

J Lee, S-H Chung, SC Lee, HJ Koh

Institute of Vision Research,
Department of Ophthalmology,
Yonsei University College of Medicine,
Seoul, Korea

Correspondence: HJ Koh,
Institute of Vision Research,
Department of Ophthalmology,
Yonsei University College of Medicine,
134 Shinchon-dong, Sodaemun-gu,
Seoul 120-752, Korea
Tel: + 82 2 2228 3570;
Fax: + 82 2 312 0541.
E-mail: hjkoh@yumc.yonsei.ac.kr

Proprietary interest: none

The authors received no financial support

Eye (2006) **20**, 1444–1446. doi:10.1038/sj.eye.6702308;
published online 3 March 2006

Sir,
Hemiretinal arterial supply by the cilioretinal artery

We read this report with interest.¹ However, we feel that the evidence that the arteriole in question is a cilioretinal artery, as opposed to an early posterior bifurcation of the central retinal artery, is not convincing.

The claim that in the early venous phase of the FFA, the dye appears faded in the upper arteriole, whereas still bright in the lower arteriole, is not sufficient evidence to support a diagnosis of a cilioretinal artery. If the dye had entered the superior arteriole earlier than the inferior, one would expect to see the superior venous filling to be at a later stage than the inferior. On the contrary, the published frame shows symmetrical lamellar flow in both the superior and inferior retinal venous circulation, suggesting that the dye entered their feeding arterioles at the same time.

An earlier frame, were it to show simultaneous filling of this arteriole and the choroid in the absence of filling of the inferior arteriole, would be conclusive, but was presumably not available.

Reference

- 1 Lewis AM, Mireskandari K. Hemiretinal arterial supply by the cilioretinal artery. *Eye* 2005; **19**(11): 1239–1240.

F Skarmoutsos and G Kyle

Department of Ophthalmology, University Hospital
Aintree, Liverpool L9 7AL, UK
Correspondence: F Skarmoutsos,
Tel: + 44 151 706 4782/ + 44 7834983755;
Fax: + 44 151 525 6086.
E-mail: skarmf@yahoo.co.uk

Eye (2006) **20**, 1446. doi:10.1038/sj.eye.6702310;
published online 10 March 2006

Sir,
Hemiretinal arterial supply by the cilioretinal artery

Skarmoutsos and Kyle have made a good point regarding the comparison of the superior and inferior venous filling in this case. The venous filling does appear to be at the same stage in the upper and lower systems. There is trilaminar flow just visible at both of the first main bifurcations of the superior and inferior hemiretinal veins. Trilaminar flow occurs at the junction of two veins with bilaminar flow, with the inner laminar of each vein joining to produce three laminae.¹ If this is a cilioretinal artery, it would have fluoresced along with the choroid 1–3 s before the central retinal artery, and in theory the venous stage would then start 1–3 s early in the superior venous system with more fluorescence present than in the picture shown.

The patient in this case had the FFA performed for unrelated reasons, and unfortunately this was the earliest frame available. Earlier frames of the fundus fluorescein angiogram would have been conclusive to identify this as a cilioretinal artery, and without these there is no definitive answer. The cilioretinal artery was an incidental finding, and we felt that it was inappropriate to repeat the FFA just for personal gain.

Reference

- 1 Johnson RN, Schatz H, McDonald HR, Ai E. Fluorescein angiography: Basic principles and interpretation. In: Ryan SJ (ed). *Retina*, 3rd edn, Mosby: Missouri, 2001, pp 875–942.

A Lewis and K Mireskandari

Western Eye Hospital, Marylebone Road,
London NW1 5QH, UK

Correspondence: A Lewis,
Tel: + 44 774 761 2414;

Fax: +44 207 886 3259.
E-mail: panda@doctors.org.uk

Eye (2006) 20, 1446–1447. doi:10.1038/sj.eye.6702311;
published online 3 March 2006

Sir,
Valsalva retinopathy associated with idiopathic thrombocytopenic purpura and positive antiphospholipid antibodies

Valsalva retinopathy may occur during a sudden increase in intraocular venous pressure caused by forced exhalation against a closed glottis.

