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Central Nervous System Bleeding After a Lumbar Puncture: Still an Ongoing Complication

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Study Design A
Data Collection B
Statistical Analysis C
Data Interpretation D
Manuscript Preparation E
Literature Search F
Funds Collection G

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Case series

Patient: Female, 84 • Male, 77
Final Diagnosis: CNS bleeding
Symptoms: Pain • subcutaneous hematomas
Medication: —
Clinical Procedure: Lumbar puncture
Specialty: Neurology

Objective: Diagnostic/therapeutic accidents





Background: A lumbar puncture is a procedure performed to uncover the state of the central nervous system by analysis of the cerebrospinal fluid. It is done also to infuse medications in the subdural space. A lumbar puncture should not cause central nervous system bleeding, but this complication is still occurring in certain cases.

Case Report: We present 2 cases where a lumbar puncture was performed in the emergency department. The first patient had severe inflammatory lower back pain and received epidural steroids through a lumbar puncture and the second case presented with the clinical picture of meningitis and a lumbar puncture was performed for diagnostic purposes. In both cases, major complications arose secondary to bleeding in the cerebrospinal fluid. The first case developed a bleeding tendency because the patient had acute renal failure and was on low molecular weight heparin. The second case had low platelet count because of myelodysplasia. Both cases bled into the subarachnoid space and subdural space resulting in compression of the cauda equine and paralysis. The bleeding eventually flowed into the posterior fossa resulting in vasospasm of the posterior circulation and infarction of the posterior cerebral arteries.

Conclusions: We concluded that both patients sustained complications from the lumbar puncture because of a bleeding tendency secondary to systemic illnesses and multiple drugs and their side effects. We recommend that patients' medical condition be well evaluated, and proper blood studies be performed prior to lumbar punctures to avoid major morbidities.

MeSH Keywords: Guideline Adherence • Hematologic Diseases • Morbidity • Spinal Puncture • Vitamin K Deficiency Bleeding

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Background

A lumbar puncture is a procedure not without risks. It is usually performed either as a diagnostic test to evaluate a patient's central nervous system for disease or as a therapeutic procedure to infuse medication into the cerebrospinal fluid. There are certain conditions where a lumbar puncture is contraindicated such as in cases with intracranial hypertension to avoid uncal herniation or cerebellar herniation [1], and in patients with bleeding disorders such as acute lymphoblastic leukemia [2]. We report on 2 cases where a lumbar puncture resulted in major morbidity and mortality even when performed following the recommended guidelines and in a university medical center.

Case Reports

Case 1

An 84-year-old female presented to the emergency department for post-traumatic sternal pain. She received nonsteroidal and opioid drugs to relieve the pain and low molecular heparin because she was on bed rest. Her neurologic examination revealed normal mental status and speech. She had normal motor power and sensation in her 4 extremities. She had no Babinski signs. Her gait was normal. Her cranial nerves were normal.

Baseline hematology and chemistry blood studies were normal and magnetic resonance imaging (MRI) of her lumbosacral spine revealed degenerative spondylarthrosis of the lumbosacral spine with degenerated discs and facet joint hypertrophy. Because of chronic lower back pain, she underwent an epidural block with lidocaine and high dose steroid. The procedure was performed at the bedside through a lumbar puncture, under local anesthesia, at the L3–L4 interspace, with the patient lying prone on her left side. The needle was inserted until it gave way upon puncturing the dura. The opening pressure was 11 cm H₂O. The procedure itself was uneventful, but less than an hour later the patient started complaining of severe headache and blurring of vision, followed by weakness in her lower extremities. The findings were attributed to the lumbar puncture. A few days later, her level of consciousness had gradually dropped, and her breathing had become slow and superficial. She was intubated. She was somnolent but grimaced to nociception. She had neck stiffness. She opened her eyes spontaneously but did not follow the light. Pupillary reflexes were preserved and symmetrical. She had no papilloedema. Oculocephalic reflex was present. She was paralyzed in the lower extremities with no response to pain. She weakly withdrew her arms to severe nociception. Deep tendon reflexes were absent, but she developed bilateral Babinski signs.

Her sensory level could not be assessed reliably. She had a lax anal sphincter.

MRI of the brain and whole spine revealed a cortical-subcortical high FLAIR signal with evidence of restricted diffusion at the level of the right and left occipital lobes and medial parietal lobes suggestive of acute bilateral posterior cerebral artery (PCA) stroke. High signal was also seen in the prepontine and basal cisterns in keeping with subarachnoid hemorrhage. magnetic resonance angiography (MRA) of the intracranial vessels showed significant decrease in the caliber of the middle cerebral artery (MCA), anterior cerebral artery (ACA) and PCA vessels bilaterally secondary to vasospasm. (Figure 1A) The MRI of the lumbar spine revealed a large intradural hematoma extending to the sacral area. There was also an epidural hematoma at the level of the L4 vertebra posteriorly, and increased signal on T1-weighted images in the epidural space consistent with blood. (Figure 1B).

