

## CASE REPORT

# A rare case of choroidal hemorrhage complicated with hypertension due to chronic renal failure

Tomoyuki Tajika<sup>1)</sup>, Hideaki Yokozeki<sup>2)</sup>, Katsuo Ishimaru<sup>3)</sup>, Takeshi Naito<sup>4)</sup>, and Hiroshi Shiota<sup>4)</sup>

<sup>1)</sup>Department of Ophthalmology, <sup>2)</sup>Department of Urology, <sup>3)</sup>Department of Internal Medicine, Tokushima Municipal Hospital ; and <sup>4)</sup>Department of Ophthalmology and Visual Neuroscience, Institute of Health Biosciences, The University of Tokushima Graduate School, Tokushima, Japan

**Abstract : Background :** Choroidal hemorrhage is usually seen as expulsive hemorrhage related to surgical invasion and is rarely observed in the absence of surgery or trauma. In this report, we describe a case of choroidal hemorrhage caused by hypertension related to chronic renal failure. **Case :** A 32-year-old man presented with sudden visual loss, eye pain and swelling in his left eye. He had a history of renal failure and hypertension. Eye examination demonstrated choroidal hemorrhage associated with ocular hypertension in his left eye and marked hypertensive retinopathy in his right eye. **Observations :** He was treated with hemodialysis and antihypertensive drugs. Upon repeat eye examinations, a gradual reabsorption of the choroidal hemorrhage was observed, although vitreous hemorrhage appeared in the left eye. The vitreous hemorrhage was treated with vitrectomy with the patient regaining good visual acuity post-surgery. **Conclusions :** This rare case suggests that patients with severe background disease need to be carefully observed. *J. Med. Invest.* 55 : 151-155, February, 2008

**Keywords :** choroidal hemorrhage, hypertension, chronic renal failure, vitrectomy

## INTRODUCTION

Choroidal hemorrhage is usually seen as expulsive hemorrhage that is related to surgical complications, or to either a perforating or blunt eye injury. This type of hemorrhage is quite rare in the absence of surgery or trauma. In this report, we describe a case of choroidal hemorrhage caused by hypertension related to chronic renal failure.

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Received for publication September 28, 2007 ; accepted November 8, 2007.

Address correspondence and reprint requests to Takeshi Naito, MD, Ph.D., Department of Ophthalmology and Visual Neuroscience, Institute of Health Biosciences, The University of Tokushima Graduate School, Kuramoto-cho, Tokushima 770-8503, Japan and Fax : +81-88-631-4848.

## CASE REPORT

A 32-year-old man presented with sudden visual loss, pain, and swelling in his left eye. He had been diagnosed with nephropathy since early childhood, and at the age of 22 years he was diagnosed with IgA nephropathy. He had no relevant family history. The subject was found to have not paid attention to his health and he experienced general fatigue, headache and slight visual loss a week before his first medical examination at our hospital. During the evening of July 28, 2003, the patient came to our hospital after an episode of coughing that resulted in sudden eye pain, swelling, and severe visual loss in his left eye.

At presentation, his blood pressure was high (264/177 mmHg) and he had severe renal failure

(blood urea nitrogen 100.1 mg/dl, serum creatinine 10.0 mg/dl). Blood tests demonstrated anemia (erythrocyte count  $2.60 \times 10^6 / \mu\text{l}$ , hemoglobin 8.4 g/dl, hematocrit 23.8%) and a slightly low platelet count ( $97 \times 10^3 / \mu\text{l}$ ). He did not have any other abnormalities in his biochemical tests. The chest X-ray (Fig. 1) demonstrated hydrothorax and an enlarged cardiac shadow.

Right eye examination revealed optic disc swelling, retinal hemorrhage and hard exudates (Fig. 2a). Left eye examination revealed lid swelling, exophthalmos and a slightly shallow anterior chamber. In addition, choroidal protrusion, retinal detachment and subretinal hemorrhage from the inferotemporal retina to the fovea were noted (Fig. 2b). Since he was confined to bed rest and underwent medical treatment for his severe renal failure and marked hypertension, we were unable to perform any further ophthalmic examinations that day.

