Chapter 7

Post Dural Puncture Headache – We Can Prevent It

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Additional information is available at the end of the chapter

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1. Introduction

Although modern anesthesiology has made great progress in the last decades, neuraxial anesthesia (NA) is still the keynote of regional blockade [1]. NA is popular for its effectiveness in producing anesthesia for providing excellent intraoperative neuromuscular paralysis and in generating analgesia for relieving postoperative pain if continuously infused [2, 3]. As the NA techniques are used popularly in clinic, post dural puncture headache (PDPH), a common iatrogenic complication resulted from post-spinal taps or accidental dural puncture (ADP) subsequent to epidural block, is frequently reported [4] and becomes a challenge to health caregivers [5]. Although the incidence of PDPH in research volunteers is ~6% [6], in patients for whom the NA is for clinical purposes the prevalence of PDPH ranges from 10% to over 80% in different aged patients underwent either epidural or spinal or combined block [7].

Investigations on the risk factors of PDPH revealed that female, age, perpendicular bevel orientation [8], previous history of PDPH [9], repeated dural puncture [10], needle gauge and design [11], and pregnancy [12] are factors substantially related with the occurrence of PDPH. The leakage of cerebrospinal fluid (CSF) was considered as the major cause of PDPH [13], whereas its real etiology is unknown. These procedure- and nonprocedure-related factors in combination determine the patterns of development of PDPH. Several procedures and methods were identified effective in treating and reducing the incidence of PDPH based on the knowledge of procedure-related factors, but whether could we prevent this morbid prior to its occurrence?

Techniques developed based on how to reduce CSF leakage are classified into either preventive or therapeutic ones. Although the results from the differently designed studies were inconsistent [4], one consensus on this topic reached is that we can prevent, at least in part, PDPH with currently available methods. Small size pencil point spinal needle [14, 15], parallel bevel orientation [8], liquid use for the loss of resistance (LOR) in epidural puncture [16], and

prophylactic epidural blood patch [17, 18] are preventive considerations in reducing PDPH. Therapeutically, intrathecal saline injection [19, 20], repeatable epidural blood patch [21], and compensatory intrathecal catheterization for drug or fluid administration [22] are means treating the on-going PDPH. Although the effectiveness of these methods is changing in different population at different ages under different clinical procedures, they are still promising for our patients.

Beside abovementioned procedure-related techniques, emerging pharmacological data support the use of analgesics. The most recent systematic review and meta-analysis [23] revealed that morphine, cosyntropin, and aminophylline are effective for reducing the incidence of PDPH with any severity, but dexamethasone on the contrary increases the risk of PDPH. For fentanyl, caffeine, and indomethacin, no conclusive evidence reached in the effectiveness and safety for preventing and treating PDPH due to the design quality and low power of the available studies. In consideration of the contribution of high body mass index (BMI) [24] and non-smoking [25] to PDPH, we thus cannot only attribute PDPH to CSF leakage. Caregivers need take careful consideration of the methods listed in this chapter to prevent and improve the clinical outcomes of this iatrogenic morbid because of its multifactorial originality.

2. History and epidemiology

First epidural blockade was reported by an American physician Dr. James Leonard Corning in 1885 [26]. The actual history of spinal anesthesia can be traced back to 1888 by German physician Dr. Heinrich Irenaeus Quincke and 1889 by Britain physician Dr. Walter Essex Wynter aspirated CSF from patients with meningitis for lowering intracranial pressure [27-29]. In 1898, Dr. Karl August Bier from Germany performed first elective spinal anesthesia for surgery [30, 31]; at the same time, Dr. Bier and his assistant they themselves experienced spinal anesthesia, and reported plus another four patients (6/9) with PDPH symptoms [32]. Since that time, analgesics, hydration, and bed rest became the basic constitutes in treating PDPH [33]; however there were still some 40% cases showed no response to these therapeutics. From the early 1970s, anesthesiologists began to use epidural blood patch (EBP) to treat severe PDPH. Until 1990, EBP was first recommended by official guideline [34].

Over the past one century, the incidence of PDPH was sharply decreased from ~70% to ~1% [35], whereas the recently reported occurrence of PDPH is still seeing a big difference in various clinical settings from different regions when diverse techniques were used in patients with different ages. A Nordic survey found the incidence of ADP in Obstetric setting is 1% [36], and 73% of the ADP patients developed PDPH [37]. The incidence of PDPH in Obstetric in Middle East is 2-4.6% [14, 38], 22.7% in Western Africa [39], 16.9% in Southeast Asia [40], 16.6% in North Europe [41], and 6% in North America [42, 43]. In non-Obstetric patients, about 18% patients developed PDPH after spinal anesthesia [44], however a lower incidence (4%) was then reported in the next year by the same group [45]. In the earlier time, another group from Denmark reported the occurrence of PDPH was 7.3% in patients underwent different types of surgeries below the diaphragm after spinal anesthe-

sia [46]. In orthopedic patients, about 1.6% experienced PDPH after continuous spinal anesthesia (CSA) or combined spinal epidural anesthesia (CSE) in South America [47]. In patients who underwent placement of an intrathecal drug delivery system (IDDS), 23% developed PDPH [48]. About 11-30.9% children with malignant disease attended for diagnostic or therapeutic lumbar puncture experienced PDPH [49, 50].

3. Risk factors

Clinical and epidemiological studies support a connection between PDPH and certain demographic factors. For adult, the frequency of PDPH was less in older age patients (51-75 years) than younger age comparisons (30-50 years) [51]. Children younger than 13 years rarely get PDPH [49, 52], but that does occur with increasing frequency in adolescents and are similar to those seen in adults [53]. To child younger than 13 years and adult older than 50 years, they have less PDPH incidence than their peers that largely may be related to the reduced CSF pressure [54, 55]. While there are some inconsistencies upon gender as an independent risk factor for the development of PDPH, a recent meta-analysis confirmed the declaration that the odds of developing a PDPH were significantly lower for male than nonpregnant female subjects with an odds ratio (OR), 0.55 and 95% confidence interval (95% CI), 0.44-0.67 [56]. Lower weight is found to be strongly associated with the higher incidence of PDPH [24] and cumulating evidence showed an inverse relationship between BMI and PDPH [57, 58] suggesting that heavier patients in general have higher intra-abdominal pressure, which in turn raises intra-epidural pressure and prevents cerebrospinal fluid from leaking when ADP occurs. New survey revealed that taller height, reduced pre-procedure intravenous hydration and lower systolic blood pressure (SBP) are novel risk factors that contribute to the pathogenesis of PDPH [59]. Although the incidence of PDPH from different countries, an indicator of racial difference, seems to be different [14, 38-43], the race itself looks unlike an independent risk factor for the PDPH that was observed in the same study [60]. Interesting findings showed that smokers had a considerably reduced rate of PDPH in comparison with non-smokers suggesting an inhibitory effect of tobacco smoking on PDPH that may be associated with the stimulation role of nicotine in dopamine neurotransmission [25].

In a more recent study, severe headache after lumbar puncture and sitting position were confirmed as predicting factors of the occurrence of PDPH, and in further sitting sampling position, history of depression, multiple effort of lumbar puncture, and high perceived stress during the procedure were found to be significantly associated with a longer duration of PDPH [61]. In the same study, migraineurs showed no change at the risk of developing PDPH compared to the non-headache subjects, and epidural puncture does not trigger migraine attacks [61]. However, there was report showing that patients had a history of chronic or recurrent headache has more chance in nearly 60% to develop PDPH than those without such a history [62]. For the multiple effort of lumbar puncture that indicates the inexperience in such clinical procedures increases the possibility of PDPH [6], but in contrast, other studies found no different between experienced and inexperienced practitioners, nor does between multiple and single dural puncture [63].

Although the leakage of CSF is regarded as the major cause of PDPH, the volume of CSF removed and its role in causing PDPH is unclear. Davignon and Dennehy reported that removal of 15-20 ml of CSF reliably caused headaches [64], but Kuntz *et al.* did not find such a causal relationship [65]. So it is hard to draw a conclusion from the available data that volume change in CSF causes PDPH. In clinical practice, the volume usually removed during diagnostic lumbar punctures or spinal anesthesia is less than 5ml that means it is not likely to be a significant factor for the PDPH. However, we cannot exclude the possibility that chronic leakage of CSF over more than 15 ml after ADP or spinal anesthesia is causative for the PDPH (see detailed pathophysiology of CSF leakage below).

Prophylactic treatment with 8 mg of dexamethasone not only increases the severity and incidence of PDPH, but is also ineffective in decreasing the prevalence of intra-operative nausea and vomiting during cesarean section indicating that dexamethasone treatment is a significant risk factor for the development of PDPH [66]. Nonetheless, hydrocortisone *i.v.* (100 mg in 2 ml 8 hourly for 48 h) was found effective in reducing PDPH following spinal anesthesia [67] suggesting that glucocorticoid with different potency and half life of action may possess different function in PDPH prevention and therapy.

Pregnancy is considered as a particular factor that relates to PDPH due to the young age, female, sometimes sitting position, pregnancy-associated depression and anxiety, and the special popularity of regional anesthesia in this population [68-70], but a meta-analysis showed that pregnancy itself does not increase the risk of PDPH [71]. For some cases of PDPH, we cannot exclude some other co-founding factors including fatigue, sleep deprivation, and night work that lead to higher incidence of ADP in clinical personnel when performing epidural analgesia. Table 1 summarizes the risk factors of PDPH.

Convincing risk factors
Young age
Female
Lower BMI
Taller height
Reduced pre-procedure intravenous hydration
Lower SBP
Non-smoking
Sitting position
History of depression
History of chronic or recurrent headache
High stress during the procedure

Multiple lumbar puncture
Dexamethasone therapy
Non-convincing risk factors
Experience level of personnel
Volume of CSF removed
Pregnancy itself
Fatigue, sleep deprivation, or night work

BMI: body mass index; SBP: systolic blood pressure; CSF: cerebrospinal fluid

Table 1. Risk factors of PDPH.

4. Anatomy of meninges

There three membranes, known as that cover the spinal cord lying within the vertebral canal. The outermost layer is the dura mater, a non-adherent, dense, and tough fibrous sheath closely applied to the inner layer of bone surrounding the spinal canal. Between the dura and the walls of the spinal canal is a potential imaginary space, the epidural space or cavum epidurale, which normally occupied by a small amount of by loose areolar tissue, fat, and the anterior and posterior plexuses of the vertebral veins. Dura mater is attached above to the margin of the foramen magnum, to the axis, and to the third cervical vertebra, and below to the level of the second sacral vertebra. In normal, a potential space known as the subdural space exists between dura mater and arachnoid mater, a thin and delicate membrane lies closely beneath the dura mater. Beneath the arachnoid mater is the pia mater that intimately applied to the spinal cord. Both the arachnoid and pia mater are continuous with the arachnoid and pia surrounding the brain. There is a space between the arachnoid mater and pia mater: the subarachnoid space, which normally is filled with CSF.

The conventional conception for the structure of spinal dura mater is that it is of elastic and collagen fibers running in the longitudinal direction. Based on this, clinical studies found the PDPH incidence is less in patients who underwent spinal anesthesia during which the dura mater was cut with a perpendicular orientation to the direction of the spinal dura fibers than those with parallel bevel orientation [8, 72]. But a more extensive electron microscopic study challenged this traditional conception of the of the anatomy of the spinal dura mater, they found that the dura mater is consisted of collages fibers that are arranged in several layers parallel to the surface, and each layer does not arranged in any specific orientation [73]. Moreover, the thickness of the posterior dura mater demonstrates big difference within and between individuals suggesting that perforation at thicker dura is less likely to result in CSF leakage than the thin dura because the thicker the dura mater, the easier the retraction after

perforation, and this inter- or intra-individual variation in dural mater thickness may be an unpredictable variable affecting the management of dural puncture [74, 75].

5. Physiology of CSF

CSF secretion in adults varies between 400 to 600 ml per day, i.e. 0.28-0.42 ml/min, and about 60%-75% of CSF is produced by the choroid plexuses of the lateral ventricles and the *tela choroidea* of the third and fourth ventricles. The total volume of CSF in the adult is ~150 ml, of which 125ml distributes in cranial and spinal subarachnoid spaces and 25 ml in the ventricles. Therefore, CSF is renewed four to five times every 24 hours in young adults. CSF circulation is a dynamic phenomenon that pulses in response to the systolic pulse wave in choroidal arteries. Ageing-related cerebral atrophy and reduction in CSF turnover enlarge the CSF compartment markedly, and aging-associated slowing down of the CSF renewal all may explain the reason why aged population had a lower incidence of PDPH [51].

