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MINI REVIEW **REGIONAL ANESTHESIA**

Intracranial subdural hematoma: a rare but serious complication following neuraxial anesthesia in obstetric anesthesia

Taizoon Q. Dhoon, Jasmin Joshi ✉, Dmitry Portnoy ✉, Govind C. Rajan ✉

Author affiliations: 101 The City Drive, Orange, CA92868, USA.
Correspondence: Taizoon Dhoon, MD; E-mail: tdhoon@uci.edu; Mobile: +1 8587754703

Abstract

Intracranial subdural hematoma (SDH) formation is an uncommon but serious complication of planned or unintentional dural puncture (DP) during straight epidural anesthesia, combined spinal-epidural, dural puncture epidural, or spinal anesthesia. Diagnosis of intracranial SDH following neuraxial anesthesia may be delayed or misdiagnosed due to rare occurrence, lack of knowledge, as well as an overlap in clinical presentation with postdural puncture headache (PDPH). Increased awareness of intracranial SDH as a complication of DP may result in earlier recognition and prevention of potentially devastating outcomes. This article reviews the relevant literature on association of neuraxial anesthesia with intracranial SDH formation.

Key words: Intracranial subdural hematoma; Anesthesia, Neuraxial; Anesthesia, Epidural; Combined spinal-epidural; Dural puncture; Anesthesia, spinal; Pregnancy; Postdural puncture headache

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1. Introduction

The spinal anesthesia revolutionized the practice of anesthesia, being easy to administer, being cheap as very less number of drugs have to be used, and with less incidence of adverse effects. Epidural anesthesia was the next, and together the spinal and epidural were called neuraxial anesthesia, or as some people prefer to call it, neuraxial analgesia. But as the quotation goes like this, "Nothing comes free, you have to pay a price", the neuraxial anesthesia also has some price to be paid if used. Spinal hypotension is the most common complication of the spinal anesthesia, although its incidence is less with epidural anesthesia. Some adverse effects and complications, although rare, but are more serious and may endanger the life of the patients. Intracranial subdural hematoma (SDH) formation is an uncommon but serious complication of planned or unintentional dural puncture (DP) during straight epidural anesthesia, combined spinal-epidural, dural puncture epidural, or spinal anesthesia. Diagnosis of intracranial SDH following neuraxial anesthesia may be delayed or misdiagnosed due to rare occurrence, lack of knowledge, as well as an overlap in clinical presentation with postdural puncture headache (PDPH). The anesthesiologists must be aware of the possibility of developing intracranial SDH as a complication of DP in susceptible patients. An early recognition may help to prevent of potentially devastating outcomes.[1] We present a review of the relevant literature on association of neuraxial anesthesia with the development of intracranial SDH.

2. Intracranial compliance

Intracranial anatomy and physiology are vital to understand intracranial SDH formation following dural puncture. The intracranial space is composed of three primary elements: the brain, cerebrospinal fluid (CSF) and blood.[2] The brain (parenchyma and intracellular fluid) measures approximately 1,400 mL in volume and is relatively non-compressible. In contrast, CSF is compressible and acts as a cushion within and around the brain.[2] The choroid plexus produces 400-500 mL of CSF daily; the arachnoid granulations regulate CSF reabsorption. CSF is produced within the intracranial

ventricles and then journeys though the foramen magnum to the spinal subarachnoid space.[2] The intracranial and extracranial compartments allow bidirectional CSF communication; each contains approximately 150 mL of CSF.[2]

The cerebral blood volume measures approximately 150 mL and is influenced by multiple factors. Local tissue environment, partial pressure of arterial carbon dioxide, vasoactive mediators and intracranial space all affect the degree of cerebral vasoconstriction or vasodilation. As the skull is inflexible, the degree of intracranial compliance is determined by the interplay of the brain, the CSF and the blood.[2] Changes in volume in one element can alter intracranial compliance and impact the others.

3. Pathophysiology of SDH formation

Intracranial SDH formation following DP is thought to be instigated by the loss of CSF though the dural hole created by the spinal needle. If the CSF rate of leak is greater than the rate of CSF production, decreased extracranial and intracranial CSF pressure can ensue. Intracranial CSF hypotension can result in compensatory blood volume expansion and meningeal vasodilation. The CSF leak can also produce intracranial hypotension and downward displacement ("sagging") of the intracranial structures, placing tension on the meninges.[3] If the cortical veins and dural veins are stretched, the vessels may tear and result in an intracranial SDH. Dural puncture can also alter cerebrospinal elasticity, resulting in increased craniocaudal compliance relative to intracranial compliance; as a result, a change from the supine to standing position may worsen intracranial hypotension and exacerbate meningeal vasodilation.[4]

3.1. Epidural Anesthesia

Epidural anesthesia has a higher incidence of intracranial SDH formation compared to spinal anesthesia due to a larger dural defect as a result of accidental puncture and a higher volume of CSF loss.[5] The rate of CSF leak following DP is influenced largely by the size of epidural needle; expectedly, a lesser leak rate was reported with smaller diameter Touhy needles.[6] Epidural needle bevel orientation also plays a role; parallel to the long axis of the spine rather than perpendicular orientation decreases the risk of CSF leakage. Parallel bevel orientation to the dural fibers constrains the size of the tear and reduces the rate of CSF leakage and headache formation.[7]

3.2. Spinal anesthesia

In contrast to epidural anesthesia, spinal anesthesia requires dural puncture in order to produce a successful anesthetic. Spinal needle selection is important in reducing the risk of CSF leakage and PDPH occurrence. Larger diameter spinal needles result in a greater dural defect, elevating the risk of PDPH and possible SDH formation.[8, 9] Pencil-point spinal (non-cutting) needles, when compared to cutting tip needles, reduce damage to the dural fibers, rate of CSF leakage and thus the incidence of PDPH.[10, 11, 12] If a cutting-tip needle is selected, the bevel should aim parallel to the long axis of the spine.

