Abstract

Objective: To describe a case of cranial subdural hematoma as an atypically sporadic and potentially devastating complication of a lumbar puncture mimicking post-dural puncture headache which may lead to delayed diagnosis.

Methods: This is a case report of chronic intracranial subdural hematoma as a complication of lumbar puncture for spinal anesthesia in a forty-eight-year-old male underwent an uneventful ureterolithotomy. In the postoperative period, the patient complained of excruciating headaches, which partially subsided with symptomatic treatment. However, after four weeks, the patient presented with worsening headache and vomiting.

Results: A contrast-enhanced computer tomography study revealed a subdural hematoma in the left frontotemporoparietal region. The patient underwent an immediate decompressive craniectomy for subdural hematoma. The postoperative period was uneventful, and the patient was discharged without any neurological deficits.

Conclusions: Intracranial complication such as subdural hematoma is rare after a dural puncture and is often masquerading as a post-dural puncture headache. Imaging studies like computerized tomography and magnetic resonance imaging are to be considered in earlier stages of non-retractable headache to rule out such rare complications.

Keywords: CT, intracranial subdural hematoma, post-dural puncture headache

Introduction

Cranial subdural hematoma is an unusually infrequent and potentially cataclysmic complication of a lumbar puncture. The prevalence of subdural hematoma as a complication after lumbar puncture is probably inaccurate due to delay in diagnosis, misdiagnosis, or under-reporting.

Case

A 48 year-old male with no comorbidities had undergone ureterolithotomy under spinal anesthesia using a twenty-five-gauge Quincke spinal needle in a single attempt. After the procedure, he developed a severe headache, which was treated as a post-dural puncture headache with bed rest, paracetamol, and caffeine. The intensity of the headache eventually decreased with this treatment. However, after four weeks, he was presented to the emergency room with a worsening excruciating headache and multiple episodes of vomiting. There was no history of trauma during this period. He was then referred to the radiology department for contrast-enhanced computerized tomogram (CT) imaging of the brain to rule out any underlying cerebral pathology.

On CT imaging, a hypo-dense subdural collection with a tiny hyper-dense component was noted on the left frontotemporoparietal region, showing a mass effect that triggered...
a midline shift (about thirteen millimeters towards the right) with compression of the ipsilateral ventricles and effacement of the sulcal spaces (Fig. 1). On the post-contrast imaging, no area of significant enhancement was seen in the adjacent areas (Fig. 2). Given the imaging findings and clinical history, the possibility of a chronic subdural hematoma with an acute component was suggested. The patient was immediately sent for an urgent neurosurgical consultation, then received decompression of subdural...
Cranial subdural hematoma is an unusually rare and potentially catastrophic complication of a lumbar puncture secondary to spinal anesthesia, myelography, discography, diagnostic lumbar puncture, and accidental dural puncture with an epidural needle. The accurate prevalence of subdural hematoma post lumbar puncture is inconclusive, probably due to delay in diagnosis, misdiagnosis, or under-reporting; however, some authors have reported an incidence of one in five million in obstetric procedures for this complication. The clinical presentations of subdural hematoma depend on age, size, site, pace of formation of the blood collection, compression of intracranial structures, and clinical state of the patient. Usually, in the initial phase, symptoms may resemble that of post-dural puncture headache with postural changes related to low cerebrovascular fluid pressure. In the second phase, there is a worsening headache with a lack of improvement with pharmacological treatment or after the provision of an epidural blood patch. Clinical manifestations ranging from alterations in consciousness level, focal neurological signs like ptosis, paresis, weakness, and language deficits are also noted.

The period of headache triggered by subdural hematoma is categorized into three patterns. The first pattern, which is the most common, is a headache that presents early (within four days) after dural puncture and lingers with subsequent onset of subdural hemorrhage. The second pattern comprises a headache that prevails early after the dural puncture which then fizzled out or attenuated briefly but resurfaced with an aggravation that is followed by the inception of subdural hemorrhage. The third pattern is a headache that does not appear early after dural puncture but emerges later with the commencement of subdural hemorrhage. Due to unknown reasons, cerebral subdural hemorrhage after spinal anesthesia occurs more regularly on the left side, as early as six hours and at the latest by twenty-nine weeks.

Risk factors for the emergence of subdural collections are cerebral atrophy, cerebral aneurysms, blood dyscrasias, platelet anti-aggregation drugs, arteriovenous malformations, pregnancy, dehydration, alcohol consumption, multiple punctures, thick bore needles, epidural needles, and fenestrations from previous punctures. Spinal anesthesia performed with fine needles of the Whitacre-, Sprotte- and Atraucan-type bevel have not been reported to prevent this complication.

Certain studies have displayed that the puncture aperture can stay for up to eighteen weeks. Extravasation of cerebrospinal fluid can reach up to two hundred and forty milliliters per day in orifices of six hundred micrometers of diameter, which causes dissipation of the autoregulation mechanisms of intracranial pressure dynamics. This condition leads to an incessantly low cerebrospinal fluid pressure and imposes traction that causes a relative ventricular collapse and a rostrocaudal movement of the central nervous system, resulting in extension of the pain-sensitive structure and intracranial subdural bridging veins. The precipitous depletion in the cerebrospinal fluid volume may also trigger adenosine receptors, leading to arterial and venous vasodilatation and subsequently presenting as the clinical symptoms of post-dural puncture headache.

If the traction expended on the bridging veins is considerable, it may disrupt its most fragile point, triggering a development of hematoma between the dura and arachnoid mater. The wall composition of the bridging subdural veins is microscopically weaker with variable thickness. Circumferential arrangement of collagen fibers, lacking the outer reinforcement by arachnoid trabeculae compared to other venous walls, makes them more vulnerable to tear. The elevation in intracranial pressure by the evolution of intracranial hematoma may compensate for the depletion in the intracranial pressure by spinal fluid leakage. A computed tomography scan or magnetic resonance imaging will authenticate the diagnosis and must be urgently executed when there is a doubt in making the diagnosis. The management of subdural hematoma is conservative, consisting of clinical observation and possible intracranial pressure monitoring or surgical evacuation. A prompt diagnosis of the hematoma can expedite the medical treatment of subdural hematoma and, thus, surgical evacuation may be circumvented. Hematomas less than five millimeters often spontaneously subside.
the risk of subdural bleeding by preventing a fall in cerebrospinal fluid pressure and has resulted in an equivocal outcome in certain cases. Acute subdural hematomas are deftly identified by a cranial computerized tomogram scan, whereas chronic intracranial lesions may need magnetic resonance imaging or cerebral angiography. As time advances, hematoma and surrounding brain tissue show similar radiographic density, making early diagnosis baffling. An expeditious subdural hematoma evacuation is usually associated with a favorable neurological outcome.

References