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Accepted for poster presentation at the RCOphth congress 2010.

Eye (2010) **24,** 1409–1410; doi:10.1038/eye.2010.63; published online 14 May 2010

Sir,

Retinal vasculitis as an early sign of bacterial post-operative endophthalmitis

Acute post-operative bacterial endophthalmitis is one of the most devastating complications of cataract surgery. Typical symptoms and signs include pain, reduced visual acuity, pan-uveitis, and hypopyon formation. This case and the images illustrate a rarely seen presentation of post-operative endophthalmitis characterised by vascular sheathing and retinal infiltrates.

Case report

A 72-year-old woman underwent right phacoemulsification with in-the-bag lens implantation. Surgery was uncomplicated; she received subconjunctival gentamicin and dexamethasone at the end of the procedure and G Tobradex QDS post-operatively. The patient was in reasonable general health with a history of hypertension but no symptoms suggestive of connective tissue disease.

Four days later the patient noticed increased floaters in her right eye. Two days after this she attended the clinic as her vision had decreased. Visual acuity was 6/24 correcting to $6/12^{-3}$. There was mild limbal injection and a moderate (+3) anterior uveitis with no hypopyon. There was moderate vitreous (+2) activity with 'snowballs' inferiorly. The optic disc appeared swollen and there were multiple intra-retinal haemorrhages, many of which had a central white core and resembled Roth's spots (Figure 1). Vascular sheathing was also noted.

Fluorescein angiography revealed disc leakage with associated staining and late leakage of the vessel walls (Figure 1). The left eye appeared normal.

A diagnosis of post-operative endophthalmitis was considered most likely. Following vitreous and aqueous biopsy, the patient received 1 mg intravitreal vancomycin, 0.4 mg amikacin, and 4 mg subconjunctival dexamethasone. She was commenced on topical dexamethasone and cycloplegics, 40 mg prednisolone, and ciprofloxacin orally. The following day, the appearance conformed to a more typical post-operative endophthalmitis with marked vitritis. Vitreous biopsy subsequently revealed Gram-positive cocci on microscopy and culture grew Staphylococcus epidermidis sensitive to amikacin and vancomycin. Investigations including CXR, FBC, ESR, CRP, ACE, autoimmune and vasculitic markers were normal. On discharge, her visual acuity was $6/9^{-2}$ corrected and the fundal appearances had improved with resolution of the retinal haemorrhages and infiltrates.

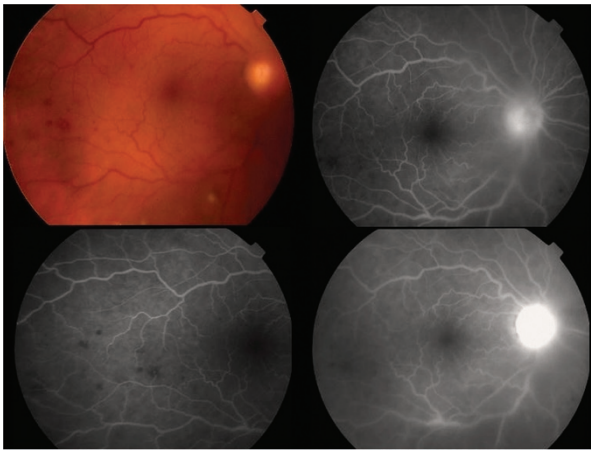

Figure 1 Colour photography shows scattered haemorrhages and Roth's spots. Fluorescein angiography demonstrates disc leakage and staining and leakage of vessel walls.

Comment

Unlike the vast majority of cases of post-operative endophthalmitis, the presentation in this patient is unusual, with the main symptom being increased floaters and clinical signs of retinal infiltrates, mild vitritis, and vascular sheathing.

There is a paucity of literature referring to posterior segment appearances in bacterial endophthalmitis.[1–3] Interestingly, animal models have shown that retinal periphlebitis often develops early in stimulated endophthalmitis, with histology providing evidence of inflammatory cell infiltration of retinal vasculature.[4]

In summary, post-operative bacterial endophthalmitis can present with atypical symptoms and unusual clinical appearance. Although these cases are rare, the authors believe it is advisable to treat with early intravitreal antibiotics rather than wait for evolution of clinical signs, considering the catastrophic outcome on visual prognosis in this condition.[5]

Conflict of interest

The authors declare no conflict of interest.

