Referred Ocular Pain Relieved by Suboccipital Injection

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Pain originating from ophthalmic disease has been well documented. A series of patients presenting with eye or peri-orbital pain attributed to cervical region dysfunction were diagnosed and treated with injections of subcutaneous lidocaine followed by triamcinolone acetonide. Twelve patients, 11 women and 1 man, ranging in age from 20 to 82 years had an evaluation including a complete eye examination, and laboratory tests and neuroimaging as dictated by the history to exclude structural abnormalities or systemic disease. All patients had marked focal suboccipital tenderness ipsilateral to the side of their headache and eye pain. A subcutaneous injection with 2% lidocaine followed by triamcinolone acetonide 40 mg was administered directly to the site of focal tenderness. After injection, five patients described total relief of pain, five patients described some degree of pain relief, and two patients had no relief of headache. Duration of pain relief ranged from several hours to 3 months. Patients may present with periorbital or eye pain as a result of disease affecting the cervical sensory roots with subsequent stimulation of the trigeminal apparatus. Subcutaneous injection of lidocaine and triamcinolone acetonide may be of help in the diagnosis of these patients and provide temporary relief.

Key words: occipital neuralgia, ocular pain, cervicogenic headache

(Headache 1995;35:101 - 103)

Head and facial pain originating from ophthalmic disease such as uveitis, acute angle-closure glaucoma, temporal arteritis, and orbital inflammatory disease has been well documented. We report a series of patients presenting with eye or periorbital pain as their primary complaint. Pain referred or originating from cervical region dysfunction was felt to be the etiology of their ophthalmic complaints.

PATIENTS AND METHODS

Patients were seen in the neuro-Ophthalmology clinic of the Cleveland Clinic Foundation, a tertiary referral center. Patients who described a pain characterized as dull and predominantly hemicranial with prominent radiation to the ipsilateral periorbital area and often the ipsilateral ear were identified (Table 1). A complete ocular examination, including slit-lamp biomicroscopy and dilated fundus examination, was performed, and found to be unremarkable for any ophthalmic etiology for the pain. Neuroimaging was performed in those with a history of head or neck trauma to exclude structural lesions. Laboratory evaluation including a Westergren sedimentation rate and a C-reactive protein measurement was performed in patients over age 55 years. In all patients, marked, focal, suboccipital tenderness could be elicited with palpation ipsilateral to the side of the periorbital pain.

Table 1.- Pain Distribution by Location

<table>
<thead>
<tr>
<th>Patient</th>
<th>Occipital</th>
<th>Temporal</th>
<th>Frontal</th>
<th>Ear</th>
<th>Eye</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td></td>
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<td>++</td>
<td>++</td>
<td>++</td>
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<tr>
<td>2</td>
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<td>+</td>
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<td>3</td>
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<td>+</td>
<td>+</td>
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<td>4</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>++</td>
<td>+</td>
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<td>6</td>
<td>+</td>
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<td>++</td>
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<td>7</td>
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<td>8</td>
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<td>-</td>
<td>-</td>
<td>+</td>
<td>-</td>
<td>+</td>
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<td>10</td>
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<td>-</td>
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<td>+</td>
<td>+</td>
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<td>12</td>
<td>+</td>
<td>-</td>
<td>-</td>
<td>+</td>
<td>+</td>
</tr>
</tbody>
</table>

Patients were given 5 mL of 2% lidocaine subcutaneously using a 25 gauge special 11/2 inch needle at the location of the palpably tender occipital scalp. This area corresponded to the region superior to the apical insertions of the sternocleido-mastoid and trapezius muscles over the occipital bone (Figure 1, site A). Patients were evaluated 15 minutes after the injection with assessment as to the subjective degree of pain relief. If pain was relieved, patients were given an injection of triamcinolone acetonide 40 mg/mL subcutaneously in the identical manner and placement as the lidocaine injection.

RESULTS

Twelve patients, 11 women and 1 man, ranging in age from 20 to 82 years (average, 55 years) were evaluated and treated. Four patients described the onset of pain as acute; seven described an insidious onset; one patient was uncertain as to the mode of onset. Nine patients characterized their pain as being constant since onset. All patients
denied symptoms consistent with the diagnosis of cluster headache. In six patients, the pain had been present for over 6 months. None had a previous diagnosis or history of migraine. Eight described worsening of the pain with neck movement. None had systemic symptoms suggestive of giant cell arteritis. Four gave a history of prior cervical or head trauma (two patients reported a "whiplash" injury sustained in a vehicular car accident, one had prior removal of an acoustic neuroma, one had undergone surgical decompression of an Arnold-Chiari malformation). An additional patient had complaints suggestive of an upper extremity radiculopathy, ipsilateral to the side of the headache.

