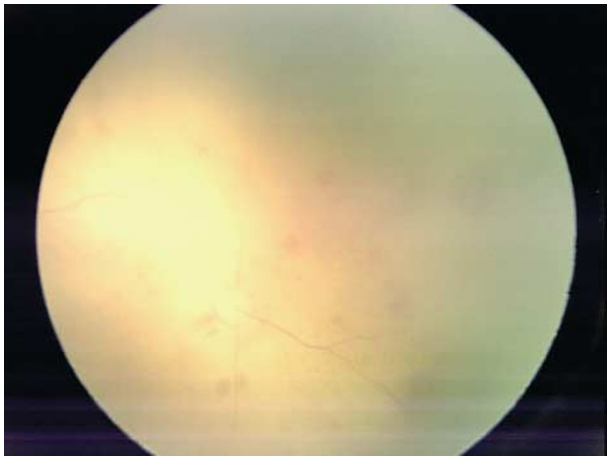

Figure 2 RetCam fundus photograph taken 70 minutes after the initial examination. There are multiple flame-shaped, 'dot' or 'blot' retinal haemorrhages that indicate bleeding at different depths within the retina.

imaging, showed widespread retinal haemorrhages in the left eye only, the right eye being normal. Repeat RetCam examination, 70 min after the first examination, documented these findings (Figure 2). The examination was atraumatic and had been carried out with the assistance of a neonatal nurse in full view of the infant's mother. The child was being continuously monitored before, during, and after examination as it required low flow nasal cannula oxygen and the records showed no evidence of distress. Cerebral ultrasound was normal as were the blood parameters. There was no bleeding from any other site and the clotting screen was normal (prothrombin time 9.2 s, INR 1.0, APTT 36 s, fibrinogen 3.3).

After 2 weeks, RetCam screening again documented no haemorrhage in either eye. When the ophthalmologist screened the baby 10 min later, without using a speculum, two haemorrhages were found in the left eye only. Assuming that these haemorrhages had been missed due to shadowing on one of the images, repeat RetCam documentation was undertaken and showed extensive haemorrhages confined to the left retina.

This incident was reported to the manufacturer, the UK distributor, and the Medical Devices Agency. No previous incidents have been recorded.

Comment

This premature child had been progressing well under neonatal special care. The infant had no indicators of a bleeding diathesis and no other signs of ocular trauma — direct or indirect. There were no known patient factors that could explain the unique observations in this case. It is likely that the haemorrhages in this baby arose from ruptured immature retinal vessels, which without smooth muscle, collagen, or elastin layers, are thought to be more susceptible to rupture in the presence of hypoxia and immature central control of circulatory autoregulation.[2] It would appear that the pressure either of the speculum used to separate the lids or of the RetCam on the globe may have caused retinal bleeding. Unintentional heavy pressure applied to the eye may have been a factor, but retinal haemorrhages caused by blunt trauma to the eye without signs of other damage is unusual and in particular there were no subconjunctival haemorrhages, which would have been expected if this had been the case. RetCam examination is very gentle relative to scleral indentation. This case emphasises how fragile the infant retinal vascular system is and how even minor pressure can cause haemorrhages in infant retinas.

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Sir,

High-altitude flight retinopathy

There have been very few reports of ocular complications from commercial high-altitude flights, despite the ever-increasing usage of long distance flying in the modern world. Reported ocular complications include anterior

ischaemic optic neuropathy,[1] worsening of diabetic macular oedema[2] and intraocular pressure problems related to gas bubbles in the vitreous cavity post-vitreoretinal surgery. We would like to report a case of haemorrhagic retinopathy associated with a long haul commercial jet flight.

