compression injuries.[9] It is caused by forcible exhalation against a closed glottis, thereby creating a sudden increase in intrathoracic/intra-abdominal pressure, which leads to rapid rise of intraocular venous pressure with rupture of perifoveal capillaries and a preretinal haemorrhage.

Purtscher's retinopathy, traumatic optic neuropathy and Valsalva retinopathy are rare entities associated with trauma that have different clinical presentations caused by dissimilar mechanisms, and it is very uncommon to observe them together in either eye as seen in our case. In spite of the severe nature of the ocular injury, it is surprising that our patient was systemically stable with no significant bony or organ injury. Compressive thoracic injury has varied ocular presentations caused by ill-understood pathophysiological mechanisms and new ocular findings associated with it continue to baffle us.

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Sir,

**Treatment of polypoidal choroidal vasculopathy with transpupillary thermotherapy: an interventional case report**

A 71-year-old Indian male patient presented to the retina–vitreous service of our tertiary care hospital with the complaints of progressive decrease in vision in the right eye over the past 6 months. Ocular examination revealed a best-corrected visual acuity of 6/12 and 6/6 (distance vision) and N18 and N6 (near vision) in the right and the left eye, respectively. Fundus examination of the left eye was unremarkable. Indirect ophthalmoscopy of the right eye revealed a serous detachment of the macula with extensive lipid exudation and an area of thickening superior to the disc suspicious of a choroidal neovascular membrane (CNVM) (Figure 1a). Fundus fluorescein angiography (FFA) demonstrated a progressively increasing irregular hyperfluorescence in that region and pooling of dye in the serous detachment in the macula but was otherwise noncontributory to the diagnosis. Indocyanine green angiography (ICGFA) was carried out that showed a branching vascular network in the inner choroid ending in two terminal hyperfluorescent polypoidal dilatations in the superior juxtapapillary region suggestive of leaking polypoidal choroidal neovascularisation (CNV) (Figure 2a). There were also two areas of ill-defined hyperfluorescence superior and superonasal to the foveola. Based on the above findings, a diagnosis of polypoidal choroidal vasculopathy (PCV) of the right eye was made.

In view of the long duration of persisting poor visual acuity, the macular involvement, and the abundant lipid suggesting an active exudative process, it was decided to treat the leaking polypoidal lesions. After informed consent, the patient underwent transpupillary thermotherapy (TTT) to the juxtapapillary vascular network inclusive of the polypoidal lesions (2 spots, 2 mm in size, 250 mW power, 1 min duration, end point: no visible reaction). Follow-up at 1 month revealed a resolved macular detachment and a reddish-orange subretinal lesion superior to the fovea that was typical of PCV (Figure 1b). The ICGFA revealed the disappearance of the treated polypoidal lesions (Figure 2b). It also showed a row of aneurysmal dilatations of the inner choroidal vascular network superior to the fovea,

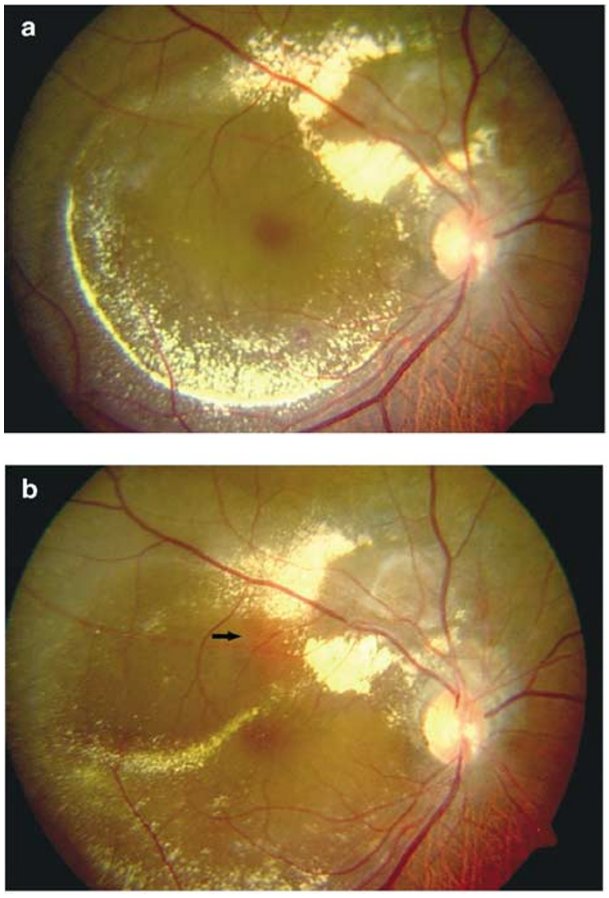


**Figure 1** (a) Pretreatment colour fundus photograph of the right eye of the patient showing the serous detachment of the macula ringed by subretinal lipid. (b) Post-treatment colour fundus photograph of the right eye at first month of follow-up of the patient showing the resolved macular detachment. A round reddish-orange lesion (black arrow) is seen superior to the fovea.

