Spontaneous Suprachoroidal Hemorrhage Associated with Age-related Macular Degeneration and Anticoagulation Therapy

Yu-Yen Chen[1], Ying-Ying Chen[1,2]*, Shwu-Jiuan Sheu[1,2]

[1]*Department of Ophthalmology, Kaohsiung Veterans General Hospital, Kaohsiung, and*
[2]*National Yang-Ming University School of Medicine, Taipei, Taiwan, R.O.C.*

Suprachoroidal hemorrhage is a rare but dreadful event. We report the case of an 86-year-old man with age-related macular degeneration (ARMD) in both eyes. He had been receiving anticoagulation therapy for several years for systemic disease. He presented with severe headache and intractable pain in his right eye. Vision was no light perception, and the intraocular pressure was 50 mmHg in the right eye despite maximal antiglaucoma medications. Slit-lamp and B-scan examination disclosed suprachoroidal hemorrhage in the right eye. Nine days later, he underwent choroidal drainage, which only relieved the symptoms for 1 day. Suprachoroidal hemorrhage recurred and evisceration was performed. This case illustrates how ARMD with anticoagulation therapy could cause spontaneous suprachoroidal hemorrhage. Therefore, anticoagulants should be meticulously prescribed with prothrombin time monitored regularly in ARMD patients. [*J Chin Med Assoc* 2009;72(7):385–387]

Key Words: age-related macular degeneration, anticoagulation therapy, choroidal drainage, spontaneous suprachoroidal hemorrhage

Introduction

Suprachoroidal hemorrhage, typically an explosive accumulation of blood within the suprachoroidal space, is an uncommon but devastating complication of intraocular surgery. Spontaneous suprachoroidal hemorrhage, which occurs unrelated to surgery, is much rarer and can also result in irreversible loss of vision. We present a patient with age-related macular degeneration (ARMD) who was under anticoagulation therapy due to systemic disease. Spontaneous suprachoroidal hemorrhage occurred and, eventually, evisceration had to be performed after failure of multiple treatment modalities.

Case Report

An 86-year-old man with hypertension had a history of transient ischemic attack and deep vein thrombosis. His blood pressure (around 134/74 mmHg) was well controlled with bisoprolol fumarate 5 mg/day. A cardiac pacemaker had been implanted 4 years previously because of bradytachycardia syndrome, a kind of sick sinus syndrome. To reduce blood clot formation and embolic stroke risks, he was placed on anticoagulation therapy of aspirin 100 mg twice a week and warfarin 2.5 mg/day for several years. His ocular history included chronic angle-closure glaucoma with bilateral laser iridotomies. After cataract operation in both eyes 6 years previously, intraocular pressure (IOP) was < 15 mmHg in both eyes without any antiglaucoma medication. However, visual acuity was only hand movement in the right eye due to ARMD with choroidal neovascularization (CNV). The fundus had drusen at the macula without CNV in the left eye, and best-corrected visual acuity was 20/25 in the left eye.

At this presentation, the patient experienced painless loss of vision in the right eye. The visual acuity was no light perception in the right eye. IOP was normal, and the anterior chamber was deep. Slit lamp

*Correspondence to: Dr Ying-Ying Chen, Department of Ophthalmology, Kaohsiung Veterans General Hospital, 386, Ta-Chung 1st Road, Kaohsiung 813, Taiwan, R.O.C.
E-mail: yychen971209@yahoo.com.tw • Received: September 8, 2008 • Accepted: April 1, 2009

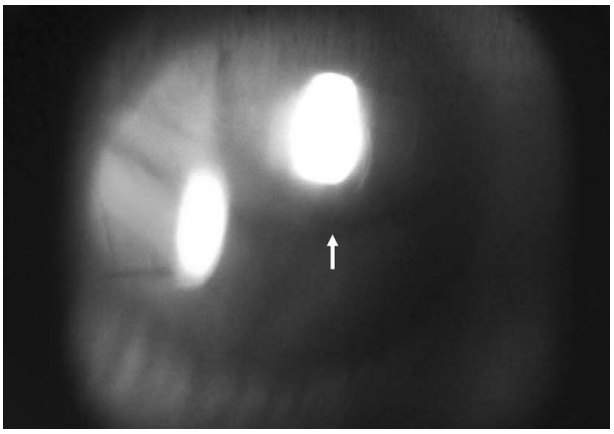

Figure 1. Slit lamp examination shows kissing retina (arrow).

