

## Chronic Subdural Hematoma Complicated Spinal Anesthesia: Report and Pilot Study of Five Surgically Treated Cases

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### Abstract

**Background:** Spinal anesthesia (SA) is generally considered a simple and safe procedure, but post spinal headache is a very common symptom appears later SA. Appearance of cranial subdural hematoma after SA is a very rare but life-threatening complication state. PDPH is the primary symptom of hematoma formation, and it should be regarded as a warning sign. Differentiation between subdural hematoma and PDPH may be difficult. Substitutional diagnoses for PDPH should be considered whenever the headache is severe, prolonged or not related to patient position. PDPH should be investigated immediately to exclude any intracranial complications.

**Objective:** The objectives of this study are to increase awareness of hemorrhagic complications following SA and assess post-SA headache to encourage early diagnosis and management. Methods: The study was a pilot study of five cases. Four cases involved postpartum patients after SA who developed small to large subdural hematomas, as assessed by full neurological examination and neurological studies, and one case involved an elderly patient with a stone ureter.

**Results:** One patient was managed conservatively, and the other four patients improved after surgical evacuation.

**Conclusions:** Alternative diagnoses to post spinal headache must be considered for severe, prolonged headaches that are not related to patient position and do not respond to bed rest and analgesia. Early assessment of PDPH and early neuroradiological evaluation are recommended to diagnose post-SA subdural hematoma for early intervention to prevent avoidable morbidity and mortality.

**Keywords:** Spinal anesthesia; Subdural hematoma; Post-dural puncture headache

### Introduction

Spinal analgesics and local anesthetics are putted into the subarachnoid space by using a sterile technique. The procedure is counted a safe and practical substitutional to general anesthesia when the surgical sites are located in the lower limbs, perineum, or lower abdominal wall [1]. Post spinal headache is a well-known drawbacks and side effect of spinal anesthesia (SA) that occurs in up to 40% of cases. Classic PDPH occurs with changes in position (i.e., posture-dependent), produces mild pain and generally responds to increased fluid intake, bed rest, and analgesics, and it is self-limited. These symptoms are likely due to a loss of cerebrospinal fluid (CSF) through the puncture site followed by a decrease in the intracranial pressure, which leads to stretching and laceration of subdural veins, even in healthy one [2]. Subsidiary factors in the development of this pathology may be the Valsalva maneuver [3], brain atrophy [4] or thrombocytopenia together with the possible thrombocytopenia that occurs in severe preeclamptic toxemia [5].

Subdural hematoma or intracerebral hemorrhage rarely underlies severe PDPH [5-9]. Chronic subdural hematomas (CSDH) primarily

occur in elderly patients and head trauma is identified in up to 50% of these cases (sometimes rather minor trauma). Other risk factors include alcohol abuse, coagulopathies (including therapeutic anticoagulation), seizures and CSF shunts [10]. Most authors agree that the true specific incidence of subdural hematoma (SDH) after spinal anesthesia is not known, and numerous cases not discovered and go unreported. This phenomenon occurs for the acute, acute-on-chronic, subacute, and chronic forms [11]. Many CSDH cases likely begin as acute subdural hemorrhage. Blood inside the subdural space stimulates an inflammatory response. Fibroblasts infiltrate the clot within several days and form neomembranes on the inner (cortical) and outer (dural) surface. This sequence is followed by neocapillary proliferation, enzymatic fibrinolysis, and liquefaction of the blood clot. Fibrin degradation products are reincorporated into new clots, which inhibit hemostasis. The balance of plasma effusion and/or rebleeding from the neomembranes and fluid reabsorption establishes the course of CSDH [12,13].

PDPH associated with CSDH is characterized by prolonged post-puncture cephalalgia, and it may develop and aggravate critical neurological dysfunction. Differentiation between PDPH and subdural hematoma headache is difficult. This case report increases awareness of these complications by describing five cases of CSDH following SA with early management to prevent avoidable morbidity and mortality.

