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Impact of migraine headaches and depression/anxiety on the incidence of post-dural puncture headache during postpartum course

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RESEARCH

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ABSTRACT

Background
Migraine headaches, anxiety disorder and depression have not been studied to determine if parturients are at risk for developing a PDPH.

Aims
This retrospective analysis is to identify and assess the risk of developing a post-dural puncture headache (PDPH) in parturients with a documented history of primary migraines and anxiety/depression.

Methods
The parturients who had accidental dural puncture (ADP) during labour epidural placement were included and further analysed for the development of a post-dural puncture headache (PDPH). We compared patient demographics and the history of depression/anxiety, as well as evaluated the patient for a history of migraine headaches and depression/anxiety.

Results
One hundred seventy-five parturients met our criteria for ADP, from which 92 (52.6 per cent) developed PDPH. A history of migraines was present in 7 of 10 (70 per cent) of patients with a PDPH, from which 4 of 7 (57.1 per cent) required an epidural blood patch treatment. A history of depression/anxiety was found in 7 of 12 (58.3 per cent) with a PDPH of which 2 of 7 (28.6 per cent) required an epidural blood patch treatment. Data analysis showed no significant difference between a history of migraines or depression/anxiety and the incidence of dural puncture headache (P=0.26 and P=0.68, respectively), nor was there an association between a history of migraines or depression/anxiety and the intensity of dural puncture headache (P=0.25 and P=0.63, respectively).

Conclusion
The results of this study indicate that in postpartum patients neither the presence of a history of migraines nor a history of depression/anxiety was associated with an increased risk of the development of PDPH or an increase in its intensity.

Key Words
Post-dural puncture headache, migraine, postpartum, depression/anxiety

What this study adds:

1. What is known about this subject?
There is a close relationship between a history of chronic headache and the development of a post-dural puncture headache (PDPH).

2. What new information is offered in this study?
The patients with a history of migraine and anxiety/depression are not more likely to experience a PDPH, and neither of these factors increased the intensity of

PDPH.

3. What are the implications for research, policy, or practice?

It warrants further studies to discover if migraines and PDPH inhibit each other.

Background

The occurrence of a postdural puncture headache (PDPH) has been well described as a complication of epidural anaesthesia and spinal anaesthesia. During epidural anaesthesia, accidental dural puncture (ADP) occurs in approximately 0.9–1.5 per cent of patients and over 50 per cent will subsequently experience a PDPH.[1,2] Patients with PDPH typically present with a non-throbbing headache within 48 hours of the dural puncture. This headache can be mild to severe, sometimes may be described as incapacitating, and may be located in the frontal and/or occipital regions. It can be exacerbated when sitting or standing and relieved when supine. In more than 90 per cent of patients the headache resolves within seven to ten days, though rarely case reports have described PDPH lasting months.[3,4]

Several demographic factors have been associated with PDPH, namely young age, female sex, needle size and needle design.[5,6] It was previously believed as well that patients with a low body mass index, a history of chronic headache, a history of PDPH, a history of anxiety, hydration status and mode of delivery increased the tendency of PDPH development.[4,7-10] In contrast, our previous retrospective study[11] and studies from other researchers[12,13] do not support the suggestion that low BMI can be a risk factor for PDPH.

Similarly, the relationship of the development of a PDPH in patients with a history of chronic headache has conflicting reports.[14,15] Migraine headaches, as one of the most common chronic headaches, has an incidence estimated to be 5 and 22 per 1000 persons /year for males and females respectively with an overall incidence of 8.1 per 1000 persons. Female gender, younger age, familial history, frequent tension headaches and high workload are considered the risk factors for a migraine.[16] The incidence of migraines varies during pregnancy and after delivery. Patients who have migraines prior to pregnancy experience alleviation in their migraines with less recurrence during pregnancy, which is thought to be due to the significant elevated oestrogen and other hormone levels. However, they tend to have an increase in recurrence after delivery, often experiencing a migraine in the first week postpartum that may be explained by the rapid decline in oestrogen level.[17]

Migraines, anxiety disorder and depression may coexist with one other.[18] Emotional stressors and dramatic hormonal changes following childbirth during postpartum may result in postpartum depression. Women with a history of depression/anxiety or a family history of mental health issues are more likely to be at an increased of postpartum depression.[19] To determine the influence of migraines and depression/anxiety on the risk of PDPH in parturients, we extended our research to assess the risk of developing PDPH in patients with a documented history of primary migraines and depression/anxiety disorders.

