Central serous chorioretinopathy (CSCR) was probably first reported by Von Grafe in 1866, and there has been a long history of clinical investigations. The disease generally affects healthy young males who are under emotional stress and is characterized by a round area of serous detachment of the neurosensory retina and retinal pigment epithelium in the macula that often spontaneously improve within 2 to 3 months. On rare occasions, variant CSCR with bullous RD occurs which is frequently misdiagnosed. We report on a case of variant CSCR with severe bullous serous retinal detachment in the left eye that was initially treated at another hospital under the misdiagnosis of rhegmatogenous retinal detachment. Because the retinal detachment developed so fast that a laser could not be applied to all leaking spots, we performed a pars plana vitrectomy, perfluorocarbon liquid-assisted external drainage, and final treatment with an endolaser. The retina was well attached after this management. (Chang Gung Med J 2003;26:777-81)

Key words: central serous chorioretinopathy, variant central serous chorioretinopathy, serous retinal detachment, perfluorocarbon liquid.
2-month history of blurred vision in his left eye. The patient had no systemic disease and had not taken steroids. He was initially treated in another hospital under the diagnosis of rhegmatogenous retinal detachment with scleral buckling and intravitreal air injection. Due to a lack of improvement, he sought help at our hospital on October 19, 2001. Results of the ophthalmologic examination showed that his visual acuity was 20/50 in the right eye and counting fingers at 15 cm in the left eye. The cornea and lens were unremarkable. The anterior chamber of his left eye was free from cells. A fundus examination revealed inferior bullous retinal detachment that partly obscured the macula in the left eye. There was a little vessel tortuosity, and the retina was somewhat unsmooth with a wavy-like appearance (Fig. 1A). An air bubble in the superior fundus was noted, but no retinal breaks were found. Some shallow subretinal fluid over the superolateral macular area was noted in the right eye (Fig. 2A). Ocular ultrasonography showed no intraocular tumor or choroidal detachment. Fluorescein angiography demonstrated multiple areas of fluorescein leakage in his left eye (Fig. 1B) and a leakage point corresponding to serous detachment in his right eye (Fig. 2B). Bilateral CSCR with severe variant CSCR in the left eye was diagnosed.

The patient was initially scheduled for laser

![Fig. 1](image1) (A) Fundus examination revealing inferior bullous retinal detachment which partly obscured the macula with wavy, mild proliferative vitreoretinopathy-like change. There was an air bubble in the superior fundus. (B) Fluorescein angiography demonstrating multiple areas of fluorescein leakage in the left eye.

![Fig. 2](image2) (A) A patch of subretinal fluid over the superolateral macular area noted in the right eye. (B) Fluorescein angiography revealing a leakage point corresponding to the ophthalmoscopic finding with some stained areas in his right eye.
treatment, however the exudative detachment developed so fast that leakage spots were all soon covered by subretinal fluid before the laser could be applied. On November 6, 2001, transscleral drainage of the subretinal fluid was performed in the left eye. Because the subretinal fluid migrated to the posterior pole in the supine position, no subretinal fluid could be drained through the external drainage site. He then was scheduled for a pars plana vitrectomy, perfluorocarbon liquid (®Perfluoron, Alcon, TX)-assisted external drainage of the subretinal fluid, and endolaser treatment the following day. During the second operation, the subretinal fluid was smoothly drained off through the external drainage site with the assistance of the perfluorocarbon liquid. As the high-density vitreous substitute displaced the subretinal fluid peripherally, subretinal fibrin-like material was noted at several sites. Diode laser photocoagulation was applied to these sites of suspected leakage. The retina was well attached postoperatively. Patches of subretinal fibrosis and chorioretinal scarring at the previous leakage spots were noted (Fig. 3). His final visual acuity 6 months after surgery was counting finger at 50 cm in the left eye.

![Fig. 3 Retina totally attached, although patches of subretinal fibrosis and chorioretinal scarring are present in the left retina.](image)

**DISCUSSION**

CSCR is a relatively benign and self-limiting disease. But the variant form of CSCR takes a longer time to resolve and may cause extreme reduction in the vision. Previous reports indicated that variant CSCR following corticosteroid therapy or an ocular operation resolved slowly with a final subnormal visual acuity. Therefore, we would like to emphasize the importance of a correct diagnosis of variant CSCR to avoid inappropriate therapy and potential adverse effects.

