posterior scleritis. Other entities like varix of vortex vein ampulla are observed intermittently implying spontaneous resolution. Extremely unusual cases of spontaneous regression of choroidal melanoma have been reported.[1,2] Even though these lesions can be differentiated routinely based on clinical examination combined with investigations including B-scan ultrasound and angiography, sometimes they can be a diagnostic dilemma.

Localized suprachoroidal haematoma can be precipitated by hypotony, inflammation, trauma, and vascular disease and can also occur spontaneously in the elderly.[3] They are usually due to the rupture of posterior ciliary arteries with resultant haematoma in the suprachoroidal space. Delayed suprachoroidal haemorrhage after cataract surgery is believed to be prolonged hypotony or it may be a small haemorrhage that might stop bleeding initially and rebleed later.[4]

Posterior scleritis may present as deep orbital pain and is commonly associated with evidence of cells in the vitreous. On B-scan ultrasound, there is evidence of lucency adjacent to the thickened sclera. These features were absent in our patient making it unlikely for it to be posterior scleritis.

Varix of vortex vein ampulla (VOVA) is undue prominence of the vortex vein ampulla due to positional kinking of the extrascleral portion of the vortex vein in a middle-aged person.[5] VOVA demonstrates positional variations and is usually seen in the nasal equatorial regions. Extensive RPE disturbance in the superior quadrant and horizontal striae are not seen in association with VOVA.

The referring diagnosis in this case was a possible choroidal melanoma as the lesion was acute (not noted at the time of cataract surgery 2 years previously), symptomatic (causing floaters), raised on B-scan ultrasound and had low internal reflectivity.

As our patient had uncomplicated cataract surgery 2 years previously and there was complete spontaneous resolution of the choroidal mass within 6 weeks of initial presentation, we believe that our case represents spontaneous resolution of a limited suprachoroidal haemorrhage.

References

1 Lambert SR, Char DH. Spontaneous regression of a choroidal melanoma. *Arch Ophthalmol* 1986; **104**: 732–734.
2 Chong CA, Gregor RJ, Augsburger JJ, Montana J. Spontaneous regression of choroidal melanoma over 8 years. *Retina* 1989; **9**: 136–138.
3 Brubaker RF. Intraocular surgery and choroidal hemorrhage. *Arch Ophthalmol* 1984; **102**: 1753–1754.
4 Gressel MG, Parrish RK, Heuer DK. Delayed non-expulsive suprachoroidal hemorrhage. *Arch Ophthalmol* 1984; **102**: 1757–1760.
5 Singh AD, De Potter P, Shields CL, Shields JA. Indocyanine green angiography and ultrasonography of a varix of vortex vein. *Arch Ophthalmol* 1993; **111**: 1283–1284.

M Gupta[1], AD Singh[1], P Stavrou[1], PA Rundle[2] and IG Rennie[1]

[1]Department of Ophthalmology
Royal Hallamshire Hospital, Glossop Road
Sheffield S10 2JF, UK

[2]Birmingham and Midland Eye Hospital
City Hospital NHS Trust
Birmingham B18 7QU, UK

Correspondence: M Gupta
Tel: +44 1142711900
Fax: +44 1142713747
E-mail: mohiteye@yahoo.co.uk

Eye (2005) **19,** 601–603. doi:10.1038/sj.eye.6701543
Published online 28 May 2004

Sir,

Exudative bullous retinal detachment after peripheral blood stem cell transplantation

Haematopoietic stem cell transplantation (HSCT) including peripheral blood stem cell transplantation (PBSCT) and bone marrow transplantation (BMT) is now well recognized as the curative treatment modality for severe aplastic anaemia and haematological malignancy. However, HSCT is associated with several complications. These result from the side effects of the treatment regimen such as high-dose chemotherapy, steroid treatment, and irradiation that induced pancytopenia after transplantation, and that of the immune reaction like graft-versus-host disease (GVHD).[1] The ocular complications of the anterior segment after BMT are common while those of the posterior segment are not frequently reported. The most commonly reported posterior segment complications are either intraretinal or vitreous haemorrhages according to the literatures.[2] We present two cases of exudative bullous retinal detachment (RD) in two patients after PBSCT who develop severe and potentially vision-threatening complications. Since our two cases died shortly after the diagnosis, bullous RD may be a poor prognostic factor for survival after PBSCT.

