

Injection of intrathecal normal saline in decreasing postdural puncture headache

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Abstract

Purpose Postdural puncture headache (PDPH) is the most common and still unresolved postoperative complication of spinal anesthesia. Although there are several positive results of intrathecal saline injection for the treatment of PDPH and prophylaxis after accidental dural puncture, the effect of deliberate intrathecal saline injection before spinal anesthesia has not been examined. The objective of our study was to evaluate the effect of prophylactic administration of intrathecal normal saline in decreasing PDPH.

Methods One hundred healthy women (ASA physical status I) of age between 18 and 35 years scheduled for elective term cesarean delivery under spinal anesthesia were included. Patients were randomly divided into two equal groups. Group C received 2.5 ml (12.5 mg) hyperbaric bupivacaine 0.5 % as a control, and group S received intrathecal normal saline 5 ml before intrathecal injection of 2.5 ml (12.5 mg) hyperbaric bupivacaine 0.5 %. The incidence and severity of PDPH were assessed after 48 h and again 3–7 days after operation.

Results Basal characteristics were statistically similar in both groups ($P > 0.05$). The incidences of moderate and severe PDPH during first postoperative 48 h were not different between the groups ($P = 0.24$). However, the frequency of PDPH after 3–7 days was statistically higher in

group C in compared with group S (16 vs. 2 %, $P = 0.03$). Totally the frequency of PDPH was higher in group C (24 vs. 2 %, $P = 0.002$).

Conclusion Administration of normal saline (5 ml) before intrathecal administration of hyperbaric bupivacaine as a preventive approach is an effective and simple way to minimize PDPH in patients undergoing cesarean section.

Keywords Anesthesia · Spinal · Postdural puncture headache · Intrathecal normal saline injection

Introduction

Spinal anesthesia remains an easy and cost-effective anesthetic technique in pregnant women, and it is often preferred to general anesthesia. However, postdural puncture headache (PDPH) is the most common postoperative complication of spinal anesthesia. Flaatten and colleagues manifested the high incidence of PDPH (24.5 %) after spinal anesthesia with a 25-G needle [1].

The traditional concept is that dural puncture causes a leak of cerebrospinal fluid (CSF) and loss of CSF pressure. The following factors are thought to influence the incidence of PDPH: age, sex, needle size, multiple dural punctures, and previous history of PDPH [2, 3]. Numerous drug therapies (caffeine therapy, both oral and intravenous theophylline, saline) are mentioned in the literature, few of which had any merit in clinical application [4–7]. Ahearn [8] and Pickering [9] injected normal saline solution into the subarachnoid space in patients suffering from PDPH as a treatment technique with some positive results. Also, Charsley and Abram [7] injected intrathecal normal saline via an epidural needle following accidental dural puncture as a prevention technique. There are numerous reports of

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research on the pathophysiology, prevention, and treatment of PDPH [10], but the effect of deliberate intrathecal saline injection before spinal anesthesia has not been examined.

The aim of this randomized study is to test the hypothesis that prophylactic administration of intrathecal saline before the injection of local anesthetic in spinal anesthesia decreases the incidence of PDPH in parturients undergoing cesarean section.

Materials and methods

The protocol was approved by the medical ethics committee of Tehran University of Medical Sciences. Written informed consent was obtained from 100 healthy women (ASA physical status I) with between 18 and 35 years of age scheduled for elective term cesarean delivery under spinal anesthesia. Parturients were excluded from the study if they had preexisting hypertension, diabetes mellitus, chronic headache, history of previous PDPH, more than two previous lumbar puncture attempts, or contraindication to spinal anesthesia. The study period extended from April to December 2009. Participants were randomly divided into two equal groups using a computer-generated table of random numbers. A wide-bore intravenous catheter (18-gauge) was inserted into a forearm vein, and all patients were hydrated with 1,000 ml intravenous lactated Ringer's solution before the spinal anesthesia. Standard monitoring was used throughout the study, including noninvasive arterial blood pressure, pulse oximetry, and electrocardiography.

Spinal anesthesia was performed with the patients in sitting position at the L3–L4 interspace using a 25-gauge Quincke needle (B. Braun Melsungen, Melsungen, Germany). The spinal needle was inserted with its bevel oriented parallel to the longitudinal axis of the back. After obtaining free-flowing cerebrospinal fluid, 2.5 ml (12.5 mg) hyperbaric bupivacaine 0.5 % was injected in group C as a control group, and injection of 5 ml intrathecal normal saline and then injection of 2.5 ml (12.5 mg) hyperbaric bupivacaine was performed in group S as a study group. On completion of spinal injection, the patient was placed in the supine position with left uterine displacement. After obtaining sensory loss at the T4–T6 dermatome, surgery was started.

