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Prevention of Postdural Puncture Headache Following Accidental Dural Puncture: Two Cases Report and Mini Literature Review

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Authors' contributions

This case report was carried out in collaboration between all authors. Authors NK, BK, CA managed the cases and wrote the case report. All authors read and approved the final manuscript.

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Case Study

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ABSTRACT

38 and 49 years old two women were admitted for total abdominal hysterectomy. Both patiens had no history of diabetes mellitus, hypertension, arrhytmia, myocardial ischemia, hyperkalemia, or local anaesthetic allergy. Also, there was no pathological finding in the preoperative laboratory evaluation. Initially, we planned combined spinal epidural anaesthesia (CSE) but because of accidental dural puncture (ADP), 15 mg hyperbaric bupivacaine 0.5% was injected into subarachnoid space through epidural needle for spinal anaesthesia. Later, the epidural needle was withdrawn into epidural space and a 20-gauge epidural catheter was easily placed into epidural space. At the end of the surgery, 10 ml of saline with 3 mg morphine was injected through epidural catheter and then continuous infusion of 10 $ml.h^{-1}$ saline was admitted via epidural catheter during 24 hours postoperatively. A second injection of 3 mg morphine in 10 ml saline was repeated on the postoperative 24 h immediately before removal of the catheter. No patient need additional analgesic treatment and no adverse effect were observed in our two cases because of epidural morphine utilization.

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Following ADP, leaving the catheter in the epidural space and the administration of morphine injection with continuous saline infusion via epidural catheter may be an alternative to reduce the post dural puncture headache (PDPH).

Keywords: Accidental dural puncture; post-dural puncture headache; epidural morphine; epidural saline.

1. INTRODUCTION

Post-dural puncture headache is one of the most common serious and debilitating complications of central neuroaxial blockade. It can ocur as a result of diagnostic or therapeutic lumbar puncture, spinal anaesthesia, and ADP during epidural analgesia [1,2].

The incidence of ADP during the initiation of epidural analgesia/anaesthesia in obstetric population is between 0.19% and 3.6% [3], the incidence of PDPH in these patients has been reported to range 50-85% [4,5].

PDPH occurs on the first or second day after dural puncture and it is not only a disabling and incapitating condition, but also has a potential for morbidity and important financial, social and psychological repercussions [6]. Conservative measures such as hydration and bed rest have a history of not being very effective [7,8]. Therefore, many treatment techniques including, epidural blood patch [3], epidural injection or infusion of saline [9-11], intrathecal injection of saline [12], continuous of intrathecal analgesia [13], and insertion of epidural catheter into the subarachnoid space through the dural hole [14] have been tried with variable success. Because results for these interventions have been mixed, there is no clear consensus as to which prophylactic measure is the most effective.

We aimed to inform our experience that effectiveness of epidural morphine injection and subsequent infusion of epidural saline in two cases and to review the other treatment techniques to prevent PDPH following ADP.

2. TWO CASES REPORT

38 and 49 years old two women were admitted for total abdominal hysterectomy at the Akdeniz University Hospital. Both patiens had no history of diabetes mellitus, hypertension, arrhytmia, myocardial ischemia, hyperkalemia, or local anaesthetic allergy. Also, there was no pathological finding in the preoperative laboratory evaluation. After establishing monitorization and infusion of 10 mlkg Ringer's lactate intravenousluy, an 18 –gauge Tuohy needle (Perifix, B Braun, Melsungen, Germany) was inserted into the L_4-L_5 interspace in the lateral decubiting position, using loss-of-resistance to saline for CSE anaesthesia. In both patients, ADP was occured at the first attempt and the free flow of cerebrospinal fluid (CSF) through the needle was seen. Because of ADP, 15 mg hyperbaric bupivacaine 0.5% (Marcaine spinal heavy; Astra Zeneca, Sodertalje, Sweden) was injected into subarachnoid space through epidural needle for spinal anaesthesia. Both of the patients were informed of this complication and its possible consequences. After explanations and agreement of the patients, it was decided the administration of morphine injection with continuous saline infusion via epidural catheter. Following spinal injection, the epidural needle was withdrawn into epidural space and a 20-gauge epidural catheter was easily placed (3 cm) into epidural space for postoperative epidural morphine and saline infusion. Following negative aspiration of blood or CSF, five ml of lidocaine 2% were injected through the catheter in order to assess the absence of intravenous injection of local anaesthetic solution. At the end of the surgery, 10 ml of saline with 3 mg morphine was injected through epidural catheter and then continuous infusion of 10 $ml.h^{-1}$ saline was admitted via epidural catheter during 24 hours postoperatively. A second injection of 3 mg morphine in 10 ml saline was repeated on the postoperative 24 h immediately before removal of the catheter. They were also recommended conservatively bed rest, iv fluid replacement. The severity of PDPH was assesed in the postoperative period according to a 10 point visual analoque scale (VAS; 0= no pain and 10= worst possible pain). The level of VAS in both patients was assessed as 2 and 3 point respectively on the second and third day during postoperative period (Table 1). No patient needs additional analgesic treatment and no adverse effect was observed in our two cases because of epidural morphine utilization. Case 1 was discharged from the hospital on the fifth day, and Case 2 was discharged from the hospital on the

fourth day of postoperative period with no headache.