CSF pressure, one part of the intracranial pressure, is the result of a dynamic equilibrium between CSF secretion, absorption and resistance to flow. Physiological values of CSF pressure vary according to individuals and study methods between 13 and 20 cmH_2O in adults and 4 and 6 cmH_2O in infants [76]. In the lumbar region in the supine position, CSF pressure ranges between 5 and 15 cmH_2O, and this pressure can increase to over 40 cmH_2O when on the vertical position [77]. In the prone position, CSF pressure changes from 8 to 21 cmH_2O, but in lateral decubitus position it reduces to 7-17 cmH_2O [78]. Besides, the normal range for lumbar CSF pressure in children is 10 to 28 cmH_2O when measured in a flexed lateral decubitus position [79]. CSF pressure is determined by parenchymal and venous pressures. Increased SBP exerts negative feedback on choroidal secretion by decreasing the pressure gradient across the blood-CSF barrier and by reducing cerebral perfusion pressure.

6. Pathophysiology of CSF leakage and pathological mechanisms

CSF leak is defined as the escape of CSF from any tear or hole in the meninges. The direct consequence of CSF leak is the drop of CSF volume and then the pressure. It is not that clear for the causal relation between CSF leak and PDPH pathogenesis, however the explanatory theory on the role of CSF leak in PDPH onset is still widely accepted. Leakage of the CSF is the most common cause of spontaneous intracranial hypotension [77]. Theoretically, lumbar puncture-induced CSF leak is consisted of two phases: acute and chronic phases. The acute phase is largely resulted from the abrupt outflow of CSF from the broken within minutes to several hours, during which the CSF pressure dives down to a lower level (~3-4 cmH_2O) that eventually leads to shifts of intracranial contents and gravitational traction on pain sensitive structures, which worsens when the patient is upright and is relieved on lying down [80]. The chronic phase refers to a stage of which started from the formation of the new CSF pressure balance (several hours to one day after dural puncture) to the complete resolution from the

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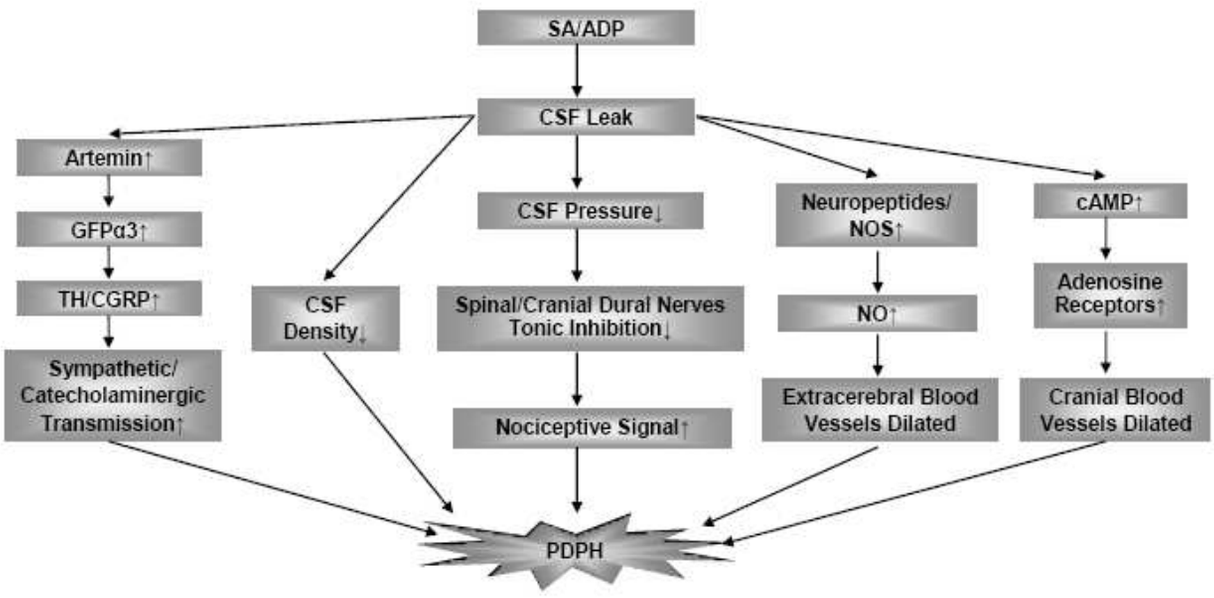
puncture (1-6 weeks). This phasic alteration in CSF leakage can explain, at least in part, why the onset of PDPH in some patients occur 1 to 7 days after ADP, but some appeared 12 days post puncture [81]. In addition, the loss of CSF may activate adenosine receptors that subsequently dilate intracranial arteries and veins and then clinical manifestations of PDPH [82]. Based on the findings that pregnancy and the immediate postpartum period are associated with the lowest CSF density [83] and the particular high incidence of PDPH in Obstetric setting [68-70], the CSF density change during the chronic leakage of CSF was also considered as a potential reason of the PDPH.

For some patients they showed "crushing" postural headache without abnormal diagnostic lumbar puncture and computed tomography (CT) angiogram suggesting that the conventional understanding on the pathogenesis of PDPH based on the over-rigorous pooled analyses needs to be reconsidered [80]. In fact, the occurrence of PDPH has its own pathological bases. For the cranial innervations, studies showed that the dura mater is heavily innervated and most likely cause intense pain [84], and abnormal distention of intracranial nerve and extracerebral blood vessels all can consequently activate the trigeminal nervous system that were thought to be the origins of headache [85]. Functional immunohistochemistry found that neuropeptides and nitric oxide synthase (NOS) are expressed in the nerve fibers of the supratentorial dura mater and the structural alterations of nitroxidergic axons innervating blood vessels of the dura mater support the idea that nitric oxide (NO) is involved in the induction of headache [86]. Therefore further studies are needed on the relationship among dura mater innervations, expression of neuropeptides and NOS, and PDPH.

Artemin, a member of the glial cell line-derived neurotrophic factor family, is a vasculature-derived growth factor shown to regulate migration of sympathetic neuroblasts and targeting of sympathetic innervation [87]. Recent evidence supports the role of artemin in cold pain [88] and inflammatory pain [89]. Moreover, the expression of artemin was detected in the smooth muscle of dural vasculature, and its receptor GFRα3 was found present in nerve fibers that closely associated with tyrosine hydroxylase (TH) or calcitonin gene-related peptide (CGRP) [90] suggesting that artemin is involved in dural afferent activity through modulating both primary afferent and sympathetic systems. In further, catecholaminergic nerve fibers innervate human cranial dura mater in density, and these nerve fibers are more abundant in the perivascular dural zone than in the intervascular zone at the basal region [91]. In collection, given TH functions as the precursor of catecholamine (norepinephrine and epinephrine) [92], a potential interaction exists among artemin, sympathetic regulation, and catecholaminergic transmission in nerves located in cranial dura mater, and this interaction may underlie the occurrence of PDPH.

Dural innervations are of importance as, like its cranial counterpart, the spinal dura mater and its nerve root sleeves may be a source of primary pain. Different types of nervous branches are given off to the spinal dura mater within the vertebral canal. The nerves in the spinal dura mater have already been described as nociceptive sensory fibers [93-96], and they also belong to sympathetic vasomotor [97, 98]. We hereby proposed that a tonic inhibition of the spinal and cranial dural nerves exists under normal CSF pressure, but this inhibition would be reduced or reversely activated by chronic leakage of CSF after lumbar puncture. However,

such reduction in tonic inhibition or/and reverse activation of the dural nerves does not determine the occurrence of PDPH, which depends on the alteration extent of the tone that can evoke nociceptive activation, i.e. it should at least reach the activation threshold. Based on this hypothesis, it can partially explain why not all patients after ADP will develop PDPH [37]. Figure 1 depicts the potential pathological mechanisms of PDPH.



SA: spinal anesthesia; ADP: accidental dural puncture; CSF: cerebrospinal fluid; GFPα3: glial-cell-line-derived neurotrophic factor family receptor alpha-3; TH: tyrosine hydroxylase; CGRP: calcitonin gene related peptide; NOS: nitric oxide synthase; NO: nitric oxide; cAMP: cyclic adenosine monophosphate; PDPH: post-dural puncture headache.

Figure 1. Pathological mechanisms of the pathogenesis of PDPH.

7. Needle gauge and tip configuration

A huge body of evidence and systematic review support the view that the diameters of the needles that pierce the spinal dura mater and the tip design, cutting-edge or pencil-point, are two key facets that determine the eventual incidence of PDPH [99]. Of the same type needles, 29G (19%) compared with 25G Quincke needle (17%) led to no reduction of PDPH [44]. Similarly, both 25G and 26G Quincke needle had same incidence of PDPH (8-9%), but in comparison, 24G Sprotte non-cutting tip needle results in a significantly lower incidence (1.5%) [100]. Patients receiving spinal anesthesia with a 27G Quincke needle suffered significantly more frequently from PDPH (6.6%) than the 27G pencil-point needle controls (1.7%) [45]. In Obstetric women, 25G Quincke, 27G Quincke and 27G Whitacre spinal needles produce 8.3%, 3.8% and 2.0% of PDPH, respectively [14], and 10% in the 25G Quincke and none in the 24G Gertie Marx spinal needle [101]. For the non-Obstetric patients, the incidences of PDPH for 27G Quincke and 27G Whitacre spinal needles were 2.7% and 0.37%, respectively [102].

However, in 33% patients reported PDPH, no statistically significant differences were found between Spinocan 22G sharp bevel needles or Whitacre 22G pencil point needle [103]. For 26G Eldor spinal needles, it was found to be better (0%) than 25G Quincke spinal needle (8.3%) for Cesarean sections to decrease the incidence of PDPH [104]. In pediatric patients, 5% in the 26G Atraucan and 4% in the 27G Whitacre spinal needle developed PDPH after spinal anesthesia for subumbilical surgery [105], and pencil point needle causes less PDPH compared to cutting point needle: 0.4% versus 4.5%, respectively [15]. Vallejo and colleagues compared the incidence of PDPH in five spinal needles and found that the PDPH were 5%, 8.7%, 4%, 2.8%, and 3.1% for 26G Atraucan, 25G Quincke, 24G Gertie Marx, 24G Sprotte, and 25G Whitacre needles, respectively in Obstetric patients [106]. Table 2 summarizes the incidence of PDPH after different spinal needles.

Needle	Incidence of PDPH	Tip Configuration	Showcase
Atraucan	5% (26G)	Combination Quincke-pencil point bevel	
Eldor	0% (26G)	Double hole pencil point	
Gertie Marx	0%-4% (24G)	Single port pencil point	
Quincke	2.7%-19% (29G-25G)	Cutting edge	
Spinocan	39% (22G)	Cutting edge	
Sprotte	1.5%-2.8% (24G)	Single port pencil point	
Whitacre	0.37%-39% (22G-27G)	Single port pencil point	

Table 2. Incidence of PDPH after different spinal needles.

8. Therapeutic strategy

The occurrence of PDPH resulted from ADP or spinal anesthesia is completely unavoidable, although we can reduce its incidence via various preventive means. Therefore, health caregivers need familiar with all potential therapeutic strategies, and treat them following different treatment protocols that are divided into four steps: conservative treatment (1st step), aggressive medical treatment (2nd step), conventional invasive management (3rd step), and aggressive invasive management (4th step) [4]. Table 3 summarizes the 4-step therapeutic maneuvers for PDPH.

8.1. First step: Conservative treatment

Initially, conservative methods are recommended for the treatment of PDPH largely because of its self-limiting property. The use of abdominal binder for PDPH patients is mainly based on its pressure transmission from the increased pressure of abdominal cavity to CSF pressure [106]. Although no powerful evidence supports this hypothesis, and the CSF pressure can change along with the intra-abdominal pressure [107], it is uncertain the consequently increased CSF pressure at early period of lumbar puncture would push more CSF exit from the broken. Conventionally, it has been suggested that PDPH would be less common if patients routinely have a period of bed rest after dural puncture because about 1-70% patients after the puncture experienced postural headache. In addition, giving supplementary fluids additional to the normal dietary intake can restore the loss of CSF. Although the degree of CSF leak does not correlate with the severity of the symptoms in a PDPH [40], it is assumed that improvements in the ratio of CSF production to CSF leak will improve the clinical picture. Dehydration can result in a decrease in CSF production [108]. However, if a patient is appropriately hydrated, and the rate of CSF production is normal, there is no evidence that overhydration will increase the rate of CSF production any further. For both bed rest and hydration, the most recent systematic review did not find convincing evidence supporting the routine bed rest after dural puncture is beneficial for the prevention of PDPH onset, and also it is still unclear whether vigorous intravenous fluid supplementation has any prophylactic or therapeutic benefit in alleviating PDPH [109].