Patients with idiopathic thrombocytopenic purpura (ITP) and positive antiphospholipid antibodies (aPL) are at particular risk for developing both bleeding and thrombotic complications.¹

Case report

A 32-year-old female patient presented with sudden visual loss in the right eye during sexual intercourse. There was no past ocular history of note but she was known to have ITP with positive aPL. On presentation,

her best corrected visual acuities (BCVA) were 6/36 in the right eye and 6/5 in the left. The anterior segments were normal, but in the right eye there was a submacular trilateral haemorrhage (subretinal, intraretinal, and subhyaloid) involving the superior arcade. (Figure 1a). Fundus biomicroscopy of the left eye was normal. Systemically, she was well and was on no medications. The blood pressure was 100/70 mmHg on presentation. All blood tests were normal except for raised IgM anticardiolipin antibody consistent with her past medical history. Her platelet count was $195 \times 10^3/\text{mm}^3$.

Within a few days, there was further reduction of vision with the BCVA dropping to 2/60. A fluorescein angiography (FA) and indocyanine green angiography (ICG) were performed. (Figure 1b–d). Both examinations showed an area of hypofluorescence corresponding to the haemorrhage but no underlying neovascularization. The diagnosis of Valsalva retinopathy was made and all management options were explained. The patient elected not to undergo any surgical treatment. On subsequent follow-up at 3 weeks, 2, 4, and 6 months, the haemorrhage gradually absorbed leaving an area of atrophy of the retinal pigment epithelium (RPE) under the right fovea and an epiretinal membrane along the superior arcade (Figure 2a–d). Her BCVA remained at 6/60.

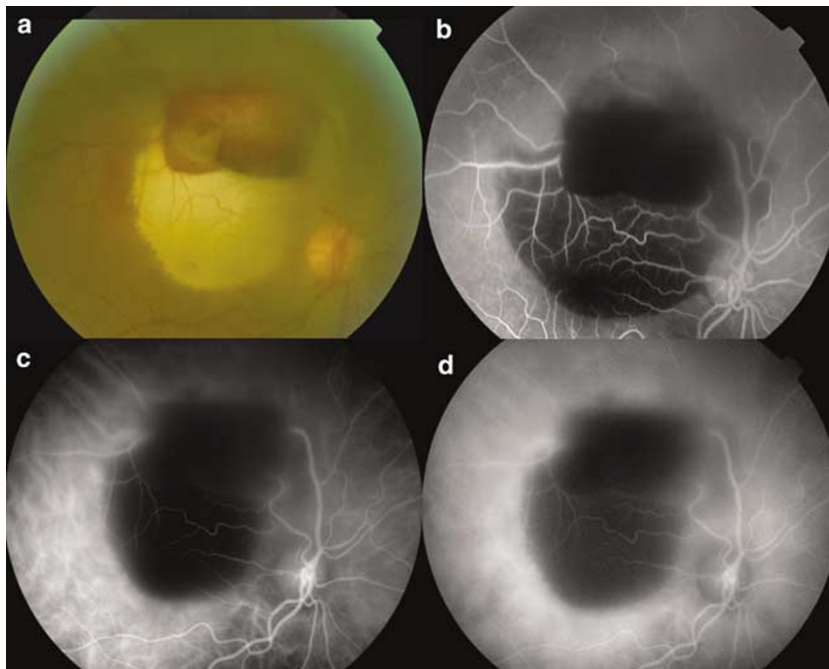


Figure 1 (a) Colour fundus picture on presentation showing the trilateral submacular haemorrhage extending up to the superior arcade. (b) Fluorescein angiography showing an area of hypofluorescence corresponding to the haemorrhage. (c, d) Indocyanine green angiography showing an area of hypofluorescence corresponding to the haemorrhage but no underlying neovascularization.