

# Rapid Onset of Guillain-Barré Syndrome After an Obstetric Epidural Block

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Reports of acute onset of Guillain-Barré syndrome (GBS) after epidural anesthesia/analgesia after labor and cesarean delivery has raised concern of a correlation between GBS and the use of neuraxial anesthesia. We present a patient who developed bilateral lower extremity weakness and paraparesis within hours after removal of an epidural catheter for cesarean delivery. The clinical diagnosis was highly suggestive for GBS after magnetic resonance imaging, cerebrospinal fluid findings, electromyogram, and nerve conduction studies. We discuss the pathophysiological mechanisms suggested in previous case reports and describe the relationship between epidural analgesia and GBS. (Anesth Analg 2013;XXX:00–00.)

Guillain-Barré syndrome (GBS) has been reported to occur during pregnancy and acutely after labor in patients with no preexisting neurological symptoms.<sup>1,2</sup> The relationship, however, between GBS and epidural anesthesia remains unclear.

We present a case in which the patient had no neurological symptoms before delivery, but complained of lingering paraparesis and rapid onset of progressing weakness within hours after cessation of a prolonged epidural block for cesarean delivery. The symptoms escalated rapidly, and the diagnosis of GBS was made. The patient improved after plasmapheresis treatment recovering slowly, but after 2 years mild balance instability remained.

The University of South Florida IRB has approved this case report and gave written permission for the authors to publish the report.

## CASE DESCRIPTION

An 18-year-old G1P0 parturient at 41 weeks of gestation, ASA physical status II, 100 kg, 160 cm was admitted to the hospital for induction of labor for postdate pregnancy. The patient's recent history included asymptomatic group-B streptococcal vaginal infection identified 4 weeks before admission, for which penicillin was administered during active labor.

On the day of admission, cervidil (prostaglandin E2), followed by a low-dose oxytocin infusion titrated to effect (total of 10 units administered over 24 hours), was administered to induce progression of labor. On the second

admission day, and on patient request, an epidural catheter was placed with the patient in the lateral decubitus position, via a 17-gauge Tuohy needle and was inserted successfully at the L3-L4 level on the first attempt. A test dose of local anesthetic solution (3 mL of 1% lidocaine with 1:200,000 epinephrine) was injected, and the patient exhibited no signs of intrathecal or intravascular placement. Subsequently, a continuous infusion of 0.2% ropivacaine and 2 µg/mL of fentanyl was administered for labor analgesia at a rate of 8 mL/h without excessive motor block. The highest sensory level experienced by the patient was at T10.

During labor (admission day 2), the patient developed a fever (oral temperature 38.3°C), which was then attributed to chorioamnionitis. Laboratory results on admission day 3 revealed a white blood cell count of 15,100/mcL (normal 9.6–10.2), significantly higher than admission values (8000/mcL). To manage these symptoms, the patient received ampicillin 2 g IV, gentamicin 160 mg IV, and clindamycin 900 mg IV during labor and continued for 48 hours.

On the morning of admission day 3, the decision was made to proceed with a cesarean delivery because of arrest of cervical dilation and the suspected chorioamnionitis. After confirming analgesic level at T10, the patient was prepared for cesarean delivery. Ancef 2 g IV was infused perioperatively. The duration of surgery was 35 minutes and uneventful. There was an estimated blood loss of 800 mL, and 2200 mL of crystalloid was infused. A 3505 g living infant was delivered in the vertex position with Apgar scores of 8 and 9 at 1 and 5 minutes, respectively, cord pH of 7.26, and base deficit of 5 mEq/L.

After approximately 28 hours of use, the epidural catheter was removed with the tip intact, in the postanesthesia care unit, while residual analgesia was receding. On postoperative day 1, 12 hours after the cesarean delivery, the patient complained of lower extremity numbness and weakness, sharp neck and upper back pain, and urinary and fecal incontinence. At this time, and for the rest of her admission, the patient was afebrile. The anesthesia team initially believed this might be a residual effect from the neuraxial block; however, symptoms progressively increased in severity.

The next day, after consultation, a neurologist found profoundly decreased strength graded 0 to 1 of 5 symmetrically in muscle groups of both lower extremities and

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limited pedal movement. The strength and tone of her upper extremities were preserved, with the exception of 2 of 4 in the brachioradialis muscles. Sensation to light touch was decreased from approximately T11 and below. Deep tendon reflexes could not be elicited in the lower extremities. There were no neurologic complaints/deficits above the neck, or dermatomal or myotomal distribution defects. Likewise, no radicular/segmental sensory or motor deficits were noted in the distribution of her peripheral nerves. Anal and sphincter tone was intact.

