clothing. He also described a bearded doctor. These figures were static, smoothly approached him and were almost constantly appearing except when he abolished them. Each apparition lasted 10 s to 1 min. The hallucinations subsided after 8 days.

Ultrasound demonstrated reversed left ophthalmic artery flow and some anomalous arterial flow within his left postero-superior orbit. Intracranial MR angiography and MR orbits excluded an arteriovenous malformation but did show increased vascularity within this region, consistent with collateral vasculature between the extra- and intra-cranial circulations. MRI brain confirmed an old left hemisphere subcortical infarct and demonstrated no occipital abnormalities.

Comment

Our patient's risk factors for NA-AION included vascular comorbidity, hypermetropic discs, and a state of probable low flow within the left collateralized cerebral circulation.

Monocular visual hallucinations are very infrequent. A retrospective study of phantom eye syndrome found 7 of 112 patients experienced complex visual hallucinations after unilateral enucleation, although it is unclear whether these phenomena were subjectively monocular.[2] Uhthoff (1899) probably provided the original description,[3] highlighted by the recent case report in *Eye*.[1]

CBS varies in semiology. Single hallucinations may last from few seconds to hours. Subjects tend to see people, animals, buildings, and scenery that can be static or moving. When moving, the hallucinations tend to do so en masse.[4] Most subjects have reduced vision secondary to ocular disease. Some diagnostic criteria require the absence of central visual or cortical lesions but most require hallucinations with intact sensorium and insight.[4]

Certain pathophysiological mechanisms have been proposed in order to explain CBS. The perceptual release theory[5] postulates the disinhibition of higher cortical activity resulting from a reduction in the afferent stimulus. This releases previously suppressed subconscious perceptual imagery in the form of hallucinations. The phantom vision theory is related to the deafferentation model[6,7] and postulates spontaneous higher visual cortical discharges in response to the loss of visual input.[8] It is unclear how these theories apply to monocular complex hallucinations since a cortically generated process should be represented bilaterally, unless there is a mechanism which suppresses the monocular representation for the normal eye.

Hallucination abolition on eye closure is sometimes explained by secondary normalization of sensory inputs. In phantom limb syndromes, for example, the painful symptoms of an amputated arm may diminish when a mirror is placed such that the patient views the existing arm on the other side.[9] By covering his affected eye, which had some residual vision, our patient theoretically also normalized his sensory input. This could explain why his hallucinations vanished even with his affected eye open.

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Sir,

Occlusive retinal vasculitis in a patient with ankylosing spondylitis

Ankylosing spondylitis is a seronegative arthropathy which typically involves sacroiliac joints. We described