Due to the ethical consideration, pre-operative communication will let the patients know that PDPH is a common iatrogenic complication, and the subsequent problems include the inability to perform daily activities, an extended length of stay (LOS) at hospital, and a higher visiting rate to the emergency room after discharge. All these will raise patients' anxiety to their possibly miserable experiences after regional anesthesia, and the psychological stress will be exacerbated if PDPH eventually occurred. Therefore, psychological support will help PDPH patients more precisely understand: 1) PDPH is a self-terminating process; 2) many medical procedures can alleviate and treat PDPH; 3) active cooperation with clinicians will promote resolution; 4) keep normal diet; and 5) think solutions with faith but not with fear. So try to comfort or reassure PDPH patients psychologically will enhance their confidence to the treatments, and improve the outcomes [110].

Symptomatic analgesia was also used as the conservative management of PDPH. Oral acetaminophen (1000 mg) along with fluid administration was suggested [4, 111], whereas the actual effect is unknown. Prophylactic administration of acetaminophen (500 mg)-caffeine combination did not prevent PDPH [112]. Non-steroidal anti-inflammatory drugs (NSAIDs), the most popular over-the-counter drugs for analgesia also can be used for PDPH treatment [32]. Antiemetics combined with other analgesics were suggested for headache or migraine, but whether such medication performs effective function in PDPH is not elucidated. Dexamethasone, a traditional anti-inflammatory glucocorticoid, is found possessing antiemetic effect for postoperative patients [113], and also was used in migraine treatment [114], but for PDPH no convincing evidence was found [23]. Erol reported that gabapentin, a gamma-aminobutryic acid (GABA) analog, significantly reduced pain, nausea and vomiting compared

to ergotamine/caffeine combinations in patients with PDPH [115] suggesting that gabapentin exerts function in PDPH patients through both analgesic and antiemetic effect.

8.2. Second step: Aggressive medical treatment

Aggressive medical treatments that include subarachnoid catheter left *in situ*, occipital nerve block, intravenous methylxanthines, and symptomatic therapies are recommended once the conservative management was not that effective in treating PDPH. Leaving a subarachnoid catheter *in situ* after spinal anesthesia or ADP has at least three benefits: 1) mechanical blockade of the CSF leak: intrathecal (*i.t.*) catheter left *in situ* for 24 h closes the hole in the arachnoid dura preventing the leakage of CSF; 2) indirect inflammation: catheter-evoked inflammatory responses helping closing the broken; 3) therapeutic administration: convenient drug or fluid infusion or injection through the emplaced catheter for postoperative analgesia or artificial CSF supplementation. This is particularly effective in reducing the incidence of PDPH (~14%) after ADP with large gauge epidural needles than those without catheter placement (70-85%) [22]. Of this technique, Kuczkowski and Benumof composed a 5-step protocol for PDPH prevention and treatment following ADP [22], and their subsequent investigation on it proved more effective than ever in conquering the incidence of PDPH to 6.6% [116]. Similarly, subsequent catheter placement into the epidural space after ADP in cesarean delivery and leaving the catheter for postoperative analgesia for 36-72 h reduced the incidence of PDPH significantly (7.1% compared to 58% in non-catheterized patients) [117]. However, attention needs to be paid on this procedure due to the potential risk of catheter-associated infection [118] and cauda equina syndrome [119, 120]. In addition, placement of the catheter needs informed consent and should be reconsidered for it causes discomfort especially when prolonged retention is intended [32].

Since the first case has been reported on the successful treatment of PDPH with occipital nerve block (ONB) [121], several other PDPH patients from different institutes were presented after treatment with ONB [122, 123]. The sensory fibers of the greater occipital nerve (GON) originate from the C2 and C3 segments of the spinal cord, and its cutaneous sensory distribution extends over the posterior part of the head, spreading anteriorly to the vertex towards the area supplied by the ophthalmic division of the trigeminal nerve [124]. The lesser occipital nerve (LON) arises from the lateral branch of the ventral ramus of the second cervical nerve. Near the cranium it perforates the deep fascia, and is continued upward along the side of the head behind the auricula, supplying the skin and communicating with the GON [125]. In migraine patients, a unilateral greater ONB can initiate an inhibitory process that shuts down several symptom generators which alleviates allodynia first then headache [126]). Moreover, physical compression of LON causes migraine [127]. In PDPH patients, nerve stimulator-guided bilateral blockade of the GON and LON were found effective in controlling PDPH symptoms [128].

Methylxanthines, a group of derivatives of xanthine, act on adenosine receptors nonselectively as antagonists that in turn lead to vasoconstriction, which exactly negate the compensatory cerebral vasodilation that occurs in response to loss of CSF volume, a theoretical cause that has been implicated in the pathogenesis of PDPH [82]. In addition, methylxanthines can

activate sodium-potassium pumps [129] that are involved in the regulation of CSF production [130], which may finally lead to headache relief. There are three major methylxanthines include aminophylline, caffeine, and theophylline that are widely used in patients who suffered PDPH. In a randomized trial, 1 mg/kg aminophylline *i.v.* given immediately after umbilical cord clamping significantly reduced the incidence of PDPH from 23.3% (no aminophylline injection) to 5% in Cesarean patients underwent spinal anesthesia, and the severe headache after 48 hours was also markedly lower (3%) than the control (11%) [131]. Although caffeine was considered as a potential drug in relieving PDPH, but currently available evidence does not endorse its therapeutic value for PDPH because no valid pharmacological rationale for this drug exists, which majorly due to the clinical trials are few in number, small in sample size, weak or flawed in methodology, and non-effectiveness, contradictory, and even confliction in results [132]. Furthermore, prophylactic oral multidose caffeine-paracetamol also cannot prevent PDPH [112] suggesting that the use of caffeine in PDPH treatment needs to be evaluated carefully and herein I do not recommend it. However, oral theophylline was found effective in treating PDPH painful symptom [133], and studies compared the analgesic efficacy of theophylline infused *i.v.* to placebo found the therapy significantly decreased the painfulness of PDPH and it was suggested as an easy, rapid, minimally invasive, an effective treatment for PDPH [134]. If methylxanthines are ready to use in managing PDPH, the side effects for these types of drugs should be bear in mind like the central nervous system (CNS) stimulation, seizures, gastric irritation, and cardiac dysrhythmia when patients had psychiatric history, gastroenterological and cardiovascular problems [135, 136].

Beside abovementioned analgesics in the conservative treatment, more potent pharmacological analgesics are recommended in this aggressive medical treatment phase. Cosyntropin, an adrenocorticotropic hormone (ACTH) analog with less antigenic than the naturally occurring hormone, was used successfully to treat PDPH. Cosyntropin functions through: 1) stimulating the adrenal cortex to secrete glucocorticoids, mineralocorticoids, and androgens; 2) activating adenyl cyclase, which increases intracellular cAMP that can promote CSF production; and 3) increasing β-endorphins in the CNS which subsequently leads to an increase in the pain threshold. The doses of cosyntropin changed from 0.25 mg to 1.0 mg *i.v.* or intramuscularly (*i.m.*), and the headache was controlled by 80%-100%, and incidence of PDPH and the need of EBP were reduced, and the time from ADP to occurrence of PDPH was prolonged [137-141]. The side effects of ACTH administration include infection, mood elevation, and intracranial hemorrhage [142].

Although epinephrine was initially regarded as a vasoconstrictor in neuraxial analgesia, its analgesic effect in the spinal cord is rarely investigated in human being. Accumulating data support the analgesic role of spinal noradrenergic transmission in various pain conditions [143]. Controversial results were reported for the possible effect of epinephrine added into local anesthetics and opioids on the incidence of PDPH. Using continuous intrathecal patient-controlled analgesia (PCA) consisting fentanyl, bupivacaine and epinephrine completely prevented the occurrence of PDPH in Cesarean patients after ADP [144], but other studies did not find correlation between occurrence of PDPH and type of local anesthetics or additives including epinephrine and opioids [145]. Opioids are still the mainstay of pain management,

and intrethecal morphine was also found to be effective in controlling PDPH [23]. While dexamethasone was found no beneficial to PDPH patients, and even is a risk factor for the development of PDPH [66], hydrocortisone *i.v.* (100 mg in 2 ml 8 hourly for 48 h) on the contrary was found effective in reducing PDPH following spinal anesthesia [67].

Gabapentin and pregabalin are two GABA analogs that have been reported to be useful in the management of epilepsy and neuropathic pain. For PDPH patients, increasing number of cases were reported after successful treatment with gabapentin or pregabalin [146-150]. Sumatriptan, a serotonin type 1-D receptor agonist, was reported effective in the treatment of PDPH, with complete resolution of symptoms [151], but Connelly *et al.* did not find significant relief in headache [152]. Methergine is used widely in the management of refractory headache and migraine [153, 154], and also it was found to be an effective drug in alleviate PDPH [155], although the actual efficacy of its single use in this context is not sure [156]. The antidepressant mirtazapine has a net positive effect on noradrenergic neurotransmission, and was reported effective in relieving PDPH [157]. In sum, although these satellite cases treated with above-mentioned medications showed effective in managing PDPH, their clinical application needs to be evaluated substantially before prescribed for PDPH.

8.3. Third step: Conventional invasive management

When medical therapies in the step 2 fail for relief of PDPH and when the symptoms of PDPH is debilitating or severe, management should move on to procedural invasive therapies. The most widely used conventional invasive procedure is blood patch, and some alternative materials like hydroxyethyl starch and saline will be discussed if EBP is contraindicated. EBP is a treatment of choice for PDPH with high success rate and low incidence of complications [158].

8.3.1. Theoretical basis of EBP

As early 1960s, Gormley found that the incidence of PDPH in patients with "bloody taps" were comparably lower than those with only saline taps [159], and then the thought of EBP began to develop. Until 1970, the use of EBP became popular, and in 1990, first official guideline recommended EBP for PDPH treatment [34]. Theoretically, EBP is assumed to work by increasing CSF pressure and stimulating fibrin and platelet formation, and secondly, the introduced blood into the epidural space will clot and exerts tamponade effect by occluding the perforation that subsequently will prevent further leakage of CSF.

8.3.2. EBP technique

EBP has the same technique as the epidural anesthesia. Modern epidural kits are disposable and are sterilized package that includes all equipments and drugs without preservative. The epidural needle (usually Tuohy needle) is typically 16-18G, 8cm long with surface markings at 1cm intervals, and has a blunt bevel with a 15-30 degree curve at the tip (the Huber tip). Wings attached at the junction of the needle shaft with the hub, which allow better control of the needle as it is advanced. In general, a traditional glass syringe with an easily slide plunger

is used to identify the epidural space. The newer commercially available disposable epidural packs contain a plastic syringe with a plunger that has very low resistance. For EBP, there is no need to use epidural catheter for continuous medication.

To identify the epidural space, several methods can be used like loss of resistance (LOR) and hanging drop technique. Of the hanging drop technique, it has been abandoned by modern anesthesia. Given the reports on the better anesthesia quality and the possible complications of large amounts of air injected into the epidural space and surrounding structures [160], therefore the LOR to saline is preferred in EBP. Even though the EBP technique can be performed with the patient either in the sitting or lateral decubitus position, the latter is the preference due to the sitting position causes more incidence of PDPH [78]. On this position, the patient should be encouraged to adopt a curled up position, as this tends to open the spaces between the spinous processes and facilitates the identification of the intervertebral spaces. After the back has been prepared with sterile solution and draped in sterile fashion, the desired level is selected.

There are two approaches for the epidural needle insertion: midline or paramedian approach. The midline approach needs insert the epidural needle through the supraspinous ligament, and then advance the needle into the interspinous ligament, until distinct sensation of increased resistance is felt as the needle passes into the ligamentum flavum. For the paramedian approach, the inserting point of the epidural needle is 1-2 cm lateral to the spinous processes, and then insert and advance the needle perpendicularly to the skin until the lamina or pedicle is encountered, and then redirect it approximately 30° cephalad and 15° medially in an attempt to "walk the needle" off the lamina, at which point the needle should be in close proximity to the ligamentum flavum. After felt the resistance from the ligamentum flavum, the needle is then advanced further using LOR to saline.