A lower extremity venous Doppler examination revealed no evidence of deep venous thrombosis; however, prophylactic anticoagulation was started with enoxaparin 40 mg subcutaneously to prevent venous thromboembolism. On postoperative day 2, a magnetic resonance imaging (MRI) of the spine, with and without gadolinium contrast, demonstrated no sign of spinal cord compression or hematoma. A lumbar puncture of cerebral spinal fluid performed on postoperative day 5 revealed: xanthochromia, protein 102 mg/dL (normal range 15–45), red blood cells 550 mm<sup>3</sup>, nucleated cells 6 mm<sup>3</sup> (normal range 0–5), and immunoglobulin 13.0 mg/dL (normal range 0.48–5.86). There were no increases in white blood cells (lymphocytes 60% and monocytes 16%) or glucose (42 mg/dL). That same evening, an electromyography examination and a nerve conduction study exhibited upper and lower limbs within normal limits except for absent left superficial peroneal sensory nerve action potential and bilateral absent H-reflexes. The results of the electrophysiologic studies in combination with the albuminocytologic dissociation in the cerebrospinal fluid were most consistent with a variant of GBS;<sup>3</sup> however, the patient refused further diagnostic testing.

On postoperative day 5, the patient complained of shortness of breath and tachycardia was noted. A computed tomography pulmonary angiogram revealed no evidence of pulmonary embolus; however, a nonspecific enlargement of axillary lymph nodes bilaterally was noted. On postoperative day 7, plasmapheresis treatments were initiated.<sup>4</sup> Five treatments were completed in the following 10 consecutive days, and a physical therapy regimen was initiated, leading to modest improvements in motor function, but the patient remained nonambulatory.

On postoperative day 14, the patient was discharged to inpatient rehabilitation, and after 3 months was able to walk with the aid of a walker and an orthotic on the right knee. Seven months later, the patient's gait had further improved, but she was still unable to walk unassisted. 2 years later, the patient's mild balance instability remained.

## DISCUSSION

GBS is an eponym for a heterogeneous group of immune-mediated peripheral neuropathies. There are 3 major subtypes of GBS: acute inflammatory demyelinating polyneuropathy, acute motor axonal neuropathy, and acute motor and sensory axonal neuropathy (AMSAN). Our patient presented with a rapidly developing syndrome most consistent with AMSAN.<sup>5</sup> The electrodiagnostic findings do not fulfill all of the diagnostic criteria for AMSAN, but this is not uncommon early in the course of the disease. The patient did exhibit absent H-reflexes, which is the most

sensitive indicator of early GBS.<sup>6</sup> The patient's subsequent clinical course was also consistent with AMSAN, in which some patients have a prolonged recovery over the course of months to years.<sup>7</sup>

Table 1 summarizes our review of 8 patients previously free from progressive demyelinating syndromes who developed GBS after the administration of neuraxial anesthesia.<sup>8–12</sup> Steiner et al.<sup>8</sup> proposed that the interaction between injection of the local anesthetic drug and insertion of the epidural catheter mediated a "trauma" to local nerve roots that may have initiated a cascade of neurological events resulting in demyelinating neuropathy. The case described by Rosenberg and Stacey<sup>9</sup> supports the same timeframe between epidural catheter removal and symptoms. Bamberger and Thys<sup>10</sup> describe a case in which sudden postoperative paralysis mimicked an acute spinal cord compression syndrome in its appearance despite a negative MRI evaluation. Yun et al.<sup>11</sup> suggest their patient's apparent gastrointestinal infection or the mental and physical stress of the epidural may have triggered an immune response leading to GBS. Gautier et al.<sup>12</sup> described a primigravida patient presenting with sensory deficits 24 hours after epidural anesthesia for vaginal delivery and suggest the stress of pregnancy to be the cause.

In two-thirds of GBS cases, a prior infection of the upper respiratory tract or gastroenteritis was identified and thought to be the triggering agent in the development of an abnormal immune response 2 to 4 weeks after the onset.<sup>13</sup> The most common infectious agents include Epstein-Barr virus, Mycoplasma pneumoniae, Campylobacter jejuni, and Cytomegalovirus.<sup>13</sup> These agents are believed to induce antibody production against specific gangliosides and glycolipids. In some infections, the antibodies produced bind to both the intended antigen and to the neuronal gangliosides resulting in diffuse neurological damage, through "molecular mimicry."<sup>13–15</sup> However, in our review of 8 cases, only 1 patient developing GBS after epidural analgesia had any documented signs of an underlying infection (case 7), suggesting the possibility that they were undetected or dormant. In this case, we believe that the patient's group-B streptococcal infection prepartum or another undiagnosed intrapartum infection may have served as a triggering agent via molecular mimicry to lead to the acute polyneuropathy.

