

# Blindness from suprachoroidal haemorrhage in two patients with age-related macular degeneration on systemic anticoagulation therapy or an antiplatelet agent

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## Clinical record

### Patient 1

A 90-year-old man with bilateral neovascular age-related macular degeneration (AMD) presented with sudden onset of painful visual loss in his left eye. Visual acuity in his right eye was 1/60 due to scarring associated with AMD, and visual acuity in his left eye had deteriorated to hand movements; it had previously been 6/120. He had been taking warfarin for about 2 years for atrial fibrillation and a transient ischaemic attack. The international normalised ratio (INR) had previously been measured almost weekly, but as it had been stable at 2.5–2.9 (target INR, 2.0–3.0), it had not been measured for 6 weeks.

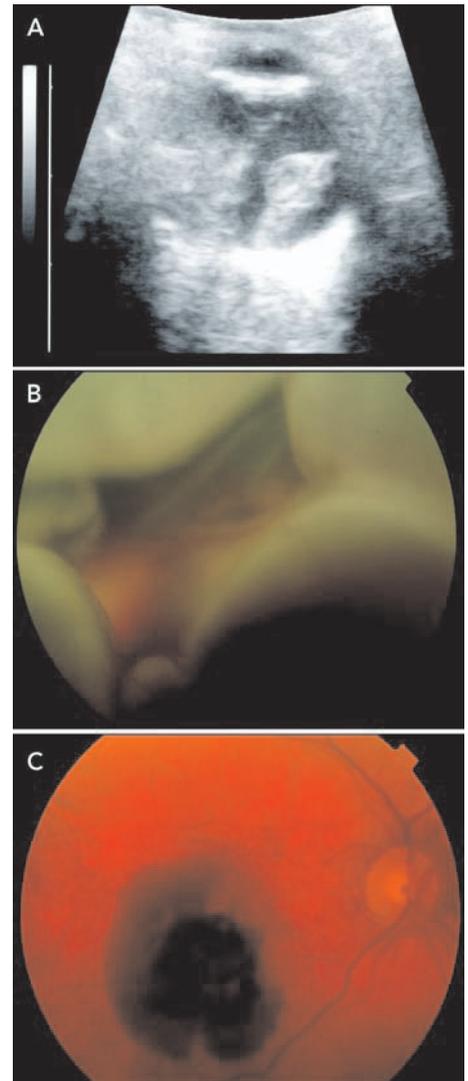
B-mode ultrasonography and dilated fundus examination revealed massive suprachoroidal haemorrhage (Figures A and B). The INR was 6.0, which was reversed with intravenous administration of two units of fresh frozen plasma and 1 mg of vitamin K. Visual acuity in the patient's left eye deteriorated to light perception. As there were no signs of spontaneous improvement and this had been his better eye, surgery was performed to drain the haemorrhage. After surgery, the haemorrhage decreased in size but his vision did not improve.

### Patient 2

An 82-year-old man presented with a 3-week history of a shadow in his left eye. He had been taking low-dose aspirin for ischaemic heart disease and stroke, but had no known ocular history. On examination, visual acuity was 6/5 and hand movements in his right and left eye, respectively, and massive subretinal haemorrhage was noted in the left eye. Non-neovascular AMD was present in his right eye, and the haemorrhage in his left eye was presumed to be due to a combination of neovascular AMD and the effects of aspirin. He was offered surgery, but declined as there was little chance of improvement in central vision. Subsequently, he gradually lost all vision (including light perception) in his left eye. Aspirin was continued because of his significant cardiovascular history. Over the following year, neovascular AMD developed in his right eye and visual acuity dropped from 6/5 to 6/36 (Figure C). He was treated with regular intravitreal ranibizumab injections, and his vision stabilised.

However, several months later, vision in his right eye suddenly deteriorated to vague perception of light. On examination of his right eye, there was no view of the fundus due to a dense cataract and vitreous haemorrhage, but B-mode ultrasonography once again revealed massive choroidal, subretinal and vitreous haemorrhage. The patient was still taking low-dose aspirin therapy, which was subsequently ceased. In an attempt to preserve vision in his only seeing eye, he underwent vitrectomy and silicone oil insertion, but there was no improvement and he subsequently lost light perception in his right eye.