Case reports

Case I

A 42-year-old woman was diagnosed of acute myeloid leukaemia (AML) in April 2001 with initial presentations of fever and sore throat. Prior to allogenic PBSCT, remission induction chemotherapy was administered with standard cytosine arabinoside and idarubicin. She underwent an allogenic PBSCT in September 2001. After 3 months, multiple skin eruptions over abdomen, bilateral palms, and forearms were noted. Dry mouth with lichen planus of oral mucosa was also found. Lip biopsy showed inflammatory cells infiltration in basal layer of buccal mucosa tissue. Chronic GVHD was suspected and she was started with cyclosporine, azathioprine, and prednisolone (60 mg daily for 6 days; the dose tapered therefore). She suffered a relapse of AML later in April 2002, was successfully treated with high-dose chemotherapy including cytosine arabinoside, etoposide, and mitoxantrone and experienced second remission. One palpable mass lesion over the right side of the neck was accidentally noted in November 2002 and the results of biopsy revealed blast cells. However, she developed a second relapse of AML, proved by bone marrow cytology simultaneously.

During the second relapse of AML, she complained of blurred vision of left eye and dryness of both eyes $1\frac{1}{2}$ years after allogenic PBSCT. Ophthalmologic examination showed corrected visual acuity of 6/6 in the right eye and 3/60 in the left eye. The anterior segments were unremarkable except conjunctival keratinization on both eyes. Funduscopy showed serous retinal detachment with whitish dots in the subretinal fluid involving the macula in the left eye, while the right eye was normal. Fluorescein angiography revealed exudative retinal detachment with multiple fine pinpoint fluorescein leakage at retinal pigment epithelium level in the left eye (Figure 1). Ultrasonogram of the left eye showed retinal detachment with diffuse choroid thickening. Her general condition got worse with

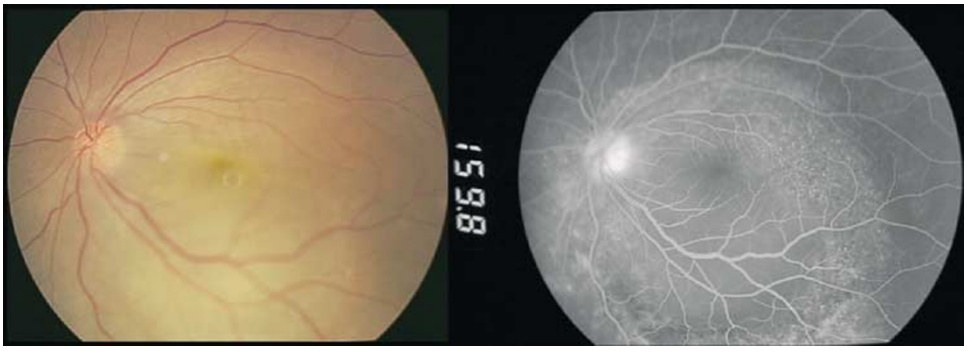

Figure 1 (Case 1) Funduscopy (left) of case 1 showed serous retinal detachment involving the macula in the left eye. Fluorescein angiography (right) revealed exudative retinal detachment with multiple fine pinpoint fluorescein leakage in the left eye. The right eye was normal.

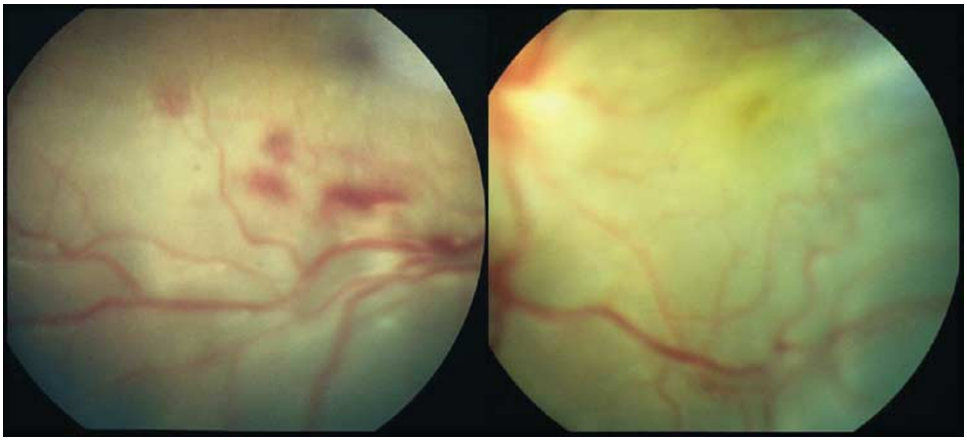

Figure 2 (Case 2) Fundus photographs of the right eye (left) and the left eye (right) showed bilateral serous bullous RD involving the macula and the inferior portion with intraretinal haemorrhage most prominent in the right eye.

neutropenic fever and respiratory distress developed a few days after the ophthalmic consultation. Subsequently, she died of heart arrest 17 months after allogenic PBSCT.