Method

This study was approved by the Institutional Review Board of Albert Einstein College of Medicine, which is a teaching institution comprised of a primary and specialty care network. Most of parturients in our institution received spinal or epidural procedures for labor analgesia or surgical delivery. Study subjects were identified through the review and analysis of medical databases and case logs of the Department of Obstetric Anesthesiology between January 2011 and June 2015. All data of ADPs that were witnessed and documented in the medical record after lumbar epidural placement for labor analgesia were collected and recorded. The patients who were discharged and then returned to the Emergency Department for management of refractory PDPH have been followed.

All vaginal deliveries requiring epidural for labour analgesia were included. Prior to epidural placement for labour pain management, all parturients were well hydrated with 500 to 1000ml of crystalloid fluids. A 17g Tuohy needle was inserted epidurally via the midline approach. An ADP was defined as those parturients who had a lumbar epidural placed and cerebrospinal fluid (CSF) was noted from the needle or aspirated from the epidural catheter. Subsequent epidural catheter placement occurred at a different spinal level. The patients with an intrathecal catheter who utilized spinal or combined spinal/ epidural anaesthesia for labour analgesia were excluded. Parturients who were prophylactically treated with epidural or intrathecal normal saline, immediate administration of non-steroidal anti-inflammatory agents after ADP or immediate epidural blood patch (EBP) for PDPH were also excluded.

The data collected was further classified using characteristics including patient age, American Society of Anesthesiologists physical status classification score (ASA),

body mass index (BMI), documented prior migraine history and documented prior depression/anxiety history. Additional data was then collected on those patients who developed a PDPH after ADP and those who required epidural blood patch as treatment.

The primary outcome of this study is the development of PDPH after ADP during epidural placement using criteria outlined by the International Classification of Headache Disorders (ICHD-II).[3] (1) dural puncture performed, (2) headache that worsens within fifteen minutes of standing or sitting with one or more of the following: tinnitus, neck stiffness, photophobia, hypacusia and/or nausea, (3) headache develops within five days of dural puncture, (4) headache spontaneously resolves within one week, or within forty-eight hours of an epidural blood patch. The severity of the PDPH was categorized based on EBP management requirements as either severe/disabling or non-disabling. Those patients requiring an EBP for PDPH management were defined as severe/disabling whereas those patients who received only conservative and supportive treatments were defined as non-disabling headaches.

Statistical analysis

Data analysis was conducted with SPSS 17.0 statistical software package. Subjects were divided into two groups based on the presence or absence of PDPH following ADP. Chi-square analysis was performed to assess two primary outcome variables of the presence of PDPH and intensity of PDPH (requirement of EBP) with migraine and depression/anxiety. In all statistical analyses, $p<0.05$ was considered significant.

Results

There were 208 parturients who had a vaginal delivery associated with an ADP between January 2011 and June 2015 of which 175 met the study criteria. A total of 92 out of 175 patients (52.6%) developed PDPH following ADP with an epidural needle. Of the patients with PDPH, 34 patients (37.0%) needed EBP due to a persistent headache. 7 of 10 (70%) of patients with a PDPH had a history of migraines, from which 4 of 7 (57.1%) required epidural blood patch treatment. There were 7 of 12 (58.3%) of patients with PDPH who had a history of depression/anxiety, from which 2 of 7 (28.6%) required an epidural blood patch treatment. The mean age of PDPH was 28.13±5.91 years and the mean age of paturients requiring EBP was 28.62±6.06. The mean age of paturients with a history of migraines who developed PDPH was 28.75±3.59 and the mean age of those with a prior migraine history requiring EBP after PDPH was 27.5±0.71. The mean age of paturients with a history of depression/anxiety who developed PDPH was 28.67±5.47and the mean age of these patients who required EBP after PDPH development was 22.00±1.41 (table 1). Demographic information of patient who developed a PDPH and who required EBP based on age, ASA classification and BMI are presented in Table 2 and Table 3.

Patients who developed a PDPH and asymptomatic patients as well as those who required EBP versus those who did not require EBP were risk stratified based on the history of migraine and depression/anxiety (Table 4 and Table 5). The results did not show a statistically significant difference in the incidence of PDPH following ADP in patients with a history of prior migraines ($p=0.256$). There was no statistically significant difference in the incidence of EBP requirement in same patient group ($p=0.0.25$).

