Approach to Headache in Post LSCS Patient

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Headache is one of the most common symptoms that are encountered in the postpartum period. The postpartum period is characterized usually by various life changes such as sleep deprivation, irregular food intake, and dehydration. All of these factors can be associated potentially with a headache. Hormonal fluctuations in oestrogen levels and headaches (particularly migraine headache) tend to recur in the postpartum period. The increasing use of regional techniques for operative delivery as well as labour analgesia is associated with post-dural puncture headaches (PDPH). Lastly, pregnancy-associated conditions such as pre-eclampsia and posterior reversible encephalopathy syndrome (PRES); and intracranial conditions such as cerebral venous thrombosis need to be identified and treated. Thus headache in the puerperal period often presents a diagnostic challenge to the anaesthetic and obstetric teams.

Incidence - The reported incidence of postpartum headache ranges from 11% to 80%. Goldszmidt et al reported a 39% incidence of headache in the first 7 days after delivery. Stella et al showed that the most common reason for persistent headache 24 hours after delivery is tension-type headache and/or migraine headache. These findings are contrary to the common dictum that postpartum headache usually is caused by a spinal headache. Goldszmidt et al, also reported that primary headaches (tension or migraine) are nearly 20 times more frequent than a secondary headache (postdural puncture). Tension-type/migraine comprised the most common cause (47%), followed by preeclampsia/eclampsia (24%) and PDPH (16%).

Classification of postpartum headache -
Headache can be classified as primary or secondary (Table 1). Migraine headaches, tension-type headaches, and cluster headaches are considered to be primary headache. A common, but under reported, cause of a primary headache in the puerperium includes orgasmic headache. The proposed triggers of puerperal orgasmic headache are hormonal (elevated oestrogen, prolactin, and oxytocin levels) and neurologic (autonomic nervous system
regulation of orgasm) in origin. Secondary headaches include headaches that are the result of regional anaesthesia complications, obstetric complications, or neurologic lesions\(^1\)

**Evaluation of postpartum headache**

The evaluation of postpartum headache should be performed in a stepwise fashion and requires a multidisciplinary approach (Figure 1).

**Figure-1- Evaluation of postpartum headache**

The typical parturient who complains of a postpartum headache usually is instructed to take analgesics. Those who continue to have persistent headache, despite the use of analgesics, require evaluation and treatment. Subsequent treatment will depend on their history, clinical findings, and presence or absence of associated neurologic symptoms or deficits. Patients without focal neurologic deficit and without findings that are consistent with preeclampsia should be considered initially to have tension-type or migraine headache. Since most of the
caesarean sections are done using spinal anaesthesia invariably the anaesthesiologist will be called in for further evaluation and management. Severe unrelenting headaches with focal neurologic signs usually require neurologic imaging and then referral to neurologists.

<table>
<thead>
<tr>
<th>TABLE -1 Differential diagnosis of postpartum headache</th>
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<tbody>
<tr>
<td><strong>Primary headache</strong></td>
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<tr>
<td>Migraine</td>
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<tr>
<td>Tension-type headache (includes benign ordinary headache)</td>
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<tr>
<td>Orgasmic headache</td>
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<tr>
<td><strong>Secondary</strong></td>
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<tr>
<td>Postdural puncture headache</td>
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<tr>
<td>Preeclampsia</td>
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<tr>
<td>Cerebral venous thrombosis</td>
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<tr>
<td>Stroke (ischemic or haemorrhagic)*</td>
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<td>Ruptured aneurysm</td>
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<tr>
<td>Hypertensive encephalopathy or bleeding</td>
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<tr>
<td>Cerebral arterial thrombosis or embolism</td>
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<tr>
<td>PRES</td>
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<tr>
<td>Postpartum cerebral angiopathy*/Call-Fleming syndrome</td>
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<tr>
<td>Pituitary apoplexy</td>
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<tr>
<td>Pseudotumor cerebri</td>
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<tr>
<td>Subarachnoid haemorrhage*</td>
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<td>Meningitis</td>
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<td>* Life-threatening.</td>
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**PDPH - Clinical presentation**

