A case report on intracranial subdural hematoma following spinal anesthesia: A rare complication of a common procedure

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Case Report

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Abstract

Introduction: Intracranial subdural hematoma is a rare complication of lumbar puncture and spinal anesthesia. The presenting symptoms mimic other benign conditions such as post dural puncture headache which often led to mis-and delayed-diagnoses. With high degree of suspicion and proper intervention fatal outcomes can be avoided.

Case presentation: A 36-year-old HIV+ primigravida mother underwent a cesarean section under spinal anesthesia for an indication of non-reassuring fetal heart rate two weeks back. After the procedure, she reported mild occipital headache which was precipitated by upright postural. With a diagnosis of post dural puncture headache, conservative management was started and her headache subsequently improved. On day four, both the mother and her baby discharged home. A week later the patient developed occipital headache which gradually worsen and become unremitting with associated diplopia, blurred vision, neck pain and vomiting which forced her to visit the emergency department at Tikur Anbessa Specialized Hospital. Except moderate nuchal rigidity her physical examination was unremarkable. Brain imaging was requested and revealed bilateral acute and subacute subdural hematoma without mass effect. After neurosurgical consultation, conservative management with bed rest, hydration and oral analgesics was initiated. On day ten her headache and other symptoms significantly improved. She was discharged with outpatient follow-up.

Conclusion: Clinicians should be vigilant about the evolving nature of post dural puncture headache and the possibility of intracranial subdural hematoma when patients presented with persistent or worsening headache which becomes non-positional following dural puncture and spinal anesthesia. Early detection and proper intervention are imperative to avoid permanent disability or mortality.

Introduction

Being an alternative to general anesthesia, spinal anesthesia has been preferred in a standard obstetrics procedure with multiple benefits. Lumbar puncture for spinal anesthesia has been associated with a well-documented complications such as low back pain, headache, radiculopathy, and infection. However, serious, life-threatening complications such as subdural hematoma (SDH) and other intracranial hemorrhages are rare occurrences with an incidence of 1 in 500,000 to 1,000,000 (1). Due to symptomatic mimicry and lack of hallmark clinical feature, post dural puncture and spinal anesthesia associated SDH is often under-detected and misdiagnosed as a benign headache by most clinicians (2). Therefore, high index of clinical suspicion and early recognition of the warning signs will enable health providers to promptly diagnose and timely intervene which prevents catastrophic outcomes. In this case report, we aimed to discuss about a HIV positive mother from Ethiopia who was diagnosed with intracranial subdural hematoma presented as a delayed complication of dural puncture and spinal anesthesia performed for cesarean section two weeks prior to her presentation.

Case Presentation
A 36-year-old right-handed primigravida mother who was diagnosed with HIV infection three month into her pregnancy with baseline CD4 count of 560 cells/mm$^3$ was immediately started on highly active antiretroviral therapy (HAART). Her prenatal period was smooth. At 40th weeks of gestational age she went into spontaneous labor but due to non-reassuring fetal heart rate her obstetrician performed caesarian section under a spinal anesthesia. The outcome was a healthy male neonate weighing 3250 grams. On the 1st postpartum day, the mother started complaining of new onset mild global headache which worsen with upright position and the diagnoses of post dural puncture headache was considered. Conservative management was started including bed rest, oral rehydration, and oral analgesic (paracetamol) and her headache resolved by day four. The mother and her neonate discharged home in a stable condition. One week after discharge she started to have an insidious onset mild occipital headache which gradually becomes more intense and non-positional that interfere with her activity of daily living. In the morning of her hospital admission, she experienced severe non remitting bilateral frontal and occipital headache associated by blurred vision, diplopia, neck pain and two episodes of non-projectile vomiting. Otherwise, she denied any trauma, limb weakness, gait imbalance, dizziness, auditory disturbance, loss of consciousness or prior headache disorders. She has no personal or family history of headache disorder, bleeding tendency, thrombotic events, joint pain, skin lesion or similar illness. She denied use of medications such as anticoagulants and antiplatelets. She has been married for 5 years and work as a secretary.

