Intracranial pressure following optic nerve decompression for benign intracranial hypertension

Case report


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The authors report the case of a patient with benign intracranial hypertension and severe papilledema, who underwent surgery for bilateral optic nerve sheath decompression. No change in the intracranial pressure (ICP) was seen during continuous recording performed before and after the operation. This case supports the contention that the decrease in papilledema and the visual improvements seen following this operation are more likely to be due to decrease in optic nerve sheath pressure than to a generalized decrease in ICP, as suggested by other authors.

KEY WORDS • benign intracranial hypertension • intracranial pressure • optic nerve sheath decompression • papilledema • pseudotumor cerebri

B enign intracranial hypertension or pseudotumor cerebri usually follows a self-limiting course, and the affected patients have minimal long-term side-effects. However, in a small percentage of cases, severe papilledema may lead to visual deterioration and visual failure.

In addition to conservative medical treatment, such as encouraging weight loss and the administration of steroids and glycerol, various surgical techniques have been tried to ameliorate this sequela of raised intracranial pressure (ICP). These procedures have included bilateral subtemporal decompression, lumboperitoneal shunting, and optic nerve decompression. Optic nerve decompression has been shown to reverse visual deterioration in patients with benign intracranial hypertension. Whether this is due to a direct release of pressure on the head of the optic nerve, or to a lowering of ICP following a leakage of cerebrospinal fluid (CSF) from the windows cut in the dural sheath surrounding the optic nerve, has not been unequivocally demonstrated.

This paper reports a patient with benign intracranial hypertension and visual failure. Bilateral optic nerve sheath decompression procedures were performed, and the ICP was measured continuously for 24 hours before and 48 hours after the operation.

Case Report

This 51-year-old obese woman was referred to us for evaluation following 14 months of constant headache.

Examination. She was found to be mildly hypertensive and had bilateral papilledema. Her corrected visual acuity was 6/12 in the right eye and 6/6 in the left eye, with normal visual fields. A computerized tomography brain scan was normal and, at lumbar puncture, CSF pressure was 330 mm H₂O, with a normal fluid analysis. Despite commencing a weight reduction diet and glycerol, 15 ml 4 times per day, her optic discs became progressively more swollen over the next 6 months, and she developed transient visual obscurations. Bilateral optic nerve sheath decompression procedures were performed (J.E.K.G.).

Operation. Details of the operation have been given fully in previous reports, and are described in brief...
Fig. 1. Upper: Preoperative intracranial pressure (ICP) tracing showing baseline systolic pressure of about 30 mm Hg. Lower: The postoperative ICP tracing was not significantly different from the preoperative tracing.

here. After the insertion of an eyelid speculum, a fornix-based conjunctival flap of 180° was fashioned on the nasal side of the globe, and radial incisions were made at each end of the flap. Traction sutures were then looped beneath the tendons of the vertical rectus muscles. The conjunctiva and Tenon’s capsule were reflected to expose the medial rectus muscle, which was detached from the globe and marked with a suture. A modified three-bladed tracheal dilator was inserted medial to the globe, so that the paired blades just rested against the globe and displaced it laterally, and the unpaired blade, which had been modified by the addition of a hook, retracted Tenon’s capsule toward the medial orbital wall.

Firm anterolateral traction of the vertical rectus sutures prolapsed the globe, retracted it laterally, and exposed the optic nerve in the depths of the wound. The operation was continued using the operating microscope. The short ciliary vessels were displaced to expose the dural sheath, which was incised with straight alligator stapedectomy scissors.

As the dura and arachnoid are adherent,2,12 the subarachnoid space was entered with ease. A 3 × 5 mm window was excised from the sheath. On both sides, CSF was noted to flow freely from the dural windows. The retractor was then removed, the medial rectus muscle was reattached to the globe, and the conjunctiva closed. Antibiotic ointment was instilled into the conjunctival sac, and the eye was covered for 24 hours.