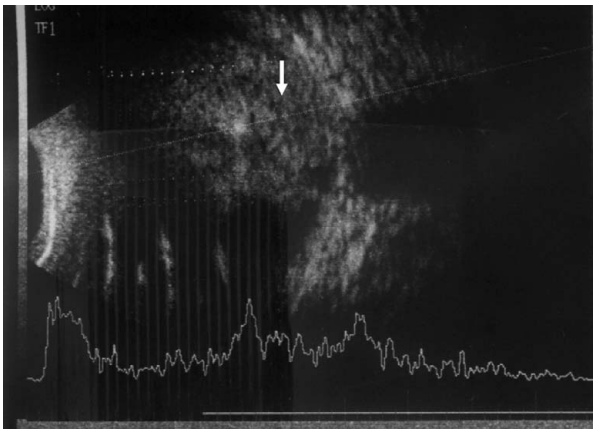

Figure 2. B-scan reveals suprachoroidal hemorrhage (arrow) and vitreous hemorrhage.

and funduscopic examination showed kissing retina with subretinal hemorrhage at the macula (Figure 1). His blood pressure was 132/72 mmHg. Two days later, he presented to the emergency department with the chief complaint of severe headache and right ocular pain. IOP had elevated to 45 mmHg in the right eye. The anterior chamber was shallow and the fundus could not be seen because of dense vitreous hemorrhage. After administration of antiglaucoma drugs, including rescula, alphagan, trusopt, mannitol, isobide and diamox, IOP declined to 35 mmHg in the right eye, but soon increased to 50 mmHg. The pain was much exacerbated; the patient described the pain as like a bomb exploding in his eye, causing an intolerable shearing force.

On examination, the anterior chamber was almost flat. Iris bombe on the temporal side implied that a mass was pushing the lens-iris diaphragm forward. The mass comprised vitreous hemorrhage and blood clot behind the posterior chamber intraocular lens. B-scan confirmed the characteristics of massive suprachoroidal hemorrhage with breakthrough vitreous hemorrhage (Figure 2). Laboratory evaluation revealed a prothrombin time of 22.0 seconds (control, 10.6 seconds) with an international normalized ratio (INR) of 2.3, which was within the therapeutic range. It was noteworthy that his blood pressure remained stable throughout the clinical course.

After consulting with the cardiologist and hematologist, the anticoagulation agents were withheld. Nine days after the onset of suprachoroidal hemorrhage, external sclerotomy was performed and a large amount of red-black liquefied blood was drained. Following the procedure, the anterior chamber was deep, IOP dropped to 35 mmHg, and his symptoms were relieved (Figure 3). However, after just 1 day, the pain recurred and IOP was elevated to 53 mmHg in the right eye. The anterior chamber was flat again, revealing a recurrent suprachoroidal hemorrhage. After understanding the limited effect of every possible treatment modality, the patient underwent evisceration to remove the non-functional and painful eye.

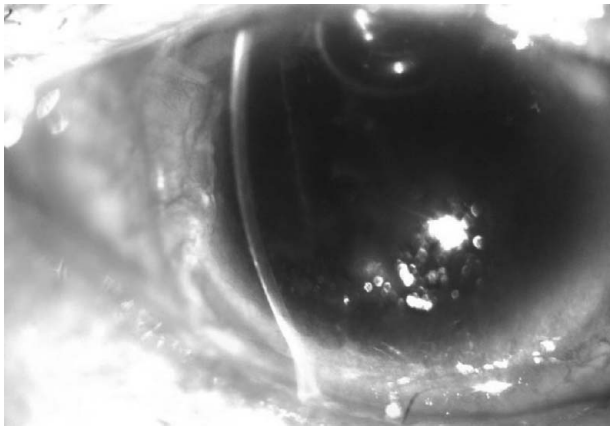

Figure 3. After suprachoroidal drainage, the anterior chamber was deep.

Discussion

Spontaneous suprachoroidal hemorrhage is an extremely rare event that has previously been described only in isolated case reports.[1–6] Risk factors include hypertension, systemic anticoagulation, advanced age and ARMD. Visual prognosis is usually poor. Yang et al[6] presented 5 eyes with spontaneous suprachoroidal hemorrhage. Four of them had vision of no light perception at the final follow-up even after surgical drainage to relieve high IOP and pain. Our patient had all the risk factors predisposing him to spontaneous

suprachoroidal hemorrhage. We tried every possible management option in vain, and the eye had to be eviscerated. This is disappointing for ophthalmologists but reflects the devastating prognosis of spontaneous suprachoroidal hemorrhage.

