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CASE REPORT

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Postpartum subdural hematoma following neuraxial anesthesia: A case report and review of literature

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Abstract
A healthy 34 year old primigravida delivered by cesarean section due to arrest of labor. Spinal anesthesia was performed without any problem via 25 Gauge Quincke needle between L3-L4 interspace. Postpartum first day, the patient had a post-dural puncture headache which eased by bed rest, aggressive intravenous hydration, caffeine and theophylline. Low molecular weight heparin therapy (enoxaparin 40 mg/day) was administered for prophylaxis of venous thromboembolism. On the sixth postoperative day, the patient referred again with violent headache. Magnetic resonance imaging confirmed subacute bilateral fronto-parieto-temporal subdural hematomas measured 3.5 mm on the left hemisphere and 5 mm on the right hemisphere. The case managed conservatively and recovered completely. Our report reviews 63 cases which complicated by postpartum subdural hematoma due to neuraxial anesthesia. Subdural hematoma should be remembered in the presence of resistant headache, additional neurological symptoms and predisposing factors since early diagnose and treatment is life-saving.

Keywords: Subdural hematoma, neuraxial anesthesia, labor, delivery, postpartum, cesarean section

Introduction

Neuraxial anesthesia is one of the indispensable elements in obstetrics practice. Spinal and epidural anesthesia and analgesia are widespreadly used for pain control during cesarean section and labor. The advantages of neuraxial anesthesia in cesarean delivery are absence of neonatal depression respiratory, less fetal neurodevelopmental disorders, less blood losses and better post-operative pain control [1]. However, it is not completely risk-free method for labor analgesia. Several complications may occur intrapartum or postpartum including hypotension (most common, 30-40%), failed block (4.7-15.4%), localized pain (such as backache, 18-19%), post-dural puncture headache (PDPH, 10-40%), neck stiffness, bradycardia, fever, decreased cardiac output, fetal bradycardia, fetal acidosis, itching, nausea, vomiting, Horner's syndrome, cranial nerve deficits, convulsion, toxicity, anaphylactic reactions, seizure, cardiac arrest, spinal hematoma, spinal cord injury, sensory loss, paraplegia, persistent neuropathy, transient neurologic symptoms, high spinal (1%), epidural abscess, puncture site infection, meningitis, intracranial hypotension and intracranial (epidural, subdural or subarachnoid) bleeding [1,2]. We present a case of subacute subdural hematoma (SDH) after cesarean delivery with spinal anesthesia and discuss the literature of subdural hematomas after neuraxial labor anesthesia.

Case Report

A healthful 34 year old primigravida was admitted for labor induction due to oligohydramniyos on the 39th week of gestation. She was administered by dinoprostone, slow release of prostaglandin E2 (Propess) vaginally. The next day, cesarean section decision was made due to arrest of labor. The process of the spinal anesthesia (2 ml of hyperbaric bupivacaine, 25 Gauge Quincke needle, interspace L3-L4, no paraesthesia during the puncture and clear cerebrospinal fluid) was normal. The operation was performed without problems with the birth of a 3360 g male without abnormalities and 10 Apgar score at the 5th minute. For prophylaxis of venous thromboembolism, enoxaparin 40 mg/day was started. Twenty four hours after the delivery, the patient described of a postural headache which her anesthesiologist recommended bed rest, aggressive intravenous hydration, caffeine and theophylline. There were no abnormal findings in her neurological examination. The parturient was discharged on the second postpartum day since pain relief. On the sixth day of postpartum, the parturient presented to the emergency department of our hospital with a serious headache that did not diminish

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with supine position. Her neurological examination showed no problem. Magnetic resonance imaging showed bilateral fronto-parieto-temporal subdural hematomas measured 3.5 mm on the left hemisphere and 5 mm on the right hemisphere (Figure 1a). There was neither associated mass effect nor midline shift. Her neurologist and neurosurgeon suggested a conservative treatment including bed rest, aggressive intravenous hydration, caffeine and theophylline. Enoxaparin treatment was stopped due to SDH however wearing of anti embolic stockings was continued. Her headache was completely passed on the thirteenth day of postpartum. Minimal resolution of bilateral fronto-parieto-temporal SDH (measuring 2.5 mm in thickness on the left and 4.7 mm thickness on the right side) was observed on MRI (Figure 1b). The patient was discharged on fourteenth day of postpartum. Following week, her examination and MRI was normal (Figure 1c).