8.3.3. Contraindications of EBP

Although the performance of EBP has the same contraindications that normally apply to epidural anesthesia, it also has some particular concerns. General absolute contraindications include: 1) patient refusal; 2) coagulopathy; 3) therapeutic anticoagulation; 4) skin infection at injection site; 5) localized sepsis in lumbar area; 6) raised intracranial pressure; 7) hypovolemia; 8) unexplained neurological symptoms; 9) active neurological disease; and 10) generalized sepsis. General relative contraindications include: 1) uncooperation; 2) pre-existing neurological disorders; 3) fixed cardiac output states; 4) anatomical abnormalities of vertebral column; and 5) prophylactic low dose heparin. Particular contraindications of EBP: 1) raised white cell count and pyrexia; 2) human immunodeficiency virus (HIV)-positive patients with other active bacterial or viral illnesses; 3) oncology (EBP in these patients may raises the potential for seeding the neuraxis with neoplastic cells).

Special attention should be paid when anticoagulants are used: 1) full anticoagulation with warfarin or standard heparin is absolute contraindication to EBP; 2) partial anticoagulation with low molecular weight heparin (LMWH) or low dose warfarin (International Normalized Ratio, INR < 1.5) is relative contraindication; 3) for low dose standard heparin (Minihep), wait for 4 h after a dose before performing EBP, and it should not be given until 1 h following blood

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patch; 4) allow a 12 h interval between LMWH administration and EBP; 5) NSAIDs including aspirin do not increase the risk of epidural hematoma; 6) avoid EBP for 24 h when fibrinolytic and thrombolytic drugs are used, and check clotting prior to needle insertion; 7) EBP needs avoid when patients were diagnosed thrombocytopenia especially when the platelet count is less than 100 000/mm^3.

8.3.4. EBP procedures

Once the EBP was determined to be applied, following procedures should be scheduled.

1. Give a full explanation of the cause of the headache, the reasons for performing an EBP, the technique, potential hazards and anticipated success rate;
2. Obtain informed consent;
3. Move patient to fully equipped work area;
4. Undertake under the direct supervision of a consultant or senior physician in the operating room with an assistant;
5. Lie flat for an hour before the EBP procedure (that may improve its efficacy by reducing the volume of CSF in the extradural space);
6. Secure *i.v.* access for fluid titration;
7. Two operators are required, both scrubbed, gowned and masked;
8. Position patient in lateral position;
9. Operator 1: sterilize skin over back, drape and perform epidural puncture at the same level as previous puncture or one level below;
10. Operator 2: simultaneously sterilize skin over antecubital fossa, drape and perform venepuncture withdrawing 20 ml of blood;
11. Blood is handed to operator 1 who injects blood via epidural needle until either the patient complains of a tightness in the buttocks or lower back, or until 20 ml is injected;
12. If back or leg pain (due to arachnoid irritation) occurs, stop injecting and wait a few seconds. If pain persists, abandon procedure. If a catheter has been used, remove it immediately after injection is complete;
13. Inject remaining blood into blood culture bottles for culture and sensitivity;
14. Nurse patient supine for 1-2 h, then mobilize cautiously;
15. Keep the patient under close review. If symptoms have not completely resolved, refer to consultant, and a repeat blood patch may be required;
16. Record procedures in medical book;
17. Refer the patient visit anesthetic clinic 2-4 weeks.

8.3.5. Distribution of the blood patch

No consensus reached as to the precise volume of blood required for EBP, but it is now recognized that the 2-3 ml of blood originally described by Gormley [159] is inadequate, and that 20-30 ml of blood is more likely to ensure success. However there was successful case treated with larger volumes of blood up to 60 ml in patients with spontaneous intracranial hypotension [161].

Several studies reported the distribution of the blood patch in the epidural space using radiolabelled red blood cells [162] or magnetic resonance imaging (MRI) scanning [163]. After injection, the blood spread caudally and cephalad regardless of the direction of the bevel of the needle, and also the blood can reach to the anterior epidural space circumferentially, and also can pass into the paravertebral space. When injecting 14 ml of blood, the highest level it can reach is six spinal segments, and caudally three segments. It is presumed that the compression of the thecal space that elevates the subarachnoid pressure for the first 3 h contributes to the rapid resolution of the headache. About 7-13 h after the EBP, there will be a thick layer of mature clot over the dorsal part of the thecal sac formed due to the procoagulant effect of CSF [164, 165].

8.3.6. PDPH outcomes of EBP

The reported success rate of the EBP technique is 70-98% if carried out more than 24 h after the dural puncture [36, 166]. Kokki et al. reported that EBP performed later than 48h following lumbar puncture or ADP is effective in parturients with postdural puncture symptoms [167]. If the first EBP failed to relieve the headache, repeated EBP can be used with the same success rate each. In a randomized controlled trial (RCT) in which a "sham" procedure was assigned as the comparison to the EBP, 11 out of 12 patients (92%) reported successful relief for the first EBP application, and the twelfth being relieved by a second procedure, whereas the sham-treated patients reported no benefit from the procedure (168). In another RCT, the respective successful rates of EBP and conservative treatments were 42% and 10% at day 1, and 84% and 14% at day 7 in PDPH patients. For those without recovery, the severity of headache was mild in all EDBP patients, but moderate or severe in conservatively treated patients (169). If the headache persists or debilitates after several attempts, other invasive maneuvers should be considered as appeared in the step 4.

8.3.7. Complications of EBP

While EBP is an effective treatment with a low complication rate, it is also an invasive method that can cause permanent neurological sequelae such as early and late back pain, radiculopathy, spinal-subdural hematoma, spinal-epiarachnoid hematoma, intrathecal hematoma, arachnoiditis, and infection. In consideration of headache as a common symptom of PDPH and cerebral venous thrombosis (CVT), therefore it is hard to distinguish them especially after EBP [170-175] suggesting that it should be carefully evaluated before EBP was planned in patients with altered coagulation state (see above *Contraindications of EBP*), and when possible treatments that would affect the coagulation were ready to be given. Besides, rare cases were

reported that EBP may cause epidural scarring that eventually results in slow spread of epidural local anesthetics, unilateral block and low efficacy if later epidural block was performed [176].

8.3.8. Prophylactic EBP

There were studies suggesting the use of prophylactic EBP in preventing PDPH, but the data were conflicting. Reported cases including patients underwent post-myelogram [177], and spinal anesthesia and ADP with an epidural needle [178, 179], have confirmed the benefit of prophylactic patching. Nonetheless, other studies found prophylactic EBP cannot decrease the incidence of PDPH or reduce the need for criteria-directed therapeutic epidural patch for parturients after ADP, but it can shorten the duration of PDPH symptoms [18]. One possible explanation for the failure of EBP is that the pressure gradient between the thecal and epidural space may be high enough immediately after blood injection which leads to patch separation from the site of the perforation. Therefore a greater volume of blood may be needed to produce a successful patch [32].

8.3.9. EBP for Jehovah's Witness

Due to Biblical interpretation principles, Jehovah's Witness patients do not authorize even autologous transfusions because blood removed from the body lost the continuity [180]. In the early time, alternative methods like epidural saline or Dextron were suggested [181]. Until 2003, Silva Lde *et al.* reported a closed system, through which two Jehovah's Witness patients with PDPH were treated successfully with autologous EBP [182]. Since then several cases were reported treated with the closed system successfully [183-185]. For this system, in brief, it includes two serum catheters cut in 60 cm segments, one two-way connection, one three-way tap and one 20 ml syringe. The system was assembled to allow one connection to venepuncture needle (20G), one connection to the three-way tap, and the remaining two ways were connected to a 20 ml syringe and to the other serum catheter segment, which would be connected to the epidural needle. After approval by the ethical committee, and informed consent by the patient, the system was filled with saline (6 ml). After epidural puncture and intravenous puncture, the already described system was connected to the epidural needle. Occluding the epidural needle way by moving three-way tap, 20 ml venous blood was aspirated to the syringe and venous catheter way was occluded. Then, epidural needle way was opened and syringe's content was injected in the epidural space. After removal of the epidural needle and system, venous access was maintained for fluid infusion. Patient remained at rest for 2 h and discharged with the recommendation to visit the clinic in case of recurrence.

8.3.10. Alternative maneuvers to EBP

For PDPH patients with absolute contraindications, alternative methods are suggested. In theory, epidural injection of other materials like saline or hydroxyethyl starch (HES) would produce the same mass effect, and restore normal CSF dynamics. Advocates of the epidural saline patch [32] include: 1) a single 30 ml bolus of epidural saline after development of headache; 2) 10-120 ml of saline injected as a bolus via the caudal epidural space; 3) 1.0-1.5 l of

epidural Hartmann's solution over 24 h, starting on the first day after dural puncture; 4) 35 ml/h of epidural saline or Hartmann's solution for 24-48 h. However, large volume of saline should be avoided in case intraocular hemorrhages through a precipitous rise in intracranial pressure [186]. Kara et al. reported a successful pediatric case with PDPH treated with epidural saline patch [187]. Epidural HES patch was also found effective in treating PDPH when patients contraindicated to EBP like bacteremia and leukemia [188, 189]. Although there were successful cases managed with other epidural materials, it is still not conclusive for their clinical use due to the lack of high quality evidence.

8.4. Forth step: Aggressive and invasive management

When epidural patch with blood, saline or HES failed to resolve the headache, the diagnosis needs to be reevaluated and more aggressive methods can be considered. These invasive managements only apply to those with persistent, severe and debilitating headache after treatment using above means in the step 3.

Fibrin glue, also known as fibrin sealant, is a biological adhesive made up of fibrinogen and thrombin that are applied to the tissue sites to glue them together or block bleed by creating a fibrin clot [190]. Fibrin glue is used frequently in repairing cranial dural perforations to block CSF leak after intradural procedures [191]. There are successful PDPH cases treated with epidural fibrin glue injected through percutaneous CT guidance or blindly in patients [192, 193] and animal models [194]. Moreover, the effectiveness of fibrin glue in sealing the hole and stopping the leakage of CSF has been studies using *in vitro* model of postdural puncture leakage and got supportive results for its application under this condition [195]. However, conflicting cases reported that such artificial formulation had a risk of the development of aseptic meningitis [196], and in further it has been warned against the application of fibrin glue when they were used in CNS because fibrin glue contains tranexamic acid (t-AMCA) which may cause severe nervous complications [197].

Surgery was considered as the last option for the treatment of PDPH if all abovementioned methods failed to resolve it. Neurosurgical procedure can be performed to identify and suture the hole in dura mater under the operating microscope. In one refractory PDPH patient in whom the headache lasted over two year, surgical repair successfully resolved the headache immediately, and the patient was rapidly mobilized from bed without orders for bed rest or any further precautions [198]. Before performing surgeries, the exact location of the CSF leak should be identified. Several medical diagnostic techniques are currently available to help detect the CSF leakage: dynamic CT myelography for fast-flow CSF leak, delayed CT myelography or magnetic resonance myelography for slow-flow leak.

9. Preventive strategy

Although the effectiveness of the prophylactic EBP is controversial [18, 176, 177], there are studies found advocating role for other strategies in reducing the incidence of PDPH. Deliberate intrathecal saline injection (5 ml) before spinal administration of hyperbaric bupivacaine

as a preventive approach is an effective and simple way to minimize PDPH in patients undergoing cesarean section (the incidence is 2% versus 16% without prophylactic saline) [20]. Subsequent spinal catheterization with epidural catheter following ADP can be used to prevent extra leakage of CSF, and then prevents PDPH [22, 116, 199]. Preventive epidural morphine 3mg given after the end of anesthesia and another 3 mg given on the following day reduced the incidence of PDPH from 48% to 12% [200]. Preventive administration of cosyntropin after ADP in parturients was associated with significant reduction in the incidence of PDPH and need for EBP and significant prolongation of the time from ADP to occurrence of PDPH [141]. Orally used prophylactic frovatriptan 2.5 mg/diet for 5 days markedly decreased the occurrence of PDPH [201]. Other methods like prophylactic administration of caffeine, magnesium, aminophylline, dexamethasone, or intravenous fluid infusion all cannot reduce the incidence of PDPH [202, 203].