The relevant blood studies at that time were platelet count 261 000 and creatinine of 3.25 mg/dL (normal 0.6–1.2 mg/dL). Electrolytes, prothrombin time, international normalized ratio (INR), and liver function tests were normal 3 days prior to the procedure, but not tested on the same day.

The patient had developed acute renal failure secondary to high dose intravenous nonsteroidal anti-inflammatory drugs (NSAID) and urinary retention secondary to continuous opioid drugs. Furthermore, she had been anticoagulated by low-molecular-weight heparin (LMWH) for several days, the effect of which may have become enhanced by the patient's renal failure. This condition may have resulted in an epidural and subdural bleeding during the lumbar puncture. When the bleeding occurred and the cauda equine was compressed, the patient developed progressive paralysis of the lower extremities and compression of the innervation of the sphincters. When the patient was placed in the recumbent position, the blood receded to the cervical cord and the base of the skull, filling the cisterns and causing vasospasm of the circle of Willis. This eventually led to cerebral infarction predominantly in the distribution of the PCAs. The cerebral infarctions and edema caused the patient's altered level of consciousness and paralysis.

Case 2

A 77-year-old male suffering from chronic myelodysplastic syndrome, who was maintained on repeated transfusions, was brought to the emergency department by his family because of several days' history of somnolence, lethargy, generalized fatigue, and confusion. There was no history of fever or chills, headaches, or abdominal pain, nausea, vomiting, or diarrhea. In the emergency department, his blood pressure was 180/95 mmHg, pulse was 82 beats per minute, oxygen

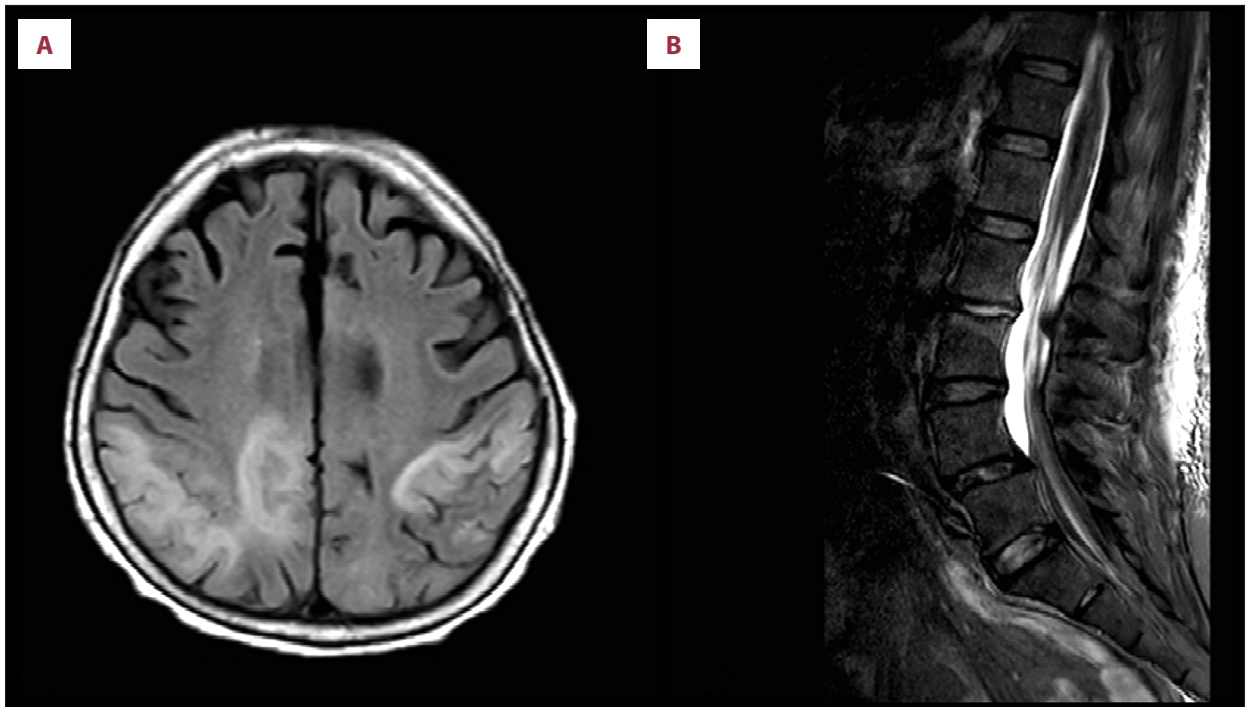


Figure 1. (A) Magnetic resonance imaging (MRI) of the brain: axial T2-weighted FLAIR image revealing bilateral hemispheric infarcts and subarachnoid hemorrhage. (B) MRI of the lumbosacral spine: STIR image revealing intradural and epidural hematoma and clumping of the nerve roots in the cauda equine.

