CASE REPORT

WAS IT CRITICAL ILLNESS NEUROPRAXIA IN
A POSTPARTUM PATIENT WHEREIN NEUROLOGICAL
SYMPTOMS AND SIGNS SPONTANEOUSLY
RESOLVED WITHIN 24 HRS?

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Abstract

Critical illness polyneuropathy is a well-known condition that is among the etiologies underlying the commonly diagnosed neuromuscular weakness among intensive care unit patients. As similar to critical illness myopathy, critical illness polyneuropathy typically has onset, progression and resolution (complete or incomplete) over a period of days to weeks to months. However, we herein present a postpartum septic shock patient whose neurological symptoms developed and then resolved over a period of 24-hours, pointing to the possibility of transient occurrence of critical illness neuropraxia (CIN).

Keywords: critical illness polyneuropathy; critical illness myopathy; critical illness neuropraxia; postpartum septic shock; peripheral nervous tissue hypoperfusion; spinal cord concussion

Introduction

Critical illness polyneuropathy is a well-known condition that is among the etiologies underlying the commonly diagnosed neuromuscular weakness among intensive care unit (ICU) patients1,2. As similar to critical illness myopathy, critical illness polyneuropathy typically has onset, progression and resolution (complete or incomplete) over a period of days to weeks to months. However, after informed and written consent, we herein present a postpartum septic shock patient whose neurological symptoms developed and then resolved over a period of 24-hours, pointing to the possibility of transient occurrence of critical illness neuropraxia (CIN).

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Case Presentation

A 20-year-old full term pregnant patient presented for induction of labor. The patient had undergone a spontaneous vaginal delivery with analgesic support from uneventful labor epidural placement and continuous epidural analgesic regimen. The epidural catheter was removed immediately post-delivery per protocol. The next morning, during postpartum day-one clinical rounds, the patient did not complain of any neurological symptoms; however oral temperature peaking at 40.0 degrees Celsius, leucocytosis (22.1 K/Cumm) with neutrophilia (18.6 K/CUMM), and subsequent blood culture positive for Streptococcus pyogenes (Group A Strep) were observed. As puerperal sepsis was suspected, an antibiotic course of ampicillin, clindamycin and gentamicin was started. During the subsequent twelve hours, the patient reported new onset weakness in both lower extremities with inability to walk. The patient began to exhibit deteriorated vital signs with systolic blood pressures 80s-90s mmHg, heart rates 140s-150s beats per minute, respiratory rates 20s-30s breaths per minute, and oral temperatures peaking at 41.4 degrees Celsius. Neurology and ICU teams were consulted. As per the neurologist’s evaluation of patient’s muscle power, left hip flexors were 3/5, left hip extensors were 4/5, left knee flexors were 3/5, left knee extensors were 4/5, left foot dorsiflexors were 3/5 and left foot plantar flexors were 3/5. Hypotonicity in both lower extremities was observed. Fine touch, pin prick, temperature sensation, vibration and proprioception were decreased in left leg below the first lumbar dermatome. Knee reflexes were subnormal bilaterally, while ankle reflex was subnormal only on right side. Urgent magnetic resonance imaging of lumbar spine ruled out any epidural hematoma or abscess. The leucocytosis (31.9 K/CUMM) with neutrophilia (22.0 K/CUMM) worsened and a high anion-gap (17 millimoles/liter) lactic acidosis (5.9 millimoles/liter) was observed. The patient was diagnosed with septic shock and transferred to ICU where non-rebreather oxygen therapy, norepinephrine titration for hemodynamic stability, aggressive intravenous fluids and antibiotic therapy with vancomycin and cefepime were initiated. Over the next twelve hours following the initiation of septic shock management, patient’s vital signs had stabilized with correction of high anion-gap lactic acidosis; the neurological symptoms spontaneously resolved with minimal proximal weakness (4/5 power in hip flexors and extensors). As per the neurologist’s feedback, although the patient had family history of multiple sclerosis, the symptoms were more closely related to peripheral neurological processes which apparently turned out to be transient. Computed-tomography of abdomen-pelvis ruled out any obvious intraabdominal infectious pathology. Piperacillin-tazobactam and tobramycin were later added but antibiotic regimen was subsequently changed to pencillin-G-potassium over the next 48 hours as per the blood culture and sensitivity reports and infectious diseases’ team’s recommendations. On postpartum day-seven, the patient was discharged to home after being transferred on oral amoxicillin therapy.

