SCIENTIFIC JOURNAL
CEREM-GO

DOI 10.37951/2675-5009.2025v6i16.155
ISSN 2675-5009
e25155
SCIENTIFIC ARTICLE - CASE REPORT

FATAL ACUTE SUBDURAL HEMATOMA AFTER SPINAL ANESTHESIA - CASE REPORT

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ABSTRACT

Post-spinal anesthesia headache is a complication with a prevalence that varies between 1/500,000 and 1/1,000,000 and its occurrence points to a pressure gradient between the intracranial subarachnoid and spinal spaces due to the presence of a dural injury caused by the puncture. The persistence of the barometric gradient, however, can determine greater separation of the cerebral surface from the dura mater, with rupture of the bridging veins and formation of acute subdural hematoma (aSDH), a condition associated with high morbidity and mortality. aSDH after spinal anesthesia is a rare and potentially fatal complication. In the literature, it is observed that the majority of aSDHs after dural puncture did not result in exuberant mass effects or major clinical changes, meaning that urgent surgical treatment was not necessary, with the institution of clinical treatment for subsequent surgical surgery in a hematoma already showing signs of urgency chronification. In this reported case, it is evident that a patient had a head CT upon admission within the normal age range, however, she developed neurological changes and a new imaging examination showed a large aSDH with great clinical repercussions, and in the evaluation by neurosurgery, she presented signs of brain death without the possibility of neurosurgical treatment due to the impossibility of reversing the condition or providing any benefit to the patient.

Keywords: Brain death, Post-spinal anesthesia headache, Acute subdural hematoma, Headache disorders, Secondary, Brain edema.

INTRODUCTION

Post-spinal anesthesia headache is a complication with a prevalence that varies between 1/500,000 and 1/1,000,0001 and its occurrence points to a pressure gradient between the intracranial subarachnoid and spinal spaces caused by the puncture. The persistence of the barometric gradient, however, can determine greater separation of the cerebral surface from the dura mater, with rupture of the bridging veins and formation of acute subdural hematoma (aSDH) between the dura

mater and the subarachnoid space[2], a condition associated with high morbidity and mortality. The following are risk factors for the occurrence of hematoma after lumbar puncture: excessive drainage of cerebrospinal fluid (>250 ml); use of a traumatic needle or needle of inadequate diameter; cerebral atrophy and use of anticoagulants. In elderly patients, the subdural space is wider due to senescent cerebral atrophy, which results in a greater propensity to hemorrhage due to rupture of the bridging veins. aSDH mimics the occurrence of post-dural puncture headache, except for the fact that it presents with non-postural pain.[3] It is therefore considered an important differential diagnosis in the presence of unfavorable neurological outcomes in patients undergoing spinal anesthesia. We describe the case of a young patient who underwent orthopedic surgery on the lower limb under spinal anesthesia, who developed spontaneous aSDH. This is a rare complication, but avoidable with an adequate syndromic diagnosis. The literature on this topic is scarce and, to date, few cases have been reported with a fatal outcome.

CASE REPORT

In Female patient, 44 years old, previously healthy, was admitted to Goiás Emergency Hospital (HUGO) in July 2023, at night, with a history of multiple trauma due to a motorcycle accident (collision with a stopped car) 30 minutes ago. Reports she falling to the right side. Denies ejection or loss of helmet at the time. She only reports pain in the upper and lower limbs on the right. She denied the use of continuous medications, illicit substances and also did not use anticoagulants or antiplatelet agents and had no history of related comorbidities. On physical examination, a score of 15 on the Glasgow Coma Scale (ECG), isochoric and photoreactive pupils and no apparent neurological deficits. She presented a 10 cm blunt injury on the anterolateral surface of the right foot with tendon exposure, with limitations in dorsiflexion movement and immobilization of the right upper limb. She underwent complementary exams, being diagnosed with a fracture of the right proximal humerus and a distal fracture of the ipsilateral exposed tibia, being approached by the orthopedics team with successful. The procedure was performed using spinal anesthesia with bupivacaine with a 22Gx3 spinal needle at the level of L3-L4 without complications. The following day, the patient began to experience symptoms of postural headache and episodes of vomiting, with a normal physical examination, and was referred for a head tomography (figure 1), which showed no changes. The patient progressed the following day with worsening nausea and vomiting, as well as a non-postural holocranial headache, and presented with re-entrant seizures without improvement with anti-seizure drugs, requiring the provision of a definitive airway, mechanical ventilation and sedoanalgesia. New investigation exams were requested, including computed tomography angiography (CTA) of the skull with contrast, with evidence of aSDH, left fronto- temporo-parietal with a deviation of approximately 1.41 cm from the midline and cerebral edema more pronounced on the left (figure 2). During the inspection of the scalp, no signs of local hematoma were seen resulting from a fall from a bed, for example. On neurological examination, she presented bilateral fixed mydriasis and absence of brainstem reflexes, such as: oculocephalic, corneal-palpebral and cough reflexes. Sedation is discontinued for subsequent neurological assessment and laboratory tests are requested. She had a hydroelectrolyte disorder with kalaemia of 3.1 mmol/L, natremia of 159 mmol/L, without acid-base disorder with arterial pH within normal limits, however, with arterial lactate of 17.5 mg/dL. She was admitted to an intensive care unit for correction of metabolic changes and neurological surveillance. Comatose patient without sedation for 18 hours, absence of brainstem reflexes during clinical

