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Surgical Treatment of Greater Occipital Neuralgia: an Appraisal of Strategies

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Summary

The surgical treatment of greater occipital neuralgia often involves diagnostic anesthetic blockade, followed by chemical or surgical ablation of the greater occipital nerve. The anatomy of this region was studied in microdissections of 2 cadaver specimens. The diagnosis and management of a series of 5 patients with greater occipital neuralgia is discussed. Two patients were treated with atlanto-epistrophic ligament decompression of the C2 dorsal root ganglion and nerve; four patients had C2 ganglionotomy performed. All patients in this series had immediate complete relief of pain following surgery. Patients were followed for a mean of 24 months (range 7–33 months). One patient had a recurrence of her original pain after 26 months following atlanto-epistrophic ligament decompression and required re-operation in the form of bilateral C2 ganglionotomy. All patients experienced transient nausea and dizziness in the several days following surgery. One patient had an incisional cerebrospinal fluid leak.

Microsurgical C2 gangliotomy is advocated as the preferred surgical treatment of greater occipital neuralgia of idiopathic origin.

Keywords: Occipital neuralgia; rhizotomy; ganglionotomy.

Introduction

Greater occipital neuralgia is characterized by a lancinating pain extending from the suboccipital region up to the cranial vertex. The pain is less often described as including or consisting of a dull aching. It may be idiopathic, or may appear following a history of cervical trauma. Neck movement may aggravate the pain.

Some underlying medical diseases have been reported as causes of this entity. These include neurosyphilis[11], temporal arteritis[8], vascular compression[6], C1–2 arthrosis[4], inflammatory disorders and postherpetic neuralgia[15], and occipital adenopathy[3, 5]. Other diagnoses that must be excluded are migraine, and glossopharyngeal, trigeminal, or geniculate neuralgia. These diagnoses may be excluded by anatomic location which is quite specific for occipital neuralgia in producing a suboccipital and vertex location of pain. Migraine may be excluded by different temporal patterns and characteristic involvement of the entire hemicranium.

The treatments for this disorder have included diagnostic nerve blocks in the occipital area, with subsequent neurolytic injections directed at destruction of the nerve, or surgical peripheral neurectomy of the greater occipital nerve[5]; local anesthetic blocks of the second cervical ganglion[1]; C2 or C3 ganglionectomy[14]; and percutaneous radiofrequency lesions of the C2 ganglion or root[13]. A treatment recently proposed was atlanto-epistrophic ligament decompression[10], which refers to division of the condensation of the atlanto-axial interlaminar ligament that engulfs the C2 ganglion and surrounding venous plexus.

The present work was undertaken to investigate the efficacy of this treatment, as well as surgical C2 ganglionotomy in the treatment of greater occipital neuralgia. In addition, cadaveric microdissection was performed to help elucidate the anatomy of the region of the C2 ganglion.

Methods

Laboratory

Two formalin fixed cadaver specimens were obtained. Surgical microdissection was performed through a midline incision, and the C2 dorsal root ganglion was exposed (see Fig. 1). In all specimens, there was a dense vertebral venous plexus which enveloped the ganglion. This varied in size between sides and between specimens. The ganglion was enclosed within a layer of connective tissue, which in some portions was quite thin. The distances to the adjacent bony

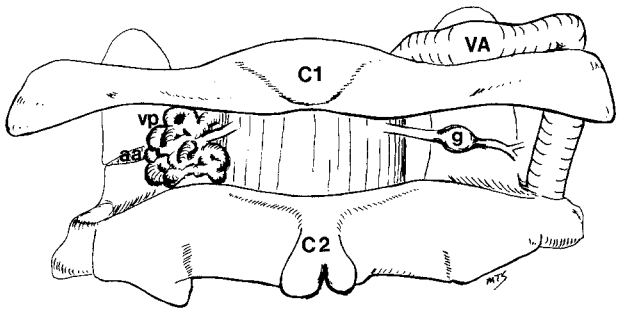

Fig. 1 a. Illustration showing the C 2 dorsal root ganglion and its relationship to the vertebral artery (*VA*), C 1 arch, and the atlantoaxial joint (*aa*) ventral to the ganglion. The venous plexus (*vp*) that encases the ganglion is shown on the right