He was able to leave his bed on the following day (July 29), and we continued his ophthalmic examinations. His corrected visual acuity (decimal) was 0.6 in the right eye and 0.01 in the left eye. Intraocular pressure was 18 mmHg in the right eye and 34 mmHg in the left eye. The ocular findings were almost exactly the same as noted on the previous day. As there were a few cells observed in the an-

terior chamber, topical treatment was started with 0.5% timolol and 0.1% betamethasone eye drops. Hemodialysis and antihypertensive treatment were started for his severe renal failure, and the ophthalmic medical treatments continued until there was stabilization of his general condition.

On the third day (July 30), choroidal protrusion started to decline while there was an increase in his subretinal and vitreous hemorrhage. The choroidal hemorrhage seemed to diffuse into the subretinal space and the vitreous cavity.

On the 6th day (August 2), the fundus became invisible due to the vitreous hemorrhage. B-mode echography showed an irregular protrusive lesion on the inferotemporal fundus, which appeared to be a choroidal hematoma associated with retinal detachment (Fig. 3). Because his general condition was unstable, we observed his eyes carefully, as we were afraid of a recurrence of the choroidal hem-

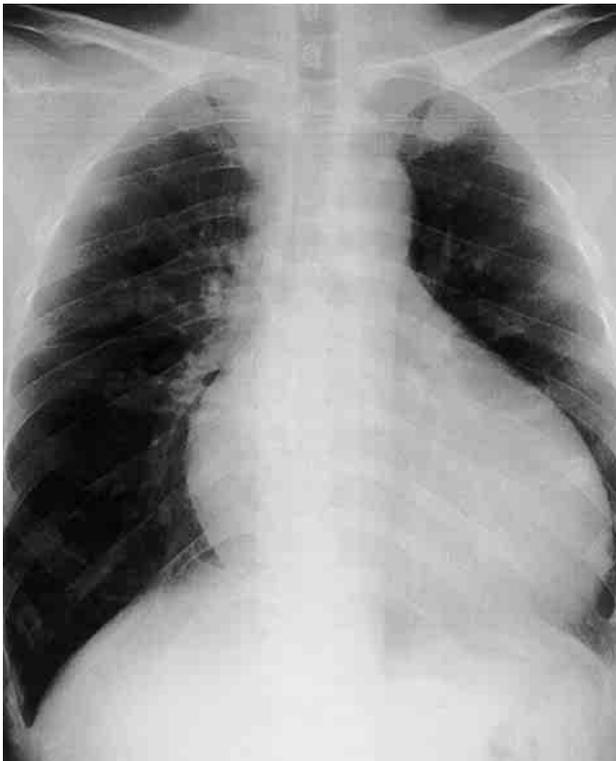


Fig.1 The chest X-ray demonstrated hydrothorax and an enlarged cardiac shadow.

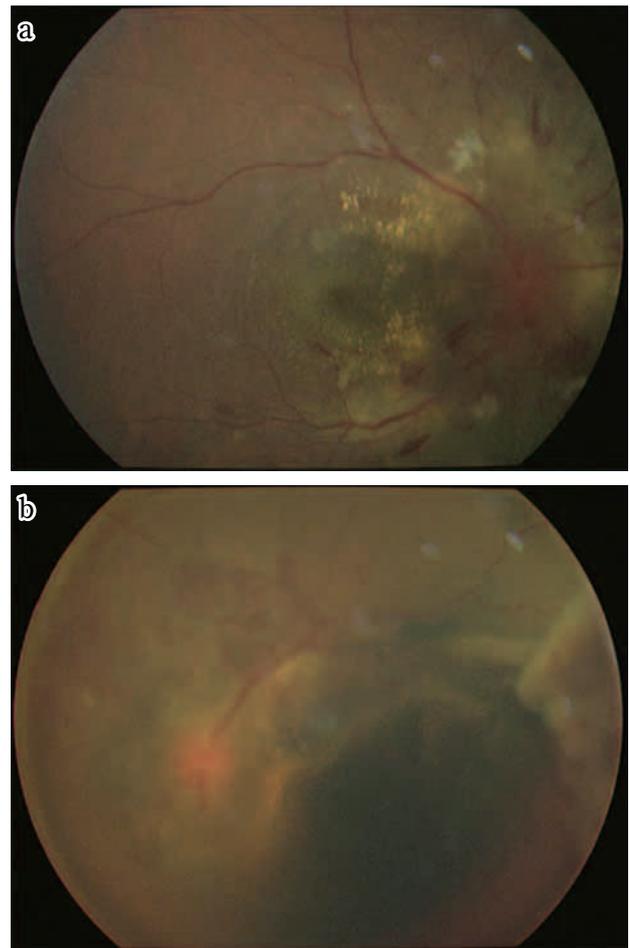


Fig.2 Fundus photographs

a. Right eye examination revealed optic disc swelling, retinal hemorrhage and hard exudates.  
b. Left eye examination revealed choroidal protrusion, retinal detachment and subretinal hemorrhage from the inferotemporal retina to the fovea.

