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CASE REPORT

Intravitreal bevacizumab for the treatment of choroidal neovascularization secondary to pseudotumor cerebri

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Abstract *Background* In pseudotumor cerebri (PTC), elevated intracranial pressure (ICP) results in papilledema and, rarely, choroidal neovascularization (CNV). Pseudotumor cerebri-induced CNV often regresses following medical or surgical ICP reduction, but additional treatments, such as photocoagulation, photodynamic therapy, peri-ocular steroid injections and/or subretinal surgery, may be necessary. Anti-angiogenic intravitreal injections have been shown to cause regression of both CNV and optic nerve edema. *Case report* We describe a patient with PTC and CNV whose CNV regressed and vision normalized after a single intravitreal injection of bevacizumab (Avastin; Genentech, San Francisco, CA).

Keywords Bevacizumab · Choroidal neovascularization · Pseudotumor cerebri

Case report

A 30-year-old woman presented with transient vision obscurations (TVO) in her left eye. The best-corrected visual acuity (BCVA) was 20/20 in the right eye (OD) and 20/25 in the left eye (OS), with a relative afferent pupillary defect (RAPD) OS. Funduscopy revealed 1+ papilledema OD and 3+ OS, with a peripapillary choroidal neovascularization (CNV) and hemorrhages OS. Neuroimaging results of the brain were normal. A lumbar puncture opening pressure was 33.5 cm of water, and cerebrospinal fluid analyses were normal. She was diagnosed with PTC and started on acetazolamide 500 mg twice per day. A week later, the TVO improved, but the BCVA was 20/25 OD and 20/400 OS. The papilledema was unchanged, but there was extension of the CNV toward the foveal avascular zone OS as well as an increase in the amount of subretinal fluid (Fig. 1).

The patient was treated with an intravitreal injection of bevacizumab (1.25 mg in 0.05 ml) OS. One week later, the BCVA was 20/20 OD and 20/50 OS, with a trace RAPD OS. Nine weeks later, the BCVA was 20/20 OD and 20/25 OS, and the RAPD had resolved. The papilledema had improved in each eye, and the CNV had regressed, with a residual fibrotic scar temporal to the optic nerve OS (Fig. 2). Her visual acuity, visual field and fundus examination have remained stable for 10 months following the single injection of bevacizumab.

Discussion

Pseudotumor cerebri has an estimated incidence of 0.9 per 100,000, with approximately 0.5% of these

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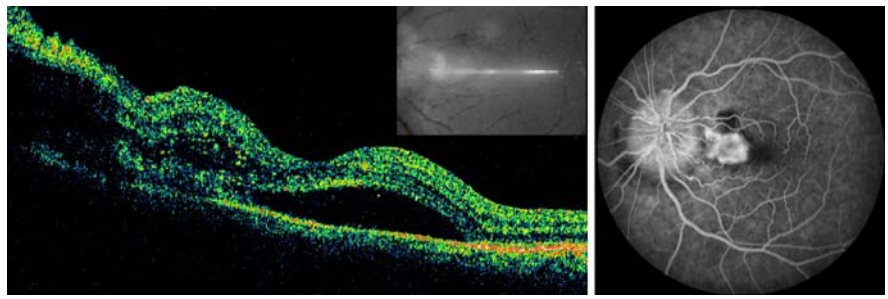

Fig. 1 Choroidal neovascularization prior to bevacizumab injection. The location of the juxtafoveal CNV prior to intravitreal injection of bevacizumab as identified by optical coherence tomography (*left*) and fluorescein angiography (*right*)

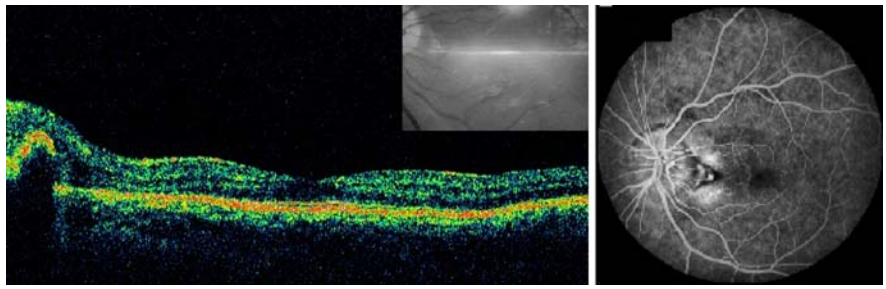

Fig. 2 Choroidal neovascularization after intravitreal bevacizumab. Decreased optic nerve swelling and fibrotic remnants of the choroidal neovascularization, as identified with both optical coherence tomography 8 months after treatment *(left)* and fluorescein angiography 2 months after treatment (*right*)

patients developing CNV [1, 2]. There have been 19 reported cases of PTC with CNV formation [2–9]. Treatments have included medical or surgical lowering of the ICP, photocoagulation, photodynamic therapy, peri-ocular steroid injections and subretinal surgery. Despite treatment, the final BCVA declines as the CNV progresses from its peripapillary origin toward the fovea. This may be due to CNV, macular edema, hemorrhage, damage to the papillo-macular nerve fiber bundle secondary to laser treatment, or any combination of these. Of the four juxtafoveal CNV cases that have been reported, all were treated with laser photocoagulation, with the BCVA decreasing from 20/20 initially to 20/25, 20/40, 20/200 and 20/200, respectively [2, 6, 7]. In contrast, our patient also had a juxtafoveal CNV with a pretreatment BCVA of 20/400. However, after receiving one intravitreal bevacizumab injection, the BCVA returned to normal.

One hallmark feature of PTC is papilledema, which may contribute to CNV formation through different mechanisms. Disruption of Bruch's membrane allows the ingrowth of vessels from the choriocapillaris, and this dehiscence may result from optic nerve swelling. Such a mechanism would be supported by the increased proportion of peripapillary CNV cases reported. Optic nerve swelling also disrupts axoplasmic flow, creating a hypoxic environment [8]. This may allow upregulation of angiogenic factors. In one study of radiation-induced optic neuropathy, optic nerve edema was shown to resolve after intravitreal bevacizumab [5].

To our knowledge, this is the first patient with PTC and CNV that received an intravitreal bevacizumab injection. Three months after a single treatment, swelling of the CNV and optic nerve resolved. In addition, the BCVA returned to baseline and has remained stable for 10 months, in contrast to the decreased BCVA in the previously reported cases of PTC with juxtafoveal CNV. Therefore, further investigation of intravitreal bevacizumab injections for PTC with CNV is warranted.

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