Case Reports in Ophthalmology
Case Rep Ophthalmol 2010;1:90–93
DOI: 10.1159/000322235
Published online: November 6, 2010

ISSN 1663–2699
www.karger.com/cop

Central Serous Chorioretinopathy after Rhinoplasty

Marilita M. Moschos Konstantinos Droutsas
Ioannis Margetis

Department of Ophthalmology, University of Athens, Athens, Greece

Key Words
Rhinoplasty · Central serous chorioretinopathy · Optical coherence tomography

Abstract
We report a case of central serous chorioretinopathy after rhinoplasty for deviation of the nasal septum in a 23-year-old Caucasian man. The patient complained of deterioration of vision in the right eye 4 days after rhinoplasty. At presentation, visual acuity of the right eye was 6/9 with metamorphopsia. Fluorescein angiography revealed a focal retinal pigment epithelium leakage and optical coherence tomography an increase in macular thickness to 245 μm. The left eye was normal. One month after the operation, without any treatment, visual acuity returned to 6/6, the leakage of the retinal pigment epithelium disappeared and the macular thickness returned to 183 μm. To the best of our knowledge, central serous chorioretinopathy after rhinoplasty has not been previously reported. This case report shows a possible association between the postoperative stress and central serous chorioretinopathy. Moreover, it widens the spectrum of drugs associated with the occurrence of the disease.

Introduction

Central serous chorioretinopathy (CSCR) is a chronic idiopathic condition affecting young to middle-aged patients with men affected more commonly than women. It is characterized by serous detachment of the neurosensory retina, with focal or multifocal areas of leakage of the retinal pigment epithelium (RPE) affecting the macular area. The exact cause of CSCR is not clearly understood. Nevertheless, it is associated with psychological stress and systemic corticosteroid therapy, which causes hyperpermeability of choriocapillaries, leading to dysfunctional degeneration of the RPE [1, 2].

Marilita M. Moschos
Department of Ophthalmology, University of Athens, 6 Ikarias Street, Ekali
GR–14578 Athens (Greece)
Tel. +30 694 488 7319, Fax +30 210 412 2139, E-Mail mihmoschos@gmail.com

Case Rep Ophthalmol 2010;1:90–93
DOI: 10.1159/000322235
Published online: November 6, 2010

ISSN 1663–2699
www.karger.com/cop

Case Report

A 23-year-old man presented with a sudden decrease of visual acuity of the right eye (RE) 4 days after rhinoplasty for a deviation of the nasal septum. General anesthesia was induced with propofol 2–5 mg/kg i.v. and sufentanil 0.5 μg/kg i.v. For the first 2 postoperative days, xylometazoline drops were applied topically. No corticosteroids, antibiotics or other postoperative treatment was used. At presentation, best-corrected visual acuity of the RE was 6/9 with metamorphopsia. Fundus examination revealed a well-delineated serous elevation of the macula and fluorescein angiography (FA) showed a focal RPE leakage. The optical coherence tomography (OCT) sections through the macula depicted a mild neurosensory retinal detachment with an increase in retinal thickness equal to 245 μm (**fig. 1**). The left eye was normal. Two weeks later, without any treatment, a gradual improvement of the symptoms was assessed. One month after the operation, visual acuity of the RE returned to 6/6 and the neurosensory retinal detachment disappeared. Also, FA revealed the disappearance of RPE leakage and the retinal thickness of the macula measured with OCT was 183 μm (**fig. 2**).

Discussion

CSCR is a multifactorial disease with controversial etiopathology and it is usually associated with steroid use, pregnancy and stress. The exact mechanism by which glucocorticoids are involved in the development of CSCR is still unknown. It is possible that a constitutionally determined susceptibility of the posterior blood-retinal barrier must be present so that, under the effect of exogenous or endogenous glucocorticoids, CSCR develops [3]. Nevertheless, Kapetanios et al. [4] found elevated values of urinary free cortisol in patients suffering from CSCR not exposed to exogenous glucocorticoids and without biological signs of endogenous Cushing's syndrome. Psychologic stress has been known to produce hypothalamic-pituitary-adrenal axis abnormalities like endogenous cortical excess, which has been found in some patients with CSCR [5]. Additionally, some psychopharmacologic and other medications may be risk factors. In fact, the use of imidazole derivatives like oxy- and xylometazoline, which directly stimulate alpha-receptors and are more alpha-2-selective, are associated with the manifestation of CSCR [6, 7].

In our case, the use of xylometazoline may have enhanced the post-traumatic stress and induced adrenergic stimulation leading to an increase in the choroidal circulation and alteration of the pump action of the RPE, which is related with the development of the CSCR. The disappearance of the macular edema after the discontinuation of xylometazoline supports this hypothesis. Contrary to this hypothesis, Riva et al. [8] state that adrenergic stimulation surely increases blood pressure. However, the simultaneous increase in choroidal resistance has no effect on choroidal blood flow, which will be regulated to be maintained constant.

In summary, we present a case of CSCR after rhinoplasty, which to the best of our knowledge has not been previously reported. This case shows a possible association between the postoperative stress or the use of xylometazoline and CSCR and widens the spectrum of drugs associated with the occurrence of the disease.

Disclore Statement

The authors have no conflict of interest.

