by a reticular pattern of yellow-orange pigmentation in her left fundus (fig 1). Nummular areas of RPE atrophy with pinpoint staining were revealed by fluorescein angiography, along with shallow neurosensory detachments overlying an attenuated RPE by optical coherence tomography/scanning laser ophthalmoscope (OCT/SLO) examination (fig 1). In addition, 20 MHz B-scan ultrasound showed diffuse unilateral choroidal thickening. High frequency (35 MHz) B-scan ultrasound revealed an anterior uveal metastasis causing narrowing of the angle, and a small overlying exudative retinal detachment in her left eye (fig 2). These findings were secondary to both anterior uveal small cell lung cancer and ipsilateral unilateral DUMP. Her right eye was normal. The patient returned at a 4 month follow up and did not have DUMP in the fellow eye.

Comment

There has been some controversy regarding the aetiology of RPE loss as specific antibodies—of the sort classically associated with autoimmune retinopathies such as cancer associated retinopathy (CAR) and melanoma associated retinopathy (MAR)—have not been discovered in BDUMP. Some speculate that the increased metabolic demand of hyperproliferating melanocytes leads to retinal hypoxia and the progression of cataract with RPE dysfunction.[9] Others have suggested that the RPE loss is a result of a distinct paraneoplastic process independent of what leads to melanocytic proliferation.[4] Histopathological specimens of BDUMP have shown benign proliferation of melanocytes within the choroid with widespread dysfunction and necrosis of the overlying RPE (even in areas with minimal melanocytic proliferation).[2 10] Gass theorised that toxic or immunological factors liberated by the interaction of a systemic carcinoma with normal melanocytes of the uveal tract are responsible for this extensive degeneration.[2]

The uveal metastasis in our patient was in close proximity to the melanocytes of the ipsilateral affected RPE and can explain the local production of factors previously described by Gass. Clinically, one sees islands of atrophic RPE lying in a sea of ''orange-pigment'' lipofuscin laden retina (fig 1). The complete absence of any signs of diffuse uveal melanocytic proliferation in the right eye suggests that systemic factors were not responsible in our case. Though the aetiology of the RPE toxicity remains unclear, this case shows that BDUMP does not have to be bilateral. Therefore, it should be called diffuse uveal melanocytic proliferation (DUMP).

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