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Case Report

Report of cerebral vasospasm as a complication of intracranial subarachnoid hemorrhage following traumatic lumbar puncture

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ABSTRACT

Background: This case report is the first documented and illustrated case of the identification and treatment of intracranial vasospasm as a sequalae of traumatic lumbar puncture (LP). LP is a routine procedure performed for both diagnostic and therapeutic purposes. Although rare, this procedure has risks and complications that should be considered before performing.

Case Description: A 58-year-old male was found to have intracranial subarachnoid hemorrhage (SAH) 2 days after a traumatic LP which occurred in the setting of subtherapeutic international normalized ratio. During his hospitalization, the patient developed both clinical and radiographic signs of vasospasm. He was taken for angiography, which demonstrated significant vasospasm of bilateral middle cerebral arteries and bilateral anterior cerebral arteries. All vasospasms resolved and the patient improved clinically after intra-arterial spasmolytic therapy.

Conclusion: LP is a routine procedure with complications that are often overlooked. The authors describe intracranial vasospasm from traumatic LP before correction of patient's coagulopathy. Cases with similar hemorrhage occurring in the spine resulting in non-aneurysmal SAH and vasospasm were reviewed.

Keywords: Cerebral vasospasm, Lumbar puncture, Spinal hematoma

INTRODUCTION

The lumbar puncture (LP) was first reported by Quinke in the late 19th century. Today, this technique has become an essential tool providing both diagnostic and therapeutic purpose. It is routinely used for the diagnosis of a variety of conditions including meningitis, encephalitis, subarachnoid hemorrhage (SAH), central nervous system vasculitis, autoimmune conditions, paraneoplastic syndromes, and some intracranial tumors.[5]

Understanding lumbar spine anatomy is vital in performing a safe LP. As the spinal needle is inserted in the lumbar spine, it will pierce the skin, subcutaneous tissue, supraspinous

ligament, interspinous ligament, ligamentum flavum, epidural space, dura, arachnoid, and ultimately into the subarachnoid space.[4,11]

Contraindications to performing a LP include infection near the site of the LP, posterior fossa mass with increased intracranial pressure, and coagulopathy. Risks of LP include spinal headache with intracranial hypotension, nerve root irritation, infection, brain stem herniation, and bleeding complications.[2] The epidural venous plexus and radicular vessels that follow the nerve roots are the primary cause of traumatic LPs. Rare complications of both intracranial and spinal hemorrhage have been reported with traumatic LPs.[1,3,7] However, only one other case of non-aneurysmal SAH with vasospasm has been reported as a complication secondary to LP. Furthermore, it is essential to address any coagulopathy; a patient may have before attempting a LP.

CASE DESCRIPTION

A 58-year-old male with medical history of autoimmune deficiency syndrome, hypertension, diabetes mellitus, polysubstance abuse disorder, psychogenic movement disorder, and antiphospholipid antibody syndrome presented to emergency department (ED) with altered mental status.

Initial computed tomography (CT) head without contrast was negative for hemorrhage. The ED proceeded with performing a LP to obtain cerebrospinal fluid (CSF) for the diagnostic purpose of ruling out meningitis and HIV encephalitis. The LP yielded 20cc of bloody CSF. Of note, patient was on Coumadin for antiphospholipid syndrome and had an international normalized ratio of 2.0 and prothrombin time of 19.8. The ED failed to reverse his warfarin before performing the LP and the patient developed headache soon after, which sustained for the next few days. A second CT of the head without contrast was completed 48 h after the LP, for sustained headache and development of somnolence, which demonstrated diffuse SAH with intraventricular extension [Figure 1]. The patient was transferred to the intensive care unit for close neurological monitoring.