Case report

A 24-year-old white female presented with a 2-day history of bilateral blurred vision, which started on arriving home from Thailand, after a long-haul commercial jet flight lasting 11 h. She was in good general health, and only had a mild intermittent headache. There was no family history of a bleeding tendency. Of note, she had participated in a no-decompression sea dive to a depth of 18 m for 1 h, 6 days before. She had also taken 2 Paludrine (proguanil hydrochloride) tablets for antimalaria prophylaxis 4 days previously. At presentation, her visual acuity was 6/36 in the right eye, 6/60 in the left, with no improvement with refraction. Anterior segment and intraocular pressure examination was normal. There was a left relative afferent pupillary defect. Funduscopy revealed bilateral posterior pole retinal haemorrhages with slightly dilated, tortuous, segmented retinal veins with mild arteriovenous crossing changes (Figure 1). Humphrey visual field analysis showed bilateral central scotomas. A fluorescein angiogram (Figure 2) demonstrated blocked fluorescence secondary to the retinal haemorrhages. There was no significant perifoveal ischaemia or vascular leakage. Her blood pressure was 100/75 mmHg and urinalysis was normal. A full blood count and coagulation screen were normal, and the ESR was 15 mm/h. Plasma viscosity was mildly elevated at 1.50 (range 1.19–1.43). Blood films for malaria parasites were clear. Other investigations included urea & electrolytes, glucose, Ca^{2+}, lipids, c-reactive protein, immunoglobulin electrophoresis, autoantibody screen, serum homocysteine and methionine levels, protein C, protein S, anti-thrombin III, factor V Leiden mutation, activated protein C resistance, and lupus anticoagulant. These were all normal.

Vision progressively improved spontaneously and 6 weeks later, her vision measured 6/6 in both eyes. The retinal haemorrhages had also cleared and the pupil reactions and retinal vasculature had returned to normal. The patient felt her vision was now completely normal.

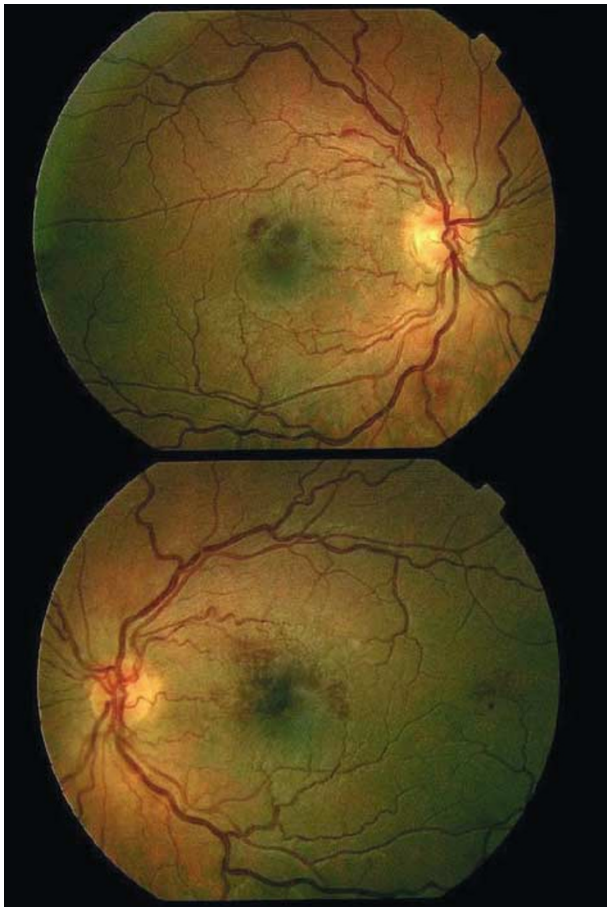

Figure 1 Fundus photographs showing bilateral posterior pole retinal haemorrhages.

Comment

There are numerous causes of retinal haemorrhages. Some of the more common ones include diabetic retinopathy, hypertensive retinopathy, retinal vein occlusions and vasculitis. Haemorrhagic retinopathy can also be due to more unusual causes like high altitudes, Valsalva manoeuver, Purtscher's retinopathy, bleeding tendencies, and Terson's syndrome.

Our patient had taken a commercial long-haul flight prior to onset of her symptoms. With commercial aircraft, cabin pressure is not maintained at sea level pressure during most of the flight.[3] Cabin pressure can be as high as 9000 f altitude equivalent, especially in the newer aircraft designs. This represents a fall in inspired PO_2 of approximately 30%, leading to a subsequent fall in alveolar oxygen tension and arterial oxygen tension. One article looking at a series of patients with benign idiopathic haemorrhagic retinopathy, had one case where a patient developed reduced vision and retinal haemorrhages after flying in a pressurized aircraft.[4] The pathology, however, was restricted to one eye. This may still represent altitude retinopathy secondary to a commercial flight.[5]