consultations, developed hemodynamic instability requiring vasoactive amine (norepinephrine) and invasive blood pressure monitoring. The brain death protocol was initiated at 10 pm on 11/07, fulfilling clinical criteria: core temperature 36.1 ° C, blood pressure 130 x 90 mmHg, SatO2 99%, PaCo2 37 mmHg. She followed with hemodynamic stress in the following days, with norepinephrine at 2.8 mcg/kg/min and vasopressin at 0.04 IU/min, on mechanical ventilation in volume-control mode with FiO2 100%, tidal volume of 380 ml and PEEP 7 , presenting arterial pH with metabolic acidosis and hypokalemia. The patient was compensated from a hydroelectrolytic and acid-base point of view, and an apnea test was performed uneventfully. However, in the evaluation with daily transcranial Doppler, showed residual arterial blood flow. Nine days after admission, brain death was confirmed with transcranial Doppler confirming the absence of cerebral blood perfusion and family members were informed about the death, who chose not to donate the organs.

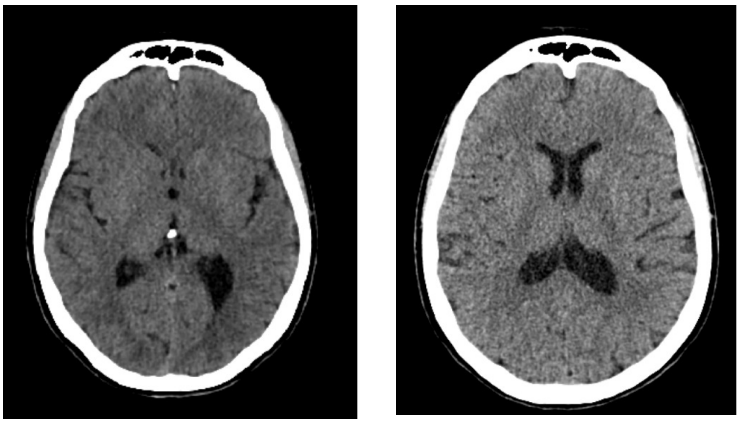

Figure 1. Cranial tomography on admission (07/07/2023) and on 08/07/2023 without changes.

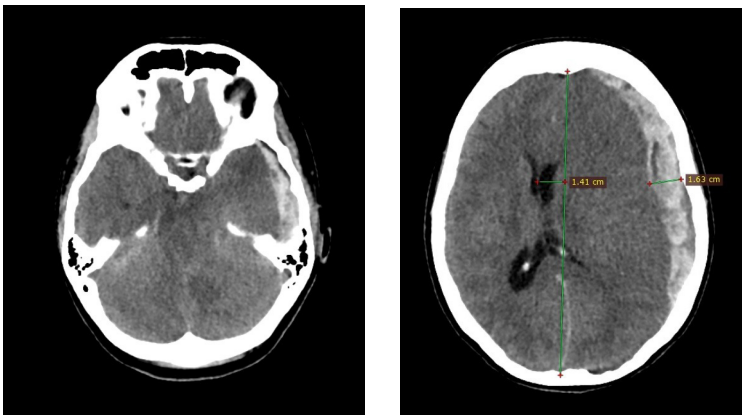

Figure 2. Head CT on the second day after admission showing acute left fronto-temporo-parietal subdural hematoma with midline shift of 1.41 cm, diffuse cerebral edema with compression of the cisterns and effacement of cerebral grooves and fissures.