## Patients and Methods

The study was a pilot study. The hospital ethical committee approved this study, and written informed consent was acquired from each patient. We investigated five cases of cranial subdural hematoma in patients who underwent SA. Four patients received SA for cesarean sections, and one patient received SA for a stone ureter. The age range of the four cesarean section patients was 20 to 35 years. The male urological patient was 65 years old. The primary complaint in all cases was headache. One patient developed abducent nerve palsy, and another developed hemiparesis. All symptoms presented after SA. Management was conservative or surgical. Patients received an initial treatment for post-SA headache of bed rest, fluids and analgesics. Surgical evacuation was mandatory for patients who midline shifts, exhibited no or poor response. Four patients underwent surgical evacuation, and one patient was treated conservatively in the Department of Neurosurgery, Aswan University Hospital from May 2014 to May 2017.

## Patient evaluations

Patients with persistent symptoms that did not respond to medical treatment post-SA were readmitted for a clinical evaluation that included thorough medical, neurological, and ophthalmological examinations. Complete blood count, prothrombin time and concentration, international ratio (INR) and hepatic function were measured to assess whether the coagulopathy was within a normal value. Symptoms of elevated intracranial pressure were assessed for all patients using the same questionnaire for headache, vomiting, visual impairment, and pulsatile tinnitus. Ophthalmic assessment included fundus examination and visual field testing. The presenting clinical manifestations were recorded.

## Diagnostic modalities

All patients were examined using without contrast-enhanced brain computed tomography (CT) scans and without contrast-enhanced brain magnetic resonance imaging (MRI). Imaging revealed subacute or chronic subdural hematoma with or without midline shift. Surgery: Surgical treatment of CSDH included the creation of two burr holes and irrigation with tepid saline until the fluid was clear.

## Follow-up

Clinical follow-up was performed for headache, hemiparesis and abducent nerve palsy.

## Results

Four patients who developed chronic subdural hemorrhage after SA underwent surgical evacuation, and one case was conservatively managed. All cases improved clinically as manifested by increased intracranial tension.

A 35-year-old woman was brought to our emergency department (ED) suffering from headache and numbness in her cheek and upper right arm. She received an elective cesarean section with spinal anesthesia in our hospital 28 days prior to presentation to our ED. The headache was diffuse with vague characteristics. This patient's pregnancy was normal and well controlled. She had no history of hypertension, blood disease, infection, neurological disease, migraine, malignancy, head trauma, coagulation abnormalities or anti-coagulant taken drugs. The spinal anesthesia was administered *via* puncture of

the L4-L5 space with a 22-gauge spinal needle and injection of 12.5 mg of 0.5% heavy bupivacaine, as described in the patient's record. The puncture was successful on the first attempt, and the course of anesthesia was without incident. Her intraoperative vital signs were normal, and the surgery was completed uneventfully. Full recovery was achieved, and she was discharged two days after the surgery. She had no headache at the time of discharge. The patient developed a mild diffuse headache 7 to 10 days later that did not subside with analgesia and bed rest. The severity of the headache gradually increased, and numbness developed in the left upper limb and cheek, at which time the patient was brought to the ED complaining of an intense generalized headache that was present at all times and worsened upon sitting upright. She was conscious and oriented. Fundus examination was normal. She had no neck rigidity or Kerning's sign. The motor force of limbs was 5/5, her BP was 120/80 mmHg, and no fever was detected. Laboratory data revealed normal white blood cell count, hemoglobin, platelet count, PT, PTT and INR. CT scan was performed and revealed a hypo-dense crescent-shaped space-occupying lesion in the left parietal lobe (Figure 1). The diagnosis was confirmed with an MRI, which showed hyper-intensity in the subdural left parietal lobe. The patient was admitted to the neurosurgery ward after emergent consultation with a neurosurgeon, and evacuation was achieved using burr-hole placement. She was discharged to her home four days later, and she had an uneventful recovery with full resolution of her symptoms. Follow-up MRI and CT scans were normal, and she remains fit and well.



Figure 1: Left parietal subdural hematoma.

