



Bilateral multifocal serous retinal detachments after multiple trauma: a case report

Obojestranski večžariščni serozni odstopi mrežnice po multiplih poškodbah: prikaz primera

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Abstract

We present a patient with bilateral multifocal serous retinal detachments after multiple trauma in a car accident without preceding corticosteroid treatment. A 37-year-old patient was admitted to our hospital after he had suffered multiple body injuries in a car accident. After being woken from an induced coma, he complained of blurry vision in his left eye, so he underwent a complete ophthalmic examination – several serous retinal detachments were found at the posterior pole of both eyes, along with thickened choroids. As there was no intraocular inflammation, accelerated hypertension or other changes indicating other causes of serous retinal detachments, and since we observed a steady spontaneous subretinal fluid resorption, we classified our case as an atypical central serous chorioretinopathy. Alternatively, serous retinal detachments could have occurred due to transient alterations in choroidal circulation following cerebral injury. In conclusion, this case report adds new data on the occurrence of serous retinal detachments and discusses their possible aetiology in the trauma setting. Care must be taken not to miss any other underlying pathology needing treatment.

Izvleček

Članek predstavi primer bolnika, pri katerem smo po multiplih poškodbah v prometni nesreči ugotavljali tudi obojestranske večžariščne serozne odstope mrežnice, čeprav se poškodovanec ni zdravil s sistemskimi kortikosteroidi. 37-letni bolnik je bil sprejet v našo bolnišnico po prometni nesreči, v kateri je utrpel več telesnih poškodb. Po prebuditvi iz umetne kome je navajal meglen vid na levo oko. Temeljit očesni pregled je pokazal obojestranske posteriorne serozne odstope mrežnice ob zadebeljenih žilnicah. Glede na odsotnost znotrajočesnega vnetja, maligne hipertenzije ali drugih sprememb, ki bi lahko kazale na druge vzroke seroznih odstopov mrežnice, in ker se je tekočina pod mrežnico počasi spontano resorbirala, smo postavili diagnozo atipična centralna serozna horioretinopatija. Serozni odstopi mrežnice bi lahko bili tudi posledica

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prehodnih sprememb v prekrvitvi žilnice po poškodbi možganov. Opisani primer dopolnjuje znanje o pojavnosti seroznih odstopov mrežnice; poleg poročila o primeru konkretnega poškodovanca predstavljamo možne vzroke zanje v povezavi s poškodbami. Pozorni moramo biti, da ne spregledamo morebitnih drugih vzrokov, ki bi morda potrebovali zdravljenje.

1 Introduction

Central serous chorioretinopathy (CSCR) is a macular disease of mainly adult men, characterized by subretinal fluid accumulation at the posterior pole of the retina, causing serous retinal detachment. The exact mechanism of CSCR is unknown, but it is thought to be due to choroidal capillaries hyperpermeability and associated retinal pigment dysfunction. Although considered idiopathic, it has been linked to diverse factors, including arterial hypertension, H. pylori infection, type-A personality type, steroid usage, and autoimmune disease (1).

It has been shown that not only steroid treatment (exogenous steroids) but also conditions that increase endogenous steroids (Cushing syndrome, major stress) can cause CSCR (1). We report an unusual case presenting as bilateral multifocal serous retinal detachments after multiple trauma in a car accident.

2 Case presentation

A 37-year-old Caucasian male was admitted to our hospital after being injured in a car accident. Head CT showed a left frontobasal haematoma, left orbital roof fracture with an intraorbital haematoma, a focal subarachnoid haemorrhage in the left insula, and a few smaller intracerebral haemorrhagic contusions on the right side of the brain, mainly deeper in the temporal lobe. He also suffered lung contusion, sternum fracture, and fractures of transverse processes of vertebrae L1, L2, and L3. In the acute setting, left lateral canthotomy was made due to proptosis of the left eye.