DISCUSSION

The Intracranial subdural hematoma post-spinal anesthesia is a complication considered rare and potentially fatal after a dural puncture procedure, thus, there are not many cases reported in the medical literature.[4] Therefore, this article aims to contribute with an unusual and fatal report, being useful to highlight the importance of clinical suspicion and early diagnosis of this complication to which patients are subject to avoid possible catastrophic outcomes.[5]

In most of the articles analyzed, it was observed that the majority of acute subdural hematomas after spinal anesthesia did not result in exuberant mass effects and little deviation from the midline, therefore, with symptoms that were not significant from a neurological point of view, such as: headache, nausea , vomiting, dizziness, diplopia, not requiring urgent surgical treatment, clinical treatment and neurological surveillance for subsequent surgical drainage of an already chronic hematoma were instituted. In this reported case, we observe a young patient, victim of polytrauma with mild cranial trauma with head tomography on admission with no evidence of changes, but she developed a convulsive status, fixed mydriasis with a new head CT indicating aSDH with large deviation from the midline and erasure of cerebral grooves and fissures. A situation in which urgent decompressive craniectomy would be the only treatment, however, the patient already presented with an absence of brainstem reflexes, progressing to brain death 6 days later. This demonstrates the severity of this complication, which can lead to clear signs of intracranial hypertension and the need for urgent surgery.

The aspect of temporality draws attention, as in most cases patients presented aSDH 48 hours[6] after spinal anesthesia and in other cases there was no well-defined pattern between the onset of symptoms and the identification of bleeding. Furthermore, the importance of early diagnosis of postural headache and initiation of appropriate treatment is highlighted, from rest, intravenous hydration and analgesia to blood patch treatment to treat symptoms of intracranial hypotension.[7] A change in headache patterns to non-postural, without improvement with the supine position, associated with intense nausea and vomiting, change in the level of consciousness, visual blurring, contralateral hemiparesis and anisocoria[2,3,5], constitute alarm signals that require assessment and imaging quickly to exclude intracranial pathologies. On head CT, the aSDH appears as a hyperdense crescent-shaped lamina across the cerebral convexity, not respecting the limits of the cranial sutures.

From a pathophysiological point of view, one of the most accepted theories is based on the reduction in intracranial pressure caused by dural puncture associated with hypovolemia of cerebral blood flow, which leads to distension and consequent rupture of the bridging veins located in the subdural space.[8] In a previously healthy patient like the one in the case presented and without neurological changes, with a head CT examination on admission within normal limits, post-spinal anesthesia headache must be suspected as the most common change or even more serious cases, such as pathologies intracranial. From this pathophysiological concept, it is understood that the catastrophic evolution presented by this patient is precisely due to the changes in cerebral hydrodynamics caused by spinal anesthesia, which in a certain way affected the mechanisms for regulating intracranial pressure and cerebral blood flow, culminating in the formation of an acute hematoma in the subdural space quickly and aggressively, resulting in significant mass effect and compression of vital structures, such as the brain stem.

The treatment of aSDH can range from conservative treatment to surgical drainage of the hematoma through decompressive craniectomy, which is indicated in cases of unilateral or bilateral cerebral edema, more specifically when it falls into the Marhsall III or IV classifications.[9]

CONCLUSION

Placental Post-spinal anesthesia headache is a common complication, with incidence rates that can reach 70% of cases, however, the occurrence of aSDH is a rare and potentially fatal outcome that involves early diagnosis and treatment for greater patient survival.

Most of the cases reported were pregnant patients undergoing spinal anesthesia for obstetric procedures, with aSDH occurring in other types of surgery being much less common, as in the case described above in a patient with orthopedic pathology. Also noteworthy, is the evolution of the clinical condition to death, which was not observed in most studies published in the literature. Therefore, this report serves as an alert to differentiate cases of post-dural puncture headache with a benign course from other cases with severe neurological symptoms due to spontaneous aSDH. New studies are needed to identify the incidence of this complication, risk factors and the best therapeutic approach.

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Library Review: Izabella Goulart
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Received: 02/02/25. Accepted: 25/03/25. Published in: 23/04/2025.