In our case, differential diagnoses include an acute spinal cord process such as compression or transverse myelitis. Enhancement of nerve roots with gadolinium on lumbosacral MRI is 83% sensitive to acute GBS;<sup>16</sup> however, our MRI of the spinal cord revealed no evidence of compression. Transverse myelitis of the thoracic or lumbar cord may not be visible and may be associated with areflexia early in the course of the disorder. However, the subsequent clinical course is dominated by upper motor neuron signs, such as hyperreflexia, which were absent in the case presented. A chemical toxicity from the prolonged infusion of ropivacaine<sup>17</sup> or a contaminant is conceivable, but the patient lacked any of the other signs associated with these conditions. Other potential etiologies of acute polyneuropathies include paraneoplastic syndrome and vasculitis; however, these would not be expected to demonstrate spontaneous recovery, and no other signs of these conditions have been apparent in the 2 years of follow-up.

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**Table 1. Review of Patients Developing Guillain-Barré Syndrome After Epidural Analgesia**

Case subject	Author (reference)	Age/sex	Initial infection	Procedure	First symptom	Time between epidural and first neurological symptoms	Treatment	Outcome
1	Steiner et al. <sup>8</sup>	29/F	—	Spontaneous delivery using epidural anesthesia	Acroparesthesias, weakness in the arms and legs, constipation, urgency and frequency, dysphagia	1 wk postpartum	Steroid treatment; discontinued after 3 mo	Recovery was slow. One-half year later, patient was medication free and asymptomatic
2	Steiner et al. <sup>8</sup>	60/M	—	Inguinal herniorrhaphy	Weakness of the legs and paresthesias	2 wk postoperatively	Steroid treatment	Patient was discharged after 25 d free of neurological abnormalities but global areflexia persisted
3	Steiner et al. <sup>8</sup>	70/M	—	Inguinal hernia repair	Weakness of the legs and paresthesias	10 d postoperatively	Steroid treatment	Discharged after 1 mo asymptomatic
4	Steiner et al. <sup>8</sup>	25/M	—	Acute prolapse of hemorrhoids	Weakness of the legs and paresthesias of soles and hands, dysphagia, dyspnea, and bifacial weakness	1 wk postoperatively	Steroid treatment	Regained strength within 2 wk
5	Rosenberg and Stacey <sup>9</sup>	58/M	—	Bronchoscopy, excopagocopy, transabdominal Nissen fundoplication, and thoracomy	Dull low back pain. Motor reflexes in legs lost, and a left facial droop was noted.	9 d postoperatively	Treated with plasmapheresis and methylprednisolone	Improvement was noted 1 mo later
6	Bamberger and Thys <sup>10</sup>	63/M	—	Exploratory laparotomy, open partial pancreateomy, splenectomy	Weakness, flaccid paralysis, loss of sensation and reflexes of the right upper extremity	2 h postoperatively	Methylprednisolone	Patient discharged home on postoperative day 11 with slow and steady progress.
7	Yun et al. <sup>11</sup>	26/M	Diarrhea 10 d before hospital visit	Radiating pain to the right lower limb: lumbar herniated intervertebral disc	Powerlessness in both lower limbs with increasing weakness	4 d after epidural block	Immunoglobulin for 5 d	Complete recovery from paralysis after 6 mo. Patient discharged after 10 d of treatment after showing improvement
8	Gautier et al. <sup>12</sup>	20/F	—	Obstetrical epidural anesthetic for labor	Left facial palsy and acroparesthesias, global weakness of all 4 limbs	24 h postpartum	Plasmapheresis	Patient regained strength and could walk within 2 wk. Patient was discharged 25 d later.

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## CONCLUSION

In conclusion, no direct link between neuraxial anesthesia and GBS can be confirmed. Understanding the differential diagnoses of the electrodiagnostic findings and collaborating with the neurology team helped provide proper care for our patient. ■■

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# AUTHOR QUERIES

## **AUTHOR PLEASE ANSWER ALL QUERIES**

AQ1—Please approve the correspondence address details.

AQ2—Please clarify whether “one-half year” refers to “one and a half year” in Table 1.

AQ3—Please clarify whether “parethesias” can be changed to “paresthesias” in Table 1.

AQ4—Please clarify whether “excopagoscopy” can be changed to “esophagoscopy” in Table 1.

AQ5—Please clarify whether “thoracomy” can be changed to “thoracotomy” in Table 1.

AQ6—“Acroparathesias” has been changed to “acroparesthesias” in Table 1. Please approve.

AQ7—Please approve the edits made to reference 4.