

Epidural corticosteroid blockade in cervicogenic headache

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Abstract. – Cervicogenic headache (CGH) is a relatively common form of headache stemming from the neck structures which presents some pathophysiological condition probably linked together with various pain-producing factors. This report presents a series of 9 patients suffering from cervicogenic headache and the results achieved by means of epidural steroid (methylprednisolone 40 mg) injection into the epidural cervical space (C6-C7 or C7-T1) level. The effectiveness of this diagnostic blockade was compared with the results obtained using the same procedure in 6 chronic tension headache (CTH) patients. A sharp decrease in the Numeric Intensity Scale (NIS) and in the Drug Consumption Index (DCI) values were observed after the diagnostic procedure in CGH patients. The short-term (12 hours) and medium-term (4 weeks) marked clinical improvement obtained in CGH patients may increase the number of available diagnostic tools which can be used to identify these underestimated patients population. The long-term effectiveness of this approach in cervicogenic patients is being evaluated over time.

Key Words

Cervicogenic headache, Cervical spine, Epidural space, Methylprednisolone.

Introduction

The problem of pain originating in the neck region and not related to degenerative situations of the cervical column was first raised by Hunter and Mayfield in 1949¹. Only more recently it has been hypothesized that one could define a new and distinct clinical entity which Sjaastad and coll in 1983 named "cervicogenic headache"^{2,3}.

The 1988 International Headache Society's classification of headaches, which employs a mathematical approach to arrive at diagnosis, does have an entry on headaches of cervical origin, but cervicogenic headache (CGH) is not yet exactly codified⁴.

CGH represents a frequent cause of pain starting from the C2/C3 levels with radiation to the occipital area, to the vertex of the head (C2 level), to the peri-oculo-frontal-auricular-temporal area, and sometimes even to the mandibular and neck region (C3 level)⁵. The pattern of pain from the neck can also follow other directions, for example, to the shoulder and the ipsilateral arm. There may be some clinical variability in the type of pain, which is often continuous, non-pulsating, non-burning, with some radicular characteristics. Additionally, there may be problems to the sense of balance and autonomic functions. Often there are periodic recurrences of paroxysmal attacks which are asymmetrically, monolaterally localized, and there is frequent concomitance of other pains in the osteoarticular apparatus. Additionally, variation of position and/or movement of the cervical column at the onset of pain is an important distinguishing feature⁶.

CGH seems to be based more on a functional disorder than on degenerative lesions of the cervical spine⁷⁻⁹. All the above mentioned clinical observations appear to be closely dependent on a very low pain pressure threshold in the occipital part of the head, with reproduction of pain in the site of pain predominance¹⁰.

The differential diagnosis of this form of headache creates difficulties because of similar signs and symptoms common to various pain syndromes strictly located within the

same unilateral head region¹¹. The pattern of response of CGH to certain diagnostic tests serve to mark this headache as a distinct entity. These diagnostic criteria can be used to define similarities and differences with the various other identified headache syndromes.

Current diagnostic approach to CGH utilizes great occipital nerve (GON) blockade¹², anesthetic block of C2 root¹³, blink reflex latency¹⁴ or pharmacological agents unabling to induce or suppress CGH crises^{15,16}.

Corticosteroids are used in nerve blocks in combination with local anesthetic or adrenergic blocking agents, such as guanethidine in various pain syndromes, such as in nociceptive pain, neuropathic pain, sympathetic-mediated pain, malignant pain, and visceral pain. Local blocks are usually reversible and may provide prolonged pain relief by breaking the pain cycle¹⁷.

The first aim of the present study was to ascertain the efficacy of epidural steroids in CGH, trying to expand the diagnostic tools now available for this type of headache. The second aim was to assess the short-term duration of the efficacy of the above mentioned epidural block.

