

Letter to the Editor

Promising visual improvement of cystoid macular oedema by hyperbaric oxygen therapy

Erik C. Jansen¹ and Niels Vesti Nielsen²

¹Hyperbaric Unit, Department of Anaesthesia and Operation Services, Rigshospitalet, Copenhagen University Hospital, Copenhagen, Denmark

²Eye Clinic, Centre for Head and Orthopaedics, Rigshospitalet, Copenhagen University Hospital, Copenhagen, Denmark

doi: 10.1111/j.1600-0420.2004.00305.x

Sir,

Cystoid macular oedema occurs as a frequent complication in diabetic retinopathy, retinal vein occlusions, uveitis and postoperatively in cataract surgery. In general, macular oedema is most prevalent in age-related macular degeneration (AMD)

Ischaemia and/or inflammation may pathologically explain the development of macular oedema associated with these disorders. No effective, specific treatment by laser, systemic or topical steroids, haemodilution or surgery is available.

Hyperbaric oxygen (HBO) treatment has been used in extraocular ischaemic disorders. The hypothesis of hypoxia as a predominant cause of cystoid macular oedema prompted us to start HBO therapy in two patients with chronic macular cystoid oedema refractory to other treatment modalities. Therefore, we present here these two patients, each of whom experienced longlasting visual improvement after HBO.

The first patient was a 57-year-old man with type 2 diabetes, with diabetic onset in 1998. He had suffered the complication of preproliferative retinopathy and severe cystoid maculopathy for 6 months prior to presentation. His visual acuity (VA) was 0.5 and he had pronounced metamorphopsia and reading disability. He had a stabilized glycaemic metabolism and normal blood pressure.

The second patient was a 56-year-old man with cystoid maculopathy complicated with idiopathic juxtafoveal retinal telangiectasis (group 1A) on the right eye, with VA 0.2 excentric, paracentral scotoma and reading blindness. The patient had a well treated arterial hypertension.

Both patients were given a series of five HBO treatments of pure oxygen for

90 min at a pressure of 2.4 bars absolute in a pressure chamber.

Before and after the daily HBO treatment, each patient was tested for visual function by Amsler chart, reading ability and VA (Snellen chart). They also underwent an ophthalmological examination which included testing of the central visual field, biomicroscopy of the anterior eye segment, assessing intraocular pressure, direct and indirect ophthalmoscopy, fundus colour photography, fluorescein angiography (FA) and ocular coherence tomography (OCT).

The diabetes patient improved increasingly during the treatment sessions, both subjectively and objectively, as shown in Fig. 1. Visual acuity in the right eye improved from 0.5 to 1.0, he achieved normal reading vision and metamorphopsia disappeared. Follow-up at 8 months showed the improvements to be unchanged.

The second patient experienced visual improvement after 3 days, with VA improving from 0.2 to >0.9, normal reading vision and no metamorphopsia. Fluorescein angiography and

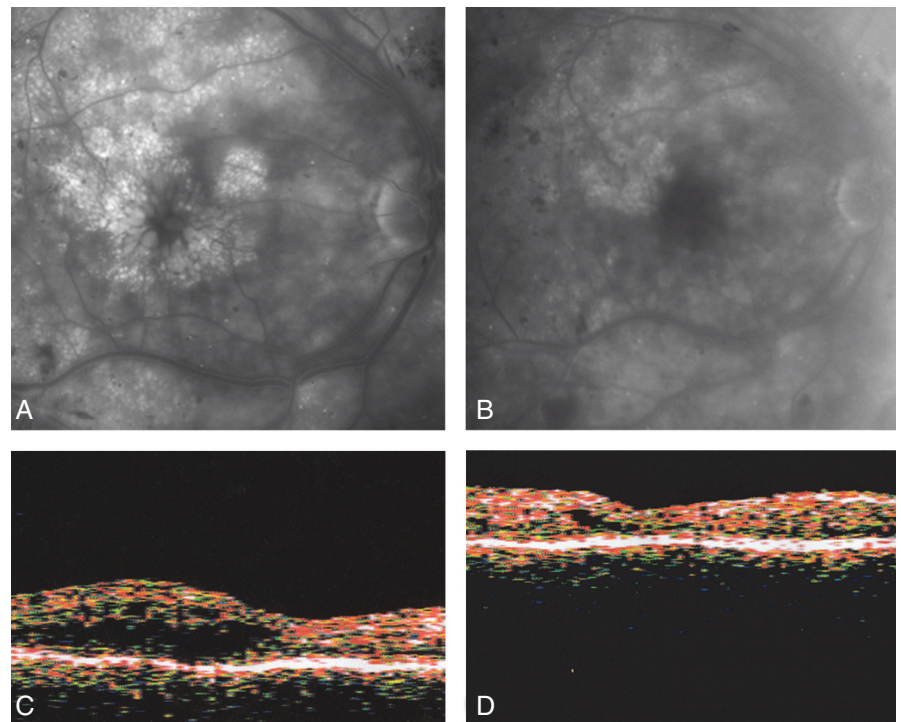


Fig. 1. Late phase fluorescein angiography (FA) of the right eye of the diabetes patient at day 0 (A) and day 5 (B) after HBO treatment. The cystoid oedema has nearly disappeared. Optical coherence tomography (OCT) of the same eye at day 0 (C) and day 3 (D) after HBO treatment. The cystoid oedema is reduced.

