Clinical Policy: Optic Nerve Decompression Surgery

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Effective Date: 09/16
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Coding Implications
Revision Log

See Important Reminder at the end of this policy for important regulatory and legal information.

Description

Optic nerve sheath decompression involves direct decompression (fenestration) of the optic nerve sheaths just behind the globe. The approach and technique for an optic nerve sheath fenestration varies. This policy describes the medical necessity requirements for optic nerve decompression surgery.

Policy/Criteria

I. It is the policy of health plans affiliated with Centene Corporation® that optic nerve sheath decompression surgery is **medically necessary** for the treatment of papilledema accompanying idiopathic pseudotumor cerebri (increased intracranial pressure) for either of the following:

- **A.** Visual function that is severely impaired or continues to deteriorate, despite aggressive medical management (e.g., Diamox [acetazolamide], furosemide, and corticosteroids); or
- **B.** Incapacitating headaches.

II. It is the policy of health plans affiliated with Centene Corporation® that optic nerve sheath decompression surgery is **investigational** for the treatment of non-arteritic anterior ischemic optic neuropathy (NAION) or traumatic optic neuropathy.

Background

Optic nerve sheath decompression surgery is typically performed in instances of papilledema due to idiopathic intracranial hypertension (IIH), in which the main symptom is rapid and/or progressive vision loss rather than headache. The effect is normally limited to the ipsilateral optic nerve, although occasionally the procedure appears to have a filtration effect, resulting in improvements in headaches and contralateral disc edema, as well.

Idiopathic intracranial hypertension

Idiopathic intracranial hypertension (IIH), also known as pseudotumor cerebri, is a disorder defined by clinical criteria which include symptoms and signs isolated to those produced by increased intracranial pressure (e.g., headache, papilledema, vision loss), elevated intracranial pressure with normal cerebrospinal fluid composition, and no other cause of intracranial hypertension evident on neuroimaging or other evaluations. The incidence of IIH in the general population is thought to be about 1 per 100,000. In obese, young females the incidence of IIH is about 20 per 100,000. IIH occurs in men and children, as well, but with substantially lower frequency. Weight is not usually a factor in men and in children under 10 years of age. Many individuals suffer from intractable, disabling headaches, and there is a risk of severe, permanent vision loss. Individuals with mild vision loss have an associated reduction in quality of life. Recommendations for the treatment of IIH are limited due to a lack of randomized controlled trials. In addition, the natural history of untreated IIH is uncertain.

The goals of treatment are to detect and prevent vision loss, to reduce the intracranial pressure, and to relieve headache. Medical treatment consists of first line treatment with Diamox (acetazolamide), which inhibits choroid plexus carbonic anhydrase and reduces cerebrospinal fluid production by 50 to 60%. Furosemide (Lasix®) and corticosteroids can be added. Surgery is reserved for patients whose visual function is severely impaired or continues to deteriorate despite aggressive medical management. Those who suffer incapacitating headaches may also be candidates for surgery.

Surgical options include optic nerve sheath decompression and lumboperitoneal shunting. However, prevailing opinion seems to favor the former. This procedure has been found to be highly effective for relief of papilledema. In fact, following a unilateral procedure, most patients have improvement in bilateral disc swelling and in severity of headache. Stabilization or improvement of vision occurs in an estimated 85 to 100% of patients. Visual function is greatly improved in patients with acute rather than chronic papilledema. Thus, in patients with significant visual loss, waiting a prolonged period for a response to medical therapy may not be warranted. Optic nerve sheath decompression also may improve visual function in patients with progressive visual loss despite functioning lumboperitoneal shunts.

Nonarteritic anterior ischemic optic neuropathy
Nonarteritic anterior ischemic optic neuropathy (NAION) is the most common form of ischemic optic neuropathy. It is an idiopathic, ischemic insult of the optic nerve head characterized by acute, monocular, painless visual loss with optic disc swelling. Visual function can be impaired through decreased central visual acuity or peripheral field loss, or both. The typical presentation is sudden onset of painless monocular vision loss, often upon awakening