Materials and Methods

Patients selection. Nine CGH patients (8 females, 1 male; mean age 42.3 ± 8.4) were recruited from among the outpatients of our Headache Centre and were diagnosed accordingly to criteria proposed by Sjaastad et al in 1990¹⁸. The clinical diagnosis in CGH group has been confirmed by GON or C2 blockades^{12,13}. The duration of the disease varies from 3 to 12 years. The studied CGH patients suffered from a continuous form of the disease from at least 6 months and take painkillers on a regular basis. The patients suffering from pain syndromes other than CGH were excluded. The control group consisted in 6 sex- and age-matched (5 females, 1 male; mean age 39.6 ± 9.7 ; duration range of the disease 5-16 years) chronic tension headache (CTH) in-patients. In consideration of the potential risk of the diagnostic technique no placebo (f.e. saline solution) control group has been used in this study. Before the study, the permission of our University Ethi-

cal Committee was obtained, and a written informed consent was signed for all subjects admitted to the study. Before admission to the trial each of the CGH selected patients was hospitalized, then routine hematological functions including blood coagulation patterns, a consultant cardiologist visit with ECG and a radiographic examination of cervical spine. Head acceleration trauma have been referred in 2 out of 9 CGH patients and in 1 out of 6 CTH patients.

In order to examine the CGH and CTH in-patients during an unmasked condition of their head pain, each patient underwent a 72-hour washout period from analgesics and anti-inflammatory medications.

Diagnostic procedure. The technical description of cervical epidural blockade in our series of CGH patients was as follows¹⁹. An intravenous cannula was positioned and 500 ml of saline solution infused before performing the epidural puncture. With the patients in sitting position and the neck flexed, the spinous process of C7 and the interspinous space C6-C7 (or C7-T1) were palpated; a cutaneous infiltration anesthesia was obtained with Mepivacain 1% (Astra, Milano) 3 ml and sodium bicarbonate 0.1 mEq/ml 1 ml. In a midline approach, a 18 G Tuohy (Braun Melsungen AG, Melsungen, FRG) needle with syringe containing saline solution was inserted in the interspace C6-C7 (or C7-T1) while proceeding with the needle, then the syringe was firmly pressed until appreciating the loss of resistance to injection after exceeding the ligamentum flavum with the needle bevel. Then 40 mg of methylprednisolone in 3-4 ml of saline were injected in the cervical epidural space. The needle was extracted and the patients maintained the sitting position for a few minutes. No major complication of this technique were seen in our study. During the injection phase all patients felt mild pain in the right side of the neck, even if the solution was injected slowly. This neck pain resolved spontaneously in about ten minutes. In addition all patients who underwent to this procedure felt occipital burning or tingling or sore pain sensation and searing sensation in the face after the cervical epidural injection and they had progressive resolution of this minor side-effect in 15-30 minutes after methylprednisolone injection.

Data collection. Twelve hours after the epidural diagnostic test each patient was allowed, if necessary, to take analgesics or anti-inflammatory drugs in order to assess the effect of the block both directly on the pain as well as indirectly on the total amount of daily analgesic intake. The pain characteristics were recorded by the patients in a daily headache diary, where the the quality (no pain, moderate pain, worst possible pain) and intensity (0-10 scale) of pain could be recorded utilizing Numeric Intensity Scale (NIS). The amount of any analgesic or anti-inflammatory medications taken during the study period has been also recorded in order to evaluate the Drug Consumption Index (DCI). We provided each patient with diaries for the full 4-week period of the study.

This protocol was approved by Ethics Committee of the University “La Sapienza” Medical School prior of starting the trial. The short-term diagnostic value of the epidural steroid injection in CGH and CTH patients was evaluated (NIS ± SEM) every hour for

the first half day (ANOVA for repeated measures within the same group, NPAR1WAY between different groups). The weekly means (± SEM) of the registered items (NIS, DCI) over time were compared by means of ANOVA for repeated measures and Duncan t-test.

Results

No serious side effects have been reported in the studied patients after epidural steroid injection, except 1 CGH and 2 CTH patients who experienced a flushing sensation in the face, lasted between 45 min and 4 hours after injection.

All patients who entered the study to treat their CGH attacks completed the study and reported their headache diary during the final visit at the 4 week after the epidural steroid injection.