The ICP was monitored continuously by means of a subarachnoid screw connected by a 150-cm saline-filled tube to a Hewlett Packard transducer and then to a Hewlett Packard amplifier connected to a continuous paper recorder.* Zero reference point for the

* Model 1280C transducer was connected to a 78200 series patient monitor with a Model 78205B amplifier, manufactured by Hewlett Packard Corp., San Diego, California. This was connected to a Hewlett Packard 7803B monitor scope and to a Rikadenki single-channel recorder, Model B-104 (modified by the Royal Melbourne Hospital Electronics Department for two-channel multiplex operation) manufactured by Rikadenki, Kogyo Co., Ltd., Tokyo, Japan.
Optic nerve decompression for pseudotumor

transducer was taken as a point 3 cm anterior to the
external auditory meatus, with the patient lying su-
prine. The zero drift and gain drift of the system were
checked and corrected at least every 6 hours if
ecessary.

Very little correction was required at any time.
Preoperatively, the ICP had a baseline systolic value
of around 30 mm Hg. The amplitude of swing was 8
to 10 mm Hg. B-waves were present for approxi-
ately 10% of the monitoring time, and these rose to
a height of 45 mm Hg (Fig. 1 upper). After the bilateral
optic nerve sheath decompression, the ICP did not
change significantly (Fig. 1 lower).

Postoperative Course. The patient was continued on
glycerol, the bilateral papilledema subsided rapidly,
and the obscurations of vision improved. The cor-
rected visual acuity was 6/6 in both eyes 2 months
postoperatively.

Discussion

Benign intracranial hypertension usually runs a
self-limiting course with a good prognosis. However,
a number of long-term sequelae have been noted,
including visual failure, empty sella syndrome, and endocrine abnormalities.

Visual acuity may be reduced in up to half of the
patients with benign intracranial hypertension, but
the long-term prognosis with regard to vision is subject to
variation in opinion. Rabinowicz, et al., noted no
significant deterioration in the visual function of pa-
tients with papilledema following benign intracranial
hypertension who were observed from 3 to 6 years.
However, other authors report a low but significant
level of severe permanent visual impairment.

It is quite evident that elevation of CSF pressure
plays a significant role in the production of papil-
ledema. Hayreh studied the pathogenesis of edema of the optic disc. He produced edema after
introducing a rubber balloon into the cranial cavity
of 35 rhesus monkeys. He not only established that
the raised ICP was essential in the production of edema of the disc, but he also showed the site of
action of this raised ICP. To this end, a window was
cut in the sheath of the optic nerve on one side to
relieve the pressure in the sheath space. This led to
the disappearance of edema of the disc on that side or
prevented the development of that edema. The con-
tralateral side did not show a similar response. Hay-
reh postulated from this that a rise of pressure in
the sheath of the optic nerve was essential for production of edema of the disc.

De Wecker first described the procedure of optic
nerve decompression for visual failure. Since then this
technique has been reported by other authors.

The exact mechanism of action of optic nerve de-
compression is still unclear. It has been suggested
that the ICP is lowered due to a leak of CSF from the
dural windows cut in the nerve sheaths. This hypothe-
sis would explain the relief of symptoms noted to
occur following optic nerve decompression, and the
improvement of papilledema in the contralateral eye
that has been reported following unilateral decom-
pression, although neither of these findings are
universal. Hayreh found that unilateral decom-
pression rarely lowered the lumbar CSF pressure
or relieved contralateral papilledema in monkeys.
However, he did show that incision of the optic nerve
sheath relieved the pressure of CSF on the optic nerve.
This local effect of decreased pressure on the optic
nerve is an alternative explanation for the mechanism
of optic nerve sheath decompression for papilledema
in benign intracranial hypertension.

This case study would indicate that there is no drop
in ICP following optic nerve sheath decompression.
Rather, the improvement in papilledema seems to be
due to the local effect of decreased pressure on the
optic nerve head.

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