Discussion

This retrospective cohort study of PDPH complements the very few prior studies of the experiences of parturients with a history of migraine headaches or depression/anxiety. Our major findings indicated the history of migraines or the history of anxiety/ depression disorders had no effect on the development of PDPH following an ADP. They were neither associated with an increased risk of post-dural puncture headache nor the risk of a disabling post-dural puncture headache.

PDPH, as an idiopathic intracranial hypotension headache, is a very common complication in parturients after ADP. Several pathophysiologic mechanisms have been suggested for the development of PDPH although the cause remains unclear. The best documented explanation is that a dural tear from an epidural or spinal needle results in the leakage of cerebrospinal fluid (CSF) and intracranial hypotension.[4] CSF volume loss from a dural puncture creates a downward traction to the meninges, leading to hypersensitivity to substance P and a compensatory vasodilation of intracranial vessels.[20] An additional explanation is that PDPH may develop from a combination of CSF hypotension with meningeal inflammation and other factors.[21]

The pathophysiological explanation for the development of migraines has been postulated to involve inflammation, vasodilation of intracranial and cephalic arteries, leakage of plasma protein from blood vessels and mast cell degranulation.[22] The release of neuropeptides such as calcitonin-gene related peptide, substance P and vasoactive intestinal peptide in response to the stimulation of the trigeminal nerve sensory branch leads to neurogenic

inflammation around nearby meningeal vessels. This conducts a pain stimulus via the trigeminal nerve to the central nervous system and further dilates the dural vessels to increase blood flow.[23,24] Although the mechanism for the development of migraines have some similarity to PDPH, it is currently thought to be more complex and multifactorial.[25]

In our study, the numerous variables that might have confounding influences [4,7-10 ,26-,28] on data pertaining to the development of PDPH had been removed. We limited our inclusion to well hydrated patients who underwent epidural placement for labour analgesia, utilizing a 17 gauge Tuohy needle, who delivered vaginally and never received prophylactic treatment for the prevention of PDPH development.

We compared patient demographics, as well as evaluated patient history of prior migraine headaches as a possible risk factor for the development or augmentation of PDPH. We found that although 7 of the 10 patients with a prior history of migraine headaches developed PDPH, this result was not statistically significant (p=0.256) and thus those with a history of migraines are not likely at an increased risk for the development of a PDPH. A subset analysis from our study of patients with a history of migraines also found no correlation between the history of migraines and the intensity of PDPH, classified as that requiring or not requiring an epidural blood patch (57% *vs.* 43%, p=0.250).

A similar conclusion was drawn from another study which implied that the history of migraines had no association with PDPH development.[29] Another larger prospective study by Oosterhout et al., evaluated the risk of developing PDPH in 160 non-parturients with a known recent migraine attack undergoing lumbar puncture and then compared that with age and gender matched healthy patients. They elicited that patients with migraines actually had a significantly lower incidence of PDPH.[15] Interestingly, a case of resolution of migraine attacks during long-lasting PDPH has been reported. It was suggestive of the trigeminal nerve may be refractory to various stimuli and desensitized after long standing activation and failure to further dilate the vessels when the cerebral vessels already being extensively dilated.[30] Presumably, migraine and PDPH as two different types of headache may have mutual inhibition.

Psychiatric comorbidities, especially depression and anxiety, have been well recognized in patients with migraines.[18,31] Recent studies have discovered that both headache and anxiety involve a coordination of different neurotransmitters, such as serotonin and norepinephrine. The serotonin norepinephrine reuptake inhibitors (SNRIs) have shown to be efficacious on treating either on anxiety disorders or modulating on the modulation of pain symptoms.[32,33] One of the antidepressants, mirtazapine, has been used to relieve PDPH by constriction of dilated cerebral vessels probably due to activation of 5-HT 1 receptors and a net positive effect on noradrenergic neurotransmission, as well as through potentiation of the endogenous opioid systems by acting as a 5-HT 2/3 receptor antagonist.[34,35]

Very little research has been done to evaluate depression/anxiety as a specific risk factor for PDPH. Mantarova's study illustrated that patients who presented in an anxious state showed an increased incidence of PDPH in non-parturients,[9] although the data about the relationship between PDPH and symptoms of anxiety/depression are still limited.