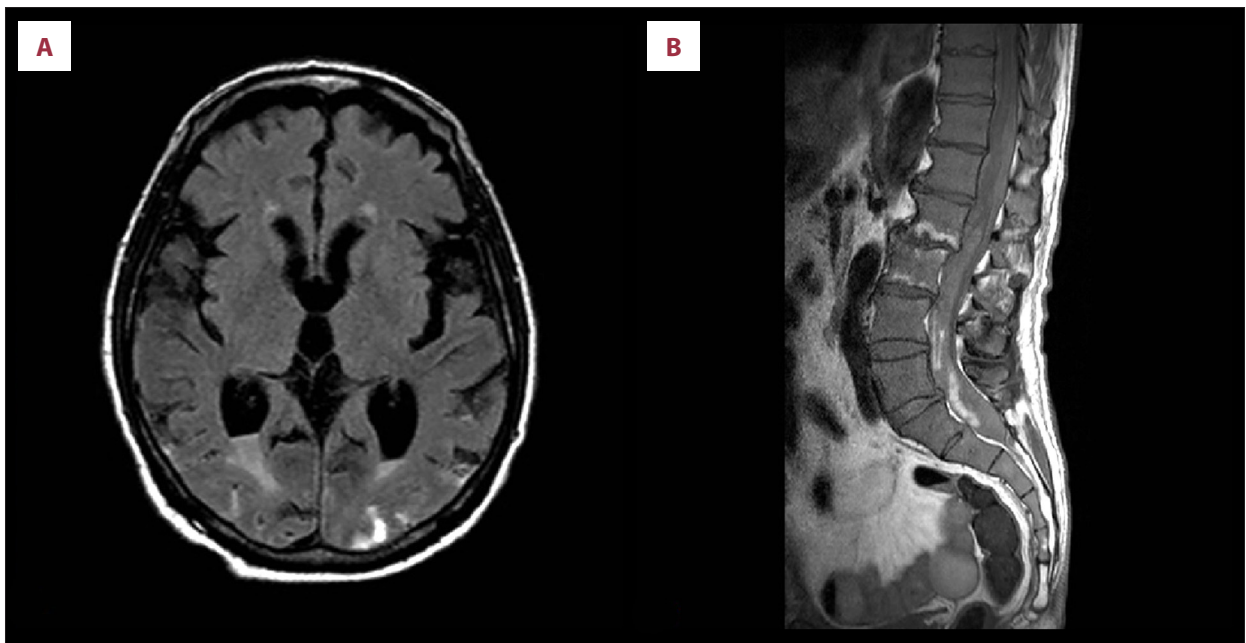


Figure 2. (A) Magnetic resonance imaging (MRI) of the brain: axial T2-weighted FLAIR image revealing diffuse subarachnoid hemorrhage and blood in the dependent portion of the lateral ventricles. (B) MRI of the lumbosacral spine: sagittal T1-weighted image revealing blood surrounding the spinal cord and cauda equine nerve roots.

saturation 100%, respiratory rate 20 breaths per minute, and temperature orally was 37°C. He looked pale and fatigued, but alert and responsive. His speech was normal. He had clear lung

sounds and a soft abdomen. He had multiple large subcutaneous ecchymosis all over his body. He had normal cranial nerves and no neck stiffness. He had normal and symmetrical motor

power in the muscles of his upper and lower extremities. Deep tendon reflexes were symmetrically depressed in the 4 limbs. He had no Babinski signs and no cerebellar signs. Computed tomography (CT) scan of the brain was normal. White blood cell (WBC) count was 2000 cell/mm³, hemoglobin was 11 gm/dL, hematocrit was 33%, platelet count was 71 000/mm³, creatinine was 1.0 mg/dL, sodium was 124 mmol/L, potassium was 4.3 mmol/L, INR was 1.1, and prothrombin time was 31 seconds (normal 27–39 seconds). The rest of the routine chemistry studies were normal.

A lumbar puncture was performed in the emergency department with a 22-gauge needle, under local anesthesia, at the L3–L4 interspace, with the patient lying prone on his left side. The needle was inserted until it gave way upon puncturing the dura. The opening pressure was 23 cm H₂O. The results revealed WBC of 92 cells/mm³ with 99% lymphocytes and 1% monocytes, red blood cell (RBC) of 4 cells/mm³, protein of 0.96 gm/L, sugar was 38 mg/dL with blood sugar 107 mg/dL, and a positive PCR for herpes simplex virus type 2. The patient was started on intravenous acyclovir drip.

