Post lumbar puncture leg weakness mimicking cauda equina syndrome

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Case report

A 60-year-old man was scheduled for his third cycle of intrathecal Methotrexate as prophylaxis central nervous system treatment for Stage 1Ae diffuse large B cell lymphoma of the left nasal cavity. His previous lumbar puncture (LP)s were technically difficult. Due to his prior traumatic experience, it was decided that more analgesia be administered locally to alleviate his pain and anxiety.

The patient was well built and slim, mobilised independently prior to LP. He had neither medical problems nor medications prior to his diagnosis and had no allergies. He had normal platelet count and coagulation screen.

Under aseptic technique, standard landmarks were identified. The patient was positioned in the left lateral position. L3 and L4 disc space interfaces were identified. Skin and subcutaneous tissue was infiltrated with eight millilitre (ml) of one percent Lignocaine using a 21 gauge needle. Routine aspiration was performed during administration of local anaesthetic with no venous flashback.

LP was performed using a 22 gauge tuohy needle with a single pass using the same tract. The patient tolerated the procedure well. Five ml of initial bloody cerebrospinal (CSF) fluid eventually became colourless. CSF fluid was sent for analysis (Table 1) after which 12.5mg in 5ml of intrathecal Methotrexate was administered by a certified doctor as per protocol. There was no resistance on intrathecal chemotherapy administration.

The patient described sensation of gradual onset paraesthesia followed by bilateral persistent leg weakness upon removal of the LP needle. He denied any back pain, nausea, or headache. His vital signs were stable. Lower limb neurology showed reduced tone bilaterally & absent reflexes. Babinski reflex was down going. Power on hip flexion, & knee extension was 4/5. Power on foot dorsiflexion, plantar flexion & knee flexion was 3/5.

<table>
<thead>
<tr>
<th>CSF fluid Constituents</th>
<th>Result</th>
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</thead>
<tbody>
<tr>
<td>Macroscopic appearance</td>
<td>Clear and colourless</td>
</tr>
<tr>
<td>White Blood Cell Count</td>
<td>Tube one: &lt;1x10⁶/L</td>
</tr>
<tr>
<td></td>
<td>Tube two: 1 x 10⁶/L</td>
</tr>
<tr>
<td></td>
<td>Tube three: &lt;1 x 10⁶/L</td>
</tr>
<tr>
<td>Red Blood Cell Count</td>
<td>Tube one: 80 x 10⁶/L</td>
</tr>
<tr>
<td></td>
<td>Tube two: 70 x 10⁶/L</td>
</tr>
<tr>
<td></td>
<td>Tube three: 56 x 10⁶/L</td>
</tr>
<tr>
<td>Chemistry:</td>
<td></td>
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<tr>
<td>CSF Glucose</td>
<td>2.8 (2.8 – 4.4 mmol/L)</td>
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<tr>
<td>CSF Protein</td>
<td>0.18 (0.15–0.45) g/L</td>
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<tr>
<td>Bacterial Culture</td>
<td>No growth</td>
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</tbody>
</table>
Perianal sensation and tone were absent. Sensation to light touch and pin prick was diminished below L3 with a sensory level at L3. Feet were cool to touch with intact pulses. Upper limb neurology was normal. Abdomen was soft and non tender with no peritonism. No bleeding was noted from the LP puncture site.

Our patient was kept under close observation. His leg weakness began to improve two hours post LP. However, he was still unsafe to weight bear and had to be admitted to the ward for further observation. His neurological weakness & bladder function fully recovered six hours post LP and he was subsequently discharged home. No post LP imaging was performed as his neurology was improving with complete resolution. He declined subsequent intrathecal chemotherapy administrations. He has now had complete response following four cycles of R-CHOP chemotherapy, and has completed radiation therapy to the nasal cavity for consolidation therapy. He remains on routine surveillance.

**Discussion**

In more than 5% of cases, back ache and shooting pain down the legs occur during LP. This usually resolves upon completion of the procedure. Bleeding may occur more commonly due to either intrinsic factors, for example thrombocytopenia & deranged coagulation profile or extrinsic factors for example anticoagulants & antiplatelet agents. This may be immediate or delayed. Post lumbar puncture headache is described to occur between 13–32% with a median duration of five days.1 Cauda Equina Syndrome (CES) is an exceedingly rare complication post LP, ie, between 0.01 and 0.7%.2 Differential diagnosis of CES in this case include lignocaine neurotoxicity, spinal dural AV fistula (SDAVF) & epidural hematoma.