*A: B-mode ultrasonogram of Patient 1's left eye, showing massive suprachoroidal haemorrhage. B: Fundus photograph of Patient 1's left eye, showing massive suprachoroidal haemorrhage bulging forward (therefore largely out of focus). C: Fundus photograph of Patient 2's right eye, showing right neovascular AMD with subretinal haemorrhage, prior to development of massive suprachoroidal, subretinal and vitreous haemorrhage.*



Age-related macular degeneration affects about one-third of people aged over 75 years.<sup>1</sup> Of those with AMD, 10%–15% develop the neovascular (“wet”) form,<sup>2</sup> characterised by abnormal new blood vessel formation in the choroid, under the retina. These abnormal vascular membranes are prone to rupture, leading to subretinal bleeding, fibrous scar formation and severe visual loss.

Many older patients who have AMD also take medications that can exacerbate or promote bleeding, such as anticoagulants or antiplatelet agents. In rare cases, intraocular bleeding — in the form of subretinal, suprachoroidal, or vitreous haemorrhage —

can be catastrophic and blinding. Previous reports link systemic anticoagulation therapy to intraocular haemorrhage and blindness in AMD patients,<sup>3–7</sup> including a recent report in this Journal.<sup>8</sup> In two of these reports, patients taking warfarin had very high INRs (4.1 in one case,<sup>3</sup> 6.3 in the other<sup>4</sup>).

Other reports link systemic anticoagulation therapy to spontaneous suprachoroidal haemorrhage, even in the absence of neovascular AMD.<sup>9–11</sup> Additionally, patients with neovascular AMD can develop massive submacular haemorrhage, even if they are not taking antiplatelet or anticoagulant agents. Unfortunately, as many patients with AMD have one eye with poor visual acuity due to

**Lessons from practice**

- Patients taking warfarin who develop neovascular age-related macular degeneration (AMD) should maintain an international normalised ratio (INR) at the lower end of the recommended range.
- INR monitoring should be more frequent for patients with neovascular AMD.
- If a patient has only one functioning eye, an ophthalmologist should assess the risk of developing neovascular AMD in the seeing eye before, or soon after, commencing warfarin or an antiplatelet agent, including aspirin.
- Ophthalmologists should ask all patients whether they take warfarin, and should communicate to the treating doctor whether this patient has, or is at high risk of developing, neovascular AMD. ♦

macular scarring, it is all the more catastrophic when a massive haemorrhage leads to blindness in their “good” eye.

We suggest that the risk of catastrophic bleeding may be stratified, with the greatest risk being associated with anticoagulants (eg, warfarin), followed by antiplatelet agents (eg, clopidogrel and ticlopidine, with aspirin conferring a lower risk). In addition, we would expect a greater risk with combination therapy comprising simultaneous use of multiple antiplatelet and/or anticoagulant agents. In the cases reported here, we suggest that haemorrhage is likely to have been more severe and blinding than it would have been in a patient who was not taking those medications.

Although these medications should not be withheld in cases where they are warranted to reduce cardiovascular morbidity and mortality, they are not without risk, and a careful risk–benefit analysis should be performed for each patient before they are prescribed. For example, the risk of stroke is different among patients with simple atrial fibrillation compared with aortic valve replacement.

It has been previously noted that if a patient has only one functioning eye, the patient’s general practitioner or cardiologist should seek an ophthalmologist’s opinion to assess the risk of neovascular AMD in the seeing eye before, or soon after, commencing warfarin.<sup>12</sup> Further, ophthalmologists should ask their patients whether they take warfarin, and should communicate to the treating doctor whether a patient has, or is at high risk of developing, neovascular AMD.<sup>12</sup>

Patients taking warfarin who develop neovascular AMD should be advised to maintain an INR at the lower end of the recom-

mended range.<sup>3</sup> In addition, we recommend that it would be prudent for INR monitoring to be at the more frequent end of the spectrum.

**Competing interests**

None identified.

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