4. PDPH signs and symptoms

The incidence of PDPH is highly variable, depending on the size and the type of the needle involved in dural puncture. Small size pencil-point spinal needles are associated with PDPH in less than 0.5% of cases, whereas large bore beveled needles are responsible in more than 50% of the cases.[1-2] If DP occurs with an epidural needle, PDPH may be reported in 50-86% of the patients.[3] Up to 26% of unintentional DP's are missed at the time of initial epidural placement.[4] PDPH typically appears 24-72 hours following dural puncture; however, cases have been reported up to 2 weeks later. The most prominent feature of PDPH is a bilateral frontal and occipital headache aggravated by upright position and improved by lying supine. Atypical presentation may include neck, shoulder and axial back pain. Associated signs and symptoms include nausea and vomiting, visual disturbances, tinnitus, hypoacusis, vertigo and photophobia.[13,14] While most of the cases of PDPH resolve within 7-12 days, it has been reported to develop into SDH if left untreated.[15] Severe PDPH often requires an epidural blood patch (EBP), involving an administration of autologous blood into the epidural space. Timely diagnosis and effective treatment of PDPH with an EBP may minimize CSF leak, improve intracranial pressure and reduce the risk of SDH formation.[13, 14, 16, 17]

5. SDH signs and symptoms

PDPH as a result of unintentional DP during epidural anesthesia is rare, with a rate of 0.19 to 4.4%.[10] However, the true number may be higher as the symptoms of intracranial SDH may mirror and overlap those of PDPH.[1] Intracranial SDH following DP often presents within two to seven days. [18, 19] Patients with SDH present with a broad range of signs and symptoms, from no symptoms to neurological conditions such as headaches, vertigo, balance disturbances, contralateral paresis, speech difficulties, seizures, confusion, and change in consciousness.[20] Headaches from intracranial SDH may be persistent in nature or exacerbated when changing from supine to upright position, similar to headaches associated with PDPH.[1, 21, 22] Clinicians should maintain a high index of suspicion for SDH when a headache

continues for over 7 days, changes from postural to non-postural in nature, or is accompanied by neurological deficits.

Computed tomography is a simple and effective way to evaluate the presence of intracranial SDH. Intracranial SDH can be disastrous, requiring neurosurgical intervention and has a potential to result in disability or death.[8, 19, 20]

Intracranial SDH formation has been associated with young age, female sex, use of antiplatelet agents, coagulation disorders, arteriovenous malformation, head trauma, multiple dural punctures, chronic hypertension, pregnancy, and particularly with preeclampsia.[1,10, 13]

SDH development in the obstetric population is not fully understood, and it is very rare with an incidence of approximately 1 per 500,000.[20, 22] Institutions with a disproportionate number of high-risk deliveries may have a higher incidence ranging from 0.0026 to 1.1%.[22] Obstetric patients may be more susceptible to PDPH and intracranial SDH due to changes in dural elasticity or physiological changes attributed to pregnancy.[20] In addition, parturients who developed PDPH after childbirth had a small yet statistically significant increase in intracranial SDH formation.[3] Parturients who underwent a cesarean delivery displayed a decreased risk of PDPH and intracranial SDH formation; in the absence of pushing during vaginal delivery, less CSF may have been lost.[13]

6. Management

Management of SDH is determined by severity, hematoma size, and mass effect. Small hematomas (clot thickness less than 10 mm, midline shift less than 5 mm, absence of pupillary abnormalities and intracranial hypertension) are managed with supportive care and typically resolve without intervention.[18] Surgery is recommended for larger hematomas with neurologic deficits. [18]

7. Conclusion

Epidural and spinal anesthetics are effective, generally safe and widespread procedures in obstetric anesthesia practice. Nonetheless, anesthesiologists, obstetricians and other healthcare providers must be aware that although intracranial SDH is rare, but it is a potentially catastrophic complication associated with dural puncture, whether intentional or accidental. Clinicians should maintain a high index of suspicion for SDH when a headache persists over 7 days, changes from postural to non-postural in nature, or is accompanied by neurological deficits. Clinicians must also be aware of the potentially similar and overlapping presentations of both PDPH as well as the intracranial SDH. In suspected cases, early multidisciplinary approach including neurosurgical consultation and brain imaging are essential to reduce the severe neurological sequelae.

8. Conflict of interest

None declared by the authors

9. Authors' contribution

TD & JJ- Concept, conduction of the study work and manuscript editing
DP & GR- Concept, manuscript editing

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