10. Recommendations for clinicians

The occurrence of PDPH is determined by multiple factors including patient's demographic variables, caregiver's aspects, procedure-related factors, and post-accidental strategies. It is unclear which of them weighs over the others and what the accurate weight for each factor is in contributing to the onset of the headache. An arbitrary predictive curve of the incidence of PDPH to its risk factors herein is modeled and depicted in Figure 2 to show the association between the headache and different risk factors, and also give a potential prediction of the PDPH occurrence. In this model, each risk factor is scored "1", and all currently identified risk factors are summed up and in total get a scoring "10". Of the PDPH incidence changes from "0" to "100". For example, a 24-year non-smoking full-term pregnant woman with depression history was assigned to spinal anesthesia for Cesarean section by a third-year resident under the supervision by his consultant, so this woman had a risk scoring of "6", and the probability for the PDPH occurrence is ~32%.

Risk factor	Scoring
Young age	1
Female	1
Lower BMI	1
Taller height	1
Non-smoking	1
History of depression	1
History of chronic or recurrent headache	1
Experience level of personnel	1
Pregnancy itself	1
Fatigue, sleep deprivation, or night work	1
Total	**10**

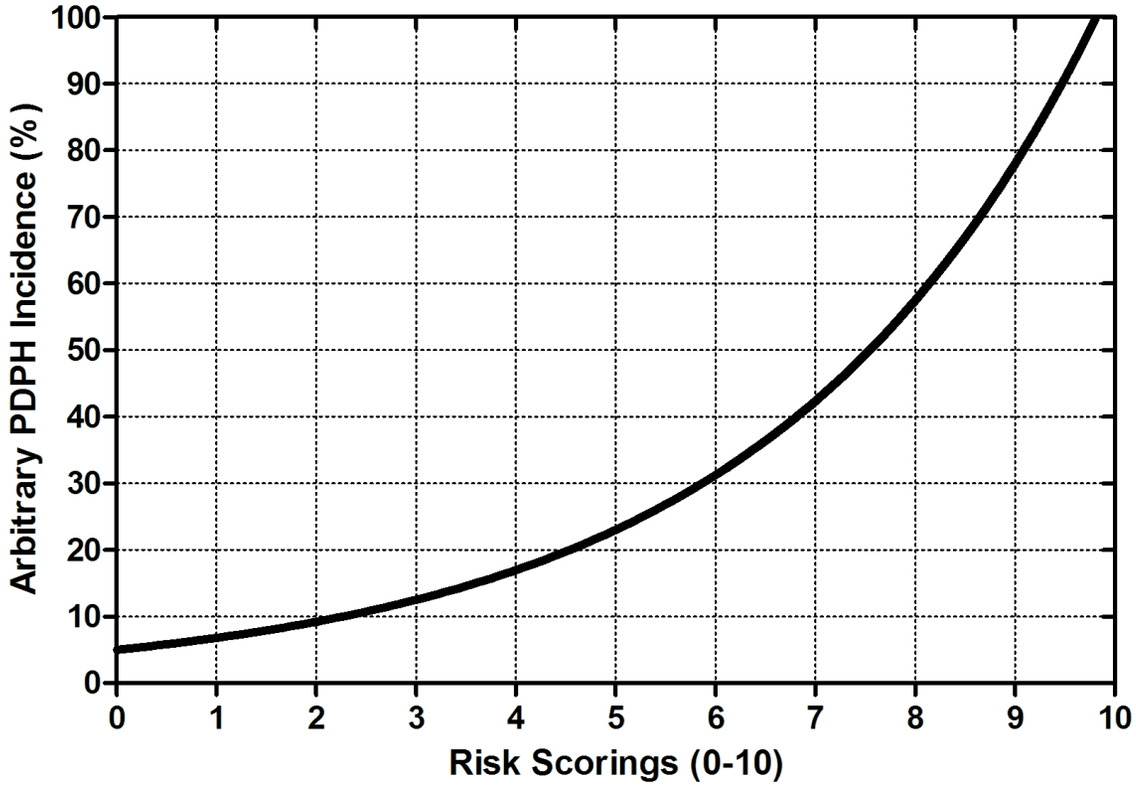

Figure 2. PDPH risk predictive curve.

Beside the risk predictive model, we need bear in mind following recommendations before and during performing spinal anesthesia.

1. Soothe patients psychologically to release stress;
2. Advance corresponding knowledge of the health care personnel;
3. Be energetic and active when performing procedures;
4. Hydrate patients prior procedures at least 500 ml;
5. Stabilize blood pressure at individual's physiological level;
6. Use lateral decubitus position for procedures;
7. Avoid repeat attempts of lumbar puncture;
8. No dexamethasone any time;
9. Reduce the volume of CSF withdrawn.

For the strategies after spinal anesthesia or ADP, refer to Table 3 for the therapeutic means and section 9 for the preventive maneuvers.

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Conservative treatment (1st step)
Abdominal binder
Bed rest
Intravenous hydration
Psychological support
Symptomatic analgesia
Acetaminophen
Antiemetics
Dexamethasone
Gabapentin
Non-steroidal anti-inflammatory drugs (NSAIDs)
Aggressive medical treatment (2nd step)
Intravenous methylxanthines
Aminophylline
Caffeine
Theophylline
Occipital nerve block
Symptomatic therapies
Adrenocorticotropic hormone (ACTH) *i.v.*, *i.m.*
Epinephrine *i.t.*
Hydrocortisone *i.v.*
Methergine *i.v.*
Mirtazapine *o.l.*
Opioids *i.t.*
Pregabalin/Gabapentin *i.v.*, *o.l.*
Sumatriptan *s.c.*
Subarachnoid catheter left *in situ*
Conventional invasive management (3rd step)
Epidural blood patch (EBP)
Epidural saline patch
Epidural hydroxyethyl starch patch
Aggressive and invasive management (4th step)
Epidural fibrin glue
Invasive surgery

i.m.: intramascular; i.t.: intrathecal; i.v.: intravenous; o.l.: oral; s.c.: subcutaneous

Table 3. Therapeutic strategies for PDPH.

11. Concluding remarks

As a well known iatrogenic complication, PDPH has its special morbidity that affects patient's daily life, even though it carries self-terminating characteristic. Based on its particular risk factors and special pathophysiological concerns, many preventive and therapeutic methods are developed, although they need to be verified by further quality studies. While we cannot find a once-for-all method for the perplexing headache, we can individually assess the patient and predict the risk of PDPH using the arbitrary predictive model in combination with the prior-procedure preventive strategies. The key to avoid such an annoying morbid is to bear all associated concerns in mind and keep alert when facing a patient ready to spinal anesthesia or epidural puncture, and actively seek and carry out effective remediation once PDPH occurred. Yes, PDPH, we can prevent it only if we paid our attention carefully on this issue.

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References

[1] Barbosa FT, Castro AA, de Miranda CT. Neuraxial anesthesia compared to general anesthesia for procedures on the lower half of the body: systematic review of systematic reviews. Revista Brasileira de Anestesiologia 2012;62(2) 235-243.

[2] Cwik J. Postoperative considerations of neuraxial anesthesia. Anesthesiology Clinics 2012;30(3) 433-443.

[3] Walker SM, Yaksh TL. Neuraxial analgesia in neonates and infants: a review of clinical and preclinical strategies for the development of safety and efficacy data. Anesthesia & Analgesia 2012;115(3) 638-662.

[4] Bezov D, Ashina S, Lipton R. Post-dural puncture headache: Part II--prevention, management, and prognosis. Headache 2010;50(9) 1482-1498.

[5] Gaiser RR. Postdural puncture headache: a headache for the patient and a headache for the anesthesiologist. Current Opinion in Anaesthesiology 2013;26(3) 296-303.

[6] de Almeida SM, Shumaker SD, LeBlanc SK, Delaney P, Marquie-Beck J, Ueland S, Alexander T, Ellis RJ. Incidence of post-dural puncture headache in research volunteers. Headache 2011;51(10) 1503-1510.

[7] Bezov D, Lipton RB, Ashina S. Post-dural puncture headache: part I diagnosis, epidemiology, etiology, and pathophysiology. Headache 2010;50(7) 1144-1152.

[8] Amorim JA, Gomes de Barros MV, Valença MM. Post-dural (post-lumbar) puncture headache: risk factors and clinical features. Cephalalgia 2012;32(12) 916-923.

[9] Amorim JA, Valença MM. Postdural puncture headache is a risk factor for new postdural puncture headache. Cephalalgia 2008;28(1) 5-8.

[10] Seeberger MD, Kaufmann M, Staender S, Schneider M, Scheidegger D. Repeated dural punctures increase the incidence of postdural puncture headache. Anesthesia & Analgesia 1996;82(2) 302-305.

[11] O'Connor G, Gingrich R, Moffat M. The effect of spinal needle design, size, and penetration angle on dural puncture cerebral spinal fluid loss. American Association of Nurse Anesthetists Journal 2007;75(2) 111-116.

[12] Kuczkowski KM. Post-dural puncture headache in the obstetric patient: an old problem. New solutions. Minerva Anestesiologica 2004;70(12) 823-830.

[13] Westbrook JL, Uncles DR, Sitzman BT, Carrie LE. Comparison of the force required for dural puncture with different spinal needles and subsequent leakage of cerebrospinal fluid. Anesthesia & Analgesia 1994;79(4) 769-772.

[14] Shaikh JM, Memon A, Memon MA, Khan M. Post dural puncture headache after spinal anaesthesia for caesarean section: a comparison of 25 g Quincke, 27 g Quincke and 27 g Whitacre spinal needles. Journal of Ayub Medical College Abbottabad 2008;20(3) 10-13.

[15] Apiliogullari S, Duman A, Gok F, Akillioglu I. Spinal needle design and size affect the incidence of postdural puncture headache in children. Paediatric Anaesthesia 2010;20(2) 177-182.

[16] Heesen M, Klöhr S, Rossaint R, van de Velde M, Straube S. Can the incidence of accidental dural puncture in laboring women be reduced? A systematic review and meta-analysis. Minerva Anestesiologica 2013; 79(10) 1187-1197.

[17] Duffy PJ, Crosby ET. The epidural blood patch. Resolving the controversies. Canadian Journal of Anaesthesia 1999;46(9) 878-886.

[18] Scavone BM, Wong CA, Sullivan JT, Yaghmour E, Sherwani SS, McCarthy RJ. Efficacy of a prophylactic epidural blood patch in preventing post dural puncture headache in parturients after inadvertent dural puncture. Anesthesiology 2004;101(6) 1422-1427.

[19] Charsley MM, Abram SE. The injection of intrathecal normal saline reduces the severity of postdural puncture headache. Regional Anesthesia and Pain Medicine 2001;26(4) 301-305.

[20] Faridi Tazeh-Kand N, Eslami B, Ghorbany Marzony S, Abolhassani R, Mohammadian K. Injection of intrathecal normal saline in decreasing postdural puncture headache. Journal of Anesthesia 2013; Doi: 10.1007/s00540-013-1683-8

[21] Boonmak P, Boonmak S. Epidural blood patching for preventing and treating postdural puncture headache. The Cochrane Database of Systematic Reviews 2010;(1) CD001791.

[22] Kuczkowski KM, Benumof JL. Decrease in the incidence of post-dural puncture headache: maintaining CSF volume. Acta anaesthesiologica Scandinavica 2003;47(1) 98-100.

[23] Basurto Ona X, Uriona Tuma SM, Martínez García L, Solà I, Bonfill Cosp X. Drug therapy for preventing post-dural puncture headache. The Cochrane Database of Systematic Reviews 2013;(2) CD001792.

[24] Singh S, Chaudry SY, Phelps AL, Vallejo MC. A 5-year audit of accidental dural punctures, postdural puncture headaches, and failed regional anesthetics at a tertiary-care medical center. ScientificWorldJournal 2009;9 715-722.

[25] Dodge HS, Ekhator NN, Jefferson-Wilson L, Fischer M, Jansen I, Horn PS, Hurford WE, Geracioti TD. Cigarette smokers have reduced risk for post-dural puncture headache. Pain Physician 2013;16(1) E25-E30.

[26] Corning JL. Spinal anaesthesia and local medication of the cord. New York Medical Journal 1885; 42 483-485.

[27] Wynter WE. Four cases of tubercular meningitis in which paracentesis was performed for the relief of fluid pressure. The Lancet 1891; 1 981-982.

[28] Quincke HI. Ueber hydrocephalus. Verhandlung des Congress Innere Medizin (X) 1891; 321-339.