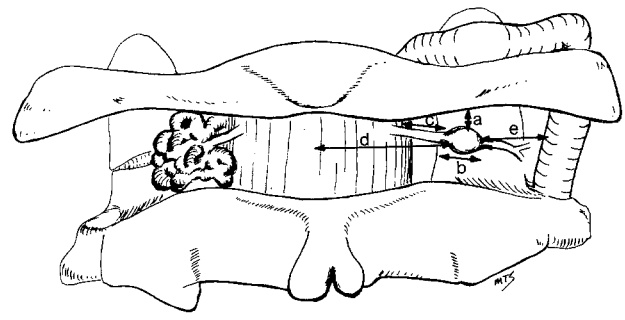

Fig. 1 b. The distances from the C 2 dorsal root ganglion to the adjacent structures are summarized. The distance from the rostral surface of the ganglion to inferior arch of C 1 (*a*) was 2 mm. The average width of the ganglion (*b*) was 4.5 mm. The length of the root from its dural exit to the medial portion of the ganglion (*c*) was 2–4 mm. The average distance from the midline measured along the circumference of the spinal cord to the proximal aspect of the ganglion was 15 mm. The distance from the lateral aspect of the ganglion to the medial surface of the vertebral artery which was situated ventro-lateral to the ganglion (*e*) was 17 mm

structures which could compress the ganglion were measured. The effects of neck rotation, and neck flexion and extension were noted on the relationship of the ganglion to adjacent bony elements.

Clinical Series

Five patients underwent a total of 6 surgical treatments for greater occipital neuralgia. All patients were female, with mean age of 39 years. Four patients had previous surgical treatment by peripheral neurectomy with return of the pain to intolerable severity within a mean time period of 11 months (range 2–24 months). Four patients presented with typical lancinating pain alone, while two had a dull aching component. Cervical spine X-rays were normal in every case. The ages, characterization and location of pain, previous tratment, duration of complaints, presence of sensory deficit, history of antecedent trauma and surgical procedure are summarized in Table 1. In the two patients with a history of trauma, there was no fracture. The exact latency period between trauma and the onset of the occipital neuralgia was difficult to determine as both patients had considerable pain that was different immediately after the injury. In both patients, the pain syndrome was recognized and treated within twelve months. Patient CH had a neurofibroma resected from the parietal area of the scalp on the side ipsilateral to her pain. Patient NS has a suboccipital incision from previous surgery for vagoglossopharyngeal neuralgia.

Two patients were treated with atlanto-epistrophic ligament decompression of the C 2 dorsal root ganglion; 3 patients treated with C 2 ganglionotomy. Surgery was performed on the patients in the prone position, with the head held in slight flexion in Mayfield pins. A midline incision was made to allow exposure from foramen magnum to C 3. The paraspinal muscles were dissected subperiosteally

Table 1. *Pre-operative Clinical Data*

Patient: age/sex	Pain: location/ character	History of trauma	Response to block	Sensory deficit	Previous surgery	Duration of symptoms
TW 45/F	bilateral/ lancinating	head/neck beatings	relief	R/hypalgesia	R/neurectomy	8 years
CG 45/F	bilateral/ lancinating	closed head injury	relief	anesthesia	bilateral neurectomy	14 years
CH 37/F	R/dull	none	relief	none	neurofibroma resection	1,5 years
CM 24/F	bilateral/ steady ache	none	relief	L/hypalgesia	bilateral neurectomy	6 years
NS[a] 47/F	L/lancinating	sub-occipital incision	relief	anesthesia	neurectomy	7 years

[a] Co-existing glossopharyngeal neuralgia.

from the arch of C 1 and C 2 to permit wide exposure. The ganglion was exposed by opening the atlanto-epistrophic ligament. The vertebral venous plexus was cauterized and divided sufficiently to permit exposure of the ganglion. In the 2 decompression cases, closure was then performed. In the ganglionotomy cases, the C 2 ganglion was cauterized, and then divided with microscissors near its proximal junction with the dorsal root. There was one case in which the ring of C 1 was partly removed inferiorly to facilitate the exposure. This was not required in any other case.

