

HYDROCORTISONE IN POST-DURAL PUNCTURE HEADACHE

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Abstract

Background: Headache after spinal anesthesia is a common complication in patients undergoing this procedure. In this study we evaluated the efficacy of intravenous hydrocortisone in the treatment of headache after spinal anesthesia in women who have undergone cesarean section.

Methods: Sixty patients with headache after spinal anesthesia were included. Patients randomly allocated into two groups, 30 patients received only conventional therapy (complete bed rest, hydration, acetaminophen and pethidine). Other 30 patients received conventional therapy plus intravenous hydrocortisone (200 mg first, then 100 mg TID for 48 hours). Mean (\pm SD) of headache intensity at 0, 6, 24, and 48 hours after beginning of treatment was measured using visual analog scale.

Results: There was no significant difference in headache intensity between two groups before beginning of treatment. After 6 hours, the mean of headache intensity in 30 patients treated conventionally was 6.63 (\pm 1.35) while it was 2.77 (\pm 1.07) in other patients received intravenous hydrocortisone too ($p < 0.001$). After 24 hours, mean headache intensity was 3.87 (\pm 1.63) in conventionally treated group versus 0.73 (\pm 0.74) in

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hydrocortisone group ($p < 0.001$). After 48 hours, mean headache intensity was $1.87 (\pm 0.93)$ in conventionally treated group versus $0.63 (\pm 0.61)$ in hydrocortisone group ($p = 0.001$).

Conclusions: This study showed the therapeutic effects of intravenous hydrocortisone in reducing headache after spinal anesthesia in women who underwent cesarean section. Its mechanism of action is yet to be determined.

Keywords: Spinal anesthesia, headache, hydrocortisone, corticosteroids.

Introduction

Headache after spinal anesthesia, post-dural puncture headache (PDPH), is a common complication which might occur in patients undergoing this type of anesthesia. PDPH might also occur following lumbar puncture and myelography^{1,6}. Headache is usually described as a severe, dull, nonthrobbing pain, often fronto-occipital which is aggravated in upright and diminished in supine position⁶. It may be accompanied by nausea, vomiting or visual disturbances⁵. Headache begins one or two days after spinal anesthesia and if left untreated, usually relieved spontaneously after a week^{5,7}.

Severe headache should be differentiated from other causes of headache after delivery such as migraine, hypertension, meningitis, cortical vein thrombosis, pneumocephalus, sinusitis, intracranial pathology, and nonspecific headache⁶.

Dural puncture and subsequent cerebrospinal fluid (CSF) leakage is the most accepted mechanism for induction of headache^{4,6}. CSF leakage through dural hole and reduction in CSF pressure lessens the cushioning effect of brain, allowing it to sag within the intracranial vault and stimulation of dural pain receptors especially in upright position³. Headache continues until dural hole repairs. It is relieved when CSF volume and pressure return to normal^{3,8}.

Current conventional and symptomatic treatments for postdural

puncture headache involve complete bed rest, hydration, analgesics (acetaminophen, nonsteroidal anti-inflammatory drugs and opiates), oral or intravenous caffeine, sumatriptan, aminophylline and ACTH^{6,8}. None of these therapeutic approaches can relieve headache completely and just help patient to endure it⁸. If headache is too severe to limit mother's activity or any evidence of cranial nerve involvement is noted, an epidural blood patch (EBP) may be performed^{1,6,9}.

In recent years along with the attention to the role of corticosteroids in management of post operation pain¹⁰ and cancer pain¹¹, some authors proposed use of these drugs to treat post dural puncture headache² and syndrome of spontaneous intracranial hypotension^{12,13}. There is scant literature of the role of steroids in post dural puncture headache².

The present study was designed to evaluate the efficacy of intravenous hydrocortisone in reducing headache intensity after spinal anesthesia in women undergoing cesarean section.

