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Neurologic Dysfunction in Postpartum Patients Caused by Hypomagnesemia

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The use of epidural analgesia to provide pain relief during labor and delivery is increasing in popularity. When a patient who received epidural analgesia develops neurologic complications in the postoperative period, the anesthesiologist is confronted with the complex problem of differential diagnosis. When faced with such a situation, one should consider other causes of neurologic dysfunction, other than those complications commonly attributed to the epidural technique. In this report, we present two patients who manifested neurologic dysfunction following epidural anesthesia that was most likely due to hypomagnesemia.

CASE REPORT 1

A 22-yr-old, 52 kg, gravida 2, para 1, A.S.A class I patient was admitted to the hospital for labor and delivery at 34 weeks of gestation. Her prior surgery included a cesarean section 3 yr prior to the present admission. She was not allergic to any medications. Physical examination was normal. Pelvic examination revealed 4.0 cm dilatation of the cervix and ruptured amniotic membranes. Her vital signs included an arterial blood pressure of 100/70 mmHg and a heart rate of 86 bpm. For pain relief, she requested lumbar epidural analgesia. Utilizing loss of resistance technique, a 17-G epidural needle was inserted into the L3-L4 interspace. During the insertion of the epidural needle, an accidental dural puncture occurred. The needle was then inserted into another epidural space and an intermittent lumbar epidural analgesia (ILEA) with 0.25% bupivacaine was administered. Eight hours later, she delivered spontaneously a healthy male infant. Twenty-four hours following the delivery, she complained of nausea, vomiting, and headache. The diagnosis at this time was spinal headache. She was advised to stay in bed and drink fluids. Because the headache persisted the next day, an autologous epidural blood patch (EBP) was performed with 10 ml of blood. During the night, 12 h following the EBP, she complained of vertigo and requested pain medications. The next morning (20 h following the EBP), she had two episodes of generalized seizures. She was given incremental doses of diazepam (20 mg total) and 500 mg of phenytoin IV to control the seizures. She remained in post-ictal coma for 2 h and then slowly became fully oriented. She denied any history of drug addiction, alcohol abuse, or seizure disorders. She had no evidence of preeclampsia. Neurological examination, CT scan of the head, drug screening, lumbar puncture and analysis of the CSF and EEG were all normal. The only abnormal laboratory finding was a low magnesium level of 1.3 meq/l (normal at this lab 1.8–2.4). Serum electrolytes were within normal limits. She was given 4 gm of magnesium sulphate iv bolus injection. Later on, she received oral magnesium supplementation. In the next 2 days, she completely recovered, and her anti-convulsive medications were slowly discontinued over a period of time. Upon discharge, her serum magnesium level was 2.0 meq/l.

CASE REPORT 2

A 22-year-old, 65 kg, gravida 1, para 0, A.S.A class I patient was admitted at term in early labor. Upon examination, her cervix was 4 cm dilated, and the membranes had ruptured. Her vital signs included an aretrial blood pressure of 110/80 mmHg and a heart rate of 92 bpm. For relief of pain, she requested lumbar epidural analgesia.

Utilizing loss of resistance technique, a 17-G epidural needle was inserted into the L3-4 interspace. A 19-G epidural catheter was then introduced through the needle without difficulty. No parasthesias were elicited on placement of the needle, catheter, or injection of the local anesthetic. Neither blood nor cerebrospinal fluid could be aspirated through the catheter. A test dose of 3 ml of 0.5% bupivacaine with epinephrine was injected. Five minutes later, an additional dose of 6 ml of 0.25% bupivacaine with epinephrine was injected. Following this, the patient experienced relief of pain. After receiving two more injections of the local anesthetic, the patient spontaneously delivered a healthy male infant. She recovered from the effect of the anesthetic in about an hour, and was released to the ward. She offered no complaints on the day following the epidural anesthesia.

Two days following the epidural anesthesia, while holding the baby,

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she noted that her left arm was trembling. Soon after that, she noted that her left lower extremity was jerking in an uncontrollable manner. She had two more similar episodes within an hour. She did not lose conciousness. Following these episodes, her vital signs remained stable. She complained that her left arm was feeling numb. Physical examination was essentially normal. She could walk without assistance. Neurologic examination done by a neurologist revealed no neurologic deficit. Even though she had denied any previous psychosomatic symptoms, a diagnosis of functional disorder was entertained, and she was carefully observed for further manifestation of neurologic dysfunction. Routine investigation in this case included determination of serum calcium and magnesium levels. Her serum calcium was 8.9 mg/dl (8.4–10.6) and magnesium was 1.5 meq/l (normal 1.8–2.4). Serum protein was 7.0 gm/dl. Other than the low serum magnesium level, all the other tests were normal. She was given vitamins with supplemental magnesium. In the next 2 days, she did not manifest any neurologic dysfunction. She was then released from the hospital. During her 6-week follow-up visit, she was noted to be symptom-free.

Discussion

A neurologic complication is likely to occur once in every 10,000–20,000 epidural anesthesias.[1] In contrast, the incidence of neurologic complications secondary to obstetric and other causes could be as high as one in 3,000 cases.[2] Non-anesthetic causes could be pathology involving CNS, spinal cord, spinal roots, nerve trunks, and peripheral nerves or due to metabolic causes.

In a review of intracranial hemorrhages secondary to rupture of an aneurysms or arteriovenous malformations, Amias[3] noted that, in three patients, hemorrhage occurred within 24 h of delivery. Neurologic deficit secondary to intracranial hemorrhage could occur in preeclamptic patients. Spontaneous epidural hematomas could occur in patients receiving anticoagulant therapy.[4] Preexisting neurologic complications, such as multiple sclerosis, may be exacerbated following regional anesthesia.‡ Compression of the lumbosacral plexus against the bony pelvis can be caused by the descent of the fetal head or, more commonly, by the use of forceps. This is probably the most frequent cause of neurologic injury from obstetric causes, and it usually manifests as either unilateral sensory loss over the leg and/or foot-drop.[1] Prolonged lithotomy position, improper positioning of the legs, and tight application of the straps can result in damage to femoral nerve, peroneal nerve, and small cutaneous branches of the foot, respectively.[5]

As these patients did not show any evidence of neurologic deficits involving lower extremities or any other areas, it was felt that their symptoms could be due to metabolic or functional causes. These patients had not received any psychiatric therapy in the past. The only abnormal laboratory finding in these cases was the decrease in serum magnesium level.

Magnesium deficiency is generally noted in chronic alcoholics, in patients with gastrointestinal disorders and in patients on chronic iv therapy. However, Wong *et al.*[6] noted a 10% incidence of hypomagnesemia (< 1.5 meq/l) in hospitalized patients. The incidence of hypomagnesemia in pregnant or postpartum patients is not known. The symptoms of hypomagnesemia include tremors, restlessness, and convulsions. The first patient in this report had generalized seizures. The second patient demonstrated fasciculations and jerky movements of the left upper and lower extremities. In both patients, other causes of neurologic dysfunctions were not discovered.

In summary, we report two unusual cases of neurologic dysfunction following epidural analgesia which were probably due to hypomagnesemia.

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