Results

Laboratory

The anatomic relations of the C 2 dorsal root ganglion are summarized in Fig. 1 which shows the measurements of the distance from the ganglion to the adjacent structures. In a neutral neck position, the distance from the rostral surface of the ganglion to the inferior surface of the C 1 vertebral arch was 2 mm. The average width of the C2 ganglion measured 4.5 mm. The length of the root from its dural exit to the medial portion of fusiform C 2 ganglion was 2–4 mm. The distance from the midline measured along the circumference of the spinal cord to the medial aspect of the ganglion averaged 15 mm. The distance from the lateral aspect of the C 2 ganglion to the medial surface of the vertebral artery was 17 mm.

Attempts were made to compress the ganglion between the arch of C 1 and the C 2 lamina during extremes of flexion, extension, and rotation. Despite a good range of movement, the ganglion was just contacted by bone in extreme extension, but it was never compressed. Extremes of neck rotation were performed to assess whether the ganglion could be compressed between the atlanto-axial joint anteriorly, and the soft tissues posteriorly. No significant approximation occurred. In order to view this, the posterior tissues were removed, however, and may have allowed more movement of the ganglion away from the joint than would normally occur in vivo.

Clinical

Surgical Treatment

All 5 surgically treated patients had immediate complete relief of pain following surgery. Patients were followed for a mean of 24 months postoperatively (range 7–33 months). One patient undergoing bilateral ganglion decompression had recurrence of her pain 26 months following her procedure. She was re-operated on and had bilateral C 2 ganglionotomy performed. She has remained pain-free during her 7 months of follow-up thus far (see Table 2). All patients experienced transient nausea and dizziness postoperatively in the several days following surgery. In all cases this lasted 1–2 days, with the exception of one case under-

Table 2. *Operation and Postoperative Clinical Data*

PT	Type of operation	Unilateral/ bilateral	Postoperative complications	Length of follow-up	Special operative notes	Relief	Repeat	Postop. vomiting, dizziness
TW	decompression	bilateral	CSF leak	33 months		initial complete	bilateral ganglion-otomy 26 months postop.	+
CG	ganglionotomy	bilateral	none	32 months		complete	–	+
CH	decompression	unilateral (R)	new hypalgesia/ hypesthesia	29 months	massive vertebral plexus	complete	–	+
CM	ganglionotomy	bilateral	none	16 months		complete	–	+
NS	ganglionotomy	unilateral (R)	none	11 months	massive vertebral plexus	incomplete (present above vertex)	–	+

going bilateral ganglionotomy in whom dizziness and nausea persisted for 1 week. There was an incisional cerebrospinal fluid leak in one patient (bilateral decompression) which resolved after 3 days of lumbar drainage. Postoperative sensation in the distribution of the greater occipital nerve remained abnormal in the 4/5 patients who had this pre-operatively. In the one patient (CH) in whom there was normal sensation preoperatively, this changed after unilateral C 2 ganglion decompression. New onset of hypalgesia and hypesthesia was present in the suboccipital region extending up to the vertex ipsilateral to the surgery.

Discussion

A series of 5 patients is described with occipital neuralgia. Previous treatment of this condition by peripheral occipital neurectomy had been performed elsewhere in 4/5 of these patients. All of these patients had experienced recurrence of their pain. All patients had relief of this pain by surgery consisting of decompression of the C 2 dorsal root ganglion and nerve (2/5), or by performing C 2 ganglionotomy (3/5). The C 2 ganglion decompression procedure has been advocated as an effective treatment in 2 patients[10]. In our series of 5 surgically treated patients, one of the 2 patients treated by C 2 ganglion and nerve decompression had a recurrence after 28 months. We did not simply re-explore and perform neurolysis of the nerve as advocated by Poletti *et al.*[10], but performed a ganglionotomy.