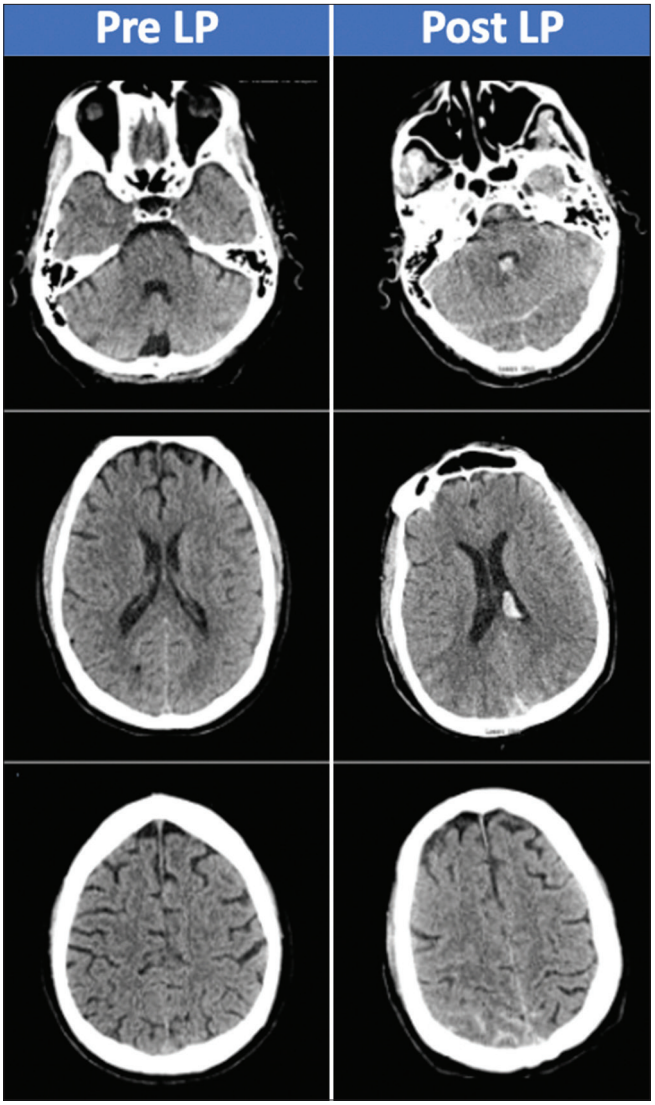

Figure 1: CT head without contrast before lumbar puncture (Pre LP) and 48 h after lumbar puncture (Post LP) demonstrated interval development of intraventricular hemorrhage in the fourth and left lateral ventricle with SAH posteriorly, most pronounced at the vertex.

The patient subsequently developed low back pain, lower extremity weakness with radiculopathy, and urinary retention. Magnetic resonance imaging of the lumbar spine demonstrated ventral extra-axial hematoma measuring 1.3 cm with compression of the cauda equina most severely at L2-L3 level [Figure 2]. Neurosurgery was consulted and the patient was taken to surgery for emergent decompression of the hematoma. Intraoperatively, the dura was tense with a durotomy noted and both intradural and extradural hematomas were evacuated.

The patient's mental status remained altered postoperatively with additional deficits of the upper extremity weakness on postbleed day 13. Transcranial Doppler (TCD) demonstrated elevated intracranial velocities (right middle cerebral arteries [MCA] velocity 251 cm/s, right posterior cerebral artery velocity 104.1 cm/s, and left MCA velocity 162.1 cm/s). Lindegaard ratio was 8.7 and 4.6 on the right and left, respectively. CT angiogram revealed moderate-to-severe narrowing of bilateral MCA and anterior cerebral arteries vessels as well as moderate narrowing of the basilar artery. Patient was started on hyperdynamic therapy and underwent a cerebral angiogram for delivery of intra-arterial spasmolytic therapy, which resolved the radiographic vasospasms [Figure 3]. Throughout his hospital course, the patient underwent one additional intra-arterial spasmolytic

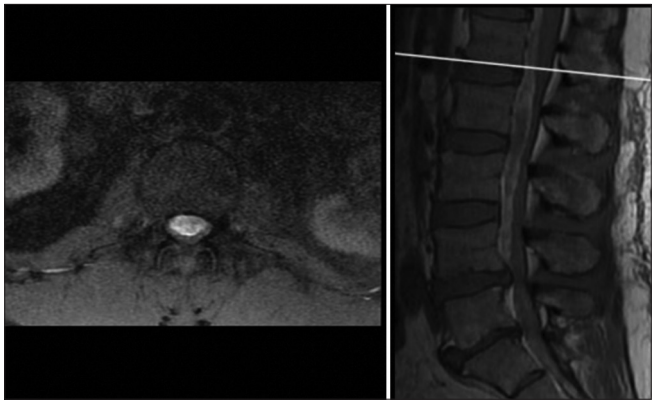

Figure 2: MRI lumbar spine with gadolinium: axial cut (left) at L1-L2 and corresponding midsagittal cut (right) demonstrates a large compressing ventral hematoma with maximal thickness at L2-L3 disc space.