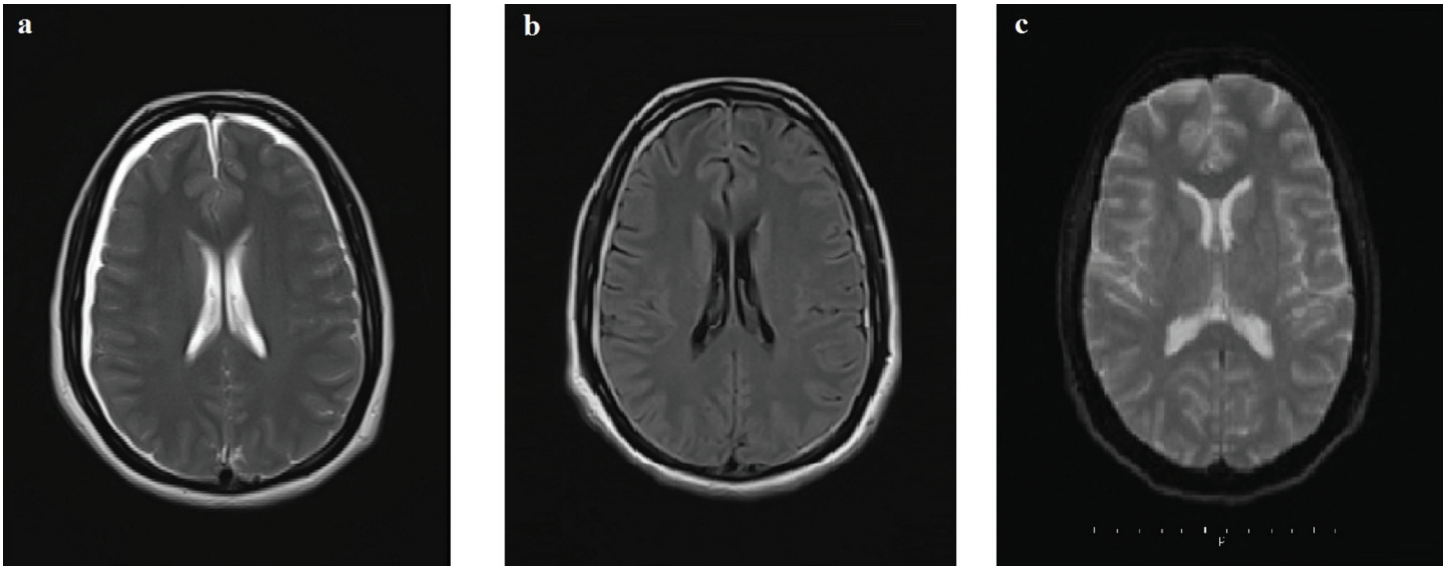

Figure 1. MRI images of the case on the sixth (a), thirteenth (b) and twenty-second (c) day of postpartum

Table 1. Patient Characteristics

		n (%)	p
Age (year)	Min-Max (Median)	19–75 (36)	
	Average ± SD	46.2 ± 13.8	
Laparoscopic cholecystectomy	Min-Max (Median)	19–75 (43)0.08	0.08
	Average ± SD	45.6 ± 14	
Conversion to open cholecystectomy	Min-Max (Median)	32–69 (51)	
	Average ± SD	52.3 ± 11	
Gender			
Laparoscopic cholecystectomy	Female	105 (89.7)	0.15
	Male	15 (10.3)	
Conversion to open cholecystectomy	Female	9 (64)	
	Male	5 (36)	
BMI (kg/m^2)			
Laparoscopic cholecystectomy	Min-Max (Median)	19.4- 39.84 (27.8)	0.08
	Average ± SD	25.85±1.62	
Conversion to open cholecystectomy	Min-Max (Median)	23.6-39.6 (31.3)	
	Average ± SD	27.8±4.83	
Time since first complain (days)			
Laparoscopic cholecystectomy	Average ± SD (Min-Max)	30±21.5 (1-120)	0.26
Conversion to open cholecystectomy	Average ± SD (Min-Max)	23.8±42.8 (1-120)	
Dominant presenting Symptoms	Pain at the upper abdomen	79 (58.9)	
	Food intolerance	27 (20.1)	
	Nausea/ Vomiting	18 (13.4)	
	Pain at the right shoulder	10 (7.4)	
Hospitalization (day)			
Laparoscopic cholecystectomy	Average ± SD (Min-Max)	1.35±0.32 (1–3)	[a]0.01*
Conversion to open cholecystectomy	Average ± SD (Min-Max)	5.15±1.99 (3-9)	