Variant CSCR appears either in healthy adults without a defined underlying cause, or more frequently in patients with systemic corticosteroid therapy. In our case, it is likely that the patient had spontaneously developed variant CSCR without a predisposing factor. The differential diagnosis of variant CSCR includes rhegmatogenous retinal detachment (RD) or serous RD due to Harada’s disease, severe hypertensive choroidopathy, posterior scleritis, multifocal choroiditis, metastatic tumor, and uveal effusion. Generally, a combination of ophthalmoscopic and fluorescein angiographic findings can help make a correct diagnosis. These include bullous inferior retinal detachment with shifting subretinal fluid and cloudy subretinal fibrinous exudates, the absence of inflammatory cells in the anterior chamber and vitreous cavity, the shifting nature of the subretinal fluid with fluctuating visual acuity, and the absence of retinal breaks. In this case, fluorescein angiography illustrated multifocal subretinal dye leakage and variable-sized areas of retinal pigment epithelial detachment. Fluorescein angiography was our major diagnostic tool owing to previous management which had masked the initial presentation and bulbous subretinal fluid which precluded an ophthalmoscopic examination. The manifestation in the contralateral eye also provided a hint of the correct diagnosis of CSCR.

From the literature, the exudative lesions of variant CSCR are self-limiting or rapidly resolve in response to photocoagulation. The final visual outcome is favorable unless the macula is involved. In our case, the subretinal fluid was accumulating so fast that it covered the all the way to the macular area and involved all the arcade area in a sitting position, which precluded application of the laser. There is one report of external drainage of the subretinal fluid in variant CSCR being used to facilitate laser delivery. In our case, however, external drainage without the help of perfluorocarbon liquid was impossible since the subretinal fluid shifted posteriorly in a supine position. A pars plana vitrectomy and injections of perfluorocarbon liquid to peripherally dis-
place the subretinal fluid proved to be very effective in the external drainage procedures. We finally applied an endolaser to all suspected leakage points and successfully prevented recurrent exudative retinal detachment. The patient’s final visual acuity improved a little, but was still poor. This was probably because of the long duration of macular detachment and subsequent subretinal fibrosis adjacent to the central retina. According to previous reports, subretinal fibrin is not a rare finding in variant CSCR, and optical coherence tomography has also revealed semitransparent subretinal fluid containing fibrin. In our case, we noted subretinal fibrin intraoperatively. A preoperative finding of a wavy, detached retina and vessel tortuosity also indicated the presence of subretinal fibrin. Subretinal fibrin is a well-known stimulus for the ingrowth of fibroblasts, which results in subretinal fibrotic scar formation. So, the surgical drainage of subretinal fluid should be carried out early to facilitate laser delivery to leakage points to lessen fibrin's ability to induce more-severe subretinal fibrosis.

Herein we report a case of variant CSCR managed with perfluorocarbon liquid-assisted external drainage and endolaser treatment. This proved to be effective and shortened the duration of retinal detachment. A correct diagnosis and prompt treatment of patients with variant CSCR are critical for better visual prognosis.

REFERENCES

使用手術治療中心漿液性視網膜病變造成之
水泡樣漿液性視網膜剝離

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漿液性視網膜剝離 (serous retinal detachment) 之成因很多，包括眼部發炎疾病如原田氏症 (Harada disease)，高血壓，老年性黃斑部病變 (age-related macular degeneration)，眼內轉移腫瘤等，只有少數病例為中心漿液性視網膜病變 (central serous chorioretinopathy) 造成。典型之中心漿液性視網膜病變可見黃斑部局部視網膜剝離，病人大多為年輕至中年男性，通常約2至3個月會自動恢復，視力預後不錯。不過會造成水泡樣漿液性視網膜剝離 (bullous serous retinal detachment) 則為少見之異型中心漿液性視網膜病變 (variant central serous chorioretinopathy)，視力預後較差，並容易被誤診為其他視網膜疾病。本病例為一雙側中心漿液性視網膜病變化病人合併左眼嚴重水泡樣漿液性視網膜剝離，先前於他院診斷為破孔性視網膜剝離 (rhegmatogenous retinal detachment)，接受手術治療無效。於我院檢查時，藉由眼部血管攝影正確診斷後，病人接受玻璃體切除術；使用重油 (perfluorocarbon liquid) 幫助網膜下液體引流及眼內裝置治療，術後視網膜完全貼合。不過視力因視網膜下纖維化而恢復不佳。（長庚醫院 2003;26:777-81）

關鍵字：中心漿液性視網膜病變，異型中心漿液性視網膜病變，漿液性視網膜剝離，重油。