On physical examination, her vital signs were stable and her general exams were unremarkable. On neurologic exam, she was conscious and communicating well. Has no papilledema, ophthalmoplegia or other cranial nerve weakness. On motor exam power, muscle stretch reflex and sensory exam were intact. She had moderate nuchal rigidity. Her laboratory results were nonrevealing (Table 1). Brain computerized tomography (CT) scan with and without contrast showed bilateral fronto-parieto-occipital crescent shaped hyperdense collections with minimal adjacent sulci effacement suggestive of bilateral acute to subacute subdural hematoma (Figure 1).

Neurosurgery unit was consulted but surgical intervention was differed as the hematoma is neither expansile nor it cause any focal neurologic deficit. Conservative management was opted and patient was transferred to the neurology ward for supportive care including bed rest, combination analgesics (acetaminophen-caffeine), oral rehydration, enteral feeding, physical therapy and frequent follow up for new onset seizure, focal neurologic deficit and any other acute clinical deteriorations. Subsequently her headache and other symptoms got better and she was discharged improved with oral analgesia after ten days of hospitalization with an appointment to the outpatient headache clinic. Two weeks later she came for follow up and all her symptoms resolved completely at this time.

**Table 1:** Patient’s laboratory test results with normal reference values
<table>
<thead>
<tr>
<th>Variables</th>
<th>Patient result</th>
<th>Normal reference values</th>
</tr>
</thead>
<tbody>
<tr>
<td>White blood cells (WBC)</td>
<td>8, 200 (N 76%, L 11.8%)</td>
<td>5000 – 11,000 cells/mL</td>
</tr>
<tr>
<td>Hemoglobin (Hgb)</td>
<td>17.4 g/dL</td>
<td>14 – 16 g/dL</td>
</tr>
<tr>
<td>Mean corpuscular volume (MCV)</td>
<td>83.1 fL</td>
<td>80-99 fL</td>
</tr>
<tr>
<td>Platelets</td>
<td>423,000 cells/mL</td>
<td>150,000 -450,000 cells/mL</td>
</tr>
<tr>
<td>Creatinine</td>
<td>0.4 mg/dL</td>
<td>0.5-1.2 mg/dL</td>
</tr>
<tr>
<td>Blood urea nitrogen</td>
<td>21 mg/dL</td>
<td>5-18 mg/dL</td>
</tr>
<tr>
<td>Alanine aminotransferase</td>
<td>17 IU/L</td>
<td>10-59 U/L</td>
</tr>
<tr>
<td>Aspartate aminotransferase</td>
<td>16 IU/L</td>
<td>10-40 U/L</td>
</tr>
<tr>
<td>Alkaline phosphatase</td>
<td>70 IU/L</td>
<td>20-140 U/L</td>
</tr>
<tr>
<td>Sodium</td>
<td>140 mmol/L</td>
<td>135-146 mmol/L</td>
</tr>
<tr>
<td>Potassium</td>
<td>3.68 mmol/L</td>
<td>3.5-4.5 mmol/L</td>
</tr>
<tr>
<td>Chloride</td>
<td>97 mmol/L</td>
<td>96-106 mmol/L</td>
</tr>
<tr>
<td>Prothrombin time (PT)</td>
<td>15.5 sec</td>
<td>12.0 -14.0 sec</td>
</tr>
<tr>
<td>Partial thromboplastin time (PTT)</td>
<td>23.6 sec</td>
<td>20-35 sec</td>
</tr>
<tr>
<td>INR</td>
<td>1.23</td>
<td>0.8-1.3</td>
</tr>
<tr>
<td>Total bilirubin</td>
<td>0.5 mg/dL</td>
<td>0.2-1.3 mg/dL</td>
</tr>
<tr>
<td>Direct bilirubin</td>
<td>0.08 mg/dL</td>
<td>0.0-0.3 mg/dL</td>
</tr>
<tr>
<td>Albumin</td>
<td>4.2 g/dL</td>
<td>3.5-5.0 g/dL</td>
</tr>
<tr>
<td>HBsAg</td>
<td>Negative</td>
<td>-</td>
</tr>
<tr>
<td>Anti HCV serology</td>
<td>Negative</td>
<td>-</td>
</tr>
</tbody>
</table>

**Discussion And Conclusions**

The lumbar puncture (LP) procedure was developed by Wynter and Quincke in 1891, who aspirated cerebrospinal fluid (CSF) from the subarachnoid space of a tuberculous meningitis patient for the treatment of raised intracranial pressure (3). Later, in 1898 a German surgeon, Karl August Bier, became the first person to inject spinal anesthesia into the SAS of his patients, his assistant and himself. Bier had a first-hand experience of post dural puncture headache (PDPH) where he attributed his symptoms to the loss of CSF (4).