[a]Student's t-test, [b]Mann–Whitney U Test, [c]Fisher's Exact Test, *p <0.05, ** p<0.001

Table 2. Comparison of ultrasonography features of laparoscopic cholecystectomy and Conversion to open cholecystectomy groups

		Laparoscopic Cholecystectomy n (%)	Conversion to Open Cholecystectomy n (%)	p
The diameter of the gallstone(s)	>1 centimeter	63 (52.5)	6 (42.8)	[c]0.57
	< 1 centimeter	57 (47.5)	8 (57.2)	
Ultrasonography features	Only Gallstone	118 (98.4)	12 (74.4)	[c]0.0002**
	Gallbladder polyp	2 (1.6)	2 (15.6)	
Size of the gallbladder	Normal	108 (90)	6 (42.8)	[c]0.0001**
	Hydropic	12 (10)	8 (57.2)	
Gallbladder wall thickness	<3 millimeter	101 (84.1)	7 (50)	[c]0.0006**
	≥3 millimeters	19 (15.9)	7 (50)	

[a]Student's t-test, [b]Mann–Whitney U Test, [c]Fisher's Exact Test, *p <0.05, ** p<0.001

Table 3. Comparison of ultrasonography features of laparoscopic cholecystectomy and conversion to open cholecystectomy groups

	Laparoscopic Cholecystectomy n (%)	Conversion to Open Cholecystectomy n (%)	p
C-reactive protein > 5mg/dl	5 (4.1)	4 (28.5)	[c]0.02*
White blood cell > 10200x103/μL	8 (6.6)	4 (28.5)	[c]0.01*
Neutrophil > 80%	7 (5.8)	3 (21.4)	[c]0.08
Lymphocyte > 50%	1 (0.8)	0 (0)	[c]1
RDW• 17.2%	24 (20)	3 (21.4)	[c] 0.5

[a]Student's t-test, [b]Mann–Whitney U Test, [c]Fisher's Exact Test, *p <0.05, ** p<0.001, • Red cell distribution width

Discussion

SDH is known as bleeding between the dura mater and arachnoid mater due to head trauma, coagulopathy, cerebral aneurysm, arteriovenous malformation, tumors, cerebral hypotension, chronic alcoholism, cardiovascular disease, or diabetes mellitus. It was first defined by Virchow, in 1857, as an internal hemorrhagic pachymeningitis. Then in 1914, Trotter described that SDH was owing to the traumatic injury of bridging veins. Symptoms vary according to size, localization, and development rate of SDH. Typical clinical symptoms include headache, nausea, vomiting, decreased level of consciousness, contralateral hemiparesis, and ipsilateral dilated pupil. SDH after neuraxial anesthesia is a very unusual but critical complication. Prevalence of SDH after spinal anesthesia is extremely low, approximately 1/220000 – 1/1000000 [3,4]. To the best of our knowledge, there are less than 100 cases of SDH due to labor anesthesia. 63% of those were due to epidural, 29% of those were due to spinal and 8% of those were due to combined spinal-epidural (CSE) anesthesia.

Pathophysiology of PDPH and SDH seem to be similar. Moreover, PDPH and intracranial hipotension syndrome (eye tearing and fifth nevre Palsy) may be considered as predisposing factors for SDH. Cerebrospinal fluid (CSF) leakage from the punctured dura may reduce the intracranial pressure and induce caudal displacement of brain and medulla spinalis. This movement is leading to headache, stretching and tearing of bridging cerebral vessels (particularly the cerebral veins and venules). In addition, intracranial hypotension due to CSF leakage could have been aggravated by pushing or operative vaginal delivery. As the walls of bridging veins are thinner in the subdural space, they are more susceptible to tearing in this region. Thus, the source of bleeding may be small artery in subarachnoid tissue or cortical veins in more serious cases. Furthermore, intracranial vessels may be more prone to stretching and tearing due to venous dilatation during pregnancy. Intracranial hypotension due to CSF leakage or intracranial hypertension due to anesthetic and surgical stress may cause rupture if there is an underlying cerebral aneurysm or arteriovenous malformation. Cerebral subdural hematomas and spontaneous arachnoid rupture may also occur spontaneously in pregnant women due to sudden intracranial pressure drop immediately after the Valsalva manoeuvre at labor [5].