A 28-year-old primiparous woman presented to the delivery ward at 37 weeks of gestation with severe preeclamptic toxemia. Her blood pressure was 170/115 mmHg on three repeated measurements. She had peripheral edema +3, proteinuria 5 gm/day and oliguria. She had no headache, abdominal pain or visual disturbances. The platelet count was 99,000  $\mu$ l/l, and coagulation (PT, PTT, and INR), serum creatinine and liver function test values were within normal ranges. She received magnesium sulfate treatment loading and infusion, and cesarean section was performed under spinal anesthesia. The needle (24 G) was inserted at the L3-L4 level on the first attempt, and 12.5 mg of 0.5% heavy bupivacaine was injected, as documented in the patient's record. Her blood pressure was well controlled in the first 24 h, her clotting function and liver enzymes were within normal ranges. The patient developed mild post spinal headache after the first postoperative day, which was treated with IV hydration, bed rest and analgesics. The patient was discharged with a persistent mild headache but was readmitted the next day for a constant and intense headache unrelated to posture. The diagnosis was reconsidered, and a neurologist was

consulted. CT of the head and MRI of the brain revealed small bilateral temporal subdural hematomas (Figure 2). She exhibited no other neurological abnormalities. The patient was managed conservatively, and a follow-up MRI on the sixth postoperative day revealed that the hematoma had resolved. She was discharged on the seventh postoperative day without further incident.

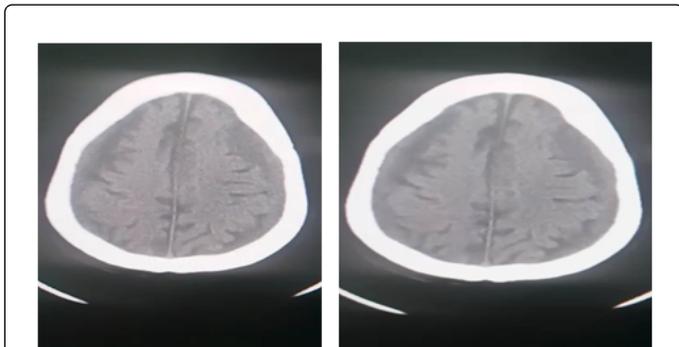


Figure 2: Small bilateral temporal subdural hematomas.

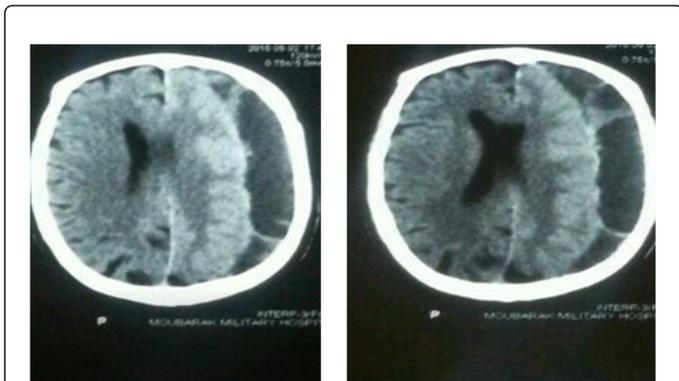


Figure 3: Left temporoparietal occipital subdural hematoma.

A 20-year-old female was admitted for urgent cesarean section with abdominal pain and vomiting. The procedure was performed under spinal anesthesia. A 22 G spinal needle was introduced through the L4-L5 interspace. Spinal anesthesia was successful on the first attempt, and 2 ml of 0.5% heavy bupivacaine was injected. The patient remained hemodynamically stable, recovered fully and was discharged on the second day after surgery. She had no headache at the time of discharge. However, she developed a severe diffuse headache one week later that did not subside with analgesics and bed rest. An intracranial lesion was suspected when she began to projectile vomit and after CT scan brain, and neurological consultation was sought. There was no history of fever, trauma or bleeding diathesis. The patient was conscious and oriented, the Glasgow coma score was 15, the fundi were normal, and there was no focal neurological deficit. She had no neck rigidity or Kerning's sign. Hemoglobin, platelet count, bleeding time, clotting time, prothrombin time and activated partial thromboplastin time (APTT) were normal. Biochemical values, including liver and renal function tests, were also normal. CT and MRI revealed a subacute subdural hematoma in the left temporoparietal occipital region with mass effect (Figure 3). The patient was managed surgically *via* the placement of two burr holes and irrigation with tepid saline until the

fluid was clear. She also received postoperative bed rest, analgesics and intravenous fluid. She was complete recovery and discharged after one week.

