Worsening of Neurologic Symptoms After Epidural Anesthesia for Labor in a Guillain-Barré Patient

Sandrine Wiertlewski, MD†*, Armelle Magot, MD*, Sophie Drapier, MD†, Jean-Marc Malinovsky, MD, PhD‡, and Yann Péron, MD, PhD*

From the *Laboratoire d’Explorations Fonctionnelles, the †Clinique Neurologique, and the ‡Département d’Anesthésie et Réanimation Chirurgicale, University Hospital, Nantes, France

We report the case of a pregnant woman presenting with Guillain-Barré syndrome in which her neurologic status worsened immediately after delivery under epidural analgesia. We believe that the anesthetic technique may have played a role in the disease progression. Because of this potential association, the risks of worsening neurologic status after regional anesthesia and alternative analgesia options should be discussed with the parturient. (Anesth Analg 2004;98:825–7)

The relationship between Guillain-Barré syndrome (GBS) and pregnancy is of special interest because of the dramatic character of GBS symptoms in the parturient. In addition, the use of neuraxial anesthesia is questionable because it might influence the course of the disease. Only a few cases have been reported in which neuraxial block was successfully used in pregnant women with GBS (1–5). We report the case of a pregnant woman presenting with GBS in which her neurologic status worsened after delivery under epidural analgesia. We believe that the anesthetic technique may have played a role in the disease progression.

Case Report

A 27-yr-old primipara was admitted to hospital in the thirty-sixth week of pregnancy with bilateral facial palsy, progressive numbness and weakness in all limbs, and inability to walk. Her symptoms developed over a period of 10 days. The patient’s recent history and pregnancy had until that point been unremarkable. Clinical examination (absent deep tendon reflexes), electromyography (reduction of nerve conduction velocity and delayed distal latencies), and cerebrospinal fluid analysis (1 cell/μL, total protein level 131 mg/dL) were consistent with a diagnosis of GBS. The patient was treated with large doses of IV immunoglobulins (Igs; 0.4 g/kg per day for 5 days). By the end of treatment, her ability to walk and mouth opening were improved, but she still had mild facial and lower limb weakness. At 41 wk of pregnancy she spontaneously delivered a live female infant using epidural analgesia that was inserted at the L2-3 interspace. A test dose of 3 mL 2% preservative-free lidocaine with 1:200,000 epinephrine was negative for signs of intrathecal or intravascular placement. Labor analgesia was induced by an initial injection of 10 mL 0.2% ropivacaine with 10 μg of sufentanil. Additional doses of 15 mL of 0.2% ropivacaine and 6 μg sufentanil were administered using a patient-controlled epidural analgesia device over a 3-h period without any continuous epidural infusion. An uneventful delivery was accomplished 4 h after initiation of the epidural anesthesia, at which point the patient had both a sensory and motor block.

The patient did not fully recover from the motor blockade induced by the epidural, and at about 12 h postpartum she was unable to walk because of increased weakness of the lower extremity. At that time she also had increased facial and upper extremity weakness. Medullar magnetic resonance imaging was normal, and nerve conduction velocities were more reduced and distal latencies were more delayed than before delivery. She was again treated with large doses of IV Igs (0.4 g/kg per day for 5 days), this time with only mild clinical improvement. An occupational/physical rehabilitation program was begun. Four months later she still had a gait disturbance and was dependent on a walker.

Discussion

The overall incidence rate of GBS is less frequent during pregnancy (6), but the risk of GBS increases for the mother during the postpartum period (7,8). GBS in the pregnant woman may worsen after delivery even if no epidural is administered for labor or cesarean delivery (9,10). The interactions between neurological disimmune diseases and pregnancy remain unclear.
From an immunologic point of view, normal pregnancy seems to be associated with a shift away from cell-mediated immunity toward increased humoral immunity. The fetal-placental unit secretes cytokines such as interleukin-10 that down-regulate the production of other cytokines mediating cellular immunity by the mother. Delivery might be associated with an inversion of this cytokine balance (11). This concept could explain why pregnancy is often associated with spontaneous remission and the postpartum period with exacerbations in disimmune neurologic diseases such as multiple sclerosis (12) or GBS. In cases where GBS worsens or develops during the postpartum period, it usually occurs about 3 weeks after delivery (3,13).

There are no established guidelines for anesthetic management of GBS parturients. GBS occurring in pregnancy is associated with an increased need for ventilatory support and an increase in maternal mortality (14,15). A regional anesthetic may be beneficial because an exaggerated hemodynamic response to labor pain may occur (16). It also avoids deleterious effects of muscle relaxants, such as cardiac arrest, which has been described after administration of succinylcholine (17). Epidural anesthesia has been successfully used for labor and cesarean delivery in patients with GBS (5), and no established case of GBS relapse related to the regional anesthesia has been reported.

Our patient did not fully recover from the epidural anesthetic, and neurological symptoms definitely worsened after delivery. Possible explanations for her worsening status include incidental immunologic changes associated with pregnancy and anesthetic toxicity. In the present case, the worsening occurred immediately after the anesthetic, without the 3-week delay usually associated with postpartum immunologic changes. Moreover, the patient responded promptly to IV IGs during the pregnancy. The same treatment during the postpartum period was much less effective. Such findings decrease the likelihood that this case was incidental or attributable to postpartum immunologic changes (3,13).

It is possible that a toxic effect of anesthetics on the nervous system may partly explain the immediate postpartum clinical worsening of our patient. Local anesthetics are responsible for morphological changes in growing neurons, impairing their growth in vitro (18). Although this experiment cannot be directly extrapolated to in vivo conditions, the detrimental effects of local anesthetics on growing or regenerating neurons must be considered. In addition, perception of labor pain in women with GBS may be reduced because of denervation; theoretically they would therefore require smaller doses of local anesthetic drugs and may achieve higher than usual spread of epidural block (4). Although worsening of the clinical status of our patient affected both the lower limbs and face, we believe that this does not necessarily preclude anesthetic toxicity as the etiology. Because the upper level of the block may have reached much higher levels than expected and systemic effects related to absorption form the epidural space might have affected peripheral nerves, the epidural may have indeed played a role.

In contrast to previous case reports describing the anesthetic management of the parturient with GBS, we report a case with neurological worsening that may have been related to the anesthetic. Because of this potential association, we believe that the risks of neurologic status worsening after regional anesthesia and alternative analgesia options should be discussed with the patient (19).

References

