causative organisms were *Haemophilus influenza* and *S. sanguis*[8] (6/13). In these cases, no direct association between the oral flora and endophthalmitis can be inferred.

The British Dental Association has advice sheets recommending the use of eye protection for both patient and practitioner during any invasive procedure. This is used primarily to protect against splatter and not specifically against penetrating injury, although the latter would almost certainly be avoided.

An increasing number of dentists are providing their patients with eye protection during routine procedures.

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No proprietary interests

Eye (2006) **20,** 981–982. doi:10.1038/sj.eye.6702096; published online 23 September 2005

Sir,

Fatal thrombotic thrombocytopenia purpura presenting with choroidal vasculopathy and serous retinal detachment

We read with interest the case reported by Ong *et al*[1] of Purtscher's retinal microangiopathy from thrombotic thrombocytopaenic purpura (TTP). Although this is a rare manifestation of an uncommon disorder, we too have recently seen a case which presented without systemic symptoms where the ocular findings were of a choroidal vasculopathy, which turned out to be fatal despite haematological intervention.

Case report

A 32-year-old Afro-Caribbean female presented with a 24 h history of left blurring of vision associated with a mild headache. Previous ophthalmic history included an undiagnosed episode of unilateral visual disturbance with systemic weakness 13 years previously. She had continued to suffer occasional visual disturbance lasting a few hours, but no weakness. She had sickle cell trait (HbS) and used the combined oral contraceptive pill.

At presentation she was systemically well with only a slight headache. Her visual acuity was 6/4 OD and 6/9 OS and colour vision and visual fields were intact. The adnexae and anterior segments were unremarkable. Fundoscopy revealed pale raised choroidal lesions in both eyes, with overlying subretinal serous fluid (Figure 1). FFA revealed patchy filling defects in the choriocapiliaris of both eyes with hotspots of increasing hyperfluorescence representing leakage in areas overlying the choroidal lesions (Figure 2).

Chest X-radiograph was normal but the blood count showed thrombocytopenia, so a haematological opinion was sought. After 6 h she developed nausea and vomiting and the following day fundoscopy of the left eye revealed a haemorrhagic choroidal detachment. Over the next 24 h she developed fluctuating visual disturbances, pyrexia, jaundice, dark urine, diarrhoea, vaginal bleeding, and malaise. Despite emergency transfer to a specialist haematological unit and subsequent plasma exchange she died of a respiratory arrest on the second day of treatment.

Comment

We describe a case of a young woman who died within a week of presenting with mild unilateral blurred vision, the ocular findings preceding systemic onset of TTP. This case is noteworthy for two reasons. First, the

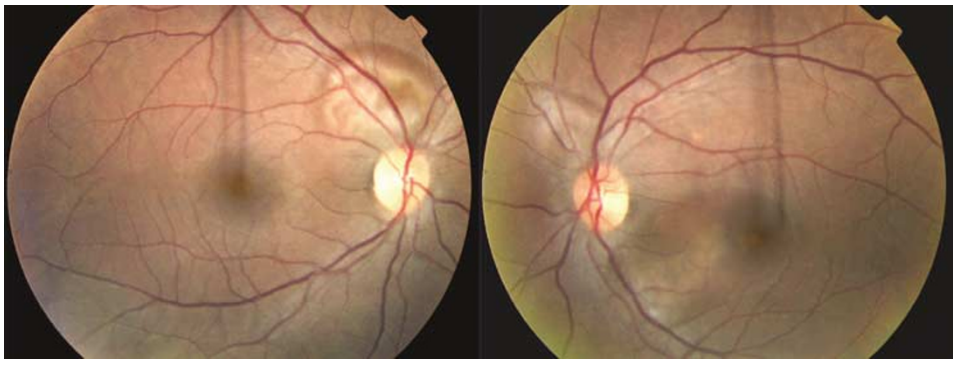

Figure 1 Colour fundus photographs showing patchy pale raised subretinal lesions with overlying subretinal fluid.

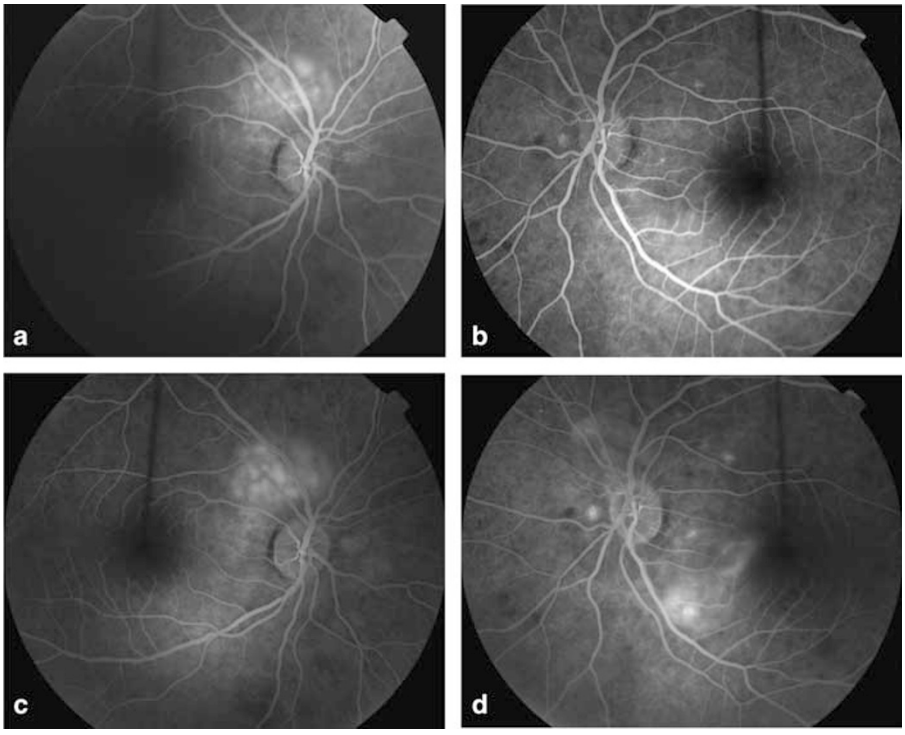

Figure 2 FFA of both eyes show patchy choroidal filling defects (a and b) and areas of hyperfluorescence overlying corresponding areas, which increase in intensity representing leakage into the subretinal space (c and d).

ophthalmologist can be the first contact with medical services, before systemic symptoms manifest. The prognosis is poor if not treated promptly and 8% of all TTP patients have ocular signs and symptoms.[2] Second, this is the first report to our knowledge where the presenting features are of choroidal vasculopathy, rather than retinal bleeding.[1–3] Although this feature has been described before, previous cases were reported to occur after systemic signs of TTP were established.[4–6]

Thrombotic thrombocytopenia purpura has an incidence of 3.7 per million, occurring most frequently in young female adults. In many cases remission is achieved and so the prevalence is increasing. TTP is caused by platelet thrombi principally in the renal and cerebral circulations and aggregations can also occur in the choroidal circulation with overlying serous fluid in the subretinal space.[4] The cause is thought to be from excessive platelet aggregation mediated by ultra-large von Willebrand Factor (ULVWF) multimers caused by a failure of the proteolytic action of metalloproteinases, such as ADAMTS 13, in affected patients.[7]

Unexplained patchy serous retinal detachment should prompt investigation for TTP with a full blood count

and, if appropriate, generate a prompt referral to haematologists—this may be life saving.

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Eye (2006) **20,** 982–984. doi:10.1038/sj.eye.6702100; published online 30 September 2005