A few hours after the lumbar puncture, the patient started complaining of severe lower back pain radiating to both thighs. This was followed by progressive weakness in the lower extremities. An MRI of the spine revealed blood within the spinal canal surrounding the spinal cord and cauda equine nerve root extending from the lower cervical spine and along the length of the thoracic spine to the sacral thecal sac. Blood cerebrospinal fluid level was seen at C6 level. A heterogeneous signal intensity was seen occupying the spinal canal at the lumbar level in keeping with acute blood causing mass effect on the conus medullaris (Figure 2B). In the days that followed, the patient became more stuporous and confused, his speech became dysarthric and incomprehensible, and he started to complain of severe headache and weakness even in his upper extremities. MRI of the brain revealed diffuse bilateral subarachnoid hemorrhage involving the dependent portions of the cerebral hemispheres as well as the posterior fossa. Layering blood was seen along the dependent portion of the lateral ventricles bilaterally (Figure 2A). Eventually, the patient developed respiratory insufficiency requiring intubation and then sustained a cardiac arrest and passed away.

Discussion

A lumbar puncture is a common procedure performed as a diagnostic test to measure intracranial pressure, or to evaluate meningeal inflammation, infection, or malignancy of the central nervous system [3]. It may be necessary in patients with bacterial meningitis not responding adequately to antibiotics because of an unusual infection such as mycobacterial or

fungal infections. It may also be important to diagnose herpetic meningoencephalitis because the treatment is specific, prolonged, and nephrotoxic. Additionally, lumbar puncture is important in diagnosing infection or subarachnoid hemorrhage in patients with acute headaches or acute altered level of consciousness [3]. Furthermore, it is sometimes therapeutic with the infusion of steroids, anesthetic agents, opiates, botulinum toxin and chemotherapy in patients with central nervous system malignancy [4].

Complications of a lumbar puncture are rare. Bacterial meningitis has been reported following non-sterile lumbar punctures [5]. Additionally, cases of intrathecal bleeding after a lumbar puncture exist and are mostly due to traumatic taps or bleeding disorders [2,6].

Complications resulting from a lumbar puncture can be mild and resolve spontaneously, such as local pain, skin infection, abscess formation, post lumbar puncture headaches, and injury to a lumbosacral spinal nerve. However, our cases are examples of major complications from a lumbar puncture. Both patients developed intraspinal hemorrhages which extended rostrally to the posterior fossa resulting in bleeding in the pre-pontine cisterns and cerebral hemispheres provoking severe vasospasm leading to ischemia and infarctions of the posterior circulation.

Most guidelines recommend that a lumbar puncture should be performed only in cases with a high suspicion of cerebral disease and with a platelet count of more than 50 000/mL and an INR of less than 1.4 [6,7]. Lumbar punctures should be performed 12 hours after the last dose of LMWH [7].

Lumbar punctures performed in our 2 patient cases abided by the guidelines but resulted in major morbidity and mortality. In retrospect, we concluded that the patients were at a higher risk than we first ascertained. The patient in our first case developed acute renal failure secondary to high dose NSAIDs. The reduced glomerular filtration rate caused a decrease in the excretion of LMWH from the kidneys, resulting in hemorrhage after the lumbar puncture. The prothrombin time and INR results that were normal 3 days prior to the lumbar puncture were not representative of the patient's propensity to bleed on the day of the lumbar puncture. In our second case, the patient had a high risk of bleeding because of myelodysplasia. Bleeding in myelodysplasia is usually due to thrombocytopenia. However, our patient developed bleeding complications despite having a platelet count greater than 50 000/mL. Another cause of bleeding in patients with myelodysplastic syndrome is platelet dysfunction. Studies have shown that patients with a normal platelet count could have a prolonged bleeding time and impaired platelet aggregometry with arachidonic acid, collagen, and epinephrine [8].

There are no guidelines that specify a platelet threshold prior to a lumbar puncture, and the evidence supporting the recommendation of raising the platelet count above 50×10^9 cells/L is “very low-quality evidence” [6–9]. The present evidence is more confusing than clear because of the different recommendations presented by different research groups [10].

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Conclusions

We conclude that the present guidelines do not insure a safe lumbar puncture procedure and thus we recommend adding platelet aggregometry studies for patients with myelodysplastic syndrome to confirm adequate primary hemostasis before performing a lumbar puncture. Furthermore, we recommend that renal function tests, liver function tests, INR, and prothrombin time be performed on the day of the lumbar puncture in patients on LMWH and NSAIDs.