Case Report

Subdural Hematoma Following Spinal Cord Stimulator Implant

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Headache following interventional procedures is a diagnostic challenge due to the multitude of possible etiologies involved. Presentation can be simple (PDPH alone) or complex (exacerbation of pre-existing chronic headache along with PDPH) or headache associated with a new onset intracranial process. Subdural hematoma is a rare complication of cranio-spinal trauma.

Cranial subdural hematoma may present in an acute, sub-acute, or chronic fashion. Diagnosis of a subdural hematoma in the wake of a PDPH is difficult, requiring a high level of suspicion. Delayed diagnosis of subdural hematoma is usually related to failure to consider it in the differential diagnosis. Thorough history, assessment of the evolution of symptoms, and imaging studies may identify the possible cause and help direct treatment. Change in the character of initial presenting symptoms may be a sign of resolution of the headache or the onset of a secondary process.

We report a case of acute intracranial subdural hematoma secondary to unintentional dural puncture during placement of a permanent spinal cord stimulator lead for refractory angina. There is need for careful follow-up of patients with a known post-dural tear. Failure to identify uncommon adverse events in patients with complicated spinal cord stimulator implantation may lead to permanent injury.

Key words: Subdural, post-dural puncture, headache, spinal cord stimulation

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ubdural hematoma is a rare complication of cranio-spinal trauma (1-4). Cranial subdural hematoma may present in an acute, subacute, or chronic fashion (1,2). The exact incidence of subdural hematoma is unknown but reports indicate an incidence of subdural hematoma in 10% of cases of intracranial hypotension regardless of cause (5). Intracranial hypotension may develop spontaneously or

as a consequence of trauma or surgery on the head and/ or spine (6-8). Presenting symptoms include orthostatic headache, neck stiffness, nausea, and vomiting which can at times resolve spontaneously without intervention. We report a case of acute intracranial subdural hematoma secondary to unintentional dural puncture during placement of a permanent spinal cord stimulator lead for refractory angina.

CASE REPORT

A 49-year-old male with unresolved chest pain presented to the Center for Interventional Pain at the University of Michigan. Pain persisted in spite of 6 angioplasties and 3 coronary stents over a 5-year period. Multiple attempts at medical management were unsuccessful in controlling anginal symptoms. Chest pain prompted multiple ER visits, and a comprehensive work up, including cardiac catheterization and perfusion scans, revealed no treatable lesion. The patient denied any abnormal bruising or bleeding tendencies. Plavix was held for 10 days prior to the procedure. Trial spinal cord stimulation for pain control was successfully performed. The decision to proceed with a permanent spinal cord stimulator implant was reached by the patient in consultation with his cardiologist and our implant team.

On the morning of surgery, the patient was admitted to the preoperative area where a pre-anesthetic assessment was completed. He was positioned prone in the operating room, with appropriate monitoring and mild sedation. The L1/L2 interspace was identified under fluoroscopy and standard surgical preparation and drapes were applied. A Tuohy needle was directed under live fluoroscopic guidance contacting the L2 lamina. Loss of resistance to air was obtained and the syringe removed. Clear fluid flowed freely from the Tuohy needle indicating intrathecal placement. The needle was withdrawn into the epidural space and a spinal lead (Medtronic Octad®) advanced using lateral fluoroscopic view to avoid intrathecal placement. Fluoroscopy in AP and lateral views confirmed correct dorsal epidural placement with the tip positioned at mid C7. Trial stimulation at this level achieved the desired dermatomal pattern. Perception threshold and discomfort threshold were also reassuring in ruling out intrathecal placement. An Implantable Pulse Generator (IPG Medtronic Restore®) was placed uneventfully in the gluteal region. Following the procedure the patient was informed about the dural puncture and the possibility of developing a post-dural puncture headache, signs, symptoms, and its conservative management.

During the post procedure phone call, the patient reported a positional headache accompanied by nausea and vomiting. This progressively worsened over the course of the day, changing in character with loss of the positional component. Due to the change in character of the headache, accompanied by continued emesis, the patient was advised to come to the emergency room (ER) for evaluation by our team. On presentation, mental status changes were noted and naloxone 0.4 mg IV was administered by the ER physician, who attributed mental status changes to an opioid overdose. This occurred prior to arrival of the implant team. Further work-up revealed an afebrile patient with non-focal neurologic findings and an absence of meningeal signs. Stat CT of the head identified the presence of a large subdural hematoma (Fig. 1). Neu-

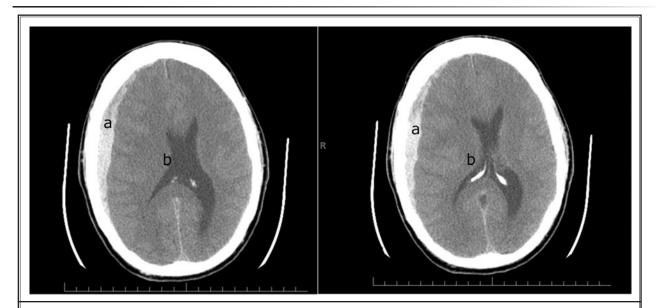


Fig. 1. Contrast enhanced CT scan of head demonstrating presence of a right-sided subdural hematoma (a) with ventricular compression and left-sided midline shift (b).

rosurgery was consulted and the patient underwent an emergent craniotomy. The remaining post-surgical course was uneventful. On post-operative day three the patient recalled falling at home one day prior to the spinal cord stimulator implant, striking his head without loss of consciousness.

