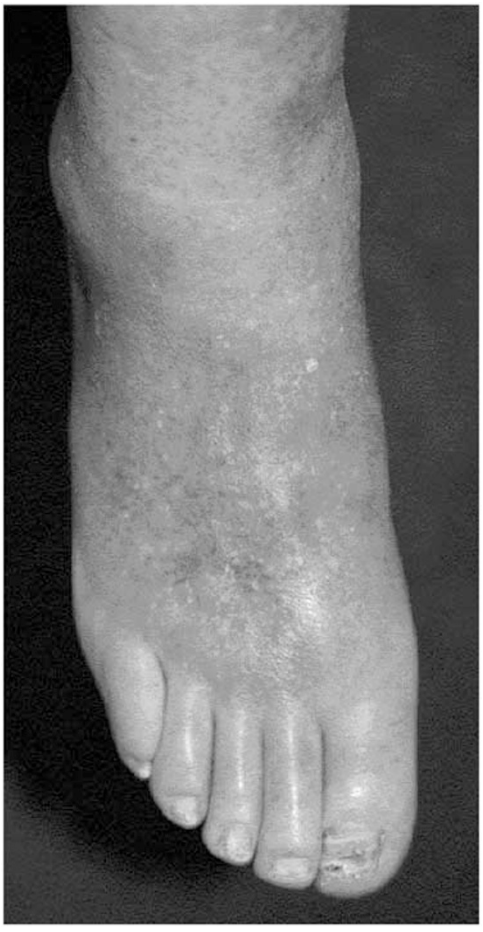

Figure 1 Photograph of right foot before intertarsal abscess debridement

collected grew *A. actinomycetemcomitans*. The abscess healed well following debridement.

Comment

The HACEK group of organisms is an unusual cause of infective endocarditis, responsible for 3% of cases.[1] Of the five reported cases of endogenous endophthalmitis due to *A. actinomycetemcomitans*, four had pre-existing heart abnormalities, three had permanent pacemakers, and two had periodontal disease.[2–6]

Our case describes this unusual cause of metastatic endophthalmitis, secondary to endocarditis, with concomitant foot abscess. There are no previous reports of macroembolic, metastatic, abscesses requiring debridement and growing positive cultures for *A. actinomycetemcomitans*. Poor glycaemic control and immunosuppression may have contributed to these novel manifestations.

References

1 Das M, Badley AD, Cockerill FR, Steckelberg JM, Wilson WR. Infective endocarditis caused by HACEK microorganisms. *Annu Rev Med* 1997; **48**: 25–33.
2 Lass JH, Varley MP, Frank KE, Speck WT. *Actinobacillus actinomycetemcomitans* endophthalmitis with subacute endocarditis. *Ann Ophthalmol* 1984; **16**(1): 54–61.
3 Donzis PB, Rapazzo JA. Endogenous *Actinobacillus actinomycetemcomitans* endophthalmitis. *Ann Ophthalmol* 1984; **16**: 858–860.
4 Ishak MA, Zablit KV, Dumas J. Endogenous endophthalmitis caused by *Actinobacillus actinomycetemcomitans*. *Can J Ophthalmol* 1986; **21**: 284–286.
5 Sullivan P, Clark WL, Kaiser P. Bilateral endogenous endophthalmitis caused by HACEK microorganisms. *Am J Ophthalmol* 2002; **133**(1): 144–145.
6 Binder MI, Chua J, Kaiser PK, Mehta N, Isada CM. *Actinobacillus actinomycetemcomitans* endogenous endophthalmitis: report of two cases and review of the literature. *Scand J Infect Dis* 2003; **35**(2): 133–136.

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Sir,

Purtscher's like retinopathy as the presenting feature of acute alcoholic pancreatitis

Purtscher's like retinopathy is a rare complication arising in patients with an established diagnosis of pancreatitis. We report a patient, with undiagnosed pancreatitis, presenting with visual loss due to Purtscher's like retinopathy.

Case report

A 31-year-old man with a history of alcohol abuse, experienced bilateral visual loss, following 24 h of epigastric pain and vomiting, precipitated by binge drinking. On presentation, 4 days later, he had a right

relative afferent pupil defect and visual acuity (VA) of counting fingers right and 6/24 left. Retinal examination revealed bilateral posterior pole cotton wool spots (CWS) and right retinal and preretinal haemorrhages, consistent with Purtscher's like retinopathy (Figure 1). Blood tests revealed a raised C-reactive protein (CRP) at 20 mg/l, raised alanine transaminase at 118 IU/l (<35), a reduced platelet count at 73×10^9/l (140–400) and a normal serum amylase. Acute alcoholic pancreatitis was diagnosed.