[29] Quincke HI. Die lumbalpunction des Hydrocephalus. Berlin Klinik Wochenschrift 1891; 28 929-233.

[30] Bier A. Versuche über Cocainisirung des Rückenmarkes (Deutsch). Deutsch Zeitschrift für Chirurgie 1899; 51 361.

[31] Marx GF. The first spinal anesthesia. Who deserves the laurels? Regional Anesthesia 1994;19(6) 429-430.

[32] Turnbull DK, Shepherd DB. Post-dural puncture headache: pathogenesis, prevention and treatment. British Journal of Anaesthesia 2003;91(5) 718-729.

[33] Mosavy SH, Shafei M. Prevention of headache consequent upon dural puncture in obstetric patient. Anaesthesia 1975;30(6) 807-809.

[34] Guidelines for the practice of obstetric anaesthesia in Nottingham. Queen's Medical Centre Nottingham, NHS Trust. June 1990. 1st Version.

[35] Waise S, Gannon D. Reducing the incidence of post-dural puncture headache. Clinical Medicine 2013;13(1) 32-34.

[36] Darvish B, Gupta A, Alahuhta S, Dahl V, Helbo-Hansen S, Thorsteinsson A, Irestedt L, Dahlgren G. Management of accidental dural puncture and post-dural puncture headache after labour: a Nordic survey. Acta anaesthesiologica Scandinavica 2011;55(1) 46-53.

[37] Van de Velde M, Schepers R, Berends N, Vandermeersch E, De Buck F. Ten years of experience with accidental dural puncture and post-dural puncture headache in a tertiary obstetric anaesthesia department. International Journal of Obstetric Anesthesia 2008;17(4) 329-335.

[38] Srivastava V, Jindal P, Sharma JP. Study of post dural puncture headache with 27G Quincke & Whitacre needles in obstetrics/non obstetrics patients. Middle East Journal of Anesthesiology 2010;20(5) 709-717.

[39] Imarengiaye C, Ekwere I. Postdural puncture headache: a cross-sectional study of incidence and severity in a new obstetric anaesthesia unit. African Journal of Medicine and Medical Science 2006;35(1) 47-51.

[40] Tejavanija S, Sithinamsuwan P, Sithinamsuwan N, Nidhinandana S, Suwantamee J. Comparison of prevalence of post-dural puncture headache between six hour- supine recumbence and early ambulation after lumbar puncture in thai patients: A randomized controlled study. Journal of the Medical Association of Thailand 2006;89(6) 814-820.

[41] L'ubuský M, Berta E, Procházka M, Marek O, Kudela M. Development of incidence of post-dural puncture headache in patients undergoing caesarean section in spinal anaesthesia at the Department of Obstetrics and Gynecology in Olomouc during 2003-2004. Casopis Lékaru Ceských 2006;145(3) 204-208.

[42] Baysinger CL, Pope JE, Lockhart EM, Mercaldo ND. The management of accidental dural puncture and postdural puncture headache: a North American survey. Journal of Clinical Anesthesia 2011;23(5) 349-360.

[43] Berger CW, Crosby ET, Grodecki W. North American survey of the management of dural puncture occurring during labour epidural analgesia. Canadian Journal of Anaesthesia 1998;45(2) 110-114.

[44] Schmittner MD, Terboven T, Dluzak M, Janke A, Limmer ME, Weiss C, Bussen DG, Burmeister MA, Beck GC. High incidence of post-dural puncture headache in patients with spinal saddle block induced with Quincke needles for anorectal surgery: a randomised clinical trial. The International Journal of Colorectal Disease 2010;25(6) 775-781.

[45] Schmittner MD, Urban N, Janke A, Weiss C, Bussen DG, Burmeister MA, Beck GC. Influence of the pre-operative time in upright sitting position and the needle type on the incidence of post-dural puncture headache (PDPH) in patients receiving a spinal saddle block for anorectal surgery. The International Journal of Colorectal Disease 2011;26(1) 97-102.

[46] Lybecker H, Møller JT, May O, Nielsen HK. Incidence and prediction of postdural puncture headache. A prospective study of 1021 spinal anesthesias. Anesthesia & Analgesia 1990;70(4) 389-394.

[47] Imbelloni LE, Gouveia MA, Cordeiro JA. Continuous spinal anesthesia versus combined spinal epidural block for major orthopedic surgery: prospective randomized study. Sao Paulo Medical Journal 2009;127(1) 7-11.

[48] Neuman SA, Eldrige JS, Qu W, Freeman ED, Hoelzer BC. Post dural puncture headache following intrathecal drug delivery system placement. Pain Physician 2013;16(2) 101-107.

[49] Wee LH, Lam F, Cranston AJ. The incidence of post dural puncture headache in children. Anaesthesia 1996;51(12) 1164-1166.

[50] Lowery S, Oliver A. Incidence of postdural puncture headache and backache following diagnostic/therapeutic lumbar puncture using a 22G cutting spinal needle, and after introduction of a 25G pencil point spinal needle. Paediatric Anaesthesia 2008;18(3) 230-234.

[51] Wadud R, Laiq N, Qureshi FA, Jan AS. The frequency of postdural puncture headache in different age groups. Journal of the College of Physicians and Surgeons-Pakistan 2006;16(6) 389-392.

[52] Bolder PM. Postlumbar puncture headache in pediatric oncology patients. Anesthesiology 1986;65(6) 696-698.

[53] Ylonen P, Kokki H. Epidural blood patch for management of postdural puncture headache in adolescents. Acta anaesthesiologica Scandinavica 2002;46(7) 794-798.

[54] Tobias JD. Postdural puncture headache in children - etiology and treatment. Clinical Pediatrics 1990;33(2) 110 -113.

[55] Tourtellotte WW, Henderson WG, Tucker RP, Gilland O, Walker JE, Kokman E. A randomized, double-blind clinical trial comparing the 22 versus 26 gauge needle in production of the post-lumbar puncture syndrome in normal individuals. Headache 1972;12(2) 73-78.

[56] Wu CL, Rowlingson AJ, Cohen SR, Michaels RK, Courpas GE, Joe EM, Liu SS. Gender and post-dural puncture headache. Anesthesiology 2006;105(3) 613-618.

[57] Peralta FM, Chalifoux LA, Stevens CD, Higgins N. Obese parturients and the incidence of postdural puncture headache after unintentional dural puncture. Anesthesiology 2011; A341.

[58] Faure E, Moreno R, Thisted R. Incidence of postdural puncture headache in morbidly obese parturients. Regional Anesthesia 1994;19(5) 361-363.

[59] Chong YFV, Tan K. A survey of lumbar puncture complications and their risk factors: the influence of height, intravenous hydration and systolic blood pressure on post-dural puncture headache. Neurology 2012; 78 (Meeting Abstracts 1) P04.250.

[60] Leibold RA, Yealy DM, Coppola M, Cantees KK. Post-dural-puncture headache: characteristics, management and prevention. Annals of Emergency Medicine 1993;22(12) 1863-1870.

[61] van Oosterhout WP, van der Plas AA, van Zwet EW, Zielman R, Ferrari MD, Terwindt GM. Postdural puncture headache in migraineurs and nonheadache subjects: a prospective study. Neurology 2013;80(10) 941-948.

[62] Clark JW. Substance P concentration and history of headache in relation to postlumbar puncture headache: towards prevention. Journal of Neurology, Neurosurgery & Psychiatry 1996;60 (6) 681-683.

[63] Flaatten H, Krakenes J, Vedeler C. Post-dural puncture related complications after diagnostic lumbar puncture, myelography and spinal anesthesia. Acta Neurologica Scandinavica 1998;98(6) 445-451.

[64] Davignon KR, Dennehy KC. Update on postdural puncture headache. International Anesthesiology Clinics 2002;40(4) 89-102.

[65] Kuntz KM, Kokmen E, Stevens JC, Miller P, Offord KP, Ho MM. Post-lumbar puncture headaches: experience in 501 consecutive procedures. Neurology 1992;42(10) 1884-1887.

[66] Yousefshahi F, Dahmardeh AR, Khajavi M, Najafi A, Khashayar P, Barkhordari K. Effect of dexamethasone on the frequency of postdural puncture headache after spinal anesthesia for cesarean section: a double-blind randomized clinical trial. Acta Neurologica Belgica 2012;112(4) 345-350.

[67] Alam MR, Rahman MA, Ershad R. Role of very short-term intravenous hydrocortisone in reducing postdural puncture headache. Journal of Anaesthesiology Clinical Pharmacology 2012;28(2) 190-193.

[68] Choi PT, Galinski SE, Lucas S, Takeuchi L, Jadad AR. Examining the evidence in anesthesia literature: a survey and evaluation of obstetrical postdural puncture headache reports. Canadian Journal of Anaesthesia 2002; 49(1) 49-56.

[69] Kuczkowski KM. Post dural puncture headache, intracranial air and obstetric anesthesia. Anaesthesist 2003; 52(9) 798-800.

[70] Choi PT, Galinski SE, Takeuchi L, Lucas S, Tamayo C, Jadad AR. PDPH is a common complication of neuraxial blockade in parturients: a meta-analysis of obstetrical studies. Canadian Journal of Anaesthesia 2003;50(5) 460-469.

[71] Morewood GH. A rational approach to the cause, prevention and treatment of post-dural puncture headache. Canadian Medical Association Journal 1993;149(8) 1087-1093.

[72] Runza M, Pietrabissa R, Mantero S, Albani A, Quaglini V, Contro R. Lumbar dura mater biomechanics: experimental characterization and scanning electron microscopy observations. Anesthesia & Analgesia 1999;88(6) 1317-1321.

[73] Reina MA, de Leon-Casasola OA, Lopez A, De Andres J, Martin S, Mora M. An in vitro study of dural lesions produced by 25-gauge Quincke and Whitacre needles evaluated by scanning electron microscopy. Regional Anesthesia and Pain Medicine 2000;25(4) 393-402.

[74] Reina MA, López-García A, Dittmann M, de Andrés JA. Structural analysis of the thickness of human dura mater with scanning electron microscopy. Revista Española de Anestesiología y Reanimación 1996;43(4) 135-137.

[75] Reina MA, López García A, de Andrés JA, Sellers F, Arrizabalaga M, Mora MJ. Thickness variation of the dural sac. Revista Española de Anestesiología y Reanimación 1999;46(8) 344-349.

[76] Sakka L, Coll G, Chazal J. Anatomy and physiology of cerebrospinal fluid. European Annals of Otorhinolaryngology, Head and Neck Diseases 2011;128(6) 309-316.

[77] Kuczkowski KM. Post-dural puncture headache in pregnant women: What have we learned? Revista Colombiana de Anestesiología 2006; 34 267-272.

[78] Schwartz KM, Luetmer PH, Hunt CH, Kotsenas AL, Diehn FE, Eckel LJ, Black DF, Lehman VT, Lindell EP. Position-related variability of CSF opening pressure measurements. AJNR American Journal of Neuroradiology 2013;34(4) 904-907.

[79] Ellis R III. Lumbar cerebrospinal fluid opening pressure measured in a flexed lateral decubitus position in children. Pediatrics 1994;93(4) 622-623.

[80] Hatfalvi BI. Postulated mechanisms for postdural puncture headache and review of laboratory models. Clinical experience. Regional Anesthesia 1995;20(4) 329-336.

[81] Reamy BV. Post-epidural headache: how late can it occur? The Journal of the American Board of Family Medicine 2009;22(2) 202-205.

[82] Fearon W. Post-lumbar puncture headache. P&S Medical Review1993.

[83] Richardson MG, Wissler RN. Density of lumbar cerebrospinal fluid in pregnant and nonpregnant humans. Anesthesiology 1996;85(2) 326-330.

[84] Kemp WJ III, Tubbs RS, Cohen-Gadol AA. The innervation of the cranial dura mater: neurosurgical case correlates and a review of the literature. World Neurosurgery 2012;78(5) 505-510.

[85] Cumberbatch MJ, Williamson DJ, Mason GS, Hill RG, Hargreaves RJ. Dural vasodilation causes a sensitization of rat caudal trigeminal neurones in vivo that is blocked by a 5-HT1B/1D agonist. British Journal of Pharmacology 1999;126(6) 1478-1486.

[86] Knyihár-Csillik E, Tajti J, Chadaide Z, Csillik B, Vécsei L. Functional immunohistochemistry of neuropeptides and nitric oxide synthase in the nerve fibers of the supratentorial dura mater in an experimental migraine model. Microscopy Research and Technique 2001;53(3) 193-211.