Classical CES is rare and occurs in 1 in 33,000 cases. For unknown reasons, women (63%) are more commonly affected than men (37%). It is classically accompanied by sciatica, saddle anaesthesia, sexual dysfunction and leg weakness. The gold standard imaging is MRI. The main causes of the syndrome include disc herniation in more than two thirds of cases, followed by tumour, spinal fractures, canal stenosis, infections, post-surgical manipulation, post-spinal anaesthesia, ankylosing spondylitis and fire arm wounds. Urgent surgical decompression is usually done 48 hours before onset of symptoms and usually improves neurological sequelae and improves prognosis.3

The initial impression in this case was that of leg weakness secondary to spinal lignocaine causing neurotoxicity as that caused by an epidural.1 This is supported by the rapid onset of symptoms and the patient’s reversible neurology. Our patient’s sensory level was below L3, and hip flexion was also noted to be weak. This is likely due to spinal lignocaine infiltrating motor nerve roots of L2 & L3 as well. A Cochrane review of local anaesthetics showed that the risk of developing transient neurologic symptoms (TNS) is about seven times higher for lignocaine compared to bupivacaine, prilocaine, procaine, ropivacaine and levobupivacaine. Despite this risk of TNS, lignocaine’s benefit outweighs its risk in comparison to other local anaesthetics as it works much more rapidly, has a shorter half-life and has a much more intense nerve blockade.5 Our patient denied any back pain or gluteal pain which made this an atypical presentation of CES. Unintentional intrathecal injection of large doses of local anaesthetic may also cause hypotension, respiratory compromise, and in rare cases, seizures and cardiac arrest, which were absent in this case.

CES after an epidural block may also be secondary to spinal cord ischemia from spasm of the anterior spinal artery or the Adamkiewicz artery. Kim et al have also described a case of transient CES, but related to a sacral schwannoma with cauda equina compression after a lumbar epidural block.6 Neurotoxicity secondary to repeated intrathecal chemotherapy administrations have been described in a few case reports. These more often cause gradual onset of neurological weakness ranging from weeks to months. In these instances, neurological weakness is much more prolonged in duration. In some instances, the neurology recovers with rehabilitation and some patients may have permanent neurological disability. In some cases, paraplegia has also been described.7
Transient leg weakness post LP has also been described post myelography, epidural steroid injection & spinal dural arteriovenous fistula (SDAVF) which were less likely given the patient's history, prior normal CT imaging of his back and relatively rapid resolution of symptoms. Nevertheless, CT is not a sensitive test for SDAVF and hence we are unable to completely exclude the presence of fistula in this patient. SDAVF is the most common vascular malformation of the spine and typically present in older men. Leg weakness exacerbated by exercise is a common manifestation of thoracic SDAVF and occurs in 43% of patients. Fistula level only corresponds to sensory level in 40% of cases and often cannot guide the level of imaging, hence warranting imaging of the entire spine when SDAVF is suspected.

Other differentials include epidural hematoma. A prospective study by Kang et al evaluating major complications of epidural anaesthesia in more than 5,000 patients only revealed one case of epidural hematoma (0.02%) and post-operative neurological deficits in 57 patients (1.12%). Spinal epidural hematoma may present with signs that include local back pain, paraparesis, sensory loss with a discernible level, and bowel or bladder incontinence. Urgent MRI of the spine is warranted if there is clinical suspicion of spinal epidural hematoma. The American College of Physicians best practice advice recommend urgent MRI imaging of the back in patients with acute low back pain who have risk factors for spinal infection (new onset of low back pain with fever and history of intravenous drug use or recent infection), risk factors for or signs of the CES (new urinary retention, faecal incontinence, or saddle anesthesia), or severe or progressive neurologic deficits. The absence of lower back pain, risk factors for bleeding and improving neurological signs gave us the reassurance to observe the patient clinically with regular neurological observations. These explanations also gave the patient reassurance.

In summary, although rare, leg weakness mimicking CES post LP needs to be recognised as a potential complication of LP. Superficial as opposed to deeper infiltration of local anaesthesia is recommended. A smaller volume of local anaesthesia infiltration, without compromising on patient’s level of comfort is also suggested. Clinical acumen is critical in identifying patients that require urgent imaging post LP, particularly in those with hemodynamic compromise and persistent or progressive neurological weakness, as they may require surgical decompression or intensive care monitoring.

Competing interests: Nil
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REFERENCES:


5. Zaric D, Pace NL, Punjasawadwong Y. Transient neurologic symptoms (TNS) following spinal anesthesia with lidocaine versus other local anesthetics Cochrane Anesthesia Group. 15 April 2009.