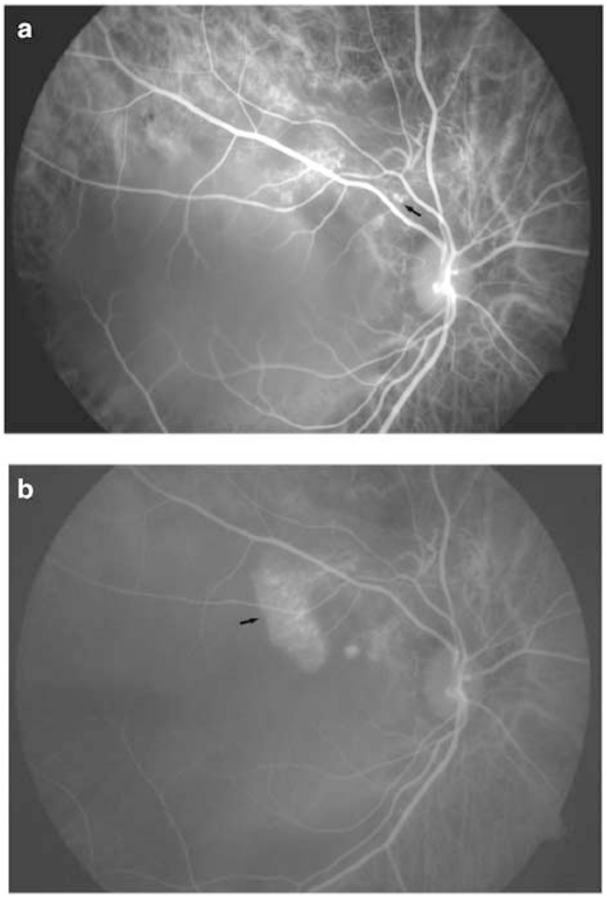


**Figure 2** (a) Pretreatment indocyanine angiogram of the right eye of the patient showing two small hyperfluorescent polypoidal lesions superior to the disc (small black arrow). (b) Post-treatment indocyanine angiogram of the right eye of the patient at first month of follow-up showing a row of hyperfluorescent polypoidal lesions with surrounding leakage (small black arrow) superior and superonasal to the fovea. Note the disappearance of the previously seen polypoidal lesions superior to the disc.

corresponding to the reddish-orange lesion and a hyperfluorescent polypoidal lesion superonasal to the fovea. These lesions corresponded to the ill-defined hyperfluorescent lesions seen in the pretreatment ICGFA, and were in all probability previously obscured by the overlying serous detachment. Alternatively, these could represent newly opened vascular channels, following closure of the treated PCV lesions. The patient's near vision had improved to N10 in the right eye, while his distant vision was maintained at 6/12. The patient's condition remained stable when last seen at 6 months of follow-up.

## Comment

PCV is a distinct form of CNV termed polypoidal CNV that is characterised by recurrent serosanguineous detachments of the macula.[1] We are aware of several reports of patients with PCV who have had spontaneous resolution due to autoinfarction.[1] However, in an attempt to restore the normal physiologic retinal pigment epithelium (RPE)–choroidal transport barriers and relationships in this case with a long-standing sensory detachment of the macula, we decided to treat the leaking polypoidal lesions.

Various management options including observation,[1] argon,[1] and diode[2] laser photocoagulation, photodynamic therapy (PDT)[3] have been advocated for this condition. However, there is no report till date of TTT as a treatment modality in this condition. We report a case of polypoidal CNV managed by TTT. This is the first report of its kind in English literature to the best of our knowledge (as per Medline search).

Gomez-Ulla *et al*[2] treated a case of PCV with diode laser and have argued that its absorption at the level of

the retinal pigment epithelium (RPE), enables the selective treatment of the choroidal lesions along with sparing of the overlying neurosensory retina. TTT that also uses the diode laser has been used successsfully to treat CNV in age-related macular degeneration (AMD).[4] It has been suggested that TTT causes closure of the choroidal vasculature by hyperthermia-induced endothelial damage.[5] We thought we could use this property of TTT and the longer wavelength of diode laser to close selectively the choroidal vascular network of the leaking juxtapapillary PCV in this case. We did not do PDT in this case since the patient could not afford it. We did not treat the hyperfluorescent lesions superior and superonasal to the fovea since they were not clearly defined in the ICGFA.

The disappearance of the leaking polypoidal lesions following treatment has been seen following laser photocoagulation and PDT as well.[1–3] Our case illustrates the efficacy of TTT in the management of PCV. We do concede the need for future prospective randomised controlled studies to establish clearly the safety and efficacy of TTT in the management of PCV.

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Sir,

**Helicoidal peripapillary degeneration**

This exceptionally rare bilateral fundus disorder was first described as choroiditis areata, in Iceland.[1,2] Later, it was renamed helicoidal peripapillary chorioretinal degeneration because of the lesion's resemblance to the propeller (helix) of an aeroplane.[3] Today, in the light of modern genetics, we believe that it is more precise to use the term dystrophy than degeneration, because it is obviously a dominantly inherited disorder.[4] A dominant form was recently mapped to chromosome 11p15.[5] Finally, a novel TEAD1 mutation is recognized as the causative allele in helicoidal peripapillary degeneration.[6]

We have had an opportunity to study three cases of inherited helicoidal peripapillary chorioretinal degeneration: a father, daughter, and son. Although this particular disorder has mostly been observed in Scandinavia, this is the first known case presented in Serbia.

***Case reports***

*Case one*

A 20-year-old female patient experienced some visual disturbances as a child and was referred to an ophthalmologist 10 years ago. Unusual atrophic peripapillary change was noticed and helicoidal peripapillary atrophy was diagnosed. Visual acuity was 6/6 for the right eye and 6/12 for the left eye.

At a routine check-up in July 2003, visual acuity had decreased to RE 6/9 with −1.75 D cyl axis 15° and LE 6/12 with −2.25 D cyl axis 170°. Intraocular pressure was normal. Discrete cataracts were observed (an unusual distribution in the anterior lens cortex) with very suggestive helicoidal peripapillary chorioretinal atrophy with tongues next to the fovea on both eyes. A fluorescein angiography was performed (Figure 1a, b).

*Case two*

Her 15-year-old brother had the same disorder, confirmed 7 years ago. His visual acuity was 6/9 with −0.75 D cyl axis 180° for both eyes, but the intraocular