[87] Damon DH, Teriele JA, Marko SB. Vascular-derived artemin: a determinant of vascular sympathetic innervation? American journal of physiology-Heart and Circulatory Physiology 2007;293(1) H266-H273.

[88] Lippoldt EK, Elmes RR, McCoy DD, Knowlton WM, McKemy DD. Artemin, a glial cell line-derived neurotrophic factor family member, induces TRPM8-dependent cold pain. The Journal of Neuroscience 2013;33(30) 12543-12552.

[89] Thornton P, Hatcher JP, Robinson I, Sargent B, Franzén B, Martino G, Kitching L, Glover CP, Anderson D, Forsmo-Bruce H, Low CP, Cusdin F, Dosanjh B, Williams W, Steffen AC, Thompson S, Eklund M, Lloyd C, Chessell I, Hughes J. Artemin-GFRα3 interactions partially contribute to acute inflammatory hypersensitivity. Neuroscience Letters 2013;545 23-28.

[90] McIlvried LA, Albers K, Gold MS. Distribution of artemin and GFRalpha3 labeled nerve fibers in the dura mater of rat: artemin and GFRalpha3 in the dura. Headache 2010;50(3) 442-450.

[91] Cavallotti D, Artico M, De Santis S, Iannetti G, Cavallotti C. Catecholaminergic innervation of the human dura mater involved in headache. Headache 1998;38(5) 352-355.

[92] Fernstrom JD, Fernstrom MH. Tyrosine, phenylalanine, and catecholamine synthesis and function in the brain. Journal of Nutrition 2007;137(6 Suppl 1) 1539S-1548S.

[93] Edgar MA, Nundy S. Innervation ofthe spinal dura mater. Journal of Neurology, Neurosurgery & Psychiatry 1966; 29 530-534.

[94] Edgar MA, Ghadially JA. Innervation of the lumbar spine. Clinical Orthopaedics and Related Research 1976; 115 35-41.

[95] Cyriax J. Dural pain. The Lancet 1978; 1 919-921.

[96] Sekiguchi Y, Konnai Y, Kikuchi S, Sugiura Y. An anatomic study of neuropeptide immunoreactivities in the lumbar dura mater after lumbar sympathectomy. Spine (Phila Pa 1976). 1996;21(8) 925-930.

[97] Bridge CJ. Innervation of spinal meninges and epidural structures. The Anatomical Record 1959; 133 553-561.

[98] Konnai Y, Honda T, Sekiguchi Y, Kikuchi S, Sugiura Y. Sensory innervation of the lumbar dura mater passing through the sympathetic trunk in rats. Spine (Phila Pa 1976) 2000;25(7) 776-782.

[99] Halpern S, Preston R. Postdural puncture headache and spinal needle design. Metaanalyses. Anesthesiology 1994;81(6) 1376-1383.

[100] Ross BK, Chadwick HS, Mancuso JJ, Benedetti C. Sprotte needle for obstetric anesthesia: decreased incidence of post dural puncture headache. Regional Anesthesia 1992;17(1) 29-33.

[101] Imarengiaye CO, Edomwonyi NP. Evaluation of 25-gauge Quincke and 24-gauge Gertie Marx needles for spinal anaesthesia for caesarean section. East African Medical Journal 2002;79(7) 379-381.

[102] Santanen U, Rautoma P, Luurila H, Erkola O, Pere P. Comparison of 27-gauge (0.41-mm) Whitacre and Quincke spinal needles with respect to post-dural puncture headache and non-dural puncture headache. Acta Anaesthesiologica Scandinavica 2004;48(4) 474-479.

[103] Luostarinen L, Heinonen T, Luostarinen M, Salmivaara A. Diagnostic lumbar puncture. Comparative study between 22-gauge pencil point and sharp bevel needle. The Journal of Headache and Pain 2005;6(5) 400-404.

[104] Tabedar S, Maharjan SK, Shrestha BR, Shrestha BM. A comparison of 25 gauge Quincke spinal needle with 26 gauge Eldor spinal needle for the elective Caesarian sections: insertion characteristics and complications. Kathmandu University Medical Journal 2003;1(4) 263-266.

[105] Kokki H, Turunen M, Heikkinen M, Reinikainen M, Laisalmi M. High success rate and low incidence of headache and neurological symptoms with two spinal needle designs in children. Acta Anaesthesiologica Scandinavica 2005;49(9) 1367-1372.

[106] Kuczkowski KM. The management of accidental dural puncture in pregnant women: what does an obstetrician need to know? Archives of gynecology and obstetrics 2007;275(2) 125-131.

[107] Bloomfield GL, Ridings PC, Blocher CR, Marmarou A, Sugerman HJ. Effects of increased intra-abdominal pressure upon intracranial and cerebral perfusion pressure before and after volume expansion. Journal of Trauma 1996;40(6) 936-943.

[108] Laterra J, Keep R, Betz LA, Goldstein GW. Blood-brain-cerebrospinal fluid barriers (Chapter 36). Basic Neurochemistry: Molecular, Cellular and Medical Aspects. 6th edition. Eds. Siegel GJ, Agranoff BW, Albers RW, Fisher SK, Uhler MD. Philadelphia: Lippincott-Raven; 1999.

[109] Arevalo-Rodriguez I, Ciapponi A, Munoz L, Roqué I Figuls M, Bonfill Cosp X. Posture and fluids for preventing post-dural puncture headache. The Cochrane Database of Systematic Reviews 2013;(7) CD009199.

[110] Mehta S, Rajaram S, Goel N. Postdural puncture headache. Advances in obstetrics and gynecology (Vol. 3). Jaypee Brothers Medical Publisher, 2011; p143-p146.

[111] Caple C. Lumbar puncture: complications and after-care. Evidence-Based Care Sheet. Cinahl Information Systems. 2012; p1-p2.

[112] Esmaoglu A, Akpinar H, Uğur F. Oral multidose caffeine-paracetamol combination is not effective for the prophylaxis of postdural puncture headache. Journal of Clinical Anesthesia 2005;17(1) 58-61.

[113] Allen TK, Jones CA, Habib AS. Dexamethasone for the prophylaxis of postoperative nausea and vomiting associated with neuraxial morphine administration: a systematic review and meta-analysis. Anesthesia & Analgesia 2012;114(4) 813-822.

[114] Soleimanpour H, Ghafouri RR, Taheraghdam A, Aghamohammadi D, Negargar S, Golzari SE, Abbasnezhad M. Effectiveness of intravenous dexamethasone versus propofol for pain relief in the migraine headache: a prospective double blind randomized clinical trial. BMC Neurology 2012;12 114.

[115] Erol DD. The analgesic and antiemetic efficacy of gabapentin or ergotamine/caffeine for the treatment of postdural puncture headache. Advances in Medical Sciences 2011;56(1) 25-29.

[116] Kuczkowski KM. Once a post-dural puncture headache patient--always post-dural puncture headache patient: an update. Acta Anaesthesiologica Belgica 2005;56(1) 23.

[117] Cesur M, Alici HA, Erdem AF, Silbir F, Celik M. Decreased incidence of headache after unintentional dural puncture in patients with cesarean delivery administered with postoperative epidural analgesia. Journal of Anesthesai 2009;23(1) 31-35.

[118] Bevacqua BK, Slucky AV, Cleary WF. Is postoperative intrathecal catheter use associated with central nervous system infection? Anesthesiology 1994;80(6) 1234-1240.

[119] Rigler ML, Drasner K, Krejcie TC, Yelich SJ, Scholnick FT, DeFontes J, Bohner D. Cauda equina syndrome after continuous spinal anesthesia. Anesthesia & Analgesia 1991;72(3) 275-281.

[120] Moore JM. Continuous spinal anesthesia. American Journal of Therapeutics 2009;16(4) 289-294.

[121] Matute E, Bonilla S, Gironés A, Planas A. Bilateral greater occipital nerve block for post-dural puncture headache. Anaesthesia 2008;63(5) 557-558.

[122] Akin Takmaz S, Unal Kantekin C, Kaymak C, Başar H. Treatment of post-dural puncture headache with bilateral greater occipital nerve block. Headache 2010;50(5) 869-872.

[123] Hamzehzadeh S, Eng C, Tran T. Occipital nerve blockade successfully treats patient with suspected post-dural puncture headache (PDPH). Regional Anesthesia and Pain Medicine Spring 2011. http://www.asra.com/display_spring_2011.php?id=137.

[124] Mueller O, Hagel V, Wrede K, Schlamann M, Hohn HP, Sure U, Gaul C. Stimulation of the greater occipital nerve: anatomical considerations and clinical implications. Pain Physician 2013;16(3) E181-E189.

[125] Madhavi C, Holla SJ. Triplication of the lesser occipital nerve. Clinical Anatomy 2004;17(8) 667-671.

[126] Young W, Cook B, Malik S, Shaw J, Oshinsky M. The first 5 minutes after greater occipital nerve block. Headache 2008;48(7) 1126-1128.

[127] Seo BF, Jung SN, Sohn WI, Kwon H. Lymph node compression of the lesser occipital nerve: a cause of migraine. Journal of Plastic, Reconstructive & Aesthetic Surgery 2011;64(12) 1657-1660.

[128] Naja Z, Al-Tannir M, El-Rajab M, Ziade F, Baraka A. Nerve stimulator-guided occipital nerve blockade for postdural puncture headache. Pain Practice 2009;9(1) 51-58.

[129] Lindinger MI, Willmets RG, Hawke TJ. Stimulation of Na+, K(+)-pump activity in skeletal muscle by methylxanthines: evidence and proposed mechanisms. Acta Physiologica Scandinavica 1996;156(3) 347-353.

[130] Speake T, Whitwell C, Kajita H, Majid A, Brown PD. Mechanisms of CSF secretion by the choroid plexus. Microscopy Research and Technique 2001;52(1) 49-59.

[131] Sadeghi SE, Abdollahifard G, Nasabi NA, Mehrabi M, Safarpour AR. Effectiveness of single dose intravenous aminophylline administration on prevention of post dural puncture headache in patients who received spinal anesthesia for elective cesarean section. World Journal of Medical Sciences 2012;7(1) 13-16.

[132] Halker RB, Demaerschalk BM, Wellik KE, Wingerchuk DM, Rubin DI, Crum BA, Dodick DW. Caffeine for the prevention and treatment of postdural puncture headache: debunking the myth. Neurologist 2007;13(5) 323-327.

[133] Schwalbe SS, Schifmiller MW, Marx GF. Theophylline for PDPH. Anesthesiology 1991; 75 A1082.

[134] Ergün U, Say B, Ozer G, Tunc T, Sen M, Tüfekcioglu S, Akin U, Ilhan MN, Inan L. Intravenous theophylline decreases post-dural puncture headaches. Journal of Clinical Neuroscience 2008;15(10) 1102-1104.

[135] Boison D. Methylxanthines, seizures, and excitotoxicity. Handbook of Experimental Pharmacology 2011;(200) 251-266.

[136] Riksen NP, Smits P, Rongen GA. The cardiovascular effects of methylxanthines. Handbook of Experimental Pharmacology 2011;(200) 413-437.

[137] Kshatri AM, Foster PA. Adrenocorticotropic hormone infusion as a novel treatment for postdural puncture headache. Regional Anesthesia 1997; 22(5) 432-434.

[138] Carter BL, Pasupuleti R. Use of intravenous cosyntropin in the treatment of postdural puncture headache. Anesthesiology 2000;92(1) 272-274.

[139] Cánovas L, Barros C, Gómez A, Castro M, Castro A. Use of intravenous tetracosactin in the treatment of postdural puncture headache: our experience in forty cases. Anesthesia & Analgesia 2002;94(5) 1369.

[140] Ghai A, Wadhera R. Adrenocorticotrophic hormone-is a single dose sufficient for post-dural puncture headache? Acta Anaesthesiologica Scandinavica 2007; 51 266.

[141] Hakim SM. Cosyntropin for prophylaxis against postdural puncture headache after accidental dural puncture. Anesthesiology 2010;113(2) 413-420.

[142] Ambrogio AG, Pecori Giraldi F, Cavagnini F. Drugs and HPA axis. Pituitary 2008;11(2) 219-229.

[143] Pertovaara A. Noradrenergic pain modulation. Progress in Neurobiology 2006;80(2) 53-83.