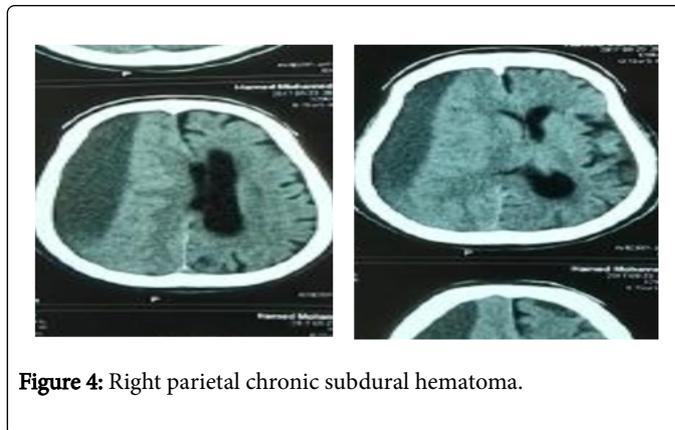


Figure 4: Right parietal chronic subdural hematoma.

A 29-year-old female was scheduled for an elective cesarean section under routine spinal anesthesia. At least four punctures at the L3-L4 level in the sitting position were reported as painful. There was no report of needle size or CSF or bloody leak from the needle in the patient's record. The anesthetic drug injected was 12.5 mg heavy Marcaine (0.5%). The patient developed a severe diffuse headache after 4 days that did not subside with analgesia and bed rest. The patient complained of dullness and mental confusion and developed abducent nerve palsy. The patient was referred to our institution for management. The patient was admitted to the neurosurgery ward after emergent consultation with a neurosurgeon. Head CT revealed a hyper-intensity in the subdural region over the right parietal lobe (Figure 4). History revealed only mild hypertension. There was no history of coagulopathy, and all laboratory investigation e.g the platelet counts, prothrombin time, activated partial thromboplastin time, fibrinogen, bleeding time, and platelet function tests were normal. Chronic hematoma evacuation was achieved using burr hole placement. She was discharged four days later and had an uneventful recovery with full resolution of her symptoms. Follow-up MRI and CT scans were normal, and she remains fit and well.

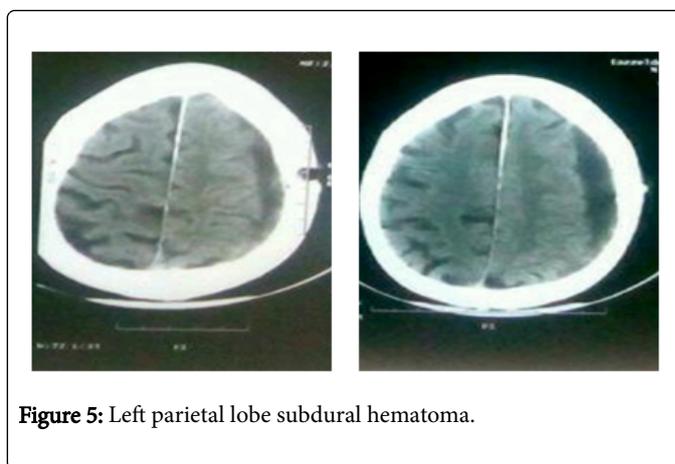


Figure 5: Left parietal lobe subdural hematoma.