The International Headache Society describes PDPH as headache that worsens within 15 min after sitting or standing and improves within 15 min after lying, with at least one of the following - neck stiffness, tinnitus, hypacusia, photophobia or nausea. There should be a history of dural puncture, the headache should develop within five days after dural puncture.
and should resolve either spontaneously within one week or within 48 h after effective treatment of cerebrospinal fluid (CSF) leak usually by epidural blood patch (EBP). The majority of PDPH present within 48-72 hrs of the procedure, but early discharge from the hospital means that the patient may be at home when the headache begins. The usual duration is 1–7 days but have been reported to persist for up to 9–12 months following dural puncture. Cranial nerve palsies have also been associated with PDPH, the most common nerve affected (92–95%) is the abducens, causing diplopia. The oculomotor and trochlear nerves may also be involved. The onset of diplopia is usually 4–10 days after dural puncture, usually preceded by PDPH. EBP is not very effective at treating diplopia, recovery occurs spontaneously in two weeks to eight months. Trigeminal and facial nerve palsies; and auditory nerve affection with hearing loss or tinnitus and post-partum blindness have all been reported to be associated with PDPH\textsuperscript{5–7}. Untreated PDPH has been associated with intracranial complications which are uncommon but can be life-threatening. With the low intracranial pressure, bridging veins between the brain and dural sinuses may be stretched and ruptures in their most fragile portion which is in the subdural space\textsuperscript{2}.

**Prevention**

1. Using the smallest gauge needles practicable is the first step in preventing PDPH. For spinal blocks, pencil point needles of 25 G or smaller should be used, especially in the obstetric population.
2. Orienting the bevel of the Qincke needle to be parallel to the fibers or using para median approach for spinal anaesthesia have been shown to reduce CSF leakage;
3. There is evidence that using saline rather air for epidural insertion lowers the incidence of dural puncture and PDPH

**TREATMENT**

**Conservative treatment**

Maintenance of hydration and prescription of simple analgesics should be done in all patients. Bed rest per se has no effect on the outcome but many patients may prefer to lie down in the position of their choice. An abdominal binder raises the intraabdominal and CSF pressure and may provide some relief; but wearing it is uncomfortable and is not in general use.

**Pharmacological treatment**

Caffeine is a central nervous system (CNS) stimulant which also has a cerebral vasoconstrictive effect. Single doses of intravenous as well as oral caffeine (250 mg and 300 mg respectively) have been shown to relieve mild PDPH but the effect is transient.
Sumatriptan is a 5-HT agonist with cerebral vasoconstrictive effects. It is used in the treatment of migraine and has been found useful for PDPH. Sumatriptan is excreted in breast milk and it is advised that breast feeding should be avoided for 12 h following exposure to this drug.

Cosyntropin or synthetic ACTH, and hydrocortisone are believed to work by stimulating the adrenal gland and increasing CSF secretion. Intravenous cosyntropin as well as hydrocortisone have been found to be effective in treating PDPH after failed EBP.

EPIDURAL BLOOD PATCH

When autologous blood is injected into the epidural space, it spreads both cephalad and caudal and increases the pressure in the epidural space, compressing the thecal sac and increasing the CSF pressure. This causes immediate relief of the headache. At the same time, the blood coagulates, helped by the procoagulant effect of the CSF, and occludes the hole in the dura, preventing further leakage of CSF. The mass effect gradually resolves over 7-13 h, leaving a mature clot in the posterior epidural space. Over the next few days, there is fibroblastic activity and collagen formation, further securing closure of the dural perforation.

Under full aseptic precautions, with the patient usually in the lateral position, the epidural space is identified, either at the level of original puncture or one space lower. Another anaesthetist, also with full aseptic precautions, performs venepuncture and hands over the blood to be injected. The volume of blood to be injected is usually 20 ml though some people inject up to 30 ml. It is slowly injected into the epidural space till the patient reports a feeling of pressure or pain in her back or legs. After the procedure, the patient is advised to lie flat for at least 2 h and avoid vigorous activity or straining for a few days.