After being woken from an induced coma on the second day after the accident, he complained of blurry vision in his left eye and was subsequently seen by a consultant ophthalmologist. Given the patient's gradual recuperation, a first thorough visual function assessment and slit-lamp examination were possible only on the 11th day after the accident. The patient complained of prolonged light and dark adaptation and had already reported some improvement in the visual acuity of both eyes. He denied any ocular pain. Best-corrected visual acuity (BCVA) was 6/9 in his right eye (OD) and

6/10 in his left eye (OS). There was no more proptosis of OS. Ishihara's test was within normal limits for both eyes. Pupillary reactions were normal bilaterally with a negative RAPD. A visual field examination (Octopus, Haag-Streit) revealed left homonymous hemianopsia. Fundus examination showed normal optic discs and multiple serous retinal detachments perifoveolarly in the right and left macula. Swept-source OCT imaging (Triton, Topcon) confirmed serous retinal detachments perifoveolarly bilaterally and showed focal areas of subtle RPE rarefaction. Outer choroidal vessels were focally dilated (pachyvessels), which correlated spatially with serous retinal detachments (Figure 1A). Subfoveolar choroidal thickness measured 607 micrometers in OD and 612 micrometers in OS (as measured with the help of a built-in caliper tool from swept-source OCT scans).

There were no cells in the vitreous body and no retinal pigment epithelial detachments. Autofluorescence retinal imaging showed multiple focal decreased autofluorescence in the posterior pole (Figure 2).

Laboratory tests showed a normal complete blood count except for mild blood-loss anaemia and slightly increased CRP (37.7 mg/L), which decreased to a normal value only a few days after the accident.

The patient's past medical history was unremarkable except for a juvenile euthyroid goitre in adolescence and a tonsillectomy in childhood. The patient was without regular medical therapy before the accident. He denied prior eye problems, stress, regular alcohol consumption, smoking, or illicit drug use.

During the patient's hospital stay, we observed an improvement of his visual acuity and a relatively fast and steady resorption of subretinal fluid (Figure 1).

At the time of the acute stage of CSCR, the patient was systemically too unwell to hold out longer-lasting diagnostic imaging such as fluorescein and indocyanine angiography. Due to convincing spontaneous improvement in serous retinal detachments, we considered it unnecessary later on. BCVA at the control examination nine weeks after the accident was 6/6 bilaterally. There was only a trace of subretinal fluid left

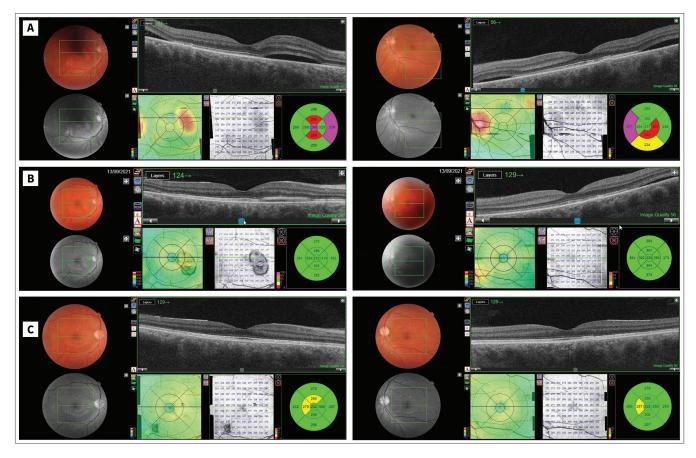


Figure 1: OCT images showing serous fluid resolution with time.

The images were taken on the 11th (A), 16th (B), and 66th (C) day after the accident; the left column represents the right eye, and the right column the left eye. On the 66th day after the accident, there was only a trace of subretinal fluid left perifoveolarly superotemporally on the left eye, not visible in the bottom right image due to a central cross-section of the OCT image. Source: archives of the Department of Ophthalmology, Izola General Hospital.

perifoveolarly superotemporally on the left eye, which had completely resolved by the following examination seven months after the accident. Homonymous hemianopsia has persisted to date.

3 Discussion

Connection of CSCR to blunt eye trauma and rarely to orbital trauma has been reported previously, usually describing an ipsilateral CSCR (2-6). Only two reports describe CSCR development in a contralateral eye (7,8). Bilateral CSCR after trauma is described only as an occurrence after systemic corticosteroid treatment for trauma-related complications like traumatic optic neuropathy or Berlin's oedema (9,10).