Table 1. VAS scores of two patients for PDPH

	Case 1	Case 2
VAS 2nd day	2	3
VAS 3rd day	3	3
VAS 4th day	0	0

3. DISCUSSION

Puncture of the dura has a potential to allow the development of excessive leakage of CSF. Excess loss of CSF leads to intracranial hypotension and a demonstrable reduction in CSF volume or pressure or both. Intrathecal hypotension may result in caudad excursion of the brain, which results in headache through traction on pain-sensitive areas of the brain and meninges. CSF loss may also cause increased cerebral blood flow and vascular dilation, resulting in a pathophysiology similar to vascular headaches [6,15].

In this two cases, we observed that epidural morphine injection and subsequent epidural saline infusion from epidural catheter after ADP was effective in decreasing the severity of PDPH.

When a PDPH occurs, there are no accepted algorithms or treatment. Conservative measures such as hydration and bed rest have a history of not being very effective to prevention of PDPH [7,8]. Therefore, numerous invasive strategies have instead been suggested to prevent PDPH. Currently, the two most widely used options following ADP are either re-siting the epidural catheter into different interspace or inserting the epidural catheter intrathecally followed by conversion to spinal analgesia [16].

Placement of spinal catheter has gained popularity following ADP [17]. However, the results from data on subsequent PDPH and epidural blood patch (EBP) rates are varied [18-20]. Some authors have suggested that intrathecal insertion of an epidural catheter at the time of ADP, with or without a continuous spinal infusion of saline, reduces the risk of PDPH and needs for therapeutic EBP [13,14,20-23] and the others did not find a significant benefit [18,19].

In a prospective controlled stuy, Russell [16] found that inserting an intrathecal catheter had no significant effect compared with repeated epidural catheter on PDPH or EBP rates following ADP during labour analgesia. In another study, inserting the epidural catheter intrathecally significantly reduced the incidence of PDPH following ADP to 42% compared with 62% in those who have the catheter re-sited epidurally [24].

The mechanism of intrathecal catheters in prevention of PDPH is that the catheter mechanically plugs the dural tear, thereby lessening or stopping the CSF leak from the subarachnoid space. Morever, as there is a continuous infusion of saline through the catheter, the fluid loss is theoritically replaced by saline. Another possible mechanism is that the spinal catheter evokes an inflammatory tissue reaction that helps to plug the dural hole. [12,14,25,26].

Some studies have demonstrated that subsequent catheter placement into the subarachnoid space through the dural puncture site after ADP, and leaving the catheter in place for more than 24 h, decreases the incidence of PDPH to less than 1% [13,14,20]. However, prolonged subarachnoid catheter placement, especially related to the microcatheter used has been associated with Cauda Equina Syndrome [27] and catastrophic complications such as accidental injection of an epidural dose of medication via subarachnoid catheter [28].

The mechanisms of leaving the epidural catheter in the epidural space in the prevention of the PDPH are varied. First, the injected solution for epidural anesthesia or analgesia may have a mass effect, resulting in thecal sac compression, thus compensating CSF pressure. Second, the injected solution and the catheter may promote an inflammatory process, facilitating closure of the dural defect. This effect would be expected to increase with the time of the catheter remaining in the epidural space and postoperative injections there. Third, it may be postulated that the compression effect of the injected volume on the dural defect may minimize the CSF leakage. Lastly, the low incidence of PDPH may be related to the use of neuroaxial opioids for postoperative analgesia in all patients [13,29,30].

Cesur and colleagues [31] investigated subsequent catheter placement into the epidural space from a different interspace after ADP and leaving the catheter for postoperative analgesia with 3 mg morphine in 15 mL saline for 36-72 h in the postoperative period. They concluded that

significant reduction of the incidence of PDPH and reduction in the indication for an EBP.

Al-metwalli RR [32] injected 3 mg morphine in 10 ml of saline through the epidural catheter which was left in situ for 24 h and then a further 3 mg before removal. The incidence of PDPH was reduced from 48% to 12% and none required an EBP. They concluded that the use of epidural morphine and subsequent epidural catheter placement is an alternative to other methods to reduce the incidence of PDPH in parturients who had ADP.

The mechanism of epidural morphine is not known. It could be due to slow systemic absorption of epidural morphine but this is difficult to support that 3 mg epidural morphine would explain this suggestion. It could be en effect of the volume of saline injected into the epidural pressure, but although the volume injected was the same in both groups, the result was in favour of the morphine group. It could be due to the sum of these two factors. Another possible mechanism is rostral spread of epidural morphine to induce central analgesia [32].

When a dural puncture was diagnosed, we injected local anaesthetic solution into subarachnoid space through epidural needle for spinal anaesthesia and then epidural needle was withdrawn into epidural space and epidural catheter was inserted to same epidural interspace to avoid further dural puncture.

