Multiple ophthalmic sequelae of arterial hypertension

Jill Wen-Chun Huang, Alison G Greene, Kirk A J Stephenson, Frank Kinsella

DESCRIPTION
A woman in her seventh decade of life presented with a 1-day history of transient left eye pain and persistent well-demarcated round black scotoma in the left superotemporal visual field after bending down. The patient’s medical history was positive for obesity, but the patient had not seen her family physician for >5 years.

Best corrected visual acuity (VA) was 6/6 in each eye. Anterior segments were unremarkable with normal intraocular pressures. There was no vitreous haemorrhage. The left fundus was remarkable for an approximately 15× disc area of subretinal haemorrhage (SRH) underlying the inferotemporal vascular arcades but sparing the fovea (figure 1A,B). There was no exudate, intraretinal or preretinal haemorrhage. Fundus fluorescein angiography (FFA, Spectralis, Heidelberg Engineering, Germany) highlighted a retinal artery macroaneurysm (RAM) along the inferotemporal arcades as well as supertemporal macular intraretinal telangiectasia (figure 1C,D). Optical coherence tomography (OCT, Spectralis) confirmed the subretinal nature and extent of SRH (figure 1E) with foveal sparing (figure 1F).

Platelet count, prothrombin time and glycated haemoglobin level were all within normal limits. Blood pressure (BP) was 160/90 mm Hg and ambulatory 24 hour BP monitoring confirmed arterial hypertension. Treatment was initiated with intravitreal anti-vascular endothelial growth factor (anti-VEGF, Ranibizumab, Novartis International AG, Switzerland) injections. Though improvement in SRH was noted (figure 2A,C) at 2 months, an inferotemporal branch retinal vein occlusion (BRVO) with cystoid macular oedema had developed reducing vision to 6/12 (figure 2D). Further, anti-VEGF injections and control of arterial hypertension were carried out.

RAMs are focal dilatations of retinal arterioles associated with hypertension.1 2 They occur in approximately 1:4500 people with a 3:1 female preponderance.2 The suggested pathophysiology is that of arteriolosclerotic damage and remodelling leading to ≥1 focal aneurysmal dilatation(s) ranging from 100 to >250 µm in diameter often occurring at arteriovenous crossings or bifurcation sites and most commonly along the temporal vascular arcades.1 Lavin et al classified RAM into quiescent (ie, incidental), exudative (ie, slow leakage with intraretinal and subretinal lipid deposition) and haemorrhagic (ie, acute bleed into subretinal space ± intraretinal/vitreous components).1 Diagnosis is clinical with adjunctive FFA or indocyanine green angiography. Visual prognosis depends on the location of the aneurysm and the status at presentation (eg, incidental vs symptomatic) with inferior lesions harbouring a more favourable prognosis. Control of systemic hypertension is critical to mitigate visual loss secondary to RAM. Ophthalmic treatment modalities include focal laser techniques and/or intravitreal anti-VEGF.4 5 Success of RAM thrombosis following laser photocoagulation may be limited (~27%) and may result in aneurysm rupture or downstream ischaemia1 while anti-VEGF therapy provides superior visual outcomes to observation.6 Management of SRH depends on location, duration and patient factors (eg, suitability for surgery). Observation of RAM-associated SRH is reasonable, particularly with inferior fovea-sparing lesions (VA ≥6/12 in 57%).7

RAMs have been reported at mean of 2.3 months following 7%–19% of BRVO, potentially as a result of the dynamic and evolving nature of these angiographic lesions.8

CONTROL OF SYSTEMIC HYPERTENSION
Blood pressure (BP) was 160/90 mm Hg and ambulatory 24 hour BP monitoring confirmed arterial hypertension. Treatment was initiated with intravitreal anti-vascular endothelial growth factor (anti-VEGF, Ranibizumab, Novartis International AG, Switzerland) injections. Though improvement in SRH was noted (figure 2A,C) at 2 months, an inferotemporal branch retinal vein occlusion (BRVO) with cystoid macular oedema had developed reducing vision to 6/12 (figure 2D). Further, anti-VEGF injections and control of arterial hypertension were carried out.

RAMs are focal dilatations of retinal arterioles associated with hypertension.1 2 They occur in approximately 1:4500 people with a 3:1 female preponderance.2 The suggested pathophysiology is that of arteriolosclerotic damage and remodelling leading to ≥1 focal aneurysmal dilatation(s) ranging from 100 to >250 µm in diameter often occurring at arteriovenous crossings or bifurcation sites and most commonly along the temporal vascular arcades.1 Lavin et al classified RAM into quiescent (ie, incidental), exudative (ie, slow leakage with intraretinal and subretinal lipid deposition) and haemorrhagic (ie, acute bleed into subretinal space ± intraretinal/vitreous components).1 Diagnosis is clinical with adjunctive FFA or indocyanine green angiography. Visual prognosis depends on the location of the aneurysm and the status at presentation (eg, incidental vs symptomatic) with inferior lesions harbouring a more favourable prognosis. Control of systemic hypertension is critical to mitigate visual loss secondary to RAM. Ophthalmic treatment modalities include focal laser techniques and/or intravitreal anti-VEGF.4 5 Success of RAM thrombosis following laser photocoagulation may be limited (~27%) and may result in aneurysm rupture or downstream ischaemia1 while anti-VEGF therapy provides superior visual outcomes to observation.6 Management of SRH depends on location, duration and patient factors (eg, suitability for surgery). Observation of RAM-associated SRH is reasonable, particularly with inferior fovea-sparing lesions (VA ≥6/12 in 57%).7

RAMs have been reported at mean of 2.3 months following 7%–19% of BRVO, potentially as a result of the dynamic and evolving nature of these angiographic lesions.8
Images in...

Learning points

► Retinal artery macroaneurysms and retinal vein occlusions are common in isolation in the >60 years age group in association with arterial hypertension.
► While uncommon, spontaneous retinal venous occlusion can occur following a macroaneurysm.
► Adequate BP control may mitigate blinding retinal complications including subretinal haemorrhage, retinal vein occlusion and retinal ischaemia.

feature of vascular remodelling, 84% of which were in ischaemic BRVOs. RAMs with subsequent BRVOs are sparsely reported. In this case, ophthalmic sequelae of hypertension occurred sequentially, highlighting the deleterious influence of uncontrolled hypertension on retinal vasculature.

Acknowledgements

The authors thank the patient for her participation.

Contributors

JWCH, AGG, KAJS, FK: manuscript drafting, revision and patient care.

Funding

The authors have not declared a specific grant for this research from any funding agency in the public, commercial or not-for-profit sectors.

Competing interests

None declared.

Patient consent for publication

Consent obtained directly from patient(s).

Provenance and peer review

Not commissioned; externally peer reviewed.

REFERENCES


Case reports provide a valuable learning resource for the scientific community and can indicate areas of interest for future research. They should not be used in isolation to guide treatment choices or public health policy.

ORCID ID

Kirk A J Stephenson http://orcid.org/0000-0002-7462-7725