Discussion

Our patient’s unusual clinical scenario brings forth multiple interesting points. Firstly, anesthesiologists are often called for evaluation and recommendation about the acute changes in peripheral neurological status in postpartum patients who have received peripartum neuraxial interventions for analgesia-anesthesia. In such scenarios, if the clinical picture is not clearly representing any direct consequence to peripartum neuraxial intervention, the anesthesia team should not hesitate to ask for a second opinion from the neurology team. Secondly, our patient’s clinical condition highlights that complications of neuraxial analgesia-anesthesia and acute pathological conditions like epidural hematoma or abscess are not the only conditions to suspect in postpartum patients; peripheral nerve diseases can also play a role in patients’ neurological symptoms, especially in critically ill patients. Finally, such neurological symptoms may NOT necessarily fit into one particular pre-defined nerve disease profile.

Peripheral neuropathy represents peripheral nerve disease/damage that often has subacute or chronic onset of symptoms except for the patients acutely suffering from Guillain-Barre syndrome like symptoms. However, these neuropathic symptoms usually resolve slowly, unlike our patient’s
neurological symptoms that resolved within 24 hours. Neuritis represents inflammation of nerves but is often associated with pain that was not observed in our patient. Transient neurologic symptoms associated with lidocaine4 supposedly represent chemical-irritant-induced pain symptoms in the absence of neurological pathology; but again, nerve-related pain was absent in our patient. Transient neurological deficits due to transient spinal cord ischemia5 may occur as similar to transient ischemic attacks happening within the brain; however, our patient’s transient paresis most likely represented peripheral nerve involvement rather than central nervous system’s involvement. It is not known whether similar mechanisms may occur with transient peripheral nerve ischemia; however, the idiopathic or functional origin of our patient’s symptoms can be easily ruled out in the presence of neurological signs (motor, sensory and deep tendon reflexes). Finally, it seems that the answer to our patient’s symptoms can be encompassed within a yet-to-be-defined transient peripheral neuropraxia which may be similar to transient cervical neuropraxia6-7, observed in the sudden hyperflexion-hyperextension injuries among contact sports athletes. This transient neuropraxia may involve any region within the spinal cord and can be referred to as spinal cord concussion8-9 with spontaneous and complete resolution within few minutes to couple of days. Analogously, our patient’s transient peripheral neuropraxia could be secondary to sepsis and septic shock precipitating peripheral nervous tissue hypoperfusion leading to CIN that responded and resolved when the inciting events (sepsis and septic shock) were corrected by vasopressors, aggressive fluid therapy and intravenous antibiotics.

On a side note, gentamicin related peripheral neuropathy and neuromuscular weakness10-12 could have been considered as the underlying etiology. However, the patient had only received the first (and only) dose of 7mg/kg-gentamicin intravenous infusion scheduled for once every 24hrs; and subsequent incidence and then resolution of acute sensory loss signs within 24-hours may rule against gentamicin as the cause of patient’s symptoms. Although patient’s thyroid function tests were normal five years back, the thyroid function was not re-evaluated in the patient most likely taking into consideration that evolving septic shock can independently affect thyroid function of septic patients13-14. Nevertheless, thyroid related myopathy seemed unlikely in the patient, considering the associated transient sensory changes along the dermatomes.

Conclusion

In summary, even though not clearly ascertained, our patient’s course of neurological symptoms seems likely to have occurred due to transient critical illness neuropraxia (CIN).
References


