ARTÍCULO DE REVISIÓN

Post-dural puncture headache in pregnant women: What have we learned?

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ABSTRACT:

The obstetric patient is at particular risk of dural puncture (and the subsequent headache) because of sex, young age, and the widespread application of regional anesthesia. The incidence of epidural needle-induced post-dural puncture headache (PDPH) in pregnant women following dural puncture with a large bore needle has been reported to range 76-85%. Although a few measures have been proposed to prevent PDPH (intrathecal injection of saline, insertion of the epidural catheter into the subarachnoid space through the dural hole, none have been shown to work with certainty to date. This article reviews the latest developments aimed at prevention and treatment of this debilitating condition.

Key Words: *Labor analgesia; epidural, combined spinal epidural; complications, dural puncture, post-dural puncture headache (PDPH); prevention.*

HISTORICAL PERSPECTIVE:

More than 100 years have passed since the initial description of the post-dural puncture headache (PDPH)[1]. However, this unique clinical entity still continues to fascinate anesthesiologists, and numerous studies on its pathophysiology, prevention, and treatment, have been published[2-22]. Using himself as subject, August Bier demonstrated spinal anesthesia (with subarachnoid injection of cocaine) one day, and «spinal headache» known today as post-dural puncture headache (PDPH) the following morning. Bier surmised that the headache was attributed to loss of cerebrospinal fluid (CSF). By the early 1900s, there were numerous reports in the medical literature of the application of spinal anesthesia with large gauge needles, with the average incidence of PDPH exceeding 50% of subjects[3]. In 1951 Whitacre developed the pencil-point needle, which led to a significant reduction in the incidence of PDPH. However, PDPH still remains a disabling complication of needle insertion into the subarachnoid space. Since those early days in 1898, we have made enormous progress in understanding this clinical entity, including its epidemiolgy, pathophysiology, clinical symptoms and treatment. This review will discuss the pathophysiology of dural puncture, incidence, presentation and treatment of PDPH with particular emphasis on the newest methods of prevention (maintaining CSF volume) of PDPH.

PATHOPHYSIOLOGY

Dura mater: anatomy

The dura mater is a dense, connective tissue layer, which is made up of elastic fibers and collagen. The classical description of the spinal dura mater (supported by histological studies) is of elastic and collagen fibers running it the longitudinal direction. Clinical studies based on this view

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confirmed that postdural puncture headache was more likely when the cutting spinal needle was oriented perpendicular to the direction of the spinal dura fibers[3]. However, recent light and electron microscopic studies, (which describe the dura mater as consisting of collages fibers arranged in several layers parallel to the surface) have contested this classical description of the anatomy of the spinal dura mater.

These new studies revealed that each layer of the dura consists of both elastic and collagen fibers that do not demonstrate any specific orientation. Interestingly, recent measurements of spinal dura thickness demonstrated that the posterior dura varies greatly in thickness within individual and between individuals. Subsequently perforation of a thick area of dura is less likely to lead to a CSF leak (and PDPH) than a perforation in a thin area, and this may in part explain the unpredictable consequences of a dural puncture with a spinal/ epidural needle[3].

Cerebrospinal fluid: physiology

About 500 ml of cerebrospinal fluid is produced per day (0.35 ml/min). CSF production occurs primarily in the choroid plexus, but there is growing evidence of extrachoroidal CSF production[4-6]. The total volume of CSF in the adult is approximately 150 ml, 50% of which is within the cranium. The CSF pressure in the lumbar region in the supine position ranges between 5 and 15 cm H_2O. On assuming the vertical position, this pressure increases to over 40 cm H_2O.

Consequences of dural puncture:

The consequences of perforation of the spinal dura are that there will be leakage of CSF. Although the loss of CSF and lowering of the CSF pressure is not disputed, the actual mechanism producing the post-dural puncture headache remains unclear[7-13]. The widely accepted theory explaining the pathophysiology of PDPH is based on the assumption of persistent leakage of CSF through the hole made by the spinal or epidural needle and decrease in CSF volume or pressure, or both, which leads to shifts of intracranial contents and traction on pain sensitive structures. Loss of CSF leads to intracranial hypotension and a demonstrable reduction in CSF volume and pressure.

The adult subarachnoid pressure of 5-15 cm H_2O may be reduced to 4 cm H_2O or less. The rate of CSF loss through the dural hole is generally greater than the rate of CSF production, particularly with needle sizes greater than 25GA. The sudden decrease in the CSF volume may also activate adenosine receptors, thus producing arterial and venous vasodilatation and subsequently clinical symptoms of PDPH. The density of CSF may also affect the incidence of headache (it has been reported that CSF density in pregnant women, who are particularly susceptible to PDPH, is significantly lower)[14].