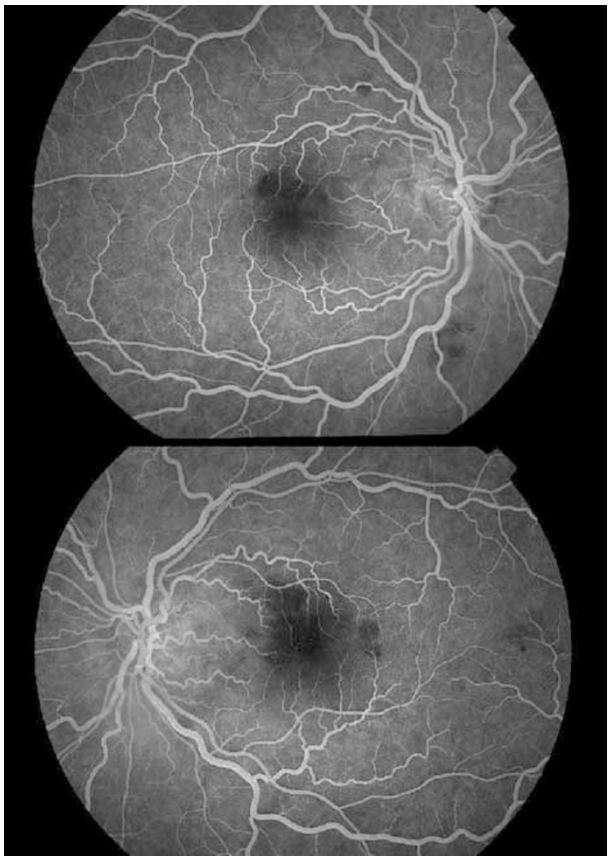

Figure 2 Fluorescein angiograms demonstrating blocked fluoresence secondary to the retinal haemorrhages.

Altitude sickness usually occurs at altitudes over 8000 f (2440 m).[6] The acute forms of high-altitude illness include acute mountain sickness, high-altitude pulmonary oedema, high-altitude cerebral oedema and high-altitude retinal haemorrhage. High-altitude retinopathy (HAR) can occur without altitude sickness (or acute mountain sickness).[7] The signs of HAR include dilated retinal vessels, diffuse or punctate retinal/preretinal haemorrhages, vitreous haemorrhage, papillary haemorrhage, peripapillary hyperemia, and papilloedema. There have been different suggestions regarding the pathogenesis of HAR, including dilatation of retinal vessels in response to hypoxia, increased venous pressure transmitted by coughing or straining, and changes in intraocular pressure during physical exertion.[8] There is also a high incidence of concomitant headache.

Our patient also had participated in a SCUBA dive, about a week before her flight. There have been reported cases of decompression sickness in commercial airliners where the flights were preceded by SCUBA diving.[9] Recommendations for a safe time interval between diving and flying vary in different diving manuals. The Manual of Civil Aviation Medicine prohibits flying within 24 h after SCUBA diving.[10] During SCUBA diving, hyperbaric conditions lead to a higher dissolved nitrogen gas content in blood and body tissues. Upon returning to normobaric conditions, nitrogen gas comes out of the blood and tissues at a certain rate. If the rate is too high, decompression sickness can result with nitrogen gas bubbles occurring in various body tissues including the central nervous system.[11] Intravascular nitrogen gas bubble formation can result in activation of the coagulation system, which may lead to increased platelet microaggregation, rouleaux formation, alterations in haematocrit and white cell rigidity, and this leads to hyperviscosity of the blood.[12] Our patient having undergone deep sea diving, although occurring the week before, may still have had relatively compromised blood values, and thus be more susceptible to the relative hypoxia over a long haul flight. This may have then contributed to her suffering from a form of HAR.

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Sir,

Massive subretinal haemorrhage secondary to age-related macular degeneration in a patient with idiopathic thrombocytopenic purpura

Idiopathic thrombocytopenic purpura (ITP) is a condition characterized by persistently low platelet counts, resulting from accelerated platelet destruction mediated by antiplatelet antibody. As ocular complication with ITP, hemianopia caused by intracranial haemorrhage, intraretinal haemorrhage caused by anaemia are reported.[1,2] An association between age-related macular degeneration (AMD) and ITP has not been reported to our knowledge. We describe a rare association between severe subretinal haemorrhage and ITP. Our case demonstrates that close ophthalmologic examination is very important in ITP.