Fluorescein angiography revealed masking from retinal haemorrhage and CWS, nonperfusion of small arterioles in the macular region, perivenous staining and venous dilatation and leakage (Figure 2).

After 7 weeks, his VA was 6/9 right and 6/6 left, with resolution of most retinal signs.

Comment

This patient's pancreatitis may have remained undiagnosed had he not developed Purtscher's like retinopathy. Pancreatitis was suspected based on the ophthalmic findings and the diagnosis was supported by the history, epigastric tenderness, raised CRP, and low platelets.[1] His serum amylase was normal because the test was performed 5 days after the onset of symptoms and levels can normalise within 3 days.[2]

As in this case, pancreatitis complicated by Purtscher's like retinopathy is usually alcohol related,[3] with few reported exceptions.[4,5] One may hypothesise that alcohol could potentiate the complement activated, leukoembolisation of retinal arterioles, thought to be responsible for the clinical picture of Purtscher's like retinopathy.[6]

This patient had mild pancreatitis, supporting the observations of others, that the development of Purtscher's like retinopathy is unrelated to the severity of pancreatitis.[7]

We believe this to be the first report of pancreatitis being diagnosed as a direct result of a patients presentation to an ophthalmologist with Purtscher's like retinopathy.

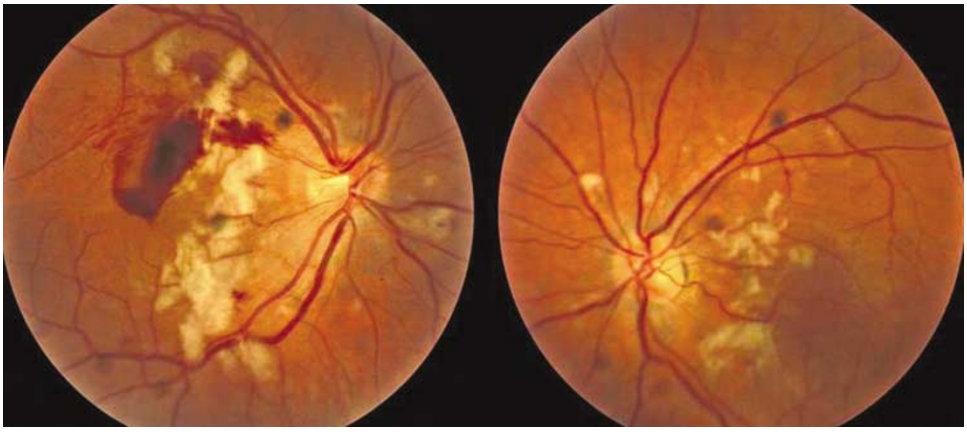

Figure 1 Right and left fundi at presentation.

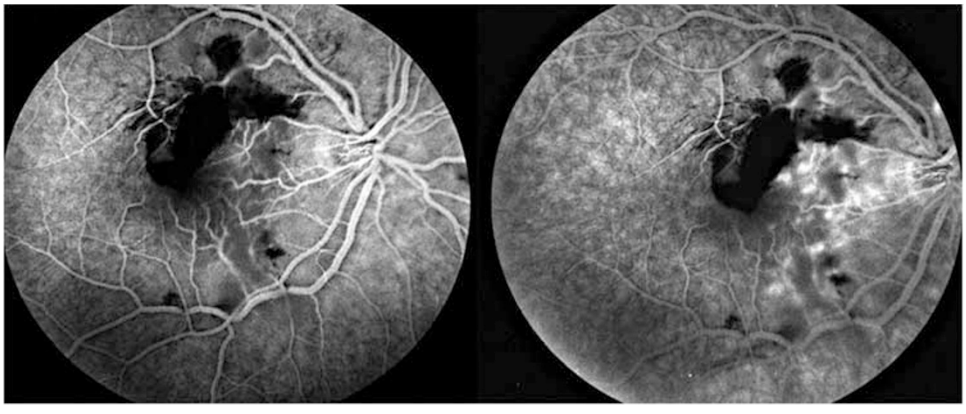

Figure 2 Early and late fluorescein angiograms of right eye at presentation.