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Eye (2010) **24,** 1410–1411; doi:10.1038/eye.2010.18; published online 5 March 2010

Sir,

Lumican and muscarinic acetylcholine receptor 1 gene polymorphisms associated with high myopia

Lin *et al*[1] report that polymorphisms in the lumican gene are associated with high myopia. They studied four SNPs in 182 highly myopic cases and 78 emmetropic controls, recruited from a sample of 3000 Taiwanese-born medical students aged 16–25 years old (Table 1a). They also published a second study addressing the same question,[2] testing five SNPs—including four of those above—in 201 high myopes and 86 emmetropic controls, also recruited from 3000 Taiwanese-born volunteers aged 16–25 years old. For the four SNPs common to both studies, the results were similar.

In neither article is the other study referred to, which suggests that the two investigations are unlikely to be an original study followed by a replication study. Thus, it seems possible that the same SNPs were genotyped twice (albeit using different methods) in a common set of subjects and reported as being independent results. If true, this would contravene established scientific practice (because it produces a false impression of the weight of evidence supporting a gene–disease association).

In another recent publication, Lin *et al*[3] genotyped four SNPs in the *CHRM1* gene in their Taiwanese case–control subjects. Genotype frequencies were hugely different ($P<10^{-7}$) between cases and controls for two SNPs (rs544978 and rs544269) suggesting strong association.[3]

Lin *et al* tested for departure from Hardy–Weinberg equilibrium (HWE), as this can be indicative of genotyping error[4] (Table 1a). We repeated these tests (R genetics HWE.test function) using the data in Table 2 of their article.[3] Our results (Table 1b) showed that both myopia-associated SNPs were not in HWE in controls and that Lin *et al*'s HWE test results were erroneous. Although departure from HWE can occur in case–control samples if the disease–polymorphism association is strong,[4] such strong association was not detected at the *CHRM1* locus in a high-myopia GWA in Japanese subjects.[5] The possibility of genotyping error weakens the evidence of an association between *CHRM1* polymorphisms and high myopia. We also noticed inconsistencies between the data reported by Lin[3] and the HapMap database, for example in the alleles of rs544978 and the allele frequencies of rs2186410 (Table 2a and b).

Table 1a HWE test *P*-values originally reported by Lin *et al*[3]

SNP	*HWE test* P-*value* (χ^2 *test)*	
	Cases	*Controls*
rs11823728	0.023	0.048
rs544978	NSD	0.012
rs2186410	NSD	NSD
rs542269	NSD	NSD

Table 1b HWE test *P*-values re-calculated from the data of Lin *et al*[3]

SNP	*HWE test* P-*value (*R *genetics HWE test)*	
	Cases	*Controls*
rs11823728	NSD	NSD
rs544978	NSD	1.867e−10
rs2186410	1.741e−11	1.450e−15
rs542269	NSD	8.231e−16

NSD, not statistically different from the null hypothesis of HWE.

Table 2a Characteristics of *CHRM1* polymorphisms reported by Lin *et al*[3]

SNP	*Alleles (frequencies)*	
	Cases	*Controls*
rs11823728	G/A (0.98 : 0.02)	G/A (0.99 : 0.01)
rs544978	G/A (0.87 : 0.13)	G/A (0.76 : 0.24)
rs2186410	G/A (0.17 : 0.84)	G/A (0.17 : 0.84)
rs542269	G/A (0.12 : 0.88)	G/A (0.15 : 0.85)

Table 2b Characteristics of *CHRM1* polymorphisms in the HapMap database

SNP	*Alleles (frequencies) HapMap HCB subjects Controls*	*HWE test* P-*value HapMap HCB subjects Controls*
rs11823728	C/T (1.00 : 0.00)	NA
rs544978	A/C (0.87 : 0.13)	1.00
rs2186410	G/A (0.87 : 0.13)	1.00
rs542269	C/T (0.13 : 0.87)	1.00

Conflict of interest

The authors declare no conflict of interest.

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