Objective

Review the newest anesthetic consideration for the parturient with an unintentional dural puncture

Syllabus

More than 100 years have passed since the initial description of the post-dural puncture headache (PDPH). However, this unique clinical entity still continues to fascinate anesthesiologists, and numerous studies on its pathophysiology, prevention, and treatment, have been published. Using himself as subject, August Bier demonstrated spinal anesthesia one day, and “spinal headache” (PDPH) the following morning. Since those early days in 1898, we have made enormous progress in understanding this clinical entity, including its epidemiology, pathophysiology, clinical symptoms and treatment.

Unintentional dural puncture complicating epidural anesthesia vary in incidence from 0.19-4.4%. 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. The incidence of epidural needle-induced PDPH in parturients has been reported to range 76-85%. PDPH is a well-established complication of procedures in which the dura mater of the spinal cord is punctured. 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 (accidental) dural puncture during epidural anesthesia.

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. 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. Richardson et al. have reported that CSF density in pregnant women, who are particularly susceptible to PDPH, is significantly lower.

The classic symptoms of PDPH consist of photophobia, nausea, vomiting, neck stiffness, tinnitus, diplopia and dizziness in addition to the often, severe cephalgia. 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. 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.

Current treatment modalities for PDPH include theophylline, caffeine, sumatriptan, epidural saline, and epidural blood patch (EBP). However, only the EBP has apparent benefits. Two theories have been proposed to explain EBP efficiency in the treatment of PDPH. 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

The incidence of epidural needle-induced post dural puncture headache (PDPH) in parturients following dural puncture with a large bore (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\(^1\), insertion of the epidural catheter into the subarachnoid space through the dural hole\(^11-15\)), none have been shown to work with certainty to date.

This lecture will describe the new intrapartum anesthetic management of cases in which the performance of epidural anesthesia in parturients was complicated by an unintentional dural puncture with an 18-GA epidural needle. In all described cases, the unintentional dural puncture was followed by: injection of the CSF in the glass syringe back into the subarachnoid space through the epidural needle\(^1\), insertion of a epidural catheter into the subarachnoid space (now referred to as an intrathecal catheter)\(^2\), injection of a small amount of preservative free saline (3-5 mL) into the subarachnoid space through the intrathecal catheter\(^3\), 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\(^4\) leaving the intrathecal catheter in-situ for a total of 12-20 hours. PDPH occurred in only one of these cases.

Our findings suggest that following unintentional dural puncture with an 18-gauge epidural needle in parturients, sequential\(^1\) 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\%\(^1\) to 14\%\(^2\).

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 Charsley et al. 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\% (the control group that did not receive the normal saline injection) to 32\%\(^11\).

Four reports have suggested that leaving the catheter in the dural hole for several hours may decrease the incidence of PDPH\(^11-14\). 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\(^12\). 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\(^13\). Third, Cohen et al.
Decrease in the Incidence Of Post-Dural Puncture Headache: Maintaining CSF Volume
Disminución de la Incidencia de Cefalea Post Punción Dural (CPPD): Manteniendo el Volum. del LCR

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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. 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.

These four reports are supported by the observation that the incidence of PDPH is near zero after continuous spinal anesthesia in nonpregnant patients. Peterson et al. found no patients with PDPH in a retrospective study of 52 consecutive cases of continuous spinal anesthesia. Kallos et al. also reported no patients with PDPH in a study of 121 patients who underwent total hip replacement under continuous spinal anesthesia.

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. 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). Still, the 55 % incidence of PDPH is moderately below the 76-85 % range of PDPH when no prophylaxis is undertaken.

At least two different mechanisms to explain the decreased incidence of PDPH after intrathecal catheter insertion have been postulated. Dennehy et al. postulated that the intrathecal catheter “plugs” the dural tear, decreasing or stopping the efflux of CSF from the subarachnoid space. Denny at al. postulated that 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. Yaksh et al. described formation of fibrin around the “chronic” (at least 5 to 7 days) intrathecal catheter at the dural tear in an experimental animal study. 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 to indicate, the relative importance of the five maneuvers performed in our study in decreasing the incidence of PDPH. We speculate 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. We conclude that the combination of injecting the CSF in the glass syringe back into the subarachnoid space through the epidural needle, passing the epidural catheter through the dural hole into the subarachnoid space, injecting of 3-5 mL of preservative free saline into the subarachnoid space through the intrathecal catheter, administering bolus and then continuous intrathecal labor analgesia through the intrathecal catheter, and then leaving the intrathecal catheter in-situ for a total of 12-20 hours appears to be a promising technique in preventing PDPH. All these five components are aimed at maintaining CSF volume.
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References