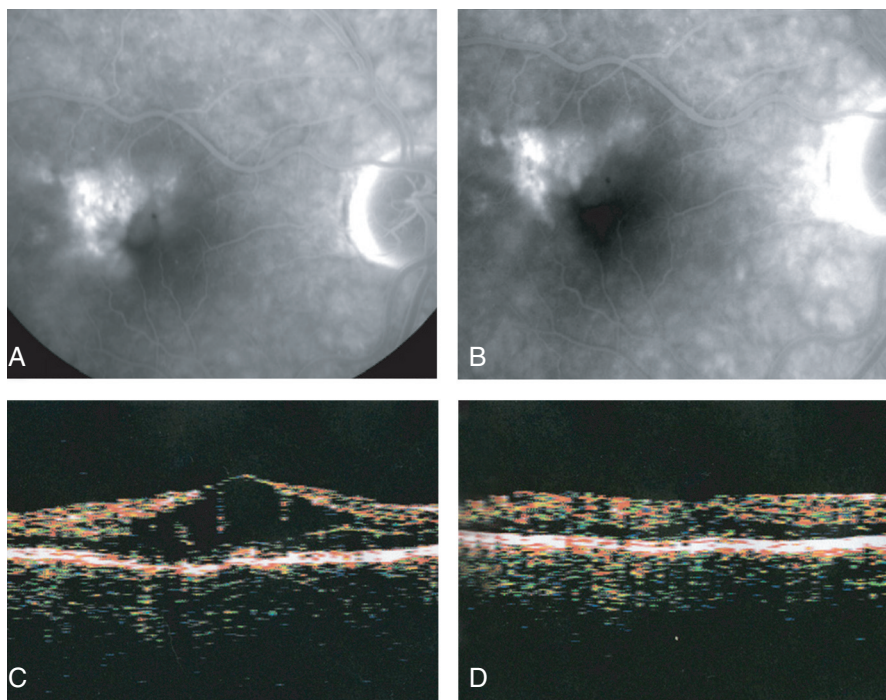


Fig. 2. Late phase FA of the right eye of the non-diabetic patient at day 0 (A) and day 5 (B) after HBO treatment. The cystoid oedema is clearly reduced. Optical coherence tomography of the same eye at day 0 (C) shows a pronounced reduction of cystoid oedema as early as 2 days (D) after beginning HBO treatment.

OCT demonstrate the results (Fig. 2). No recurrence of symptoms or macular oedema was seen at the 7-month follow-up. The patient still performs auto control by Amsler chart and his reading ability and improvements are unchanged.

Hyperbaric oxygen therapy provided an outstanding and very rapid improvement in visual function and macular oedema despite the long duration of the disorder. This report seems to be the first in the literature to include a day-by-day objective documentation by OCT showing the absorption of the macular oedema associated with improved visual function. Surprisingly, the improvement in vision

occurred shortly after the first treatment with HBO and the patients obtained normal vision after the very short series of HBO treatments. More surprisingly, the improvements have been shown to be longlasting. The therapeutic effect of HBO is caused by a 17-fold increase in the arterial oxygen tension. Several studies have shown a precapillary arterial contraction in cerebral and retinal tissue. This may reduce the oedema of the tissue. The high oxygen diffusion from the choroidal vasculature may further reduce the relative ischaemia of the tissue, improving the oxidative phosphorylation and thereby improving retinal function and reducing oedema. Hyper-

baric oxygen has been used in the short-term treatment of acute brain oedema. Further studies are needed to establish whether the improvement in vision is due solely to the treatment of the oedema or whether retinal function is also improved during HBO treatment. A previous study by Krott et al. (2002) found HBO adjunctive in the treatment of macular oedema. The authors gave an average of 15 HBO treatments and found no alteration in the oedemas of their diabetes patients, while the macular oedemas in patients with retinal vein occlusions were reduced. One might assume that the treatment should be commenced as soon as possible before fibrotic and thereby irreversible degeneration occurs. However, the present cases demonstrate that normal reading vision can be obtained for patients after a short series of HBO treatments. This finding deserves further research.

References

- Krott R, Heller R, Aisenbrey S & Bartz-Schmidt K-U (2002): Adjunctive hyperbaric oxygenation in macular oedema of vascular origin. *J Undersea Hyperbaric Med* **20**: 195–204.

Correspondence:

Dr Erik C. Jansen
 Clinic of Anaesthesia and Operation Services
 Rigshospitalet
 Blegdamsvej 9
 DK-2100 Copenhagen
 Denmark
 Tel: +45 35 45 12 57
 Fax: +45 35 45 29 50
 Email: jansen@rh.dk