Continuous pulse oximetry and arterial blood pressure were recorded every minute until delivery and then every 5 min until the end of surgery. A 25 % decrease in systolic blood pressure compared with the preoperative value was regarded as hypotension.

Onset time of analgesia was determined by the time from injection to maximum level of sensory blockade. Duration of sensory regression was defined as the time between achievement of the highest level of sensory blockade and its regression to the T10 level.

The occurrence of clinically relevant hypotension was treated with a bolus of fluid and incremented dose of ephedrine 5–10 mg IV, and bradycardia was treated with atropine 0.5 mg IV. All patients received 2,000–2,500 ml lactated Ringer's solution during the operation. After delivery and clamping of the umbilical cord, we provided intraoperative intravenous sedation with midazolam 2 mg and fentanyl 25 µg. Pain was checked objectively by the numerical rating score (NRS: 0 = no pain and 10 = the worst imaginable pain) at the time of the patient's complaint of pain, and moderate to severe pain (NRS \geq 4) was treated with incremented dose of fentanyl 25 µg IV (maximum dose, 100 µg).

Maternal age, height, weight, and duration of surgery were recorded, and the occurrence of PDPH was obtained by interview at 12, 24, and 48 h postoperatively by an anesthetic nurse who was blinded to study group assignment. If a headache was reported, the interviewer completed a second questionnaire ascertaining the onset and distribution of the headache, the effect of posture, and whether there were any visual or auditory disturbances and severity of PDPH. A headache was categorized as a PDPH if it was worse on sitting or standing and relieved or reduced by lying flat. Otherwise, the headache was recorded as a nonpostdural puncture headache. The severity of PDPH was classified by the patients as no headache, mild headache, moderate headache, and severe headache. In this study, the incidence of PDPH was compared between the groups using the total of number of patients with moderate and severe headache and patients with no and mild headache. Patients without any surgical complications and headache were discharged on the third postoperative day and evaluated by a telephone call 1 week later for any signs or symptoms of a delayed-onset headache.

If the patient was found to have PDPH, she received conservative therapy, consisting of bed rest, increased fluid intake, and analgesics. If this regimen failed to relieve the headache and if the patient agreed, an epidural blood patch was performed. Postoperative analgesia was provided by suppository diclofenac 50 mg three times per day, followed by an intravenous injection of pethidine 25 mg slowly if the patient was still in pain 10 min after diclofenac. If necessary, pethidine was repeated 4 h after the previous injection. Total postoperative pethidine consumption (mg) was recorded for 2 days.

Based on the results of Flaatten et al. [1] that showed the incidence of PDPH was about 25 % in patients who received spinal anesthesia with a 25-G needle, and our estimation based on the pilot study in 20 patients in each group, the incidence after normal saline injection would be 5 %. Therefore, we calculated that 50 patients would be required in each group to detect such difference in incidence with a power of 80 % and $\alpha = 0.05$ by using Epi Info site (<http://www.cdc.gov/epiinfo/>).

Statistical evaluations were performed with SPSS software 13 (SPSS, Chicago, IL, USA). Quantitative data were displayed as mean \pm standard deviation and qualitative data were displayed number with percentages. Student's *t* test, chi-square test, or Fisher's exact test were used when appropriate. A *P* value less than 0.05 was considered as statistically significant.

Results

Basal characteristics were statistically similar ($P > 0.05$) in both groups (Table 1).

The frequency and severity of PDPH are shown in Table 2. The frequency of PDPH in the first 48 h was not different between the two groups ($P = 0.24$). However, the frequency of PDPH after 3–7 days was statistically higher in group C when compared with group S (16 vs. 2 %, $P = 0.03$). Overall, the frequency of PDPH was higher in group C (24 vs. 2 %, $P = 0.002$). In the majority of patients, spinal anesthesia was induced with a single puncture. Two attempts were required in two patients in group C and three patients in group S. During the operation, five patients in group C and four patients in group S received fentanyl 50 μ g. Mean pethidine requirements were not different between the groups during 2 days. Table 3 presents the rate of complications in both groups.

