Neurotoxicity post-epidural anesthesia in a patient with Vitamin B12 deficiency: a case report

Christelle Ibrahim

christelle.ibrahim@lau.edu

Lebanese American University

Nancy Maalouf

Lebanese American University

Case Report

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Abstract

Background: Neurotoxicity have been reported after central neuraxial block. Some metabolic disturbances (thiamine deficiency, diabetes) were identified as contributing factor.

Case presentation: We describe a young woman with vitamin B12 deficiency who developed persistent lower extremity weakness and hypoesthesia for weeks after epidural anesthesia. Initial examination revealed bilateral distal lower extremities weakness and decreased sensations. Serial neurologic examinations showed improved motor and sensory findings. An injected lumbosacral MRI was unremarkable. Vitamin B12 level was very low. The patient received physiotherapy and Vitamin B12 supplementation. Nerve conduction study and electromyography on day-23 post-epidural anesthesia revealed evidence of peripheral nerve injury.

Conclusion: This case is the first to recognize vitamin B12 deficiency as a predisposing factor for the development of neurotoxicity following epidural anesthesia, it also emphasizes the importance of timely identification of vitamin deficiencies in patients developing neurotoxicity following central neuraxial blocks.

Introduction

Several neurologic complications are described following different types of central neuraxial blocks (CNBs) including spinal, epidural, and combined spinal epidural (CSE) anesthesia. The most reported are spinal epidural hematomas, spinal cord ischemia, cauda equina syndrome, meningitis, arachnoiditis, and nerve injury[1].

Anesthesia-related neuropathies are usually attributed to mechanical injuries, ischemia, or local toxicity. Several pathways for the latter have been suggested but no single mechanism identified as predominant. The “double crush syndrome” postulates that peri-operative stressors may trigger symptomatic neuropathies in pre-existing subclinical neurologic pathologies[2].

We report a patient with vitamin B12 deficiency who developed distal lower extremities (LEs) weakness and hypoesthesia post-epidural anesthesia (EA).

Case presentation

A 30-year-old woman (G1P1) known to have vitamin B12 deficiency on 5,000mcg of sublingual methyl-cobalamin weekly presented for normal vaginal delivery (NVD). EA was performed under sterile techniques at the L4-L5 level. The epidural space was identified by loss of resistance to fluid, the catheter placed without difficulty and taped at 9cm, and the analgesia mixture (Ropivacaine 0.2%, Fentanyl 0.2mcg/ml) administered at a rate of 10ml/hr. Hemodynamic stability was maintained throughout the procedure and delivery. As the anesthesia was more effective over the right LE, the patient was

repositioned in left lateral decubitus. Six hours after EA initiation, the patient had an uneventful NVD following 20 minutes of lithotomy position.

Neurology team was consulted ten hours after the epidural catheter removal because of persistent numbness and weakness below knees bilaterally, and urination difficulty requiring Valsalva maneuvers. Further questioning was negative for a history of coagulopathy, previous neurologic deficit, antiplatelet or anticoagulant intake.

Neurologic examination revealed decreased strength in the LEs distally more than proximally and left more than right (Table 1). Deep tendon reflexes were present symmetrically with bilateral flexor plantar responses. Sensory exam revealed decreased light touch/pinprick/cold sensation in the posterolateral legs and the feet, more severe on the left. It also showed decreased vibration and absent proprioception in the distal LEs.

An injected lumbosacral spine Magnetic Resonance Imaging (MRI) was unremarkable. Vitamin B12 level was 109 pg/mL so daily intramuscular injections were initiated (Vitamin B12 1,000mcg, B1 100mg, B6 100mg).

Neurological deficits improved gradually. Upon discharge (day-2 post-EA), the patient had mild residual weakness in the left foot (Table 1), and minimal decreased light touch and pinprick in the right sole and left posterolateral leg. The discharge diagnosis was probable neurotoxicity related to EA.