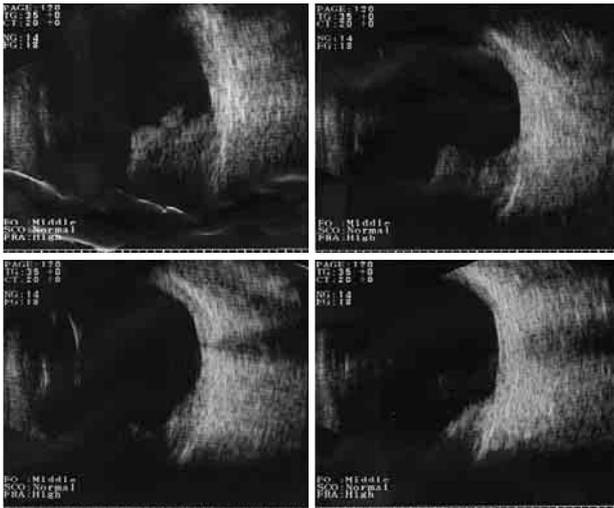


Fig.3 B-mode echography showed an irregular protrusive lesion on the inferotemporal fundus which appeared to be a choroidal hematoma associated with retinal detachment.

orrhage and the potential complications this would cause if indeed it were going to be necessary for us to have to perform surgery. Regular B-mode echography showed a gradual regression of the protrusive choroidal lesion, and there were no abnormalities in the posterior fundus. We waited for the vitreous opacity to be absorbed and followed the regression of the choroidal protrusive lesion. After hemodialysis and antihypertensive therapy, his blood pressure decreased. The optic disc swelling and retinal hemorrhage disappeared and his visual acuity improved to 1.2 in the right eye.

At the beginning of September, the protrusive choroidal lesion had almost completely disappeared although, as seen with B-mode echography, it was still slightly recognizable at the equator of the left fundus. However, the vitreous opacity did not clear up. Therefore, we performed vitrectomy without lensectomy in his left eye. During the surgery, we recognized posterior vitreous detachment, subretinal fibrosis, a protrusive lesion associated with atrophy of the pigment epithelium, and hard exudates in the inferotemporal fundus. This appeared to be an old hematoma. No retinal detachment or retinal break was recognized, but there appeared to be capillary nonperfusion in the retina. Thus, we performed panretinal endolaser photocoagulation at the end of the surgery. The postoperative course was uneventful. The choroidal protrusive lesion persisted. The hard exudates eventually disappeared and on November 27<sup>th</sup>, the visual acuity had improved to 0.8 (50 days after surgery) (Fig. 4). Since that time, the visual acuity has continued to be good.