Five patients underwent an MRI scan and one had a CT scan of the head. One patient had cervical spine x-ray films. None of the imaging studies revealed a cause for the pain. Five patients had both the Westergren sedimentation rate determined, and the C-reactive protein level determined; all were within the normal range for their respective ages.

Ten subjects experienced some degree of pain relief immediately following the injection of lidocaine. After injection with triamcinolone acetonide, these 10 patients described various durations of pain relief (Table 2). Nine had pain relief for greater than 1 week, with one individual having extended relief for 3 months. One patient, who had pain relief after lidocaine injection, eventually had a permanent nerve block.

Table 2.-Duration of Pain Relief After Suboccipital Injection

<table>
<thead>
<tr>
<th>Duration of Relief</th>
<th>No. of Patients</th>
</tr>
</thead>
<tbody>
<tr>
<td>2-3 months</td>
<td>2</td>
</tr>
<tr>
<td>1-2 months</td>
<td>2</td>
</tr>
<tr>
<td>1 week-1 month</td>
<td>6</td>
</tr>
<tr>
<td>1 day-1 week</td>
<td>0</td>
</tr>
<tr>
<td>&lt;1 day (hours)</td>
<td>1</td>
</tr>
<tr>
<td>No relief of pain</td>
<td>2</td>
</tr>
</tbody>
</table>

COMMENTS

Referral of pain from the neck to the head and face is believed to occur by one or more of multiple mechanisms.\textsuperscript{2} Stimulation of the C2 sensory root by compressive, irritative, or inflammatory mechanisms may elicit such pain.\textsuperscript{3} Additional neurogenic mechanisms include stimulation of a C1 sensory root, and stimulation of a tentorial nerve which provides an anastomosis between the trigeminal nerve and C2.\textsuperscript{4} Anatomically, the nucleus of the trigeminal spinal tract, which primarily sub-serves the transmission of pain and temperature impulses, extends to the second cervical segment and merges with the substantia gelatinosa\textsuperscript{5} (Figure 2). Experimental stimulation of the upper cervical sensory roots results in potentials being generated in the trigeminal apparatus.\textsuperscript{6} Myogenic head pain may occur if spasm of neck muscles initiates spasm in scalp musculature. Thus, cervical region disease may directly, or by referral through the trigeminal nerve, result in head pain.

While the IHS classification of headache acknowledges occipital neuralgia,\textsuperscript{7} "cervicogenic" headache has been used in the past as a nonspecific term for head pain originating from the neck. These headaches are characterized by nonexcruciating, long-lasting (without clustering), unilateral attacks with signs of neck involvement. In a series of 11 patients reported by Fredriksen et al,\textsuperscript{8} all patients had pain periorbitally, in the temporal region, and in the lower occipital region. Most described the pain as having a boring quality. Attacks could be initiated by pressure on specific neck areas. While "cervicogenic" headache does meet the Vahlquist\textsuperscript{9} and Bousser\textsuperscript{10} criteria of a migraine headache, cervicogenic headaches usually last longer, have precipitating neck mechanisms, and are typically minimally responsive to migraine medications.\textsuperscript{8}

Treatment of suboccipital neuralgia in patients with pain of the eye, orbit, or temple was documented by Knox and Mustonen.\textsuperscript{11} They used 1 mL of 2% lidocaine with the optional addition of 2 mg of a soluble corticosteroid. In keeping with our observation, they also noted "tenderness to pressure on one or both greater occipital nerves," a preponderance of women with this head pain complaint, and success with the treatment regimen.
of suboccipital injection. The authors viewed "the greater occipital neuralgia syndrome" as a result of stimulation of the descending tract of the trigeminal nerve by compression, irritation, or ischemia of the greater occipital nerve, in turn caused by tension of the posterior neck muscles.