As illustrated in Figure 1, there was a marked decrease of the NIS scores immediately after

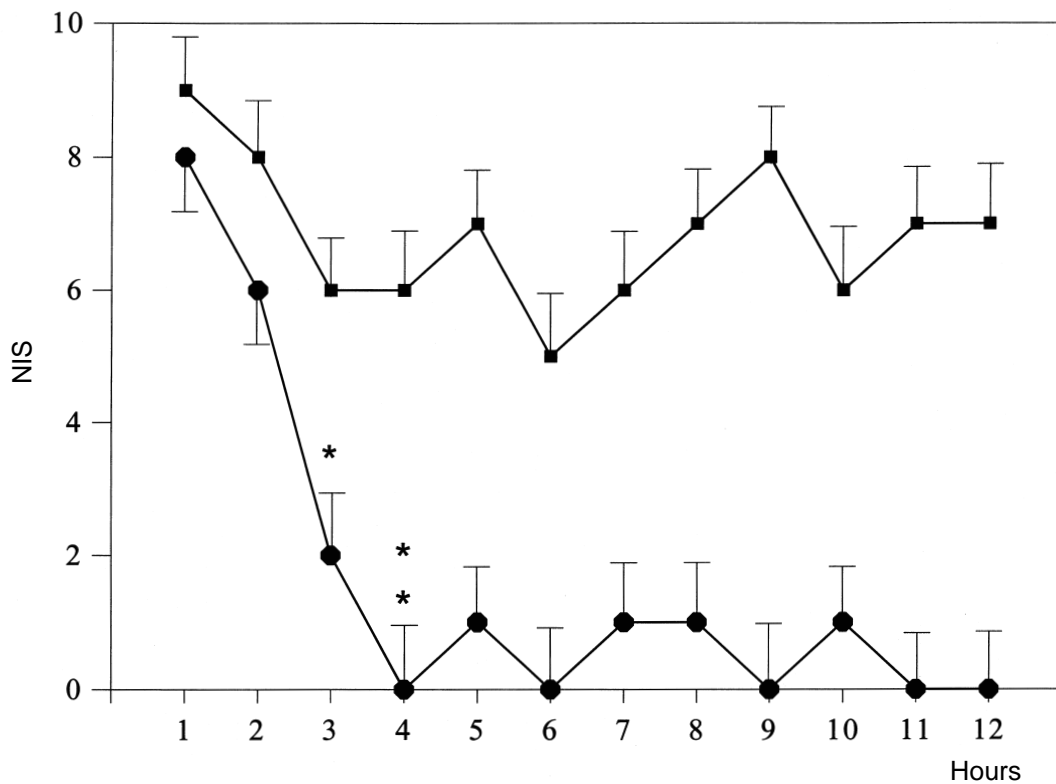


Figure 1. Mean hourly values (± SEM) of Numeric Intensity Scale (NIS) in a group of 9 patients suffering from cervicogenic headache (●) and in 6 patients suffering from chronic tension headache (■) after the cervical epidural blockade. * p < 0.02; ** p < 0.001 (One-way ANOVA and Duncan t-test).

the epidural injection, during the first twelve hours after the described diagnostic technique.

The beneficial effect in CGH group was statistically significant if compared with the NIS values obtained in control group (Figure 1). During the medium-term follow-up period this decline in the NIS was accompanied by a parallel decrease of the DCI index (Figure 2). The decline in both the NIS and DCI index began immediately after the epidural injection and remained at significantly lowered values even during the 4 week follow-up period after the epidural injection.

Discussion

The nature of pain in CGH is not still defined. However, the diagnostic effectiveness of the cervical epidural steroid blockade led us to hypothesize its targets of action.

Corticosteroids combat inflammation by inhibiting a number of proinflammatory sub-

stances and pain mediators, such as prostaglandins, leukotrienes, tumor necrosis factor and interleukin-1^{20,21}.

A scientific rationale in the use of corticosteroids as diagnostic tool of CGH could be based upon the clinical evidence that this pain syndrome initiated as an inflammatory status, mechanically induced (cervical spine acceleration trauma), of the structures of the cervical spine (vertebral articulations, ligaments, intrinsic and long muscular structures of the spine itself) continues its natural course probably by sensitizing the cervical nerve roots and initiating a pain-producing loop. This could be at least the initial event that occurs in the multifactorial CGH pathophysiology. In addition, the local microvascular ambient could amplify the inflammation process by increasing the vascular adhesion of leucocytes and the subsequent transvascular migration in the surrounding tissues²². This microvascular experimental model has been used in studying migraine²³ and dermatomyositis²⁴. In this last case pulse methylprednisolone administration