In our patient, subretinal hemorrhage preceded the massive suprachoroidal hemorrhage, implying the role of CNV in initiating the process. We hypothesized that at first, bleeding from CNV extended into the subretinal space, leading to subretinal hemorrhage. Increased vascular fragility related to advanced age and hypertension, combined with anticoagulants, further precipitated spontaneous suprachoroidal hemorrhage. Blood accumulated in these potential spaces and caused anterior displacement of the retina and choroids, which subsequently stretched the posterior ciliary artery. Then, appositional suprachoroidal hemorrhage occurred as a direct result of rupturing of the blood vessels by shear forces as the vessels entered the suprachoroidal space.

Suprachoroidal hemorrhage may present as acute angle-closure glaucoma because of forward displacement of the lens-iris diaphragm, therefore inducing IOP elevation that cannot be relieved by patent peripheral iridotomy or medical treatment. Surgical drainage is indicated for lens-cornea touch, progressive IOP elevation and intolerable pain. Previous literature recommended that drainage procedures be deferred for 7–14 days following suprachoroidal hemorrhage, when clot lysis has been completed.[7,8] In our patient, external sclerotomy was performed on the 9th day, with liquefied blood drained out smoothly. However, rebleeding occurred 1 day later. Recurrent suprachoroidal hemorrhage is uncommon after successful drainage. In this case, the recurrence may have been due to the abnormal vessels and suppressed clotting function. ARMD resulted in CNV, which bled easily and clotted with difficulty. Anticoagulants resulted in a non-selective lytic state, which may have caused persistent bleeding. A combination of these factors was likely to increase the chances of recurrent bleeding and worsen the prognosis.

Compared to previously reported cases, our patient was unique because his INR of prothrombin time was within the therapeutic range (a reference value of 2.5–3.0 is considered therapeutic). Therefore, the dosage of anticoagulation agents must be made on a case-by-case basis, considering the attendant risks and benefits. Following consultation with the cardiologist, the warfarin level was adjusted to maintain INR below 1.5. The patient was informed of the risk of bleeding in the other eye, which also had ARMD. Regular funduscopic examination and INR evaluation were performed. On the other hand, ARMD patients might require more aggressive treatment for CNV to avoid the catastrophic consequences of spontaneous suprachoroidal hemorrhage if they are on anticoagulants. However, further studies are needed to clarify this issue.

In conclusion, this case emphasizes the risk of spontaneous suprachoroidal hemorrhage in ARMD patients who are on anticoagulation therapy. Therefore, the internist must understand the potential risks and prescribe anticoagulants meticulously. These patients must be informed of the importance of regular follow-up. In addition, ophthalmologists should be aware of the hemorrhagic complications and provide prompt diagnosis.

References

1. Khawly JA, Ferrone PJ, Holck DEE. Choroidal hemorrhage associated with systemic tissue plasminogen activator. *Am J Ophthalmol* 1996;121:577–8.
2. Alexandrakis G, Chaudhry NA, Liggett PE, Weitzman M. Spontaneous suprachoroidal hemorrhage in age-related macular degeneration presenting as angle-closure glaucoma. *Retina* 1998;18:485–6.
3. Chorich LJ, Derick RJ, Chambers RB, Cahill KV, Quartetti EJ, Fry JA, Bush CA. Hemorrhagic ocular complications associated with systemic tissue thrombolytic agents. *Ophthalmology* 1998;105:428–31.
4. Wong JS. Spontaneous suprachoroidal hemorrhage in a patient receiving low-molecular-weight heparin (fraxiparine) therapy. *Aust NZ J Ophthalmol* 1997;27:433–4.
5. Knox FA, Johnston PB. Spontaneous suprachoroidal hemorrhage in a patient with age-related macular degeneration on excessive anticoagulation therapy. *Eye* 2002;16:669–70.
6. Yang SS, Fu AD, McDonald MR, MacDonald HR, Johnson RN, Ai E, Jumper JM. Massive spontaneous choroidal hemorrhage. *Retina* 2003;23:139–44.
7. Chu TG, Green RL. Suprachoroidal hemorrhage. *Surv Ophthalmol* 1999;43:471–86.
8. Scott IU, Flynn HW, Schiffman J, Smiddy WE, Murray TG, Ehlies F. Visual acuity outcomes among patients with appositional suprachoroidal hemorrhage. *Ophthalmology* 1997;104: 2039–46.