Methods and Materials

The study was approved by Tehran University Institutional Board Review and written informed consent was obtained from all involved patients. Of the 475 pregnant women who delivered in the Imam Khomeini General Hospital between March to September 2003, sixty patients (18 to 40 years, ASA I, II) and headache after spinal anesthesia for cesarean section were included. Patients with a history of cluster headache, convulsion, cerebrovascular accident, preeclampsia, eclampsia, high intracranial pressure, coagulopathy, or previous neurologic disease were excluded.

Two consultant anesthetists performed all spinal anesthesia in sitting position and midline approach with 25-gauge Quincke needle and 75 mg of lidocaine 5%. Thirty patients were randomly allocated in two groups: 30 patients were treated conventionally: complete bed rest, hydration (serum dextrose saline 3L/4h) and analgesics (acetaminophen two 325 mg tablets every 6 hour and intravenous pethidine 50m every 12-hour).

Other 30 patients were treated conventionally but received 200 mg hydrocortisone intravenously as a bolus, first, and then total of 6 doses of 100 mg hydrocortisone (every 8 hours) for 48 hours. Parturient women and observer did not know which patients had received hydrocortisone.

Mean of headache intensity was measured in all 60 patients after one minute in the upright position. A trained observer asked patients about headache severity at 0, 6, 24, and 48 hours after beginning of treatment. Using Visual Analog Scale (VAS), results were sorted into four categories (0-1, no headache; 2-4, mild headache, 5-7, moderate headache; 8-10, severe headache). T-test was used to compare mean of headache intensity between the two groups and p value <0.05 was considered significant.

Results

All 60 patients had severe headache before beginning of treatment. Mean (\pm SD) of the age, weight and height of patients were 27.1 ± 3.45 years, 69.8 ± 2.4 Kg and 162.3 ± 5.6 cm respectively.

The mean headache intensity was 9.07 ± 0.69 in the conventionally treated Group and 9.20 ± 0.71 in hydrocortisone group (VAS = 8 – 10) (Table 1). The mean (\pm SD) of headache intensity at 6, 24, and 48 hours after beginning of treatment in two groups are listed.

Table 1
Mean headache intensities at 0, 6, 24 and 48 hours following the beginning of treatment in the conventionally treated Group and the conventional treated with i.v hydrocortisone Group

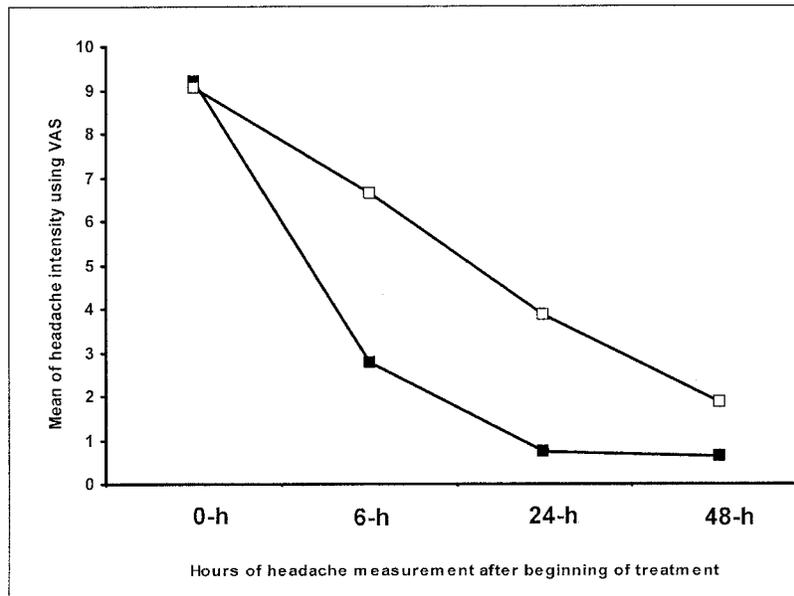
Hour/Group	Conventional treatment*	Conventional treatment + IV hydrocortisone	p-value
		Mean (SO)	
0	9.07 (0.69)	9.20 (0.71)	0.466
6	6.63 (1.35)	2.77 (1.07)	<0.001
24	3.87 (1.63)	0.73 (0.74)	<0.001
48	1.87 (0.93)	0.63 (0.61)	= 0.001

* Conventional treatment = complete bed rest, hydration, acetaminophen and pethidine.