[144] Cohen S, Amar D, Pantuck EJ, Singer N, Divon M. Decreased incidence of headache after accidental dural puncture in caesarean delivery patients receiving continuous postoperative intrathecal analgesia. Acta Anaesthesiologica Scandinavica 1994;38(7) 716-718.

[145] Etezadi F, Yousefshahi F, Khajavi M, Tanha FD, Dahmarde AR, Najafi A. Post dural puncture headache after cesarean section, a teaching hospital experience. Journal of Family and Reproductive Health 2012; 6(1) 17-21.

[146] Torres D. Gabapentin and PDPH. Acute Pain 2007; 9(2) 93.

[147] Lin YT, Sheen MJ, Huang ST, Horng HC, Cherng CH, Wong CS, Hot ST. Gabapentin relieves post-dural puncture headache--a report of two cases. Acta Anaesthesiologica Taiwanica 2007;45(1) 47-51.

[148] Wagner Y, Storr F, Cope S. Gabapentin in the treatment of post-dural puncture headache: a case series. Anaesthesia and Intensive Care 2012 Jul;40(4) 714-718.

[149] Zencirci B. Postdural puncture headache and pregabalin. Journal of Pain Research 2010;3 11-14.

[150] Huseyinoglu U, Huseyinoglu N, Hamurtekin E, Aygun H, Sulu B. Effect of pregabalin on post-dural-puncture headache following spinal anesthesia and lumbar puncture. Journal of Clinical Neuroscience 2011;18(10) 1365-1368.

[151] Carp H, Singh PJ, Vadhera R, Jayaram A. Effects of the serotonin-receptor agonist sumatriptan on post-dural puncture headache: report of six cases. Anesthesia & Analgesia 1994;79(1) 180-182.

[152] Connelly NR, Parker RK, Rahimi A, Gibson CS. Sumatriptan in patients with post-dural puncture headache. Headache 2000;40(4) 316-319.

[153] Graff-Radford SB, Bittar GT. The use of methylergonovine (Methergine) in the initial control of drug induced refractory headache. Headache 1993;33(7) 390-393.

[154] Saper JR, Evans RW. Oral methylergonovine maleate for refractory migraine and cluster headache prevention. Headache 2013;53(2) 378-381.

[155] Hakim S, Khan RM, Maroof M, Usmani H, Huda W, Jafri F. Methylergonovine maleate (methergine) relieves postdural puncture headache in obstetric patients. Acta Obstetricia et Gynecologica Scandinavica 2005;84(1) 100.

[156] Alici HA, Cesur M, Erdem AF, Ingec M, Bebek Z. Is methergine alone sufficient in relieving postdural puncture headache? Acta Obstetricia et Gynecologica Scandinavica 2006;85(5) 632-633.

[157] Sheen MJ, Ho ST. Mirtazapine relieves postdural puncture headache. Anesthesia & Analgesia 2008;107(1) 346.

[158] Sandesc D, Lupei MI, Sirbu C, Plavat C, Bedreag O, Vernic C. Conventional treatment or epidural blood patch for the treatment of different etiologies of post dural puncture headache. Acta Anaesthesiologica Belgica 2006;57 55-56.

[159] Gormley JB. Treatment of post-spinal headache. Anesthesiology 1960; 21 565-566.

[160] Sanford CL 2nd, Rodriguez RE, Schmidt J, Austin PN. Evidence for using air or fluid when identifying the epidural space. American Association of Nurse Anesthetists Journal. 2013;81(1) 23-28.

[161] Pleasure SJ, Abosch A, Friedman J, Ko NU, Barbaro N, Dillon W, Fishman RA, Poncelet AN. Spontaneous intracranial hypotension resulting in stupor caused by diencephalic compression. Neurology 1998;50(6) 1854-1857.

[162] Szeinfeld M, Ihmeidan IH, Moser MM, Machado R, Klose KJ, Serafini AN. Epidural blood patch: evaluation of the volume and spread of blood injected into the epidural space. Anesthesiology 1986; 64(6) 820-822.

[163] Beards SC, Jackson A, Griffiths AG, Horsman EL. Magnetic resonance imaging of extradural blood patches: appearances from 30 min to 18 h. British Journal of Anaesthesia 1993; 71(2) 182-8.

[164] DiGiovanni AJ, Galbert MW, Wahle WM. Epidural injection of autologous blood for ostlumbar-puncture headache. II. Additional clinical experiences and laboratory investigation. Anesthesia & Analgesia 1972; 51(2) 226-232.

[165] Cook MA, Watkins-Pitchford JM. Epidural blood patch: a rapid coagulation response. Anesthesia & Analgesia 1990; 70(5) 567-568.

[166] Abouleish E, Vega S, Blendinger I, Tio TO. Long-term follow-up of epidural blood patch. Anesthesia & Analgesia 1975; 54(4) 459-463.

[167] Seebacher J, Ribeiro V, LeGuillou JL, Lacomblez L, Henry M, Thorman F, Youl B, Bensimon G, Darbois Y, Bousser MG. Epidural blood patch in the treatment of post dural puncture headache: a double blind study. Headache 1989; 29(10) 630-632.

[168] van Kooten F, Oedit R, Bakker SL, Dippel DW. Epidural blood patch in post dural puncture headache: a randomised, observer-blind, controlled clinical trial. Journal of Neurology, Neurosurgery and Psychiatry 2008; 79(5) 553-558.

[169] Kokki M, Sjövall S, Keinänen M, Kokki H. The influence of timing on the effectiveness of epidural blood patches in parturients. International Journal of Obstetric Anesthesia 2013;22(4) 303-309.

[170] Kueper M, Goericke SL, Kastrup O. Cerebral venous thrombosis after epidural blood patch: coincidence or causal relation? A case report and review of the literature. Cephalalgia 2008;28(7) 769-773.

[171] Ghatge S, Uppugonduri S, Kamarzaman Z. Cerebral venous sinus thrombosis following accidental dural puncture and epidural blood patch. International Journal of Obstetric Anesthesia 2008;17(3) 267-270.

[172] Borum SE, Naul LG, McLeskey CH. Postpartum dural venous sinus thrombosis after postdural puncture headache and epidural blood patch. Anesthesiology 1997;86(2) 487-490.

[173] Barrett J, Alves E. Postpartum cerebral venous sinus thrombosis after dural puncture and epidural blood patch. The Journal of Emergency Medicine 2005;28(3) 341-342.

[174] Jungmann V, Werner R, Bergmann J, Daum J, Wöhrle JC, Dünnebacke J, Silomon M. Postpartum cerebral venous sinus thrombosis after epidural anaesthesia. Anaesthesist 2009;58(3) 268-272.

[175] Collier CB. Blood patches may cause scarring in the epidural space: two case reports. International Journal of Obstetric Anesthesia 2011;20(4) 347-351.

[176] Gutterman P, Bezier HS. Prophylaxis of postmyelogram headaches. Journal of Neurosurgery 1978; 49 869-871.

[177] Colonna-Romano P, Shapiro BE. Unintentional dural puncture and prophylactic epidural blood patch in obstetrics. Anesthesia & Analgesia 1989; 69(4) 522-523.

[178] Agerson AN, Scavone BM. Prophylactic epidural blood patch after unintentional dural puncture for the prevention of postdural puncture headache in parturients. Anesthesia & Analgesia 2012;115(1) 133-136.

[179] Benson KT. The Jehova's Witness patient: considerations for the anesthesiologist. Anesthesia & Analgesia 1989; 69(5) 647-656.

[180] Bearb ME, Pennant JH. Epidural blood patch in a Jehovah's Witness. Anesthesia & Analgesia 1987;66(10) 1052.

[181] Silva Lde A, de Carli D, Cangiani LM, Gonçalves Filho JB, da Silva IF. Epidural blood patch in Jehovah's Witness: two cases report. Revista Brasileira de Anestesiologia 2003;53(5) 633-639.

[182] Jagannathan N, Tetzlaff JE. Epidural blood patch in a Jehovah's Witness patient with post-dural puncture cephalgia. Canadian Journal of Anaesthesia 2005;52(1) 113.

[183] Pérez Ferrer A, Martínez B, Gredilla E, de Vicente J. Epidural blood patch in a Jehovah's witness. Revista Española de Anestesiología y Reanimación 2005;52(6) 374-375.

[184] Tanaka T, Muratani T, Kusaka Y, Minami T. Epidural blood patch for intracranial hypotension with closed system in a Jehovah's Witness. Masui 2007;56(8) 953-955.

[185] Clark CJ, Whitwell J. Intraocular haemorrahge after epidural injection. BMJ 1961; I 1612-1613.

[186] Kara I, Ciftci I, Apiliogullari S, Arun O, Duman A, Celik JB. Management of postdural puncture headache with epidural saline patch in a 10-year-old child after inguinal hernia repair: a case report. Journal of Pediatric Surgery 2012;47(10) e55-e57.

[187] Vassal O, Baud MC, Bolandard F, Bonnin M, Vielle E, Bazin JE, Chassard D. Epidural injection of hydroxyethyl starch in the management of postdural puncture headache. International Journal of Obstetric Anesthesia 2013;22(2) 153-155.

[188] Chassard D, Vassal O. Epidural injection of hydroxyethyl starch in the management of postdural puncture headache. International Journal of Obstetric Anesthesia 2013; 22(4) 353-354.

[189] Dhillon S. Fibrin sealant (evicel® [quixil®/crosseal™]): a review of its use as supportive treatment for haemostasis in surgery. Drugs 2011;71(14) 1893-1915.

[190] Chauvet D, Tran V, Mutlu G, George B, Allain JM. Study of dural suture watertightness: an in vitro comparison of different sealants. Acta Neurochirurgica (Wien) 2011;153(12) 2465-2472.

[191] Gentili ME. Epidural fibrin glue injection stops postdural puncture headache in patient with long-term intrathecal catheterization. Regional Anesthesia and Pain Medicine 2003;28(1) 70.

[192] Crul BJ, Gerritse BM, van Dongen RT, Schoonderwaldt HC. Epidural fibrin glue injection stops persistent postdural puncture headache. Anesthesiology 1999;91(2) 576-577.

[193] García-Aguado R, Gil F, Barcia JA, Aznar J, Hostalet F, Barberá J, Grau F. Prophylactic percutaneous sealing of lumbar postdural puncture hole with fibrin glue to prevent cerebrospinal fluid leakage in swine. Anesthesia & Analgesia 2000;90(4) 894-898.

[194] Gil F, García-Aguado R, Barcia JA, Guijarro E, Hostalet F, Tommasi-Rosso M, Grau F. The effect of fibrin glue patch in an in vitro model of postdural puncture leakage. Anesthesia & Analgesia 1998;87(5) 1125-1158.

[195] Schlenker M, Ringelstein EB. Epidural fibrin clot for the prevention of post-lumbar puncture headache: a new method with risks. Journal of Neurology, Neurosurgery & Psychiatry 1987;50(12) 1715.

[196] Schlag MG, Hopf R, Redl H. Convulsive seizures following subdural application of fibrin sealant containing tranexamic acid in a rat model. Neurosurgery 2000;47(6) 1463-1467.

[197] Pouskoulas CD, Taub E, Ruppen W. Successful treatment of post-dural-puncture headache with surgical dura repair two years after spinal anesthesia. Cephalalgia 2013; 33(15) 1269-1271.

[198] Jadon A, Chakraborty S, Sinha N, Agrawal R. Intrathecal catheterization by epidural catheter: management of accidental dural puncture and prophylaxis of PDPH. Indian Journal of Anaesthesia 2009;53(1) 30-34.

[199] Al-Metwalli RR. Epidural morphine injections for prevention of post dural puncture headache. Anaesthesia 2008; 63(8) 847-850.

[200] Bussone G, Tullo V, d'Onofrio F, Petretta V, Curone M, Frediani F, Tonini C, Omboni S. Frovatriptan for the prevention of postdural puncture headache. Cephalalgia 2007;27(7) 809-813.

[201] Zajac K, Zajac M, Hładki W, Jach R. Is there any point in pharmacological prophylaxis of PDPH (post-dural puncture headache) after spinal anaesthesia for Caesaren section? Przeglad lekarski 2012;69(1) 19-24.

[202] Doroudian MR, Norouzi M, Esmailie M, Tanhaeivash R. Dexamethasone in preventing post-dural puncture headache: a randomized, double-blind, placebo-controlled trial. Acta Anaesthesiologica Belgica 2011;62(3) 143-146.

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