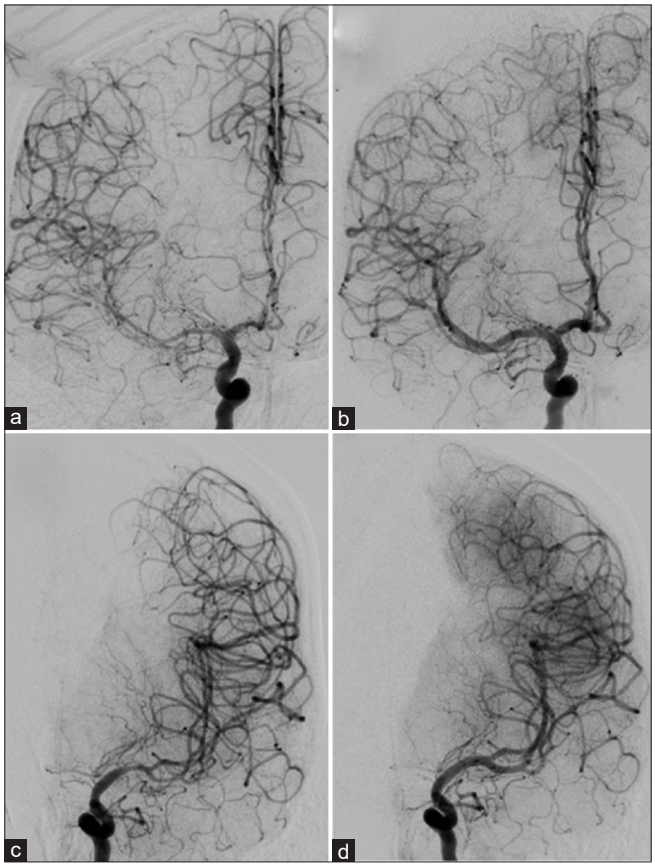

Figure 3: Cerebral angiogram, pre and post intra-arterial vasospasm treatment (a) right internal carotid artery (ICA) frontal projection pretreatment demonstrating moderate-to-severe vasospasm of the M1 and M2 segments of the MCA. ACA demonstrates mild-to-moderate vasospasm (b) right ICA frontal projection demonstrating improved vessel lumen caliber posttreatment (c) left ICA frontal projection demonstrates moderate-to-severe M1 and mild M2 vasospasm pretreatment (d) left ICA frontal projection demonstrating improved vessel lumen caliber posttreatment.

treatment for clinical vasospasm on postbleed day 15. At that time, patient had recurrent weakness of the upper extremities as well as TCD findings suggestive for vasospasm.

The patient was discharged from the hospital 45 days after admission after completing a 3-week course in the neurocritical care unit for close observation for any subsequent vasospasm and followed by rigorous inpatient physical therapy. At discharge, the patient's upper extremity weakness had resolved, but he remained paretic in the lower extremities. He was able to feed and groom himself independently and transfer himself from bedside to wheelchair (modified Rankin score [mRS] 4). At 2-year follow-up, the patient had improvement in his lower extremity strength with some deficits noted (mRS 2).

DISCUSSION

Traumatic LP in a patient taking anticoagulants is one etiology of spinal hematomas,[6,10] which may be classified as epidural, subdural, subarachnoid, or intramedullary. Diagnosis may be made radiographically, intraoperatively, or at autopsy.[8] Traumatic LP as the cause of intracranial hemorrhage or SAH is rare. Although an intradural spinal SAH and intracranial SAH could occur independently, it is more likely that the mechanisms causing one would cause the other as well. In such cases, an arterial injury is likely to have occurred causing significant bleeding and spread of blood products to the intracranial compartment. We believe that the blood products were the source of this patient's cerebral vasospasms, based on the temporal association.

There has been extensive research regarding the pathogenesis of arterial vasospasm. Some studies provide evidence that particular substances found in CSF after SAH, namely, oxyhemoglobin found in xanthochromic CSF, are the main mediators of cerebral vasospasm.[13] Within the first 24 h of SAH, there is large infiltration of white blood cells that phagocytose and breakdown red blood cells.[13] This process peaks around day 7 coinciding with the period for peak risk of cerebral vasospasm after aneurysmal SAH. This breakdown of red blood cells within the subarachnoid CSF space results in elevated levels of blood breakdown products such as vasoactive oxyhemoglobin and bilirubin.[13]

There have only been four other cases reported of spinal hematoma resulting in intracranial vasospasm as listed in [Table 1].[8,9,12,13] Three of these reports found hematoma at level of the spinal cord itself while one reported hematoma around the conus and cauda equina. In all but one case,[9] spinal hematoma associated intracranial vasospasm was also found to have intracranial hemorrhage.[8,12,13] There has been only one other reported case of LP associated intracranial vasospasm;[12] however, in this case, vasospasm

Table 1: Similar cases of spine hemorrhage resulting in intracranial vasospasm.