Discussion

Headache following interventional procedures is a diagnostic challenge due to the multitude of possible etiologies involved. Presentation can be simple (PDPH alone) or complex (exacerbation of pre-existing chronic headache along with PDPH) or headache associated with a new onset intracranial process. Thorough history, assessment of evolution of symptoms, and imaging studies may identify the possible cause and help direct treatment. Symptoms of note include location (unilateral vs. bilateral), timing (continuous vs. episodic), and associated features (nausea, vomiting, aura etc.), including the absence or presence of a postural component. Change in the character of the initial presenting symptoms may be a sign of resolution of the headache or the onset of a secondary process.

Intracranial hypotension has a number of potential causes, including head, brain, or spine surgery, trauma, and lumbar puncture (6,9-13). Intracranial hypotension can variably present as an orthostatic headache (14). Ninety-percent of headaches occur within 3 days of the dural tear (15). The nature and location of pain varies, although most cases present with a fronto-occipital headache within the first 24 – 48 hours (16). Common characteristics include exacerbation of pain with coughing, laughing, and valsalva maneuvers (17,18).Patients may experience nausea, vomiting, neck pain, dizziness, and upper extremity radicular symptoms (19). Most cases are benign and self limited resolving within 7 days (20). Atypical presentations include subdural hematoma, dementia, Parkinsonism, cranial nerve palsy, syringomyelia, seizures, coma, and death (17,21-26).

Intracranial hypotension can lead to cranial or spinal hemorrhage through bridging dural vein tears (27-29). Bridging veins empty into dural sinuses, with the thinnest segment traversing the subdural space. Traction exerted on bridging veins may lead to rupture at their weakest point in the subdural space (29). Subdural hematoma following traumatic intracranial hypotension is associated with a number of factors, including the use of large bore needles, multiple attempts, cortical atrophy, alcohol abuse, and neurologic disease (30-36). Diagnosis of a subdural hematoma in the wake of a PDPH is difficult, requiring a high level of suspicion (37). Delayed diagnosis of subdural hematoma is usually related to failure to consider it in the differential diagnosis (38-40). Following onset of a PDPH, worsening of headache, loss of positional component, disorientation, hemiplegia, and seizure warrant immediate assessment.

In this patient blunt head trauma occurred within 24 hours of the dural tear. The timing of the subdural hematoma is impossible to determine with any degree of accuracy. It is equally possible that the patient developed a sub-clinical subdural hematoma the day prior following head trauma that was exacerbated by loss of cerebrospinal fluid (CSF) pressure, or that the drop in CSF pressure relating to the dural puncture was responsible for the subdural bleed. As a history of the recent fall was not obtained on the morning of surgery, the implication of blunt head trauma was not assessed prior to the procedure. The lack of visible signs of trauma, such as bruising on the forehead or scalp, was another factor contributing to the failure to identify the traumatic event. In retrospect, postponement of surgical implantation may have been warranted in order to minimize risk to the patient.

Although an epidural blood patch was not performed in this case, a brief review of current management of PDPH follows. Treatment usually begins with conservative management. This involves bed rest, oral or intravenous fluid administration, and use of oral analgesics. Intravenous or oral caffeine, theophylline, and steroids are also used (8,41,42). Patients that do not respond to conservative measures may benefit from an epidural blood patch (43). The mechanism of effect of an epidural blood patch is currently under debate (44). The effectiveness of a single epidural blood patch is reported between 61% and 93% (43-45). Volume of blood injected ranges from 10 to 20 mL (44-47). A prophylactic blood patch after an inadvertent dural puncture with an epidural needle is controversial (44,48-50). Some authors have suggested a prophylactic blood patch may reduce the incidence of subsequent PDPH to 5% - 21% (51-53). Loeser et al reports a decrease in efficacy of epidural blood patch during the first 24 to 48 hours after a dural puncture (54). It is of note that, an epidural blood patch performed in the presence of intracranial hemorrhage can result in neurologic deterioration due to rebound intracranial hypertension (55, 56). An epidural blood patch, although initially effective in treating symptoms of PDPH, may not prevent the development of a subdural hematoma and continued vigilance is required (57,58).

Conclusion

Headache following instrumentation of the spine should be followed closely by clinicians and treated either conservatively at first or by means of recommended intervention if a post-dural leak is suspected. Change in the character of the headache should be

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evaluated urgently to avoid permanent harm to the patient, as the alteration in symptoms may be the initial presentation of a developing subdural hematoma. There is need for careful follow up of patients with a known post-dural tear. Failure to identify uncommon adverse events in patients with complicated spinal cord stimulator implantation may lead to permanent injury. Careful review of history (including review of any pre-procedure trauma) should be completed and postponement of surgery considered, in such cases.

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