Pregnant women performed neuroaxial anesthesia are at increased risk for SDH due to peripartum dehydration which could decrease the amount of CSF, postpartum diuresis, instantaneous reduce of intra-abdominal pressure, venacaval pressure at delivery, early ambulation, apprehension about labor and hormonally-induced ligamentous changes [6].

Predisposing factors for the development of SDH in obstetric anesthesia are multiple dural punctures, large dural hole, excessive CSF leakage originating from dural puncture(s), trauma, cerebral atrophy, anticoagulant use, platelet dysfunction, herbal medications, bleeding disorders, cerebral aneurysm, brain tumor, cerebrovascular events, meningovascular syphilis, dehydration and arteriovenous malformation. The recommended bed rest in PDPH treatment is leading immobilization. This immobilization further increases the risk of venous thromboembolism after cesarean delivery thus low molecular weight heparin (LMWH) therapy is indicated. However, here a dilemma occurs. The risk of development of SDH due to PDPH should be taken into account before administering LMWH therapy. 12% of patients with epidural anesthesia, 16% of patients with spinal anesthesia, 60% of patients with CSE anesthesia had platelet dysfunction or anticoagulant use. My patient had been administered LMWH (enoxaparin sodium 40 mg/day) due to increased risk of venous thromboembolism and she was complicated by a SDH. I recommend that antiplatelet and anticoagulation therapy be initiated after confirming that the patient has no PDPH after neuraxial anesthesia.

The correlation between needle size and risk of PDPH has been known for many years [7]. The needles used by a majority of ranged in size from 16 to 25 gauge. The hole in the dura mater is small when 27 or 29 gauge needles are used. SDH may develop

acutely or chronically owing to a small spinal needle, such as 27 gauge. PDPH is rarely seen due to small – size needles with a pencil – point tip.[8] Whitacre and Sprote are examples of pencil – point needles. The Quincke has a cutting tip and a higher incidence of PDPH.[1] The healing of the puncture in the dura mater is more difficult with a cutting tip. Puncture needle should be placed on the length of the spine at a parallel angle since this technique decreases the incidence of PDPH and CSF outflow. The incidence and severity of PDPH after ADP by a Tuohy needle is higher than the incidence of PDPH after spinal anesthesia.[9] ADP is observed in 58% of SDH patients with epidural anesthesia. Multiple dural puncture is present in 11% of SDH patients with spinal anesthesia. Advancement the epidural catheter into the subarachnoid space (modifying to a macrocatheter continuous spinal technique), injection of CSF from the epidural syringe back into the spinal space through the epidural needle, intrathecal injection of normal saline (10 mL), performing epidural blood patch (EBP) and prophylactic treatment of ondansetron may forestall formation of PDPH and subdural hematoma due to CSF leakage of ADP. [10] On the other hand, 33% of all postpartum SDH cases, 56% of SDH patients with spinal anesthesia and 25% of SDH patients with epidural anesthesia has no risk factors.