Case II

A 15-year-old girl was admitted in October 2002 with 1-week history of easily petechiae and menorrhagia. On examination, pancytopenia was noted and bone marrow biopsy disclosed hypocellularity. Severe aplastic anaemia was diagnosed. She had received preconditioning therapy including chemotherapy of cyclophosphamide (50 g/kg/day for 4 days), hydrocortisone (0.2 g for 3 days), and total body irradiation (TBI) (200 cGy twice for 2 days) prior to transplantation. She underwent an HLA-matched unrelated allogenic BMT in December 2002. Standard-dose of cyclosporine (66 mg twice on day 2) combined with methotrexate (15 mg daily on day 2) were given as prophylaxis for acute GVHD. Bone marrow cytology on day 14 disclosed graft failure. She was arranged a rescue HLA-mismatched allo-PBSCT from her younger sister on day 20 after initial BMT. She developed persisting high fever with mucositis, gingivitis, and oral ulcer on day 44. Acute GVHD was suspected and was treated with cyclosporine and dexamethasone (10 mg daily on days 44–74).

She reported blurred vision of both eyes for several days after rescue allo-PBSCT. Ophthalmologic examination showed corrected visual acuity of 20/400 in the right eye and 20/800 in the left eye by near chart. Examinations of anterior segment showed severe bilateral conjunctival chemosis and moderate posterior subcapsular cataract. Esotropia and lateral limitation of ocular movement in the left eye were noted. Funduscopy showed bilateral serous bullous RD involving macula and the inferior portion and intraretinal haemorrhage in the right eye (Figure 2). Her general condition deteriorated with progressive bilateral pneumonia and pulmonary haemorrhage. She was transferred to intensive care unit for respiratory failure and died of massive pulmonary haemorrhage on day 72 after PBSCT.

Comments

Recently, the availability of peripheral blood as additional sources of stem cells other than bone marrow has expanded the applicability of haematopoietic stem cell transplantation. Although PBSC transplant recipients engraft more quickly than BMT recipients, they develop more frequent late-onset chronic GVHD than marrow recipients.[3] Acute GVHD, tumour relapse, and the survival rate, however, were similar in patients receiving PBSCs or bone marrow.[3]

Our two cases presented with exudative bullous RD after PBSCT whose pathogenesis may be due to systemic steroid reaction similar to central serous chorioretinopathy,[4,5] occlusive choroidal angiopathy by disseminated intravascular coagulopathy[2] or leukaemic cells[6] or GVHD[4,7] *per se*.

In the first case with chronic GVHD, blurred vision in the left eye, neck malignancy, and AML relapse occurred simultaneously 14 months after PBSC. The fluorescein angiographic pictures of diffuse pinpoint leakage confined to the RD area of the left eye, and the negative study of the right eye, however, is dissimilar to the pictures of typical central serous chorioretinopathy that present with multiple pigment epithelial detachment bilaterally. Fawzi had reported a case of chronic GVHD with unilateral exudative RD.[4] The choroidal circulation may be compromised by the inflammatory cells during reaction of GVHD and leads to choroid hyperpermeability and multifocal CSC development.[8] Leukaemic infiltration, another cause of exudative RD, may also decrease the choriocapillaris blood flow and subsequent retinal detachment.[9] The definite diagnosis could be confirmed by retinal biopsy or autopsy.

In our second case, bilateral inferior bullous RD involving macula presented almost simultaneously with the obvious manifestations of acute GVHD such as mucositis, oral ulcer, and gingivitis. Our patient died of massive pulmonary haemorrhage prior to high-dose steroid treatment to control the acute GVHD. High-dose systemic corticosteroid and cyclosporine had been reported to resolve the conjunctival chemosis and CSCR that occurred in a patient with GVHD.[7] Most investigators believe that GVHD represents an immunological process mediated by immunocompetent donor T-lymphocytes reacting to genetically determined histocompatibility differences in the host.[10] The histopathologic changes in the choroid of patients with acute GVHD are peculiar histiocyte–like cells infiltration in the choriocapillary layer.[8]

These two patients died shortly after vision-threatening complications. Bullous RD may be a poor prognosis factor for survival after PBSCT. Although the bullous RD after PBSCT is unusual, ophthalmologists should keep in mind this complication and help seek the different underlying cause in the hope that early intervention might be life saving.

Acknowledgements

Proprietary interest: None.
Financial support: None.