References

1 Vaquero E, Casellas F, Bisbe V, Puig-Divi V, Bermejo B, Guarner L *et al*. Thrombocytopenia onset in acute episodes of pancreatitis. *Med Clin* 1995; **105**: 334–337.
2 Ellis H, Calne R, Watson C. *Lecture notes on General Surgery*, 9th ed. Blackwell: Oxford, 1998, p 267.
3 Sanders RJ, Brown GC, Brown A, Gerner EW. Purtscher's retinopathy preceding acute pancreatitis. *Ann Ophthalmol* 1992; **24**: 19–21.
4 Nunez L, Cubiella J, Moreno C, Diez MS, Sanchez E, Vega M. Purtscher's retinopathy: a rare complication of acute non-alcoholic pancreatitis. *Gastoenterol Hepatol* 2003; **26**: 541–544.
5 Bui SK, O'Brien JM, Cunningham ET. Purtscher retinopathy following drug-induced pancreatitis in an HIV-positive patient. *Retina* 2001; **21**: 542–545.
6 Jacob HS, Goldstein IM, Shapiro I, Craddock PR, Hammerschmidt DE, Weissmann G. Sudden blindness in acute pancreatitis: possible role of complement-induced retinal leukoembolisation. *Arch Intern Med* 1981; **141**: 134–136.
7 Flores MSD, Rodriguez IN, Bernal IL, Rubio MA, Ledesma AG, Acevedo JMP *et al*. Retinopathy as a systemic complication of acute pancreatitis. *Am J Gastroenterol* 1995; **90**: 321–324.

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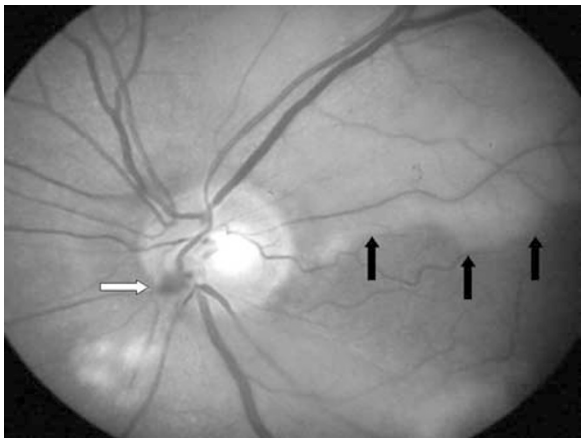

Figure 1 Demarcation line between ischaemic oedematous retina (BRAO) and healthy retina (black arrows). Prepapillary loop out of focus (white arrow).

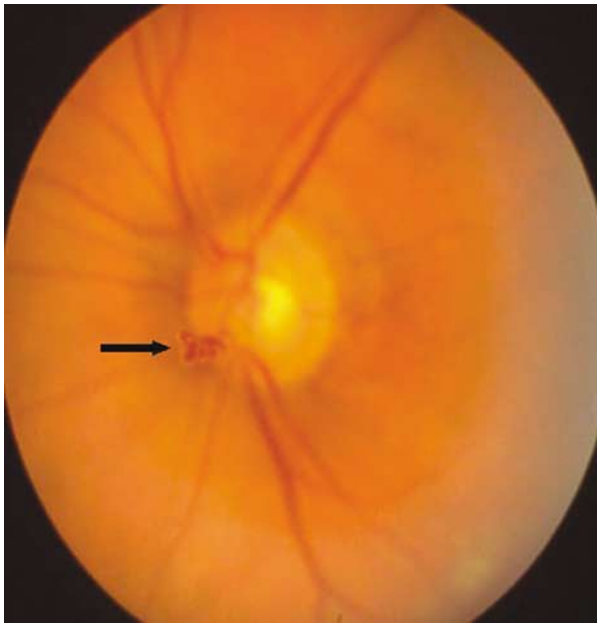

Figure 2 Prepapillary loop extending from left optic nerve (black arrow).

Sir,

Branch retinal artery obstruction in a patient with a prepapillary loop and carotid artery stenosis

Prepapillary loops (PPLs) are rare. They are usually unilateral, congenital, and uncommonly associated with complications.[1] We describe a patient with carotid artery stenosis and a congenital PPL, who developed a branch retinal artery obstruction (BRAO).

A 57-year-old male smoker presented with a 1-week history of a left inferior scotoma associated with photopsia. There was no relevant past ocular or medical history.

On examination, visual acuities were 6/6 OD, 6/4 OS, and a left afferent pupillary defect was present. On fundus examination, a PPL and findings compatible with a BRAO were detected (Figures 1 and 2). An inferior visual field defect was evident on confrontational testing. No emboli were visible. His blood pressure was 160/92 mmHg and a left carotid bruit was audible. Carotid doppler ultrasound scanning subsequently revealed a 50% stenosis of the right common carotid artery, and a