Case report

A 73-year-old woman experienced a sudden loss of vision in the left eye and are referred to our macular clinic for a detailed examination. She had been followed up for exudative AMD and received cataract surgery with intraocular lens implantation at the age of 66 years. During the referring physician's recent observation, the visual acuity of the left eye was 0.1–0.3. Ophthalmoscopic examination (Figure 1a), fluorescein angiography, (Figure 1b) and indocyanine green angiography (Figure 1c) reavealed a persistent occult choroidal neovascular membrane not suitable for treatment. She also had a systemic previous history of diabetes mellitus, hypertension, and ITP. The platelet count was $7000/mm^3$. On our ophthalmologic examination, the visual acuity became 30 cm hand movements. The dilated fundus examination of the left eye revealed 7×10 disc diameter (DD) submacular haemorrhage (Figure 1d). Although fluorescein angiography failed to detect the cause of haemorrhage, indocyanine green angiography (ICG) disclosed a hyperfluorescent lesion indicative of choroidal neovascularization (CNV). Observation was recommended. After 1 month, massive haemorrhage developed so rapidly that it reached the posterior capsule of the lens as disclosed by B-mode ultrasonography (Figure 1e) and slit-lamp biomicroscopy (Figure 1f). The visual acuity dropped to light perception. Within the following week, the patient revisited us claiming of severe headache. The visual acuity was no light perception. The ocular pressure of the left eye was 63 mmHg. Slit-lamp examination showed a narrow anterior chamber, and dilated fundus examination and B-mode ultrasonography revealed total retinal detachment (Figure 1g).

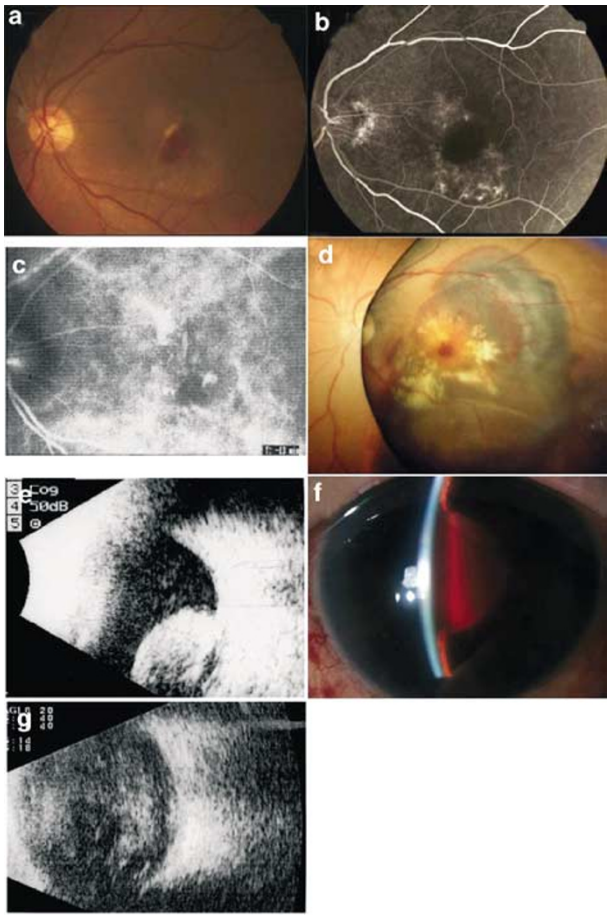

Figure 1 Dilated fundus examination (a) revealed subretinal haemorrhage in the macular area at our recent observation. Fluorescein angiography (b) and indocyanine green angiography (c) disclosed a persistent occult choroidal membrane not suitable for treatment. At initial presentation to our clinic, the dilated fundus examination disclosed a massive subretinal haemorrhage (d). After 1 month, massive subretinal haemorrhage was seen as high reflex on B-mode ultrasonography (e) and slit-lamp examination showed massive subretinal haemorrhage reaching the posterior capsule of the lens (f). At 1 week thereafter, the subretinal haemorrhage reached the posterior capsule of the lens as shown by B-mode ultrasonography (g).