References

1 Ng JS, Lam DS, Li CK, Chik KW, Cheng GP, Yuen PM, Dennis SC *et al*. Ocular complications of pediatric bone marrow transplantation. *Ophthalmology* 1999; **106**: 160–164.
2 Coskuncan NM, Jabs DA, Dunn JP, Haller JA, Green WR, Vogelsang GB *et al*. The eye in bone marrow transplantation. VI. Retinal complications. *Arch Ophthalmol* 1994; **122**: 372–379.
3 Ringdén O, Labopin M, Bacigalupo A, Arcese W, Schaefer UW, Willemze RA *et al*. *J Clin Oncol* 2002; **20**: 4655–4664.
4 Fawzi AA, Cunningham ET Jr. Central serous chorioretinopathy after bone marrow transplantation. *Am J Ophthalmol* 2001; **131**: 804–805.
5 Karashima K, Fujioka S, Harino S. Two cases of central serous chorioretinopathy treated with photocoagulation after bone marrow transplantation. *Retina* 2002; **22**: 651–653.
6 Kazuaki M, Satoshi K, Yoshihito H. Serous retinal detachment caused by leukemic choroidal infiltration during complete remission. *Br J Ophthalmol* 2000; **84**: 1318–1319.
7 Cheng LL, Kwok AK, Wat NM, Neoh EL, Jon HC, Lam DS *et al*. Graft-vs-host-disease-associated conjunctival chemosis and central serous chorioretinopathy after bone marrow transplant. *Am J Ophthalmol* 2002; **134**: 293–295.
8 Jabs DA, Hirst LW, Green WR, Tutschka PJ, Santos GW, Beschorner WE *et al*. The eye in bone marrow transplantation. II. Histopathology. *Arch Ophthalmol* 1983; **90**: 4–13.
9 Tewart MW, Gritter KA, Cohen G. Acute leukemia presenting as a unilateral exudative retinal detachment. *Retina* 1989; **9**: 110–114.
10 Lopez PF, Sternberg P Jr, Dabbs CK, Voglep WR, Crocker I, Kalin NS *et al*. Bone marrow transplant retinopathy. *Am J Ophthalmol* 1991; **112**: 635–646.

K-L Peng[1], S-J Chen[1], P-Y Lin[1], W-M Hsu[1], M-H Yang[2], C-H Tzeng[3] and P-M Chen[2]

[1]Department of Ophthalmology
Taipei Veterans General Hospital
201, Section 2, Shih-Pai Road
Taipei 112, Taiwan

[2]Division of Medical Oncology
Department of Medicine
Taipei Veterans General Hospital
201, Section 2, Shih-Pai Road
Taipei 112, Taiwan

[3]Division of Transfusion Medicine
Department of Medicine
Taipei Veterans General Hospital, Taipei, Taiwan

Correspondence: S-J Chen
Tel: +886228757325
Fax: +886228720959
E-mail: sjchen@vghtpe.gov.tw

Eye (2005) **19,** 603–606. doi:10.1038/sj.eye.6701547
Published online 27 August 2004

Sir,

Meningococcal group B meningitis associated with a focal chorioretinitis

Meningococcal meningitis is a medical emergency with numerous possible ophthalmic manifestations. We present an unusual story of unilateral visual loss associated with meningococcal meningitis.

Case report

An 18-year-old man presented with a 36 h history of headache, rigors, and malaise. There were signs of meningism and a widespread petechial rash. There was no focal neurological deficit. He was treated with intravenous cefotaxime (2 g q.i.d.). The cerebrospinal fluid showed a white cell count of 3680 per mm^3 (90% polymorphonuclear leucocytes) with Gram-negative diplococci seen on microscopy. There was no bacterial growth on culture of the CSF; however, polymerase chain reaction confirmed the presence of *Neisseria meningitides* group B DNA.

He made a good recovery; however, on the 10th day following admission he complained of blurring of the vision of the right eye with a 'ghost' image and pain in the right ankle. The visual acuity was 6/60 on the right and 6/9 on the left. There was a mild cellular reaction in the right anterior chamber. Fundoscopy showed a focal pale yellow, raised chorioretinal lesion on the right macula just superotemporal to the fovea (Figure 1a). There was no clinical effusion in the right ankle and the range of movement appeared normal.

The vision in the right eye improved gradually with no further treatment. His Snellen's acuity on the right was 6/18- after 2 weeks and continued to improve to 6/9 at 6 months. He had mild metamorphopsia on Amsler's grid testing with a small positive scotoma inferonasal to fixation. At the latest review at 18 months, he was seeing 6/5 with a pinhole on the right eye and the scotoma was unchanged. The fundus shows distortion of the foveal reflex and a small, flat, poorly defined parafoveal chorioretinal pigmentary disturbance (Figure 1b).

Comment

Ophthalmic manifestations of meningococcal meningitis include ocular motility abnormalities due to third, fourth, and sixth nerve palsies and nystagmus. Raised intracranial pressure associated with meningitis may cause papilloedema and secondary optic atrophy. Optic neuritis and papillitis are potential causes of visual loss in patients with meningitis.[1]

Meningococcal septicaemia may result in vascular obstruction secondary to disseminated intravascular coagulation with ocular features including retinal