INCIDENCE:

There is considerable variability in the incidence of PDPH, which is affected by many factors such as age, gender, pregnancy, and needle type and size (Table 1)[2-4]. In 1989 the incidence of PDPH was nearly 70%. This alarmingly high incidence of PDPH was attributable to the use of large gauge, cutting edge spinal needles. Over time the use of fine gauge spinal (Pencan, Sprotte) has produced a great reduction in the incidence of PDPH[3]. The clinical signs of PDPH may be observed following intentional dural puncture associated with the administration of spinal anesthesia or combined spinal-epidural anesthesia or unintentional dural puncture during epidural anesthesia.

Table 1
Factors affecting the incidence of PDPH

1. Age
2. Gender
3. Pregnancy
4. Needle size
5. Needle type
6. Number of attempts

Spinal anesthesia:

The incidence of headache after spinal anesthesia varies greatly between studies. The incidence is 40% with a 20GA needle; 25% with a 25GA needle; 2-10% with a 26GA needle, and less than 2% with a 29GA needle[3]. However, technical difficulties are common when spinal block is attempted with needles of 29GA or smaller. The principal factor responsible for the development of PDPH is the size of the dural perforation. Other factors such as the shape of the dural perforation and the orientation of the spinal needle have a less significant role. Therefore, a balance has to be struck between the risk of dural puncture headache and technical failure. Most experts agree that 25-26 and 27GA needles probably represent the

optimum needle size for spinal anesthesia. Clinical and laboratory studies confirmed that pencil-point needles produce fewer PDPHs than cutting edge spinal needles.

Diagnostic lumbar puncture:

Until recently, diagnostic lumbar puncture was commonly performed with a 20 or even 18GA cutting edge needle leading to a high incidence of PDPH. Although most anesthesiologists are critical of the use of large gauge needles for lumbar puncture, many neurologists still maintain that adequate flow of CSF can only be achieves with spinal needles of 22 GA or larger.

The obstetric patient:

The obstetric patient is at particular risk of dural puncture (and the subsequent headache) because of sex, young age, and the widespread application of regional anesthesia[4,7,9,10,15]. Loss of resistance to air confers a higher risk of dural puncture than loss of resistance to fluid (normal saline)[7]. Unintentional dural puncture complicating epidural anesthesia vary in incidence from 0,19-4,4%.

The incidence of epidural needle-induced PDPH in parturients has been reported to range 76-85%[4]. It has been suggested that the incidence of unintentional dural puncture during epidural anesthesia is inversely related to operator experience. However, sleep deprivation, operator fatigue and the effect of night work may be a confounding variable producing the higher incidence of unintentional dural puncture in junior personnel performing epidural analgesia.

CLINICAL SYMPTOMATHOLOGY:

PDPH is a well-established complication of procedures in which the dura mater of the spinal cord is punctured. The classic symptoms of PDPH consist of photophobia, nausea, vomiting, neck stiffness, tinnitus, diplopia and dizziness in addition to the often, severe cephalgia (Table 2). It may seem more accurate to call the clinical spectrum of symptoms that follow dural puncture, the post dural puncture syndrome (PDPS), rather than PDPH, which falsely implies the headache as the only manifestation[4]. The headache is usually severe and throbbing, frontal in origin, with radiation to the occiput, and is exacerbated by sitting or standing. The positional nature of the headache, and dramatic improvement on assuming the supine position remains the standard diagnostic criterion for this condition. In general PDPH is more common in young women, particularly in pregnancy.

Table 2
Common symptoms of PDPH

1. Photophobia
2. Náusea
3. Vomiting
4. Neck stiffness
5. Tinnitus
6. Diplopia
7. Dizziness
8. Cephalgia

The differential diagnosis of PDPH is often clear from the history of dural puncture and the presence of a severe postural headache. However, it is important to consider alternative causes of headache (Table 3).

Table 3
Differential diagnosis of PDPH in pregnant women

1. Caffeine-withdrawal headache
2. Non-specific headache
3. Migraine
4. Meningitis
5. Sinus headache
6. Pre-eclampsia
7. Drugs (amphetamine, cocaine)
8. Pneumocephalus-related headache
9. Intracranial pathology (hemorrhage, venous thrombosis)

TREATMENT:

Current treatment modalities for PDPH include theophylline, caffeine, sumatriptan, epidural saline, epidural dextran, and epidural blood patch (EBP) (Table 4). However, only the EBP has apparent benefits[3-7].