The advantage of decompression over ganglionotomy is the theoretical preservation or provision for return of sensation in the distribution of the greater occipital nerve. This is tempered by the possible risk of pain recurrence, as the series of Poletti *et al.* reports re-operation on one of their two patients[10]. The enthusiasm for ganglionotomy over decompression in the present series is explained on this basis. Patients were anxious to choose the procedure that the surgeon felt had the least likelihood of being associated with recurrence. Eighty percent of the present series consisted of patients who had sensory deficit in the distribution of the greater occipital nerve due to previous peripheral neurectomy procedures, so preservation of sensory function was not a consideration. The one patient in whom there was normal sensation pre-operatively was noted to have new onset of hypesthesia and hypalgesia in the suboccipital and occipital-vertex region after unilateral ganglion decompression. This raises the possibility of thermal injury playing some therapeutic role, as the exposure of the ganglion always involves abundant bipolar cautery to facilitate division of the venous plexus that engulfs the ganglion.

The postoperative complication of nausea and vomiting was disturbing to all patients, and lasted for as long as one week. There is evidence from axonal tracer studies that there is convergence in the dorsal medulla of the afferent fibers from the upper cervical joints with second order vestibular projections[12]. This circuitry, which was postulated to be involved in the mediation of the cervico-ocular reflex, may explain the postoperative symptoms of nausea and dizziness that were seen in all patients of the present series. The anatomical studies that have been performed[1], and the cadaver dissections of the present work have demonstrated that the environment of the C 2 ganglion is different from all others owing to the uniqueness of the C 1–2 articulation. The ganglion is not protected within a bony enclosure, but rather is housed in a ligamentous structure, the atlanto-epistrophic ligament. This is intimately associated with the vertebral venous plexus. It is conceivable that an abnormally prominent venous plexus may be etiologic in compression of the ganglion. Similarly, hypertrophy of the lamina or C 1–2 articulation combined with the fixed position of the nerve and ganglion in connective tissue could conceivably result in compression of the ganglion. Because of this special anatomy, it is conceivable that injury may occur to the C 2 nerve and ganglion that may not occur at other levels. The special predilection has been alluded to by Hunter and Mayfield[7]. A history of antecedent trauma was noted in all but one of the patients of the current series. Our anatomical studies in cadaver specimens do not confirm the feasibility of a direct bony crush of the ganglion in hyperextension. However, the compression of the ganglion by the compaction of it indirectly in neck hyperextension by the surrounding soft tissues may still be sufficient to render injury and subsequent pain.

It is concluded from this work that the disabling pain of greater occipital neuralgia can be successfully treated by consideration of the unique anatomy of this region. For patients in whom pain from the syndrome of greater occipital neuralgia is disabling to the point of interferring in their ability to perform their job or participate in recreational activities, then treatment of this condition is required. If medical therapy with analgesics, non-steroidal anti-inflammatories and finally neuralgia relieving agents such as carbamazepine proves ineffectual, then surgical treatment is indicated.

The responsiveness to peripheral greater occipital nerve block may not be predictive of therapeutic response. However, blockade of the C 2 root and ganglion should ameliorate the pain[1, 10]. While some early relief might be achieved with the surgery of atlanto-epistrophic ligament decompression, there was a recurrence of pain in 50% of the patients treated in this manner both in our series and that of Poletti *et al.*[10]. It is important to also consider evidence suggesting that the C 3 and even C 4 dorsal root ganglia may play a role in pain in the suboccipital region and that treatment directed at these structures may be warranted in some of these cases[9]. The technique of percutaneous radiofrequency lesion of the C 2 ganglion may be a reasonable alternative to surgery, but the enveloping of the C 2 ganglion by venous plexus, and the proximity to the vertebral artery creates a risk of vascular injury, as well as the possibility of spinal cord injury if the needle placement is not ideal. Based on the results of the present series and a consideration of the above factors, we advocate the performance of microsurgical C 2 ganglionotomy as the procedure of choice for idiopathic greater occipital neuralgia when medical treatment is unsuccessful.

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