Migraine headaches

Migraine headaches are defined as recurring cranial pain lasting 4–72 hours, often with typical features such as pulsating pain in a unilateral location, nausea and photophobia. The presence of focal neurological symptoms, usually preceding the headache, characterizes the subset type of migrainous headache with aura. Pregnancy can have an ameliorating effect on migraine frequency in the majority of sufferers but symptoms may recur soon after delivery, with reports of 34% within the first week postpartum and 55% within the first month. Generally the symptoms are similar to their typical pattern, although often milder and less often unilateral. It is rare for a migraine presentation to occur for the first time during the postpartum period. Recently there has been interest in the potential association between migraines and pregnancy induced hypertension. At this point in time there appears to be a
positive association which may reflect an underlying predisposition to cerebral ischemic injury\textsuperscript{11}

**POSTERIOR REVERSIBLE LEUKOENCEPHALOPATHY SYNDROME (PRES)**

PRES symptoms include headache, seizures, altered mental status, visual changes and occasionally, focal neurologic deficits\textsuperscript{12}. The neuroradiologic features of PRES include symmetric areas of cerebral edema, predominantly involving the white matter regions of the posterior circulation (occipital lobes, posterior parietal and temporal lobes). The pathophysiology of this disorder is similar to hypertensive encephalopathy, in that the cerebrovascular regulation is altered and with it loss of blood-brain integrity. The accompanying vasogenic edema can be reversed by prompt recognition and supportive therapy (cessation of provocative medications, aggressive treatment of hypertension and seizure prevention). However, irreversible cytotoxic edema with permanent neurological damage can occur if the initial disorder is not diagnosed early. This syndrome often presents in the postpartum period, usually following identification of pregnancy induced hypertension. Typical features distinguishing it from other causes of postpartum headache include associated accompanying seizures, and focal neurological deficits, such as temporary loss of vision\textsuperscript{13}

**MENINGITIS**

The severe headache of meningitis typically presents several days after delivery. It is accompanied by fever, nuchal rigidity, and positive Kernig and Brudzinski signs. Lethargy, confusion, vomiting, seizures and a skin rash also may occur. Usual pathogens include group B streptococcus, staphylococcus epidermidis, group A beta-hemolytic streptococcus, The diagnosis is confirmed by examination and culture of the CSF\textsuperscript{14}

**BRAIN TUMOR**

Postpartum headaches as a presenting feature of intracranial tumors has been reported\textsuperscript{15}The features of the headache may be dull rather than throbbing, and often do not have the noticeable improvement with supine positioning. The headache may occasionally be associated with nausea, vomiting, seizures, or focal signs. Neurologic examination may reveal evidence of increased intracranial pressure. In reviewing these case reports the atypical presentation of the headache, either with persisting headache symptoms in the supine
position, or exacerbation following epidural blood patch should prompt further neuroradiological investigations.\textsuperscript{16}

**PNEUMOCEPHALUS**
The subdural or subarachnoid injection of air used for identification of the epidural space may be associated with the sudden onset of severe headache, sometimes accompanied by neck pain, back pain, or changes in mental status.\textsuperscript{17} Headache symptoms can mimic postdural puncture headaches in that they are worse in the sitting position and may be relieved by lying down. Radiologic studies confirm the presence of intracranial air and the headache typically resolves over the first week.

**SINUSITIS**
Headache caused by inflamed paranasal sinuses is associated with purulent nasal discharge and occasionally fever. Pain may be unilateral or bilateral depending on the extent of the disease, and the skin over the affected sinus may be tender. Frontal sinus infection causes headache in the frontal region. Ethmoidal and sphenoidal sinus infections cause periorbital pain, and maxillary sinus infection may cause diffuse facial discomfort. The sinuses fill overnight, and pain typically is worse on awakening. Pain improves in the upright position, which assists drainage.\textsuperscript{18}

**CORTICAL VEIN THROMBOSIS**
Cerebral cortical vein thrombosis risk is increased in the pregnancy and has been estimated at an incidence between 10–20 per 100,000 deliveries in developed countries. The incidence appears higher in developing countries with an incidence of 450 per 100,000 deliveries in India.\textsuperscript{19} Its’ presentation can often be difficult to distinguish from postdural puncture headache, as it often has a postural component. Associated features may include focal neurological signs, seizures, and coma. Cerebral infarction may ensue if diagnosis is delayed. Diagnosis is best confirmed by Magnetic Resonance Imaging (MRI). Treatment of cortical vein thrombosis largely is symptomatic, with the aim of preventing seizures. Currently anticoagulation therapy is being evaluated, with observational and randomized trial studies indicating better outcomes.\textsuperscript{20}

**CEREBRAL INFARCTION/ISCHEMIA**
Cerebral arterial insufficiency is one of the causes of stroke in pregnancy and has an estimated incidence of 19 per 100,000 deliveries.\textsuperscript{21} Approximately half of the events occur in
the delivery and postpartum period, and the clinical presentation is often a woman with sudden onset of headache, vomiting, seizures and focal neurologic deficits. CT and MRI evaluations are often normal and require cranial doppler or angiographic investigations to diagnose the arterial ischemia or infarct.