Our case is, to the best of our knowledge, the first describing bilateral serous chorioretinopathy after trauma in the absence of preceding corticosteroid treatment. The cause could lie in the fact that our patient suffered multiple body injuries rather than only an eye or an orbital injury, which could lead to a higher increase in endogenous corticosteroids (11,12). Moreover, CSCR in our case presented multifocally perifoveolarly in both eyes and not as a monofocal subfoveolar serous retinal detachment as classically. Our patient also didn't exhibit serous pigment epithelial detachments, which are otherwise common in CSCR.

Our patient showed no signs of intraocular inflammation, accelerated hypertension or neoplastic process (choroidal infiltration or metastatic disease), indicating other potential causes of exudative retinal detachment (13-16). After excluding the latter, the question remains whether serous retinal detachments after trauma should even be categorized as central serous chorioretinopathy (17). Firstly, CSCR is considered idiopathic, i.e., without a known cause. However,

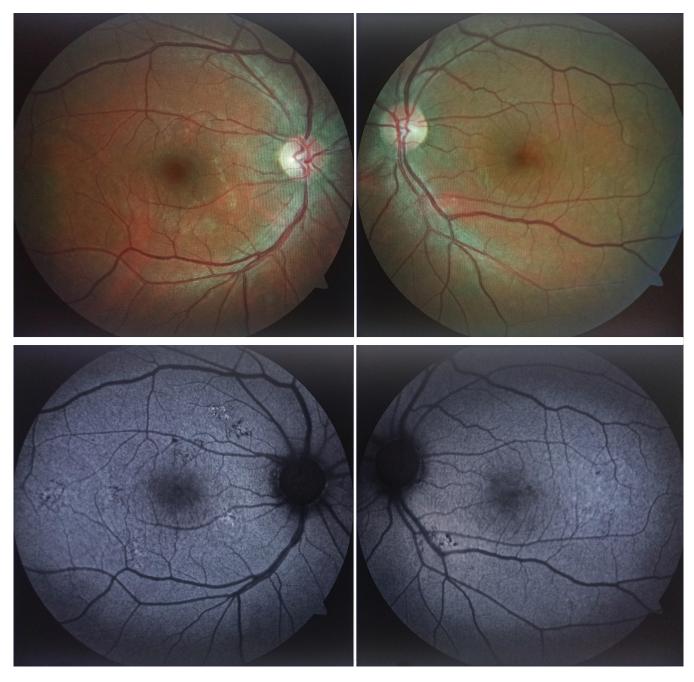


Figure 2: Colour fundus photography (the upper two) and fundus autofluorescence (the lower two images).

Images were taken on the 20th day after the accident. Autofluorescence imaging shows multiple focal hypo- and hyperautofluorescences at the posterior pole of both eyes, corresponding to serous retinal detachments faintly visible on the colour fundus photographs.

Source: archives of the Department of Ophthalmology, Izola General Hospital.

trauma could be considered as a precipitating factor rather than a direct cause of CSCR by increasing endogenous corticosteroids, which then cause CSCR in predisposed eyes (6). Secondly, CSCR has known atypical presentations, and our knowledge about them and the pachychoroid spectrum has been evolving lately. Since any other possible causes of multiple serous retinal detachments seemed clinically highly unlikely, since there was a pachychoroid present and since we observed a steady spontaneous resorption of subretinal fluid, we believe that the most appropriate characterization of our case is an atypical trauma-related CSCR presentation. Alternatively, transient alteration in choroidal circulation could have been the cause of serous retinal detachments in our case. Transiently increased intracranial pressure after head injury (due to haemorrhage or cerebral oedema) can lead to hindered venous outflow, causing choroidal congestion and fluid leakage into the subretinal space. A traumatic caroticocavernous fistula could result in the same consequences by increasing the intraorbital venous pressure (18), but this was not the case in our patient.

4 Conclusions

Our case report expands the knowledge about the possible occurrence of serous retinal detachments. Bilateral multiple serous retinal detachments after a traumatic insult in the absence of preceding corticosteroid treatment could present a case of atypical central serous chorioretinopathy or alternatively occur as a consequence of transient post-traumatic alterations in choroidal circulation. Other causes of serous retinal detachments should be excluded, and care must be taken not to miss any underlying pathology needing treatment.

Declaration of conflicting interests

The authors declare no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

Declaration of patient consent

The authors confirm that they have obtained the patient's consent to report his clinical information and images in the journal. The patient understands that his name will not be revealed.

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