

Spinal anesthesia for cesarean section in a patient with chronic inflammatory demyelinating polyradiculoneuropathy

Torsten Richter · Karl-Anton Langer ·
Thea Koch

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Abstract Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) is an inflammatory disorder of the peripheral nervous system with progressive or relapsing signs in more than one limb, ending in prolonged periods of disability. There are no guidelines for anesthesia in this uncommon paralyzing disease. This report features a 19-year-old woman with CIDP scheduled for an elective cesarean section who had prolonged recovery of motor function after the administration of spinal anesthesia. Although a partial neural block in both feet persisted for 1 day, we conclude that spinal anesthesia is acceptable for cesarean delivery in CIDP-patients when reasonable precautions have been taken.

Keywords CIDP · Cesarean section · Spinal anesthesia

Introduction

Chronic inflammatory demyelinating polyradiculoneuropathy (CIDP) is an autoimmune inflammatory disorder of the peripheral nervous system that progresses steadily in a

slow, stepwise manner or presents with repeated episodes of symmetrical extremity weakness with loss of sensation and areflexia developing over at least 2 months. Some patients present with predominantly motor, sensory, or autonomic involvement; however, most patients demonstrate a combination of these symptoms [1, 2]. The present case of a patient with CIDP with cesarean section adds some information to an area of anesthesia in which publications are extremely limited.

Case report

A 19-year-old woman, gravida 1, para 0, was scheduled for elective cesarean section at 38 weeks' gestation due to progressive CIDP. The patient weighed 82 kg, was 171 cm tall, and had no medical co-morbidities other than CIDP, which was first diagnosed at age 15 years. After the initial diagnosis she had received several cycles of intravenous immunoglobulin therapy, with almost complete remission of symptoms after each session. The last cycle of this therapy had taken place one and a half years before her admission for delivery. During the course of her pregnancy no specific treatment for CIDP was given. The patient was closely supervised by her gynecologist and neurologist. Beginning in the 25th week of pregnancy, the patient noticed a deterioration of her motor function and she developed symmetrical, distally accentuated weakness of her arms and legs. Additionally, she felt a progressive numbness in her fingertips. Detailed neurological examination confirmed a loss of reflexes, except for a remaining left patellar tendon reflex. The progression of symptoms did not involve the cranial nerves. After discussion between the obstetrician and the neurologist, cesarean section was scheduled. At the time of pre-anesthetic assessment, the

T. Richter · T. Koch
Department of Anesthesiology and Intensive Care Medicine,
University Hospital Carl Gustav Carus, Technical University
Dresden, Dresden, Germany

T. Richter (✉)
Department of Anesthesia and Intensive Care, University Hospital,
TU Dresden, Fetscherstrasse 74, 01307 Dresden, Germany
e-mail: torsten.richter@uniklinikum-dresden.de

K.-A. Langer
Department of Anesthesiology, Mafraq Hospital,
Abu Dhabi, United Arab Emirates

patient presented with muscular atrophy of her hands and feet and hypesthesia of the distal upper extremities.

After the situation had been thoroughly reconsidered with the gynecologist and neurologist, spinal anesthesia (SPA) was chosen for the cesarean section.

The subarachnoid space was punctured with a 27-gauge pencil-point spinal needle at vertebral level L 3–4. After the identification of clear liquor, 2.0 mL of hyperbaric bupivacaine 0.5% was injected intrathecally, providing a T5 sensory block after 5 min.

Before SPA the patient received 500 mL of crystalloid solution intravenously. Extended standard monitoring of the vital signs (including invasive measurement of arterial blood pressure and repeated arterial blood gas sampling) was applied continuously. To keep the blood pressure stable, an additional 1000 mL of crystalloid solution and 3 mL of Akrinor® (AWD pharma, Dresden, Germany) (=300 mg cafedrine hydrochloride and 15 mg theoadrenaline hydrochloride) were given IV intraoperatively. Standard vital signs, invasive arterial blood pressure, and arterial blood gases were monitored regularly. During the entire intraoperative period the patient showed no signs of autonomic nervous system dysfunction. A healthy, full-term baby was delivered.

Postoperatively, the sensory block started to decline after 1 h, and the SPA subsided within 4 h with a remaining partial neural block in both feet. Despite plantar flexion of both feet, the patient was not able to perform dorsal flexion as she had done before the SPA. This condition was still observed after 15 h. This additional muscle weakness was accentuated on the right side and disappeared gradually within the next few hours. Eventually, 24 h after application of the SPA, the patient presented with a neurological status similar to that assessed preoperatively. Seven days after the delivery she was transferred to the Department of Neurology for an immune modulatory treatment session, performed over 3 days, with 3 × 10 g Endobulin® (Immune Globulin Human: Baxter Deutschland GmbH BioScience, Heidelberg, Germany). The postoperative course was uneventful and the patient was discharged from the hospital 12 days after the cesarean section.

