Letters to the Editor

Proliferative retinopathy during hyperbaric oxygen treatment

A 43-year-old male with type 2 diabetes mellitus, treated with insulin for 28 years and with an HbA1c of 7.9% six months prior, suffered from bilateral plantar ulcers refractory to specialised wound care. He underwent a planned 40 sessions of hyperbaric oxygen treatment (HBOT) at 243 kPa for 90 minutes. Consent was given for this report.

The patient's ophthalmic history included bilateral proliferative diabetic retinopathy (PDR) identified on routine diabetic eye screening three years previously. This was treated with pan-retinal photocoagulation (PRP). Three months before starting HBOT, he underwent phacoemulsification and intra-ocular lens insertion of his left eye, having had the same procedure done to his right eye a year prior, without complication. He was reviewed again one week prior to his first HBOT and fundoscopy confirmed non-proliferative diabetic retinopathy (NPDR) without evidence of PDR.

The patient had a routine follow up by the ophthalmologist following his fifth HBOT when fundoscopic examination revealed pre-retinal haemorrhage, a form of PDR, in his left eye. This was treated with PRP at the time. His visual acuity, 6/9 bilaterally, had not changed, nor did he describe any changes in his visual field despite these findings.

He was seen three weeks later (following 12 further HBOT) when fundoscopy showed worsening proliferative changes, this time in both eyes. Bevacizumab was injected at the time and fill-in PRP performed the following week. His visual acuity remained unchanged in both eyes. At this point, HBOT was withheld to allow the proliferative phase of the patients' retinopathy to remit.

The potentially adverse effects of hyperbaric oxygen to the retinal vasculature of diabetic patients was postulated in 1994 following a similar experience, albeit without a baseline fundoscopic examination.¹ In particular, the concern was of accelerating the proliferative process of retinopathy with subsequent irreversible loss of vision. Thereafter, routine screening and treatment of all diabetic patients for PDR was adopted at our facility. Until now, there have been no further cases of NPDR evolving into PDR at three-month review following HBOT. Indeed, a brief literature search using the terms "retinopathy", "complications", "adverse effects", "vitreous", "hemorrhage", "haemorrhage", "hyperbaric" and "oxygen" has not found any other cases described.

In a double blind, randomised trial (meeting abstract only) of 15 diabetic patients with both NPDR and PDR, patients in neither the HBOT (243 kPa for 90 min) nor the control arm had evidence of neovascularisation nor worsening of their proliferative retinopathy at three-month follow up.²

The significance of PDR following cataract surgery has also been considered. A review article consistently found that NPDR progression occurred in up to a third of such patients. Despite this, there were no cases of NPDR progressing to PDR at 12-month follow up.³

Whether this patient's sudden progression to PDR was related to HBOT, recent cataract surgery or another unknown factor is unclear. However, the temporal relationship to 17 HBOT is difficult to explain and appears more rapid than available data regarding vascular regrowth in wound healing would suggest.

References

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Key words

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