Psychological:

PDPH during the postpartum period is almost always a complication of regional anesthesia. The obstetric patient is usually aware that her headache is an iatrogenic problem, and she may be angry, resentful and/or depressed. Headache may make it difficult to care for the newborn and to interact with other family members. It is therefore important to give the parturient a thorough explanation of the reason for the headache, the anticipated time course, and the therapeutic options available[3].

Table 4
Current treatment modalities of PDPH in
pregnant women

1. Caffeine
2. Theophylline
3. Sumatriptan
4. Epidural saline
5. Epidural dextran
6. Subarachnoid catheter
7. Epidural blood patch

Caffeine:

Caffeine is a central nervous system stimulant, which produces cerebral vasoconstriction. It is available in an oral and intravenous form. The oral preparation is well absorbed from oral mucosa with peak blood levels reached in approximately 30 minutes[3]. Caffeine easily crosses the blood-brain barrier and has a long half-life of 3-8 hours. Several studies however, showed that the beneficial effect of caffeine might be transient. Caffeine appears in breast milk in very small amounts.

Sumatriptan:

Sumatriptan is a serotonin agonist that affects predominantly type 1-D receptors. It promotes cerebral vasoconstriction in a similar way to caffeine. Sumatriptan has been advocated to the treatment of migraine and recently, for PDPH[3-7]. This drug is expensive and must be given by subcutaneous injections.

Epidural saline:

It has been speculated that an epidural injection of saline would, in theory, produce the same «mass effect» as autologous epidural blood patch, and restore normal CSF dynamics. Advocates of an epidural saline infusion (or boluses) maintain that the lumbar injection of saline raises epidural and subarachnoid pressures[8]. However, to date no studies have demonstrated either a sustained rise in CSF pressure or accelerated closure of the dural hole (tear) following administration of epidural saline[3]. It is therefore difficult to conclude from the evidence that epidural saline administration will restore normal CSF dynamics.

Epidural dextran:

It has been implied that the high viscosity and high molecular weight of dextran may slow its removal from the epidural space. However, it is unlikely that dextran would act any differently to normal saline in the epidural space[3,4]. Any pressure increase with the epidural and subarachnoid space would, like saline, be short lived. Additionally, it has been reported that dextran does not demonstrate any inflammatory response that would promote the dura healing process.

Subarachnoid catheters:

Following unintentional dural puncture with a large gauge epidural needle, it has been suggested that placement of a subarachnoid catheter through the dural hole may provoke an inflammatory reaction that will seal the puncture site[5,8,15-17,19-23]. Histological animal and human studies with long-term subarachnoid catheters confirm the presence of an inflammatory reaction at the catheter insertion site. Further studies are needed.

Epidural blood patch:

Two theories have been proposed to explain EBP efficiency in the treatment of PDPH[4,5,11,13]. The first theory suggests that the autologous blood injected in the epidural space forms a clot, which adheres to the dura mater and directly patches the hole. The second theory suggests that the volume of blood injected in the epidural space increases CSF pressure, thus reducing traction of pain sensitive brain structures, leading to relief of symptoms. The optimal volume of blood to be injected in the epidural space remains controversial.

PREVENTION:

The incidence of epidural needle-induced PDPH in parturients following dural puncture with a large bore (e.g., 18-GA) needle has been reported to range 76-85%[4]. Although a few measures have been proposed to prevent PDPH (intrathecal injection of saline, insertion of the epidural catheter into the subarachnoid space through the dural hole, none have been shown to work with certainty to date[2,3,8,12,15-23].

In two recent reports[5, 6] an unintentional dural puncture with 18 GA epidural needle in several parturients was followed by (*1*) injection of the CSF in the glass syringe back into the subarachnoid space through the epidural needle, (*2*) insertion of a epidural catheter into the subarachnoid space (now referred to as an intrathecal catheter), (*3*) injection of a small amount of preservative free saline (3-5 ml) into the subarachnoid space through the intrathecal catheter, (*4)* administration of bolus and then continuous intrathecal labor analgesia (in one patient followed by the administration of spinal anesthesia for Cesarean section) through

the intrathecal catheter, and then (*5)* leaving the intrathecal catheter in-situ for a total of 12-20 hours. Interestingly, PDPH occurred in only one of these cases[5,6].

These findings suggested that following unintentional dural puncture with an 18-gauge epidural needle in parturients, sequential (Table 5) (*1)* injection of the CSF in the glass syringe back into the subarachnoid space through the epidural needle, (*2)* insertion of a 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 bolus and then continuous intrathecal labor analgesia, and (*5)* leaving the catheter in-situ in the subarachnoid space for a total of 12-20 hours decreased the incidence of PDPH from 76-85% to 6.6 %[6].