The incidence of subdural hematoma due to neuraxial anesthesia in postpartum period is not known precisely because it is managed as PDPH if there is no neurological symptom. The incidence of PDPH increases to 51% in pregnant women particularly complicated by ADP with a large epidural needle. A typical PDPH is aggravated in the vertical position, located in the frontal or occipital region, relieved by the supine position and associated with neck stiffness, photophobia, tinnitus, diplopia, cranial nevre palcies [1]. According to the diagnostic criteria of the International Headache Society (2004, ICHD-II), the pain worsens or develops within 15 minutes after the individual sits or stands up, and it improves within a similar period after the individual lays down [11]. It develops within 5 days after the puncture and disappears spontaneously within 1 week or up to 48 hours after epidural blood patch. Of note, PDPH rarely exceeds 2 weeks and does not respond to EBP. PDPH can mimic or mask other underlying causes. Bedrest, aggressive intravenous hydration, cerebral vasoconstrictor drugs (caffeine, sumatriptan), butalbital, acetaminophen, vasopressin, theophylline, and adrenocorticotropic hormone, greater occipital nerve block, sphenopalatine ganglion block and EBP are the treatment options for PDPH [2]. In the differential diagnosis of persistent PDPH following treatment, the following should be considered: hypertension, pre-eklampsi, eklampsi, posterior reversible encephalopathy syndrome, lactation headache, migraine, cortical vein/dural sinus thrombosis, intracranial (epidural, subdural or subarachnoid) bleeding, reversible cerebral vasoconstriction syndrome, cerebral aneurysm, arteriovenous malformation, stroke, tumour, sinusitis/meningitis/encephalitis, local anaesthetic toxicity, drug-induced headaches, pneumocephalus, dehydration and caffeine withdrawal [1,2]. The findings of intracranial pressure increase due to subdural hemorrhage follows headache due to intracranial hypotension and may occur acutely or chronically. Warning signs are nonpostural headache, alterations in headache characteristics (such as resistant headache, retroorbital or frontal headache, temporal disappearance of headache), nausea, vomiting, any neurological symptoms, personality changes, neck stiffness, tinnitus, and cardiac arrest. Almost all postpartum SDH patients (97%) complain of headache. Headache may be seen alone or accompanied by other symptoms. Common symptoms except headache are following: nausea and vomiting with 24%, hemiparesis with 16%, confusion and coma with 14%, seizure with 13%, blurred vision and vision loss with 13%, disorientation with 11%, neck stiffness with 10% , tinnitus with 6%, cardiac arrest with 3%.

The duration of hematoma may be divided into three groups: (1) acute (first 3 days); (2) subacute (3-14 days); and (3) chronic (≥2 weeks). Postpartum SDH tends to be chronic (41%). The ratio of acute and subacute SDHs are similar. (31% and 33%, respectively.) Various combinations of these are observed approximately 10%. Clinical evaluation, computerized tomography (CT) and magnetic resonance imaging (MRI) can be used for diagnosis [12]. CT and MRI have advantages and disadvantages when being compared. CT is a safe, easy, fast, more accessible, and low-cost method of detecting subdural hematomas [12]. An isodense SDH can make diagnosis harder in tomography. Therefore, if CT is normal and clinical doubt persists, contrast-enhanced CT or MRI may be required. MRI is more sensitive than CT in determining the location and size of SDH [13]. SDHs are mostly bilateral hemispheric (42%). These are followed by left hemispheric (27%), right hemispheric (21%), and parafalcine (10%) SDHs.

Treatment of the SDH in postpartum period includes conservative (medical observation, anti-oedema medication), EBP or surgical (intracranial pressure monitoring, external ventricular CSF drainage, single or multiple burr-hole drainage,and craniotomy/craniectomy) techniques [12]. The efficacy of EBP treatment in reducing pain ranges from 60% to 90%. EBP has complications such as bleeding, infection, nerve injury, intracranial hypertension, repeat dural puncture, repeat failure, meningitis, arachnoiditis, and cauda equine syndrome. If second blood patch is required, success rate is up to 97%. EBP acts by blocking the CSF leak from the hole in the dura mater and increasing the CSF flow from the lumbar region to the brain. Moreover, it is thought that rapid effectiveness of EBP is due to cerebral vasoconstriction and an increase in CSF pressure. If PDPH occurs in patients dural puncture with a large needle, EBP should be performed urgently before development of SDH. Hematomas with a depth of less than 5-10 mm tend to recover spontaneously. Surgery may be inevitable if occurring a SDH >10 mm, a midline shift >5 mm, diffuse oedema, cerebral herniation or severe neurological symptoms due to brain compression [14]. Approximately 50% of postpartum SDH cases require surgery. 82% of patients with midline shift or brain compression are treated surgically. Preoperative good neurological condition, young age and early diagnosis correlates with the satisfying postoperative outcome in cases requiring surgery [15]. Although the majority of cases (86%) recover without any sequelae, SDH may result in permanent neurological deficit (8%) and death (6%) if there is a lateness in diagnosis and treatment.

In summary, we presented a parturient which was complicated with subacute SDH with persistent headache after treatment of PDPH under LMWH therapy. Postpartum SDH is an unusual but critical complication. Pregnancy itself is considered as a risk factor. SDH should be taken into account in the presence of an additional predisposing factor and warning signs since rapid diagnose and treatment is life-saving.

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