A 65-year-old male presented to our ED suffering from headache and right hemiparesis. He underwent an elective lower third ureter stone removal with spinal anesthesia in our hospital 2 weeks prior to presentation to our ED. The headache was defined as frontal that increased with sitting and standing up. He had no history of

hypertension, blood disease, infection, neurological disease, migraine, malignancy, head trauma or coagulation abnormalities prior to surgery, and all laboratory investigations were normal. The spinal anesthesia was performed *via* puncture of the L4-L5 space using a 22-gauge spinal needle and injection of 15 mg of 0.5% heavy bupivacaine, as recorded in the patient's record. The puncture was successful on the first attempt, and the course of anesthesia did not have any side effects or complication. His intraoperative vital signs were normal, and the surgery was completed uneventfully. Full recovery was achieved, and he was discharged on the third day after surgery. He had no headache at the time of discharge. The patient developed a mild diffuse headache 7 to 10 days later that did not subside with simple analgesics and bed rest. The severity of the frontal headache gradually increased and was exacerbated by sitting and standing up. Numbness developed in right half of the body the right upper, lower limbs and face, and the patient was brought to the ED. He complained of an intense generalized headache that was present at all times but worsened upon sitting upright 2 weeks postoperatively. He was conscious and oriented. Fundus examination was normal. He had no neck rigidity or Kerning's sign. The motor force of right limbs was 4/5. His BP was 120/80 mmHg, and no fever was present. Laboratory data revealed normal hemoglobin, white blood count, platelet, PT, PTT, and INR. CT revealed a hypo-dense crescent shape in the left parietal lobe (Figure 5). The diagnosis was confirmed using MRI, which exhibited hyper-intensity in the subdural left parietal lobe. The patient was admitted to the neurosurgery ward after consultation with a neurosurgeon. Evacuation was achieved using burr hole placement. He was discharged four days later and had an uneventful recovery with full resolution of his symptoms. Follow-up MRI and CT scans were normal, and he remains fit and well.

## Discussion

Subarachnoid block is an agreeable and often preferable method to do anesthesia and analgesia in obstetric patients [14,15]. The most frequent side effect of lumbar puncture is headache [16]. However, females are more susceptible to post spinal headache, which is a frequent and benign side effect of dural puncture [17,18]. The mechanism underlying post spinal headache is not fully comprehend, but the headache may be caused by excessive (<250 ml/day) leakage of CSF through the dural puncture site [19]. This CSF drainage is often prohibited causes caudal displacement of intracranial structures [20], which stretch the intracranial sensitive parts dura sinuses and blood vessels which lead to headache. The International Headache Society provided criteria to assist in the differentiation of post spinal headache from other more severe and serious complications of dural puncture [8]. These criteria note that the pain in PDPH develops within 15 min of simulating an upright position and improves within the same period after the individual reclines. PDPH develops within 5 days after the puncture and disappears spontaneously over one week or up to 48 h after an epidural blood patch is used. PDPH typically occurs soon after SA and generally subsides in several days with complete bed rest and some analgesics, and it is self-limiting. Headache that is persistent and more severe than PDPH may be due to subdural hematoma formation [21].

SDH after SA is a rare, but sometimes fatal, complication of SA. The rare but grievous complication of subdural hematoma formation should be kept in mind given the recent increased use of SA. It is difficult to exactly identify the predisposing factors of SDH because of the relative rarity of these complications. Previous studies concentrated

on CSF leakage [7]. Continued CSF leakage reduces CSF pressure [1,6,7].

Patients, who undergo SA invariably follow-up with gynecologists and general surgeons rather than the anesthesiologists who managed their anesthesia. Therefore, these clinicians should be educated about this rare complication. The diagnosis of any intracranial complication is easily made on cranial CT, and early surgical intervention is often curative if the patient exhibits a good neurological status.