Table 5
Maintaining CSF volume: the new method of prevention of PDPH

1.	Injecting the CSF in the glass syringe back into the subarachnoid space through the epidural needle
2.	Passing the epidural catheter through the dural hole into the subarachnoid space
3.	Injecting of 3-5 ml of preservative free saline into the subarachnoid space through the intrathecal catheter
4.	Administering bolus and then continuous intrathecal labor analgesia through the intrathecal catheter
5.	Leaving the subarachnoid catheter in-situ for a total of 12-20 hours

The replacement of the escaped CSF volume by injecting the small amount of CSF filling the syringe back into the subarachnoid space and 3-5 ml of preservative-free normal saline seems a low risk maneuver; however, the replacement of this small amount of CSF volume seems of questionable significance when one takes into consideration the total volume of CSF (approximately 150 ml) and the rate of production of CSF (0.35 ml/min) in the subarachnoid space. Nevertheless other studies did find 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 %[8].

Four reports have suggested that leaving the catheter in the dural hole for several hours may decrease the incidence of PDPH[8,15-17]. First, Cohen et al. reported only a 20% incidence of PDPH in a group of 10 parturients receiving continuous spinal analgesia via a 20-gauge catheter inserted after unintentional dural puncture[15]. Second, Dennehy et al. found in three patients that immediate insertion of an intrathecal catheter after inadvertent dural puncture followed by intermittent injections of either bupivacaine or lidocaine with fentanyl for analgesia during labor and delivery prevented PDPH in all three patients[16]. Third, Cohen et al. in a retrospective study found in thirteen Cesarean section patients a zero incidence of PDPH when accidental dural puncture was followed postoperatively by continuous spinal analgesia through an intrathecal catheter[17]. Fourth, Charsley et al. found in six patients that intrathecal catheter placement following accidental dural puncture, and injection of 10 ml of normal saline prior to removal of the intrathecal catheter after an unknown period of time, effectively prevented PDPH in all six patients[8].

These four reports are supported by the observation that the incidence of PDPH is near zero after continuous spinal anesthesia in nonpregnant patients[19]. Peterson et al. found no patients with PDPH in a retrospective study of 52 consecutive cases of continuous spinal anesthesia[19].

However, and very importantly to the contrary, Norris et al. in a prospective study found that inserting an intrathecal catheter for at least two hours and providing continuous spinal anesthesia after unintentional dural puncture does not greatly decrease the incidence of PDPH in parturients[19]. In this study of 56 parturients who suffered unintentional dural puncture with an 18-gauge epidural needle, 35 women had insertion of an intrathecal catheter followed by continuous spinal analgesia throughout labor and delivery, while a second group of 21 women received continuous lumbar epidural analgesia (catheter inserted following re-identification of the epidural space). The difference in PDPH between the two groups was not significant (55% in the continuous spinal analgesia group versus 53% in the continuous epidural group)[19]. Still, the 55% incidence of PDPH is moderately below the 76-85% range of PDPH[4] when no prophylaxis is undertaken.

At least two different mechanisms to explain the decreased incidence of PDPH after intrathecal catheter insertion have been postulated; first, the intrathecal catheter "plugs" the dural tear, decreasing or stopping the efflux of CSF from the subarachnoid space[16], second, inserting a catheter in the dural hole leads to an inflammatory reaction, with edema or fibrin exudates subsequently sealing the dural tear after catheter removal[21]. Others described formation of fibrin around the "chronic" (at least 5 to 7 days) intrathecal catheter at the dural tear in an experimental animal study[22]. Thus, in addition to directly plugging the dural hole, the long-term

presence of the intrathecal catheter may also promote an inflammatory response around the dural hole, which facilitates dural closure after catheter removal.

It is difficult (at this time) to indicate, the relative importance of these five maneuvers in decreasing the incidence of PDPH. The authors speculated that the immediate insertion of the epidural catheter into the subarachnoid space («short term plugging») with careful attention to minimize additional CSF loss and the prolonged presence of the catheter in the subarachnoid space («long term plugging»), seem the most likely mechanisms of prevention of continuous leakage of CSF and subsequent development of PDPH[5,23]. Further studies are needed.

Summary

The combination of (*1*) injecting the CSF in the glass syringe back into the subarachnoid space through the epidural needle, (*2*) passing the epidural catheter through the dural hole into the subarachnoid space, (*3*) injecting of 3-5 mL of preservative free saline into the subarachnoid space through the intrathecal catheter, (*4*) administering bolus and then continuous intrathecal labor analgesia through the intrathecal catheter, and then (*5*) leaving the subarachnoid catheter in-situ for a total of 12-20 hours appears to be a promising technique in preventing PDPH (Table 5). All these five components are aimed at maintaining CSF volume[5, 6, 23].

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