Discussion

We found that the incidence of PDPH in parturients was significantly less when 5 ml intrathecal normal saline was administered just before spinal injection.

Table 1 Total characteristics of the two groups

	Group S (<i>n</i> = 50)	Group C (<i>n</i> = 50)	<i>P</i> value
Age (years)	26.8 \pm 5.1	27.5 \pm 5.8	0.6
Weight (kg)	81.0 \pm 14.1	79.7 \pm 12.4	0.6
Height (cm)	164.3 \pm 4.7	162.7 \pm 4.3	0.1
Body mass index (BMI) (kg/m ²)	29.9 \pm 4.3	30.0 \pm 3.5	0.9
Onset time (min)	5.1 \pm 4.2	5.4 \pm 1.4	0.7
Duration of sensory regression (min)	141.8 \pm 70.3	123.5 \pm 16.1	0.1
Pethidine consumption (mg)	52 \pm 9.8	52 \pm 11.1	1

Group S, intrathecal normal saline 5 ml before intrathecal injection of 2.5 ml (12.5 mg) hyperbaric bupivacaine 0.5 %; group C, control

P refers to *t* test

Table 2 Number of postdural puncture headaches (PDPHs) after the first 48 h and after 3–7 days

	Group S	Group C	<i>P</i> value
First 48 h			
No or low	50 (100)	47 (94)	
Moderate or severe	0 (0)	3 (6)	
			0.24
Headache on 3rd day			
No or low	49 (98)	42 (84)	
Moderate or severe	1 (2)	8 (16)	
			0.03
Headache on 7th day			
No or low	50 (100)	46 (92)	
Moderate or severe	0 (0)	4 (8)	
			0.12
Total headache	1 (2)	12 (24)	
			0.002

P refers to χ^2 test and Fisher's exact test

Table 3 Comparison of complications in both groups

	Group S	Group C	<i>P</i> value
Nausea	10 (20)	13 (26)	0.64
Vomiting	13 (26)	13 (26)	1.00
Hypotension	48 (96)	42 (84)	0.09
Bradycardia	1 (2)	6 (12)	0.11
Number of PDPHs	0 (0)	4 (8)	0.12

P refers to χ^2 test

Although the mechanism producing PDPH is not clear, it seems the maintaining CSF volume and pressure could be effective in reducing the incidence of PDPH. One explanation for the beneficial effect of intrathecal saline is that the increased CSF pressure may result in approximation of the dura and arachnoid at the puncture site, thus sealing the defect.

The replacement of the escaped CSF volume by injecting the small amount of normal saline seems questionable when one takes into consideration the total volume of CSF (approximately 150 ml) and about 500 ml of CSF production (0.35 ml/min). Nevertheless, Charsley and Abram [7] found that the immediate injection of 10 ml intrathecal normal saline through the epidural needle after a dural puncture reduced the incidence of PDPH from 62 to 32 %.

Ahmed and colleagues [11] indicated that the decrease in CSF volume may activate adenosine receptors, which causes cerebral vasodilation and stretching of pain-sensitive cerebral structures, resulting in headache after lumbar puncture. Injection of intrathecal saline may prevent activation of the adenosine receptors and by this means may decrease the incidence and severity of PDPH.

Another study by Kuczkowski and Benumof reported in seven cases a combination of five maneuvers following unintentional dural puncture with an 18-gauge epidural needle in parturients, that is, (1) sequential injection of the CSF in the glass syringe back into the subarachnoid space through the epidural needle, (2) insertion of an epidural catheter into the subarachnoid space, (3) injection of small amount of preservative-free saline (3–5 ml) into the subarachnoid space through the intrathecal catheter, (4) administration of a bolus and then continuous intrathecal labor analgesia, and (5) leaving the catheter in situ in the subarachnoid space for a total of 12–20 h, decreased the incidence of PDPH from 76–85 [12] to 14 % [13]. It was difficult to indicate the relative importance of the five maneuvers performed in their study in decreasing the incidence of PDPH. However, the results of our study confirmed the third maneuver.

It is thought that intrathecal saline 5 ml may influence the extent and duration of the sensory and motor blockade. However, these variables were not investigated precisely, which is a limitation of this study.

In conclusion, intrathecal administration of normal saline as a preventive approach is effective, and it is a simple way to minimize PDPH in patients undergoing cesarean delivery under spinal anesthesia. However, further studies are required to confirm our findings and the effective volume of normal saline.

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