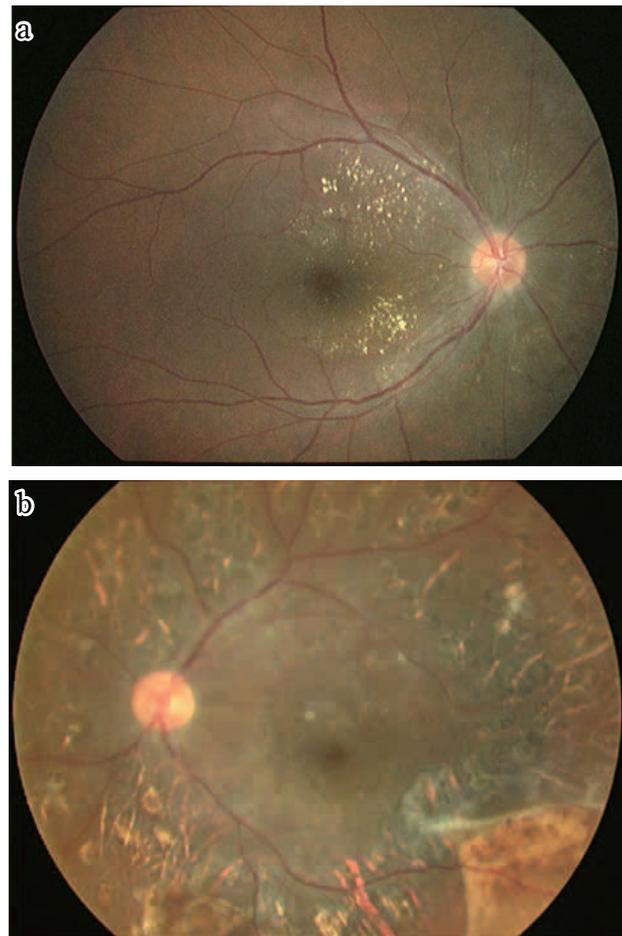


Fig.4  
a. The optic disc swelling and retinal hemorrhage disappeared and the visual acuity improved to 1.2 in the right eye.  
b. The hard exudates eventually disappeared and the visual acuity improved to 0.8 in the left eye.

## DISCUSSION

Choroidal hemorrhage is usually seen as expulsive hemorrhage related to the lowering of the intraocular pressure during intraocular surgery. Previous reports have indicated that it is related to glaucoma surgery, scleral buckling, vitreous surgery, and perforating and blunt injuries. Its occurrence without the presence of surgery or trauma is quite rare (1). The risk factors for expulsive hemorrhage include ocular risk factors (high myopia, past history of ocular injury, glaucoma, etc.) and general risk factors (hypertension, arteriosclerosis, diabetes, blood clotting abnormalities, etc.) (2). In rare cases in which choroidal hemorrhage occurred in the absence of surgery, it was found that hypertension, abnormalities in blood clotting (1, 3, 4) and anticoagulant or thrombolytic therapy (5-11) were responsible for the hemorrhage. Other ocular risk factors that can cause choroidal hemor-

rhage include age-related macular degeneration (12-14) and angle-closure glaucoma (4). On rare occasions, choroidal protrusive lesions have been misdiagnosed as choroidal tumors (15). In this report, we describe a case of choroidal hemorrhage caused by hypertension related to chronic renal failure.

This patient had chronic renal failure associated with hypertension and renal anemia. We believe his exophthalmos was due to retrobulbar hemorrhage, although this could not be confirmed by CT. Zauberman (16) has reported that congestion of the vortex vein can cause choroidal hemorrhage. In our case, the retrobulbar hemorrhage occurred first, with the subsequent high intraorbital pressure due to the retrobulbar hemorrhage, extraordinary hypertension, chronic renal failure, anemia and weakness of the blood vessels responsible for causing the choroidal hemorrhage.

In the present case, blood flowed from the choroidal hemorrhage into the vitreous cavity after a few days, making it difficult to observe the fundus due to the vitreous opacity. B-mode echography demonstrated reduction of the choroidal hematoma without retinal detachment. Therefore, we were able to observe the eye without any surgical treatment. The patient was initially treated with hemodialysis and antihypertensive medication. In addition, the vitreous hemorrhage was later treated successfully with vitrectomy, with the net result of good visual acuity being obtained. The relatively small hemorrhage in this patient also helped to contribute to the good visual acuity obtained. Indications for surgical treatment of choroidal hemorrhage depend upon whether or not there are findings of macular heterotopia or metamorphosis. When surgery is performed, a transscleral approach is used to drain the choroidal hemorrhage. Because of the hemolysis of the hematoma, the optimal time for surgery is 1-2 weeks after onset (17). In the current case, the macular heterotopia, which was recognized at the time of onset, disappeared at the point where the vitrectomy was performed. This suggests that improvement of mild heterotopia can occur without any surgical therapy.

Cases of choroidal hemorrhage without ocular surgery or trauma seem to be related to severe general risk factors such as hypertension, renal failure, abnormalities in blood clotting, and anticoagulant or thrombolytic therapy. Our case involved a relatively young patient with severe ocular complications. Based on our results, it is suggested that such

patients with severe general background disease need to be carefully observed, and that management of such cases should be based on not only ophthalmic findings, but also on the general condition of the patients.

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