LP is a common procedure performed by puncturing of the dura/arachnoid mater either for diagnostic or therapeutic purposes. One of the most common complications of LP is PDPH, which is caused by excess
CSF leakage and downward displacement of the intracranial structures with stretching of intracranial pain sensitive structures. Although 66% to 90% of PDPH occur within the first 48 to 72 hours of the procedure, it usually subsides in few days with supportive care such as bed rest and antipain (5).

Intracranial SDH is a rare but potentially fatal complication following LP and spinal anesthesia. The pathogenesis of intracranial SDH and PDPH overlaps. Beside the traction of pain sensitive intracranial structures, the sagging of brain following CSF hypotension led to traction on arachnoid mater and dural veins. This causes tear to thin walled vessels with blood extravasation and formation of SDH (2). Furthermore, the sudden drop in CSF volume may activate adenosine receptors which causes arterial and venous vasodilation and subsequent development of PDPH (6).

According to the international classification of headache disorders (ICHD-3) (7), the diagnosis of PDPH includes headache develops within 5 days of LP and accompanied by neck stiffness and/or subjective hearing symptoms. The pain remits spontaneously within 2 weeks or after sealing the leak with autologous epidural patch. However, if the headache persisted or failed to respond to conservative treatments, it is a red flag sign for a more serious cerebral lesion (3). Our case illustrates that headache that persisted or worsen following the diagnosis of PDPH necessitate emergency evaluation to rule out life threatening conditions where a high index of suspicion and early recognition of warning signs will save life.

Prior studies (8,9) identified several risk factors for intracranial SDH following spinal anesthesia which includes dehydration, pregnancy, use of anticoagulants, multiple dural punctures, large dural hole, cerebrovascular abnormalities, and brain atrophy. Other factors that might contribute to SDH but not related to LP including head trauma, tumors, cerebral aneurysm, coagulopathy, arteriovenous malformation, chronic alcoholism, and diabetes mellitus might coexist. Although HIV induced coagulopathy, vasculopathy, brain atrophy and platelet dysfunctions has been linked with spontaneous SDH (10), in our patient her recent pregnancy and LP procedure potentially explain her SDH diagnosis. Generally, in young patient with SDH screening for potential risk factors will avoid recurrence and grave prognosis.

The diagnosis of SDH is confirmed with neuroimaging studies such as CT scan or magnetic resonance imaging (MRI). If the hematoma is <1 cm in thickness, the midline shift is <5 mm without mass effect and no focal neurologic deficits warrants conservative treatment as we presented it in our case. However, acute SDH with rapid neurological deterioration or chronic SDH with mass effect requires urgent surgical evacuation of hematoma by craniotomy to relieve the intracranial pressure (11).

In conclusion, clinicians should be vigilant about the evolving nature of post dural puncture headache and the possibility of a rare and fatal complication, subdural hematoma. The clinical presentation between PDPH and SDH often overlap however change in headache pattern, failure to respond to conservative treatment and development of neurological signs prompt clinicians to carefully assess patients for possible SDH. Early detection and proper intervention are imperative to avoid permanent disability or mortality.
Abbreviations


Declarations

Ethics approval and consent to participate

Authors’ institution does not require ethical approval for publication of a case report.

Written informed consent was obtained from the patient.

Consent for publication

A written informed consent was obtained from the patient for publication of this case report and any accompanying images. A copy of the signed consent form is available on request.

Availability of data and materials:

All data generated during this report are included in this manuscript

Competing interests

The authors declares that they have no competing interests.

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Author’s contributions

YZZ conceived the idea of reporting the case. KA, BG and YZZ drafted the manuscript and collected the images, interpretation, preparation of the manuscript and collecting the pictures. All authors were involved in the clinical management of the patient.

YZZ contributed to the review and editing of the manuscript. All authors read and approved the final manuscript.

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**References**


**Figures**

**Figure 1**

Axial (A) and Coronal (B) non contrast brain CT scan showed crescent shaped hyperdense collections in bilateral frontal, parietal and occipital lobes with minimal effacement of adjacent sulci.