Case	Author	Year	Etiology of spinal hematoma	Anticoagulation	Location of spinal hematoma	Intracranial hematoma	Vasospasm
1	Shakur and Farhat[13]	2013	Spontaneous	Coumadin	T2-12 intradural	Bilateral frontoparietal subarachnoid hemorrhage	Right MCA, bilateral ACA
2	Nam *et al*.[8]	2014	Spontaneous	Heparin, dual antiplatelet	C7-T6 intradural	Bilateral parietal subarachnoid hemorrhage	Right MCA
3	Oh *et al*.[9]	2015	Spontaneous	None	C5-T4 Intradural	None	Multifocal
4	Sawaya and Sawaya[12]	2018	Lumbar puncture	None	L1-S1 intradural and epidural	Bilateral frontoparietal subarachnoid hemorrhage	Bilateral MCA, ACA, PCA
5	Current case	2022	Lumbar puncture	Coumadin	L2-3 intradural and epidural	Bilateral parietal subarachnoid hemorrhage, intraventricular hemorrhage, tentorial subdural hematoma	Bilateral MCA and ACA

was not treated and patient ultimately developed permanent quadriplegia secondary to cerebral infarction. In our case, there was only a small amount of intracranial blood along the posterior peripheral sulci and ventricular systems without evidence of increased intracranial pressure. This suggests that cerebral vasospasm had resulted from vasoactive blood breakdown products carried in the CSF. Clinical cerebral vasospasm is not classically considered among the risks of LP; however, we have outlined other cases in addition to ours that present this phenomenon. Furthermore, adequate assessment of indications and contraindications should be evaluated before proceeding with LP.

CONCLUSION

LP is not an emergent nor benign procedure. Therefore, proper indications, contraindications, and all risks should be considered before performing it, especially in a coagulopathic patient. We report a case of traumatic LP resulting in a patient developing nonaneurysmal SAH and subsequent intracranial vasospasm. This complication could have been avoided in this case if, first, the indication and necessity for LP were considered and, second, if the risk of completing the procedure with coagulopathy was considered. Our case demonstrates the successful and timely treatment necessary to improve outcomes for patients who develop this complication.

Declaration of patient consent

The authors certify that they have obtained all appropriate patient consent.

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Nil.

Conflicts of interest

There are no conflicts of interest.

REFERENCES

1. Adler MD, Comi AE, Walker AR. Acute hemorrhagic complication of diagnostic lumbar puncture. Pediatr Emerg Care 2001;17:184-8.
2. Armon C, Evans RW. Addendum to assessment: Prevention of post-lumbar puncture headaches: Report of the therapeutics and technology assessment subcommittee of the American Academy of Neurology. Neurology 2005;65:510-2.
3. Breuer AC, Tyler HR, Marzewski DJ, Rosenthal DS. Radicular vessels are the most probable source of needle-induced blood in lumbar puncture: Significance for the thrombocytopenic cancer patient. Cancer 1982;49:2168-72.
4. Chakraverty R, Pynsent P, Isaacs K. Which spinal levels are identified by palpation of the iliac crests and the posterior superior iliac spines? J Anat 2007;210:232-6.
5. Frederiks JA, Koehler PJ. The first lumbar puncture. J Hist Neurosci 1997;6:147-53.
6. Gustafsson H, Rutberg H, Bengtsson M. Spinal haematoma following epidural analgesia. Report of a patient with ankylosing spondylitis and a bleeding diathesis. Anaesthesia 1988;43:220-2.
7. Kreppel D, Antoniadis G, Seeling W. Spinal hematoma: A literature survey with meta-analysis of 613 patients. Neurosurg Rev 2003;26:1-49.
8. Nam KH, Lee JI, Choi BK, Han IH. Intracranial extension of spinal subarachnoid hematoma causing severe cerebral vasospasm. J Korean Neurosurg Soc 2014;56:527-30.
9. Oh JH, Jwa SJ, Yang TK, Lee CS, Oh K, Kang JH. Intracranial Vasospasm without Intracranial Hemorrhage due to Acute Spontaneous Spinal Subdural Hematoma. Exp Neurobiol 2015;24:366-70.
10. Perrini P, Pieri F, Montemurro N, Tiezzi G, Parenti GF. Thoracic extradural haematoma after epidural anaesthesia.

Neurol Sci 2010;31:87-8.
11. Sandoval M, Shestak W, Stürmann K, Hsu C. Optimal patient position for lumbar puncture, measured by ultrasonography. Emerg Radiol 2004;10:179-81.
12. Sawaya C, Sawaya R. Central nervous system bleeding after a lumbar puncture: Still an ongoing complication. Am J Case Rep 2018;19:1103-7.
13. Shakur SF, Farhat HI. Cerebral vasospasm with ischemia following a spontaneous spinal subarachnoid hemorrhage. Case Rep Med 2013;2013:934143.

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