Optic nerve sheath decompression surgery was reported in 1989 to be of benefit to patients with NAION. The presumed mechanism of action in optic nerve decompression surgery revolved around restoration of impaired blood flow to the optic nerve through reduction of the pressure around the nerve. Initial results of uncontrolled studies suggested that optic nerve sheath decompression was a promising treatment of progressive visual loss in patients with NAION. Other investigators who evaluated this surgical procedure reported varying degrees of success. To resolve the controversy over the effectiveness of optic nerve decompression for NAION, the National Eye Institute sponsored the Ischemic Optic Neuropathy Decompression Trial, a multicenter, randomized controlled clinical trial of optic nerve decompression surgery for patients with NAION. The study found no benefit from surgery in NAION patients with progressive visual loss; in fact, significantly more patients in the surgery group had progressive loss of vision than patients who received only careful follow-up. The investigators concluded that optic nerve decompression surgery is not an effective treatment for NAION and, in fact, may increase the risk of progressive visual loss in NAION patients. The trial was stopped early because the surgery was not helping the participants more than careful follow-up alone. Pain and double vision were harms experienced by some participants in the surgery group at one week after the surgery. The trial investigators reported that continued enrollment would be unlikely to produce results in favor of surgery.

Traumatic optic neuropathy

Traumatic optic neuropathy (TON) is an important cause of severe visual loss following blunt or penetrating head trauma. Following the initial insult, optic nerve swelling within the optic nerve canal or compression by bone fragments are thought to result in secondary retinal ganglion cell loss. Optic nerve decompression with steroids or surgical interventions or both have been advocated to improve visual prognosis in TON. However, there is no evidence that surgery improves visual outcome in traumatic optic neuropathy. The current body of evidence for TON consists mostly of small, retrospective case series.

In a retrospective review of 109 individuals with TON, Sosin et al (2016) evaluated recent institutional trends in the treatment of traumatic optic neuropathy, assessed the outcomes of different treatment strategies, and identified factors associated with improved vision. The reviewers reported management included intravenous corticosteroids alone in 8.3 percent of patients (n = 9), 56.9 percent (n = 62) underwent observation, 28.4 percent (n = 31) had surgical intervention, and 6.4 percent (n = 7) underwent surgery and corticosteroid administration. Only 19.3 percent of patients returned for follow-up. The reviewers reported optic nerve decompression has fallen out of favor at the reviewer's institution, and observation is the most common management strategy. Outcomes following corticosteroid administration and observation were comparable. Li et al (2008) reported that there was no difference (statistically) between steroids and steroids plus optic nerve decompression in treating TON. At this time, there is a lack of randomized, controlled trials evaluating the role of optic nerve decompression surgery in TON.

Coding Implications

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CPT® Codes	Description
67570	Decompression optic nerve (e.g., incision or fenestration of optic nerve sheath.

HCPCS Codes	Description
N/A	

ICD-10-CM Diagnosis Codes that Support Coverage Criteria

ICD-10-CM Code	Description
G93.2	Benign intracranial hypertension
H47.011-H47.019	Other disorders of optic (2nd) nerve and visual pathways
H47.10-H47.13	Papilledema

Reviews, Revisions, and Approvals	Date	Approval Date
Policy adopted from Health Net NMP353 Optic Nerve Decompression Surgery	08/16	09/16

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Important Reminder

This clinical policy has been developed by appropriately experienced and licensed health care professionals based on a review and consideration of currently available generally accepted standards of medical practice; peer-reviewed medical literature; government agency/program approval status; evidence-based guidelines and positions of leading national health professional organizations; views of physicians practicing in relevant clinical areas affected by this clinical policy; and other available clinical information. The Health Plan makes no representations and accepts no liability with respect to the content of any external information used or relied upon in developing this clinical policy. This clinical policy is consistent with standards of medical practice current at the time that this clinical policy was approved. “Health Plan” means a health

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This clinical policy is effective as of the date determined by the Health Plan. The date of posting may not be the effective date of this clinical policy. This clinical policy may be subject to applicable legal and regulatory requirements relating to provider notification. If there is a discrepancy between the effective date of this clinical policy and any applicable legal or regulatory requirement, the requirements of law and regulation shall govern. The Health Plan retains the right to change, amend or withdraw this clinical policy, and additional clinical policies may be developed and adopted as needed, at any time.

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Note: For Medicaid members, when state Medicaid coverage provisions conflict with the coverage provisions in this clinical policy, state Medicaid coverage provisions take precedence. Please refer to the state Medicaid manual for any coverage provisions pertaining to this clinical policy.

Note: For Medicare members, to ensure consistency with the Medicare National Coverage Determinations (NCD) and Local Coverage Determinations (LCD), all applicable NCDs, LCDs, and Medicare Coverage Articles should be reviewed prior to applying the criteria set forth in this clinical policy. Refer to the CMS website at http://www.cms.gov for additional information.