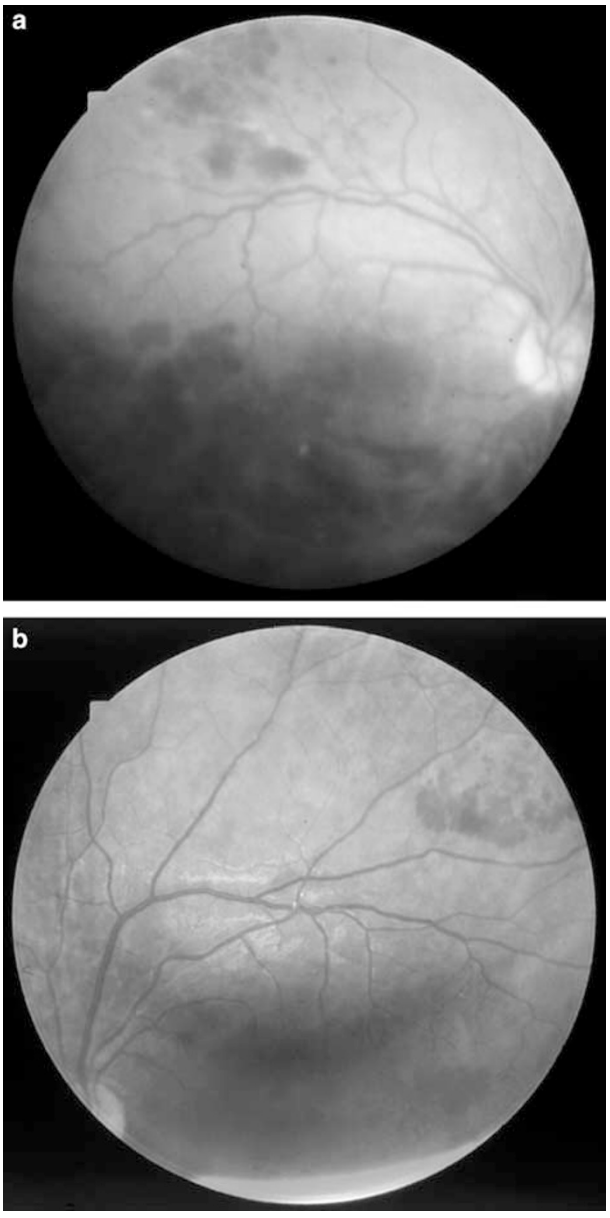

Figure 1 (a) Fundus photograph showing sheathed vessels and (b) retinal haemorrhages in both eyes.

a patient of ankylosing spondylitis with bilateral occlusive retinal vasculitis.

A 29-year-old man presented with 1 month history of decreased vision in both eyes. Visual acuity was 3/10 in the right eye and 10/10 in the left eye. There was 2+ vitreous cell in each eye. Fundus examination revealed branch retinal vein occlusion, sheathed vessels and retinal haemorrhages at the superior and inferior temporal region in both eyes with macular oedema in the right eye (Figure 1a, b). Fundus fluorescein angiography showed retinal vasculitis in both eyes (Figure 2a, b). The review of his medical history revealed that he had ankylosing spondylitis.

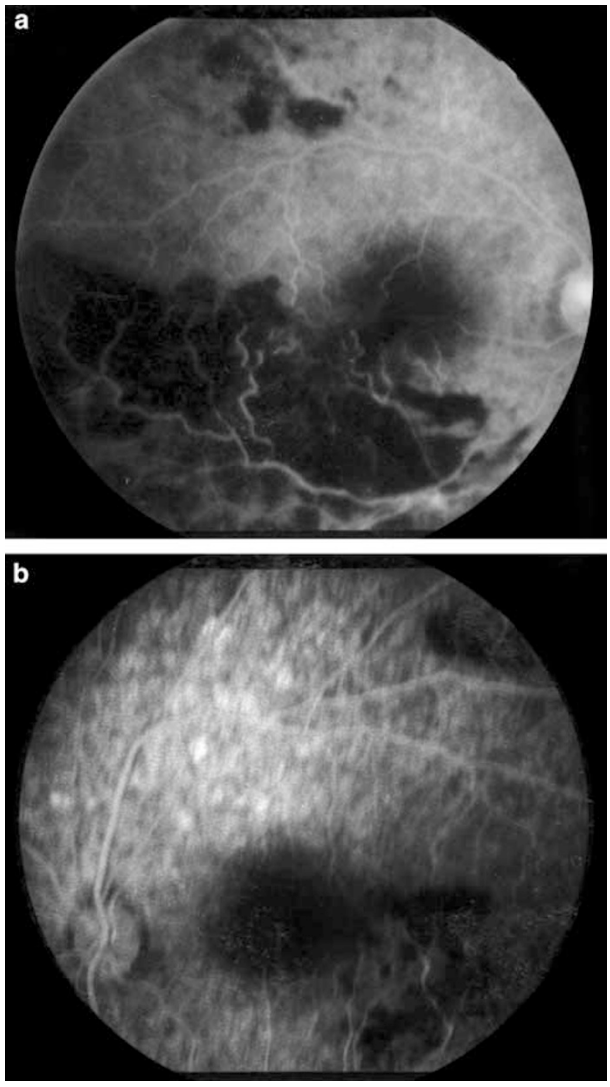

Figure 2 (a) Fluorescein angiogram showing retinal vasculitis and (b) hypofluorescence due to retinal haemorrhages in both eyes.