For prevention of PDPH, we injected 3 mg morphine in 10 ml saline and subsequently continuous infusion of 10 $ml.hr^{-1}$ saline was given via epidural catheter during 24 hours postoperatively. We repeated 3 mg morphine injection in 10ml saline and epidural catheter was removed in two patients at the 24^{th} h during postoperative period. Patients were also recommended conservatively bed rest, iv fluid replacement. The severity of PDPH in both of patients was assessed as 2 and 3 point respectively on the second and third day for postoperative period according to VAS. No patient needs additional analgesic treatment and no adverse effect was observed in our two cases because of epidural morphine utilization.

The use of epidural morphine has only been investigated in one RCT [32]. Although the use of epidural morphine has the highest risk reduction in all interventions studied, there are two case report supporting the use of epidural morphine as a prophylaxis for PDPH [33,34].

Eldor J and colleagues [35] reported six patients with PDPH in whom total relief of headache was attained using epidural injection of morphine via placed epidural catheters. The mechanism of epidural morphine injection could be due to systemic absorption of epidural morphine, but this is difficult to support. Because the small dose (3 mg) and slow systemic absorption of morphine would not explain this suggestion. Another possible mechanism is rostral spread of morphine to induce central analgesia [32].

In another study, patients who had ADP were divided into 3 groups: An epidural catheter placed at different interspace, a subarachnoid catheter placed for only labor analgesia and removed after delivery, a subarachnoid catheter left in subarachnoid place for 24 h after delivery. The incidence of PDPH was significantly less in both subarachnoid catheter groups compared with epidural catheter group [20].

Cohen S and colleagues [13] reported a decreased incidence of PDPH using continuous postoperative intrathecal analgesia in parturients who had experienced ADP following attempts at epidural anaesthesia [13]. In another study, placement of intrathecal catheter for at least 12 h and providing continuous spinal anaesthesia following ADP did not decreased the incidence of PDPH [18].

Charsley MM and Abram SE [12] investigated whether the injection of 10 ml of saline into the subarachnoid space following ADP reduced the incidence of PDP and the need for EBP. When an intrathecal catheter had been placed following a wet tap, injection of 10 ml of saline before its removal effectively prevented PDPH.

When a PDPH occurs, there are no accepted algorithms or treatment, but the nature of the defect (needle size suspected of making the puncture) and the presence and severity of symptom are important considerations. The gold standard for the treatment of severe PDPH is epidural blood patch (EBP) because it has the highest benefit-to-risk ratio and is the most effective treatment to date. It is now evident that untreated PDPH, especially when significant intrathecal hypotension is present, may lead to a higher incidence of morbidity and mortality, including chronic headache syndromes, subdural haematoma [36-38].

Two theories have been proposed to explain EBP efficiency in the treatment of PDPH [23,39]. The first theory suggests that the autologous blood injected in the epidural space forms a clot, which adheres to the dura mater and directly patches the hole. The second theory suggests that the volume of blood injected in the epidural space increases CSF pressure, thus reducing traction of pain sensitive brain structures, leading to relief of symptoms [40]. The efficacy of an EBP is influenced by its timing in relation to the initial dural puncture. Several studies show improved outcomes if the EBP is delayed [36,39,41]. In a study, the initial rate of resolution of headache did not differ significantly between parturients receiving a blood patch within 48 h of dural puncture and after 48 h. However, in the former group, the incidence of recurrent headache was significantly higher [41].

While there is a clear evidence that therapeutic EBP is effective, there is some evidence that earlier EBP (within the first 24-48 h after ADP) may be less effective. In fact the recurrence of headache can be 50% higher if EBP is completed within the first 24 h [36,42,43].

Subdural haematoma is one of the most serious complication of ADP and results from tearing intracerebral bridging veins due to caudad brain migration after CFF loss [38,44]. While evidence is lacking that the risk of subdural haematoma formation in patients with PDPH is reduced by EBP treatment, there are reports that EBP treatment itself may cause subdural haematoma [45,46]. Other serious complications of EBP include intrathecal injection and arachnoiditis [47,48], infected blood patch, facial nerve paralysis and permanent spastic paraparesis and cauda equina syndrome [49,50,51].

Prophylactic treatments (prophylactic blood patch, epidural or intrathecal saline, epidural morphine, and intrathecal catheter placement) have been used but no clear consensus exists on the best preventive measure for PDPH following ADP [3]. An EBP may be part of that treatment but administering it to all women with PDPH is not the optimal management approach [52]. Gobin J and colleagues [53] proposed a combination of these techniques: An intrathecal catheter was immediately inserted following ADP in order to avoid CSF leakage, and was kept in place for more than 24 h. Immediately after intrathecal catheter removal, a prophylactic blood patch was performed in order to increase epidural pressure and clog the dural tear.

4. CONCLUSION

Subsequent placement of a catheter into the epidural space following ADP and administration of morphine injection with saline infusion via epidural catheter may reduce the PDPH. However, a large and well-designed RCT is required to provide clinical evidence for the effectiveness of epidural morphine injection and subsequently saline infusion via epidural catheter for PDPH.

CONSENT

All authors declare that ‘written informed consent was obtained from the patients and have therefore been performed in accordance with the ethical standards laid down in the 1964 Declaration of Helsinki.

COMPETING INTERESTS

Authors have declared that no competing interests exist.

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