Table 1
Motor Power in the lower extremities day-1 and day-2 post- EA

Motor Power	Day 1		Day 2	
	Left	Right	Left	Right
Hip Flexion	4+	5-	5	5
Knee Flexion	3	3+	5	5
Knee Extension	5	5	5	5
Knee Abduction	5	5	5	5
Knee Adduction	5	5	5	5
Inversion	1	3	4+	5
Eversion	1	3	3	5
Dorsiflexion	1	3	3	5
plantarflexion	1	3	4+	5

The patient received intensive out-patient physiotherapy. Due to persistent symptoms, Nerve Conduction studies/Electromyography (NCS/EMG) was performed on day-23 post-EA and revealed normal NCS with decreased recruitment in the left L5-S1 myotomes suggesting nerve injury to the corresponding roots. Supplementation with Vitamin B12 was continued.

Discussion

Neurotoxicity has been reported after all types of CNBs. One case report described a patient who developed unilateral leg paralysis after CSE anesthesia; the neurotoxicity was diagnosed and followed up by serial examinations and NCS/EMGs[3]. Another paper reported a patient with new onset LEs weakness and paresthesia two weeks after spinal anesthesia. The patient was found to have axonal neuropathy related to thiamine deficiency and improved after thiamine supplementation[4]. A case of neurotoxicity manifesting as paresthesia was also reported after EA with Ropivacaine in a patient with diabetic sensory polyneuropathy[5].

In our case, a standard amount of ropivacaine/fentanyl was administered during the anesthesia, and the catheter was removed post-delivery. Based on the initial neurologic examination, the most likely site of injury was either the spinal cord, the nerve roots, or the peripheral nerves. CNS pathologies such as spinal hematoma, epidural abscess, anterior spinal cord syndrome, and arachnoiditis were ruled out by a negative lumbosacral MRI, while the NCS/EMG confirmed the diagnosis of anesthetic root neurotoxicity. By identifying the epidural space through the loss of resistance to fluid technique, the anesthesiologist confirmed that the tip of the epidural catheter did not enter the arachnoid space.

Based on the theory of the “double crush syndrome” and the fact that EA was found to be associated with the development of severe neurologic deficit post-operatively in 0.4% of patients with a known neuropathy[6], the patient's pre-existing vitamin B12 deficiency may have made her more susceptible to the development of neurotoxicity after receiving EA, in the setting that a standard amount of anesthetic mixture was used.

Nerve toxicity secondary to anesthesia usually appears bilaterally, but in this case the symptoms rapidly improved on the right side and remained for weeks on the left. This asymmetry is most likely attributable to the repositioning of the patient and a subsequent probable higher exposure of the left LE nerves to the anesthetics.

To our knowledge, this is the first paper reporting a neurotoxicity post-CNB associated with vitamin B12 deficiency. This case suggests that vitamin B12 deficiency may predispose patients to anesthetic nerve injury, even in the setting of a normal NCS. Additionally, it highlights the importance of identifying and treating pre-existing conditions that may predispose patients to neurological complications prior to administration of epidural anesthesia.

Abbreviations

Central neuraxial blocks (CNBs); combined spinal epidural (CSE); lower extremities (LEs); epidural anesthesia (EA); normal vaginal delivery (NVD); Magnetic Resonance Imaging (MRI); Nerve Conduction studies/Electromyography (NCS/EMG).

Declarations

Ethics approval and consent to participate

As this is a single case report an institutional review board approval was not required.

Consent for publication

A written informed consent form was obtained from the patient for the publication of this case report. A copy of the written consent is available for review by the Editor of this journal.

Availability of data and material

All data underlying the results are available as part of the article and no additional source data are required.

Competing interests

The authors have no conflict of interests to declare.

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Authors contribution

CI wrote the main manuscript text and prepared the figure with input from NM. NM supervised and validated the article. All authors read, reviewed, and approved the final manuscript.

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