Laboratory investigations including complete blood counting, prothrombine time and active thromboplastine time, protein C and S, antithrombine III, active protein C resistance, factor V leiden existence, anticardiolipin antibody M and G, homocystein, vitamin B_{12} and folic acite levels, ANA (antinuclear antibody), anti-double-stranded DNA (anti-Ds DNA) tests were normal. The patient had no oral afts and pathergy skin reaction was negative. HLA class I antigen testing revealed HLA-B27 positivity, but HLA-B5 negativity. Sacroiliac joint graphy showed periarticular sclerozis and irregular loss of the joint space. According to the clinical and laboratory examinations he did not have tuberculosis, sarcoidosis, or inflammatory bowel disease. Cardiovascular and neurological examination revealed no pathology. According to immunology and dermatology consultations, the

diagnosis of ankylosing spondylitis had comeout right. Behçet's disease was not considered.

The patient was placed on a regimen of intravenous methylprednisolone (1 g/day) for 3 days because of the existing inflammatory findings. Additionally, both eyes received retrobulber methylprednisolone acetate injection. After 3 days, the treatment was continued with a maintenance dose of oral prednizolone (20 mg/day).

Retinal vein occlusion is associated in older ages with common systemic vascular disorders such as hypertension, arteriosclerosis, and diabetes mellitus.[1] Coagulation deficiency, hyperviscosity syndromes, chronic leukemia, mitral valve prolapse, homocysteinemia, Behçet's disease, and systemic inflammatory diseases may also cause retinal vein occlusion in younger ages.[1–4] However, our case did not demonstrate any of them.

Ankylosing spondylitis is common in 20–40-year-old males and HLA-B27 antigen is present in about 96% of the patients. The joint involvement is asymmetric and periferic. Ocular involvement occurs in 20–30% of patients with ankylosing spondylitis. The ocular findings include recurrent, nongranulomatous iridocyclitis, which rarely affects vision.[5] Posterior inflammatory manifestations such as severe vitritis, papillitis, and retinal vasculitis have been described in patients with ankylosing spondylitis.[6]

In summary, we report an unusual case of ankylosing spondylitis presenting with bilateral occlusive retinal vasculitis.

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Sir,

Reply to endophthalmitis following 25-gauge vitrectomy

We read the article by Taylor and Aylward[1] with great interest. We congratulate the authors for publishing the first reported case of successful management of 25-gauge transconjunctival sutureless vitrectomy (TSV).

We agree with the authors that smaller incision, reduced operative time, lack of suture material and limited manipulation may reduce the risk of endophthalmitis with TSV. However, reduced flow rate (6 times) may facilitate bacterial foothold inside the eye during surgery.[2] Alignment of the conjunctival and scleral incisions and unsutured sclerotomies may increase the risk of postoperative hypotony allowing influx of microbes. This makes the eye more susceptible to postoperative endophthalmitis in comparison to the watertight wounds of a sutured 20-gauge sclerotomy.

To overcome this potential complication of postoperative hypotony and resultant potential increase in bacterial influx, we suggest displacing the conjunctiva superiorly while introducing the 25-gauge trocar and cannula at the initiation of the surgery. At the end of surgery, when the trocar and cannula are removed, the conjunctiva moves back inferiorly covering the sclerotomy. This misalignment of conjunctival and scleral wound prevents a continuous tract for fluid egress, hypotony and bacterial influx.[3]

We believe that at the completion of surgery, removing the superior cannulas while maintaining infusion creates high pressure within the vitreous cavity and promotes the vitreous to occlude the sclerotomy. This can reduce the incidence of postoperative hypotony. We also suggest suturing the sclerotomy with a single 10-0 vicryl suture when integrity of the wound is doubtful.

We congratulate the authors for reporting this uncommon but grave complication of 25-gauge TSV.

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