Reduction of headache intensity during 48 hours after treatment is shown in Fig. 1.

Fig. 1

Comparison between reduction of headache intensity in the two Groups (---□--- conventional treatment, ---■--- conventional + hydrocortisone treatment)



Epidural blood patch was used for one patient with severe headache in the conventionally treated Group whose headache was not resolved after 4 days therapy. No patients was re-referred to Hospital for relapse of headache and no adverse effects were noted.

Discussion

This study showed that addition of intravenous hydrocortisone to conventional treatment of patients with post dural puncture headache can reduce headache significantly. Use of hydrocortisone in the treatment of PDPH has been proposed by Toriel et al. as a case reopr². In that study, 3

patients with severe spinal headache after cesarean section, received 100 mg intravenous hydrocortisone every 8 hour for 3 days, in addition to conventional therapy. Headache disappeared completely 12 hours after last dose. The technique of anesthesia and needle gauge was not the same in these patients. In our study, however, we administered hydrocortisone to 30 patients where the technique of anesthesia and needle gauge was the same for all patients. Our results are in concordance with results of Toriel study.

Efficacy of corticosteroids in the treatment of the syndrome of spontaneous intracranial hypotension, has been shown too¹²⁻¹⁵. This syndrome is characterized by low CSF pressure (<60 mmHg at lumbar puncture), usually severe fronto-occipital headache that is aggravated in upright position and not relieved by analgesics¹⁴. The quality of headache and response to therapy is very similar to PDPH. In a recently published case report, Gentile et al. presented 3 patients with spontaneous intracranial hypotension whose headache disappeared in 2-4 weeks after treatment with oral prednisolone¹³. Other researchers have reported similar results^{12,15}.

The mechanism by which corticosteroids resolve PDPH and headache of intracranial hypotension has not been elucidated^{2,12-14}. In laboratory animals, steroids have no effect on CSF production¹³ and in clinical practice these drugs are currently used to reduce intracranial hypertension^{6,13}.

Recent studies have shown that patients with headache associated with spontaneous low CSF pressure, present a variable amount of CSF in the spinal extradural space associated with dilated epidural veins in the high cervical portion^{16,17}. It seems that CSF hypovolemia, and not intracranial hypotension, might be the more important pathogenetic mechanism of the disease¹³. Steroids may exert their clinical effect by favoring the reabsorption of CSF from the extradural space and thus increasing CSF volume¹³.

We propose another hypothesis about steroids mechanism of action. Today, there is an increasing attention to the role of steroids in

controlling the pain after surgery¹⁰ and cancer pain¹¹. Steroids suppress arachidonic acid production through lipocortin-induced phospholipase inhibition, which ultimately inhibits production of algogenic prostaglandins (PGE 2 and PGI 2) and leukotriens (LTB 4)¹⁰. Furthermore, corticosteroids block production of pro-inflammatory cytokines such as interleukine-1, interleukin-2, and tumor necrosis factor-alpha¹⁰. It seems that analgesic action of steroids in PDPH, may relate to their anti-inflammatory effects at dural puncture site. During the healing process of dural puncture site, inflammatory mediators that are secreted from immune cells, spread in CSF and stimulate pain receptors, cause headache. Steroids may suppress production of these algogenic mediators and relieve headache. Admittedly, our theory cannot explain why PDPH usually appears in the upright position and is relieved in the supine position. Since short-term use of steroids may have some adverse effects^{10,11}, safety of administration should be evaluated along with their mechanism of action in future studies.

Conclusion

This study showed the efficacy of intravenous hydrocortisone in reducing headache after spinal anesthesia in women who underwent cesarean section. Further studies are needed to consider steroid therapy as a standard treatment for post-dural puncture headache.

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