**SUBDURAL HAEMATOMA**

According to the debut symptoms it may be hard to differentiate a subdural hematoma from PDPH, the most frequent benign complication of spinal anaesthesia which improves within a few days if treated with analgesics and bed rest.

Leakage of CSF from the dural hole is the presumed mechanism postulated for PDPH as well as for subdural hematoma. Loss of CSF is believed to lower both intraspinal and intracranial pressures resulting in a caudally directed movement of the spinal cord and brain. Stretching of the pain-sensitive structures and of the intracranial subdural bridging veins occurs. The sudden decrease in the CSF volume may also activate adenosine receptors, thus producing arterial and venous vasodilatation and subsequent clinical symptoms of PDPH. If the traction exerted on the bridging veins is substantial, it may cause a rupture at their weakest point, leading to hematoma formation²².

Early recognition of intracranial subdural bleeding is crucial to start an effective treatment. Failure to make an early diagnosis of subdural hematoma may result in fatal complications.

A subdural hematoma should be suspected when the headache becomes more severe and persistent, even in the recumbent position, in association with neurologic symptoms which include vomiting, blurring of vision, drowsiness, and disorientation. The occurrence of convulsions, diplopia, and high blood pressure after birth may erroneously be interpreted as eclamptic in the absence of an imaging evaluation. Other clinical conditions need also to be ruled out: migraine, meningitis, drug-induced headache (amphetamine and nifedipine), and intracranial pathologies²³ (sinus venous thrombosis, arteriovenous malformations, etc.)

Acute treatment with hypotensive drugs and magnesium sulphate, in case of misdiagnosed dural hematoma, may lead to failure in cerebral autoregulation. Therefore in any patient showing neurological symptoms after spinal or epidural anesthesia, a CT scan should be performed before starting treatment, in order to exclude intracranial bleeding.

Based on the interval between anaesthesia and the onset of symptoms, subdural hematomas may be acute and subacute/ chronic. Most reported acute cases develop within the first 2 days. While acute bleeding becomes rapidly symptomatic, subacute/ chronic subdural
bleeding may develop over a period of days or weeks, posing diagnostic problems. A subacute subdural hematoma may act as and be confused with PDPH, causing an initial orthostatic headache, responsive to analgesics, bed rest, and fluid replacement. With time these symptoms may go through alternate phases of improvements and exacerbations, loosing relation with posture and accompanied by neurological signs. According to published studies, interval between dural puncture and recognition of a chronic hematoma is 2 to 4 weeks. Since chronic subdural hematomas heal without sequelae if treated timely, a cranial CT is justified if a suspected PDPH does not respond to conservative therapy, increasing in severity or recurring after a pain-free interval. Concerning localization of bleeding, the hematoma may involve the frontal, parietal, and temporal regions (alone or in combination), and although more frequently unilateral, it is not unusual to observe a bilateral intracranial involvement.

Acute subdural hematomas are well recognized by a cranial CT scan, whereas chronic intracranial lesions need MRI or cerebral angiography as effective neuroimaging techniques since with time hematoma and surrounding brain tissue show similar radiographic density.

Treatment for subdural hematomas may be surgical or conservative: acute subdural hematomas often cause a rapid neurological deterioration which indicates a surgical evacuation of hematoma by craniotomy or burr holes to reduce the intracranial pressure and preserve brain function. A conservative approach has been recommended for patients with chronic hematoma without mental status changes nor seizure activity, absent intracranial mass effect, and when the hematoma is <1 cm in maximum thickness, causing a midline shift <5mm.

**Conclusion**

Postdural puncture headache is one of the major causes of headache in the postpartum period. Preventive measures for PDPH include the use of smaller gauge pencil-point needles for spinal blocks; epidural needles of 18 G or less, using saline rather than air for epidural space identification and the use of ultrasound guidance. When a patient complains of severe, persistent headache following regional anaesthesia unrelieved by conservative measures, one should consider the possibility of other causes of headache. Careful follow up is mandatory in order to come up with an early diagnosis and an appropriate treatment before major complications like death or irreversible neurologic damage occur.
References –


16. MacArthur A; Differential diagnosis of postpartum headaches; Revista Mexicana de Anestesiología; Vol. 32. Supl. 1, 2009, pp S16-S23