All of our patients had focal suboccipital tenderness which, when elicited by palpation, reproduced or aggravated their periorbital pain. As outlined above, there may be multiple mechanisms which can cause referral of pain from cervical/occipital branches to the trigeminal apparatus. Four of our patients had prior head trauma or brain surgery which may have possibly stimulated the tentorial innervation. None of the patients had structural lesions which could have been considered an irritative focus for their pain. In an evaluation of cervicogenic headache, Pfaffenrath et al\textsuperscript{12} found no specific radiologic differences when comparing the cervical spine x-rays of cervicogenic headache patients with those of age-matched controls.

The therapeutic response to lidocaine and triamcinolone acetonide was measured subjectively in our study. We cannot exclude a placebo effect from the injections in some patients. However, the majority of patients claimed significant relief, although it was temporary. We advocate the procedure of suboccipital injection in patients with marked focal occipital tenderness and secondary ocular pain. A carefully obtained history and detailed examination with attention to any palpable suboccipital trigger points will assist in the diagnosis.

Acknowledgments: Figure 1 was reproduced from Principles and Practice of Pain Management (Warfield CA, McGraw-Hill, Inc; 1993). Figure 2 was adapted from Correlative Anatomy of the Nervous System (Crosby ED, Humphrey T, Lauer EW. New York: MacMillan; 1962).

REFERENCES

Letters to the Editor

Paravertebral C2 Nerve Blocks

I refer to the interesting article, "Referred Ocular Pain by Suboccipital Injection," by Brian D. Ellis, MD and Gregory S. Kosmorsky, DO, of the Cleveland Clinic (Headache 1995; 35:101-103).

In Toronto, we have been doing greater and lesser occipital nerve blocks for many years as effective relief and treatment of headaches, including "ophthalmoplegic migraine."

More recently, we have found paravertebral nerve blocks at the level of C2 to be much more efficacious. These will usually relieve not only the periorbital and retro-orbital pain of migraine, but also the blurred vision and ptosis. We perform C2 paravertebral blocks effectively as an office procedure, without x-ray control. Patients report relief of pain and visual disturbances within minutes.

Presumably, the effectiveness of both occipital nerve and C2 paravertebral blocks derives from reducing the nociceptive input into the subcaudate nucleus of the trigeminal system; the importance of which has now been demonstrated by Bogduk and others.

We believe that most headaches (both "tension" and migrainous) arise from upper cervical intervertebral joint dysfunction; usually C2-C3, but sometimes as low as C5, and occasionally contralateral where the headaches switch sides.

Such dysfunction often originates in the soft tissue myotatic elements of these joints. Although such lesions (often late posttraumatic) are not easily discernable even with modern imaging techniques, the noteworthy effectiveness of the nerve blocks suggests the importance of some kind of nociceptive input from this region.

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Possible Increased Risk of Myocardial Infarction From Calcium-Channel Blocking Medications

The national news media widely reported data suggesting that calcium-channel blockers may be dangerous in increasing the risk of heart attack. The widespread use of calcium-channel blockers in the treatment of headaches, a non-life-threatening condition, has implications not only in prescribing, but potential medicolegal risks.

The news media reported a paper presented to the American Heart Association Epidemiology & Prevention Council meeting in San Antonio, Texas on Friday, March 10, 1995. An abstract (number 18) with preliminary data was published in Circulation 1995;91(3):925. The chief author of the paper entitled, "The Risk of Incident Myocardial Infarction Associated with Antihypertensive Drug Therapies" was Bruce M. Psaty of the Cardiovascular Health Research Unit, University of Washington, Seattle.

All subjects in the study had hypertension without prior clinical cardiovascular disease. The study included 291 cases and 1240 controls. The data suggests that the relative risk of myocardial infarction in patients taking calcium-channel blockers alone is 1.25 and if combined with a diuretic 1.95. The authors recommend diuretic and beta-blocker as the first line antihypertensive agents. The relative risk among the calcium-channel blockers was not reported.

This study may or may not apply to migraine or cluster headache patients treated with calcium-channel blockers. Both the study and control group were hypertensive as opposed to the normotensive headache population. The predisposition of myocardial infarction in hypertensive patients does not apply to headache patients. We are unfamiliar with any suggestion of increased incidence of myocardial infarction in headache patients treated with calcium-channel blockers.

If readers are familiar with cases of headache patients treated with calcium-channel blockers who have experienced myocardial infarctions, please send a brief report to our office. Epidemiological studies on a headache patient population could clarify any similar adverse effects of calcium-channel blockers in this population.

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