Case Rep Ophthalmol 2010;1:90–93
DOI: 10.1159/000322235
Published online:
November 6, 2010

ISSN 1663–2699
www.karger.com/cop

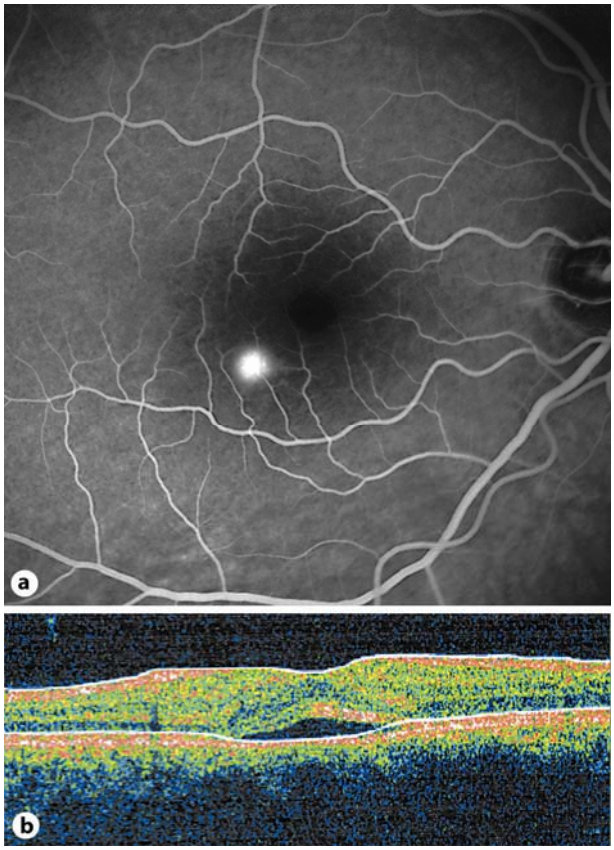

Fig. 1. At presentation, FA showed a leakage of the RPE within the macular avascular zone in the RE (**a**). OCT sections through the macula depict an increase in retinal thickness (**b**).

Case Rep Ophthalmol 2010;1:90–93
DOI: 10.1159/000322235
Published online: November 6, 2010

ISSN 1663–2699
www.karger.com/cop

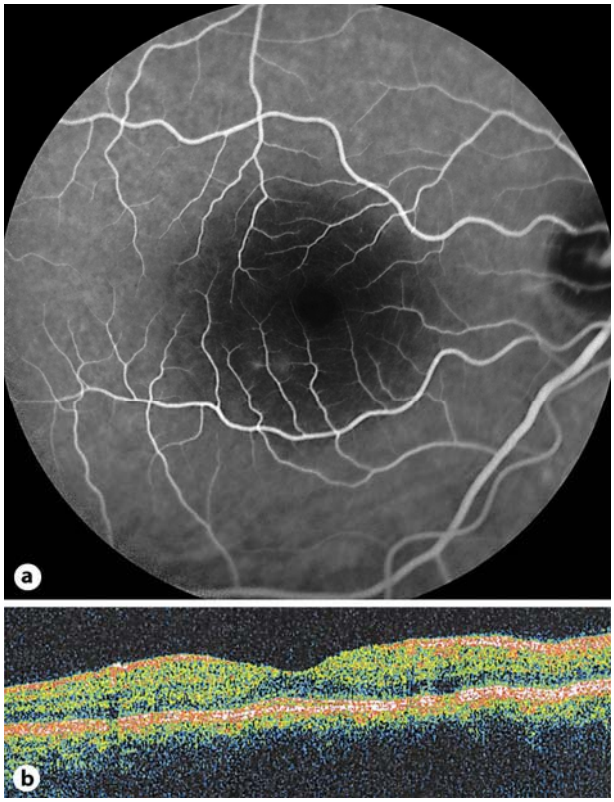

Fig. 2. One month after rhinoplasty, the leakage of the RPE in the RE disappeared (**a**). OCT sections through the macula depict a decrease in retinal thickness (**b**).

References

1 Gelber GS, Schatz H: Loss of vision due to central serous chorioretinopathy following psychological stress. Am J Psychiatry 1987;144:46–50.

2 Wakakura M, Ishikawa S: Central serous chorioretinopathy complicating systemic corticosteroid treatment. Br J Ophthalmol 1984;68:329–333.

3 Bouzas EA, Karadimas P, Pournaras CJ: Central serous chorioretinopathy and glucocorticoids. Surv Ophthalmol 2002;47:431–448.

4 Kapetanios AD, Donati G, Bouzas E, Mastorakos G, Pournaras CJ: Serum central chorioretinopathy and endogenous hypercortisolemia [in German]. Klin Monbl Augenheilkd 1998;212:343–344.

5 Garg SP, Dada T, Talwar D, Biswas NR: Endogenous cortisol profile in patients with central serous chorioretinopathy. Br J Ophthalmol 1997;81:962–964.

6 Michel JC, Pak J, Pulido D, de Venesis G: Central serous chorioretinopathy associated with sympathomimetic agents. Am J Ophthalmol 2003;136:182–185.

7 Lashmikanta M, Sayan B, Krishnapada B, Gautam B: Central serous chorioretinopathy after dacryocystorhinostomy operation on the same side. Indian J Ophthalmol 2009;57:57–58.

8 Riva CE, Titze P, Hero M, Petrig BL: Effect of acute decreases of perfusion pressure on choroidal blood flow in humans. Invest Ophthalmol Vis Sci 1997;58:1752–1760.