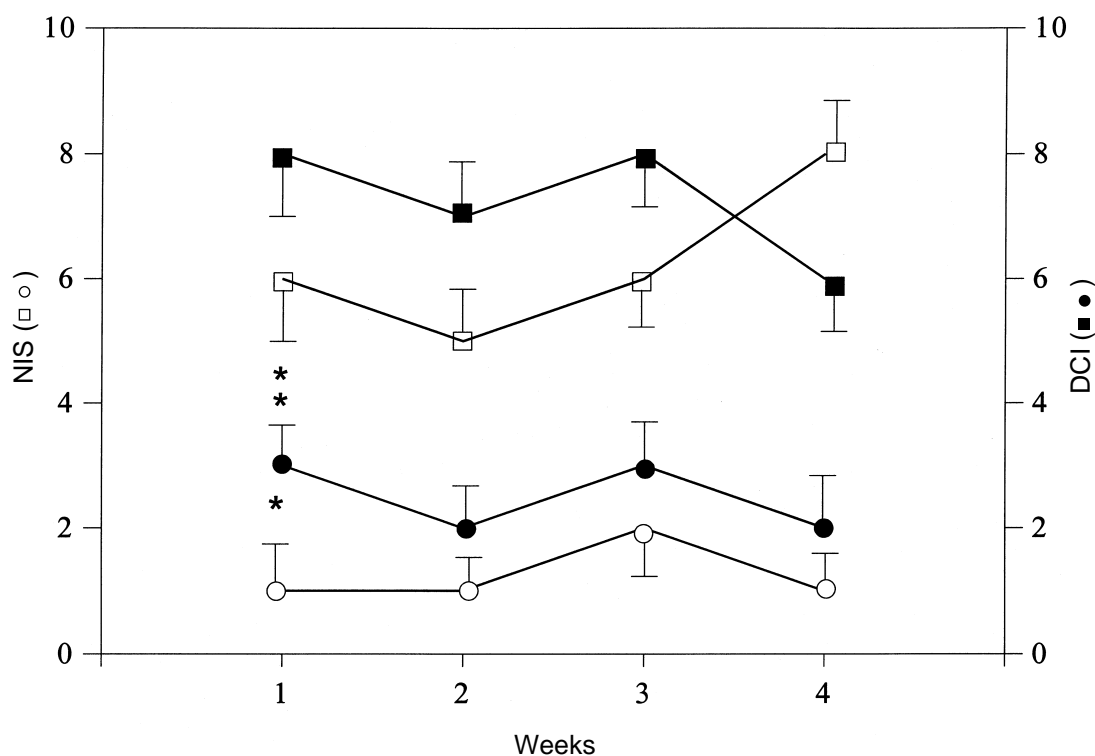


Figure 2. Mean weekly values (\pm SEM) of Numeric Intensity Scale (NIS) and Drug Consumption Index (DCI) in cervicogenic headache patients (NIS ○; DCI ●) and in 6 chronic tension headache patients (NIS □; DCI ■) during the 4 weeks following the cervical epidural blockade.

* $p < 0.02$; ** $p < 0.001$ (One-way ANOVA and Duncan t-test).

profoundly inhibits the permeability phase of vascular inflammation by reducing the adhesion molecules expression.

In addition, the reported efficacy of spinal manipulation therapy in patients suffering from cervicogenic headache^{11,25-29} appears to support the currently accepted pathophysiological hypothesis for this pain syndrome, as mainly derived from a dysfunction of the cervical neuro-musculo-skeletal system.

However, at this time, in absence of any pathological findings at C7-T1 levels, it's difficult to speculate about the anatomical background of corticosteroid action on CGH.

One way of summarizing the pros and cons of this diagnostic technique in CGH patients would be to consider that it is a reliable and fast technique, more less invasive than other proposed approaches³⁰⁻³³ and needs only of a 48-hours period of hospitalization. Lastly, from a technical point of view, this diagnostic technique is relatively safe in experienced hands, but the unintentional intrathecal injection can produce serious damages, including meningitis or chemical arachnoiditis^{34,35}.

In our study only CGH few patients (n. 4, 6, 7) needed a second epidural injection during the follow-up period. Further observations and a long-term follow-up will clarify the effectiveness or failure of this technique as possible therapeutic approach to CGH.

In the next future, when the actually confusing epidemiological data^{36,37}, the diagnostic parameters³⁸, and the therapeutic indications¹¹ provided for a CGH have been widely validated, it will be possible to give a definitive profile to this type of headache.

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