An incidence of 1 in 500,000 was reported for intracranial SDH following SA of cesarean section [9], but most authors believe that the exact incidence of SDH after dural puncture is not known, and many cases are not reported. Zeidan et al. reported that the time between lumbar puncture and symptom presentation ranged from 6 h to 29 weeks in 25 patients. Nine cases presented 25-50 days after the suspected procedure [7] within the 3 week period of our study (28 days, 48 h, 1 week, 4 days, and 2 weeks). SDH after SA occurred most frequently on the left side of the brain in the Zeidan study (13 cases), followed by 6 right-sided, 4 bilateral, and 2 intracerebral cases [7]. In our study, SDH after SA occurred in the late chronic phase in the left parietal, small bilateral temporal, left temporoparietal occipital, right parietal, and left parietal regions, which means that the bleeding was left-sided in three cases, right-sided in one case and bilateral in one case. Amorim et al. reviewed 35 cases of intracranial hemorrhage following SA and found that 15 of these patients had no known contributing factors. The most common known risk factors were pregnancy, multiple trials of punctures, use of anticoagulants drugs, intracranial vascular abnormalities, and in old age brain atrophy [13]. Most of the patients in our study were pregnant, but none exhibited early (i.e., at the time of cesarean section) or late (i.e., at presentation of subdural hematoma) bleeding disorders. The urological patient was old with brain atrophy. Most patients (89%) in the Amorim study at least one of the following symptoms presentations in addition to headache: vomiting, diplopia, squint, cognitive changes, altered mental status, hemiparesis or focal neurological signs [13]. Patients in our study reported vomiting, squinting, dullness and mental confusion, numbness in the face and upper limb and hemiparesis in addition to headache, but most patients were fully conscious and oriented (Glasgow coma score 15/15). We recommend that the presence of any of these findings should prompt the physician to search for causes other than post spinal headache. A change in the headache characteristics from positional to non-positional was also mentioned as a sign that intracranial hemorrhage (sub dural hematoma) may be complicating a simple intracranial hypotension [7].

The treatment decision is conservative or surgical. Small types of hematomas often resolve spontaneously, and the risk of surgical evacuation is not justified. Twenty-seven of the 35 cases in the Amorim study required surgical drainage [13]. Surgery was performed in 20 of the 25 patients in the Zeidan series [7]. Four cases in our study required surgical evacuation, and 1 case was managed conservatively and resolved spontaneously. Pavlin et al. reported 2 cases of subdural hematoma after spinal anesthesia that required surgical decompression [6]. The true incidence of subdural hematoma after spinal anesthesia is not known. Most patients with headache are treated without investigation. Subdural hematomas resolve spontaneously [22]. Subdural hematoma headache following lumbar puncture remains rare and most of these patients are diagnosed late, after presentation with neurological deficits. The diagnosis of SDH is 100% using CT and MRI. One patient in our study developed subdural hematoma despite the use of a narrow gauge (24 G) needle, and the hematoma increased

sufficiently in size to warrant an emergency surgical evacuation. Subdural hematomas increase in size because the capsule acts as an osmotic membrane that facilitates the diffusion of water into the hematoma.

## Conclusions

Severe and progressive headache following spinal anesthesia that does not respond to bed rest and analgesia should be a warning sign of an intracranial complication. The anesthesiologists should inform other clinicians of this rare but lethal complication of SA. Early neurosurgical consultation in these patients is recommended to facilitate early diagnosis and prompt intervention to prevent avoidable morbidity and mortality. The present study has limitations because of the small sample size. Recommendations Increased awareness of hemorrhagic side effects following SA and improved assessment of post-SA headache will aid the early diagnosis and management of atypical PDPH. The anesthesiologist should administer an epidural blood patch and refer to a neurologist for evaluation and management.

## Declarations

**Ethics approval and consent to participate:** This study was performed after approval by the hospital ethical committee, and written informed consent was obtained from each patient or relatives. Substantial or procedural ethical aspects were considered in this study. The purpose and nature of the study and the risks were explained to participants prior to study admission. The participants agreed that he/she understood the investigational nature of the study, its inherent risks and benefits, his/her rights to terminate participation in this study without affecting his/her rights to receive proper health care at the study site, whom to contact with questions about the study, and that he/she freely provided informed consent to participate.

**Informed consent:** The signed informed consent forms are a permanent part of the participant's study records and are maintained in the same manner as other records.

## Consent for Publication and Confidentiality

The confidentiality of all participants admitted to this study was protected to the fullest extent possible. The study participants will not be identified by name in any report or publication resulting from the data collected in this study.

## Availability of Data and Material

The data supporting the findings of this study are available. All authors can provide access to the data and materials.

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No funding was used in the design of the study and collection, analysis, and interpretation of data and in writing the manuscript.

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