In contrast to Mantarova's study where patients with situational anxiety were the main characteristic, our patients presented with a relatively long standing psychiatric history. We observed that there was no positive correlation between a chronic history of depression/anxiety and the development of PDPH (p=0.678) or the intensity of the PDPH (p=0.633). One possible explanation for this discovery may be that chronic neurotransmitter depletion from long standing depression/anxiety may lead to desensitization.

This is a preliminary study which was limited by our small sample size of patients with documented migraine or anxiety/depression history. The restriction of a small sample does not allowed us to further classify this patient population into further subgroups to quantify the migraine intensity. It also prevented us from separating depression and anxiety as co-variables to analyse their effects on PDPH. Further additional analysis is necessary to expand our cohort size by collecting data over a longer period of time or by coordinating with other institutions.

Conclusion

Overall, this retrospective cohort study demonstrated that patients with a history of migraine headaches and anxiety/depression are not more likely to experience a PDPH during epidural analgesia for vaginal delivery, and neither of these factors increased the intensity of PDPH. This warrants further study to discover if migraines and PDPH inhibit each other.

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PEER REVIEW

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CONFLICTS OF INTEREST

The authors declare that they have no competing interests.

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ETHICS COMMITTEE APPROVAL

This study was approved by the Institutional Review Board at the Albert Einstein School of Medicine, NY.

Table 1: Mean age of patients and Mean age of migraine and depression/anxiety

	PDPH		EBP	
	Yes	No	Yes	No
Age (M+SD, years)	28.13±5.91	27.29±5.72	28.62±6.06	27.93±5.93
Age of Migraine (M+SD, years)	28.75±3.59	28.50±7.78	27.50±0.71	30.00±5.66
Age of Depression/Anxiety (M+SD, years)	28.67±5.47	28.67±4.93	22.00±1.41	32.00±2.16

Table 2: Demographic characteristics of patients

	PDPH (n=92)	Non PDPH (n=83)	Total (n=175)
Age(y), n (%)			
<20	6 (42.9)	8 (57.1)	14
20–30	55 (52.3)	51 (47.7)	106
>30	31 (56.4)	24 (43.6)	55
ASA classification			
II	61 (50.8)	58 (49.2)	119
III	29 (54.7)	24 (45.3)	53
IV	2 (66.7)	1 (33.3)	3
BMI (kg/m^2), n (%)			
<25	7 (58.3)	5 (41.7)	12
25–29.9	32 (62.7)	19 (37.3)	51
30–34.9	22 (41.5)	31 (58.5)	53
35–39.9	13 (50.0)	13 (50.0)	26
>=40	8 (54.5)	15 (45.5)	33

Table 3: Demographic characteristics of patients with PDPH

	PDPH with EBP (n=34)	PDPH without EBP (n=58)	Total (n=92)
Age (y), n (%)			
<20	2 (33.3)	4 (66.7)	6
20–30	20 (37.0)	34 (63.0)	54
>30	12 (37.5)	20 (62.5)	32
ASA classification			
II	24 (39.3)	37 (60.7)	61
III	9 (31.0)	20 (69.0)	29
IV	1 (50.0)	1 (50.0)	2
BMI (kg/m^2), n (%)			
<25	1 (14.3)	6 (85.7)	7
25–29.9	13 (40.6)	19 (59.4)	32
30–34.9	11 (50.0)	11 (50.0)	22
35–39.9	4 (30.8)	9 (69.2)	13
>=40	5 (27.8)	13 (72.2)	18

Table 4: Migraine and depression/anxiety *vs.* PDPH and demographic characteristics of patients

	PDPH (n=92)	PDPH (n=83)	Total (n=175)	Statistics (p values)
History of migraine, n (%)				0.256
No	85 (51.5)	80 (48.5)	165	
Yes	7 (70.0)	3 (30.0)	10	
History of depression/anxiety, n (%)				0.679
No	85 (52.1)	78 (47.9)	163	
Yes	7 (58.3)	5 (41.7)	12	

Table 5: Migraine and depression/anxiety *vs.* the intensity of PDPH and demographic characteristics of patients

	PDPH with EBP (n=34)	PDPH without EBP (n=58)	Total (n=92)	Statistics (p values)
History of migraine, n (%)				0.250
No	30 (35.3)	55 (64.7)	85	
Yes	4 (57.1)	3 (42.9)	7	
History of depression/anxiety, n (%)				0.633
No	32 (37.6)	53 (62.4)	85	
Yes	2 (28.6)	5 (71.4)	7	