Discussion

The prevalence of CIDP may be underestimated because of limitations in clinical, serological, and electrophysiological diagnostic criteria. However, the disease prevalence is estimated to be 1–7.7/100,000 [3].

CIDP is an autoimmune disease that is similar to acute inflammatory demyelinating polyradiculoneuropathy or Guillain–Barré syndrome (GBS) associated with progressive sensorimotor neuropathy. In GBS and CIDP, spinal

nerve roots and peripheral nerves are infiltrated by macrophages and T cells [4]. CIDP is associated with progressive proximal and distal extremity weakness with numbness and paresthesias in both the feet and hands. The course may be steadily progressive, stepwise progressive, or relapsing-remitting. Other diagnostic features include the absence of or a reduction in deep tendon reflexes, demyelination of motor nerves consistent with electrophysiological findings (abnormal conduction velocities, conduction block, prolonged F-waves, or distal latencies), and increased protein concentrations in cerebrospinal fluid (CSF) [3]. Patients in the acute phase of CIDP show elevated levels of chemokines macrophage inflammatory protein-3 beta (MIP-3 β) in the CSF [4]. Hypertrophy of lumbar and cervical nerve roots and intercostal nerves on magnetic resonance imaging (MRI) has also been reported [5].

Among the treatments for CIDP are intravenous immunoglobulin, corticosteroids, plasmapheresis, and immunomodulating agents such as azathioprine, cyclophosphamide, cyclosporine A, interferon-alpha, and mycophenolate mofetil, as well as 10% caprylate-chromatography purified immune globulin [1, 6–8].

Because of its rarity, there are no specific guidelines for the anesthetic management of patients with CIDP. It is questionable how far experiences in the anesthesia of patients with GBS are applicable to CIDP patients. The choice of anesthesia for cesarean section should thus be considered carefully for these patients.

The use of general anesthesia in a patient with CIDP may cause the effect of muscle blockade to be prolonged [9]. Also, a depolarizing muscle relaxant may induce hyperkalemia and cardiac arrest [10]. There are no experiences with rocuronium-induced neuromuscular block, in combination with sugammadex for reversal of the neuromuscular blockade, for cesarean section in patients with CIDP [11] and no recommendations exist for sugammadex use in patients with neuromuscular diseases [12].

Considering these risks, we regarded spinal anesthesia as the procedure of choice. Although any changes in neurological status after delivery may not be related to the type of anesthesia used [13], we noticed in our patient prolonged recovery of motor function in the area supplied by the common peroneal nerve. The motor and sensory block in our patient may have produced compression palsy because of the relaxation and compression of the lateral aspect of the lower legs on the bearing surface [14]. This compression palsy might have been the result of slight pressure against the fibular head and neck, although the patient was positioned carefully. Otherwise, fluctuations in CIDP symptoms can probably result from a wide range of physiological stresses such as natural activity, ischemia, or recovery from transient ischemia [15]. Acute inflammatory demyelinating polyneuropathy relapses more frequently during the first 30

days after delivery referred to the later periods [16]. However, some observations suggest that regional anesthesia in demyelating disorders exacerbates neurological symptoms or may even trigger the disease [17, 18]. Epidural anesthesia was discussed recently concerning its connection with the worsening of symptoms in GBS [19]. However, other authors have reported the uncomplicated use of regional anesthesia without any worsening or relapse of neurological symptoms in GBS and CIDP [16, 20, 21].

Due to the theoretical and unpredictable worsening of neurological symptoms in CIDP after an immune modulatory trigger such as physiological stress (i.e., delivery, operation, and general or regional anesthesia with possible prolonged time of recovery), the pre-anesthetic evaluation in these patients should include a complete documentation of neurological status prior to regional anesthesia. Another concern in patients with demyelinating diseases is autonomic instability [22]. Therefore, we performed continuous monitoring of arterial oxygen saturation and blood pressure in our patient, related to recommendations for anesthesia in GBS [23]. There were no signs of hemodynamic instability during her stay in the hospital.

In our patient, regional anesthesia outweighed any theoretical risks and confirmed the former successful use of SPA for cesarean delivery in a patient with CIDP, although there was prolonged recovery of motor function.

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References

1. Toyka KV, Gold R. The pathogenesis of CIDP: rationale for treatment with immunomodulatory agents. *Neurology*. 2003;60:2–7.
2. Dyck PJ, Lais AC, Ohta M, Bastron JA, Okazaki H, Groover RV. Chronic inflammatory polyradiculoneuropathy. *Mayo Clin Proc*. 1975;50:621–37.
3. Latov N. Diagnosis of CIDP. *Neurology*. 2002;59:2–6.
4. Press R, Pashenkov M, Jin JP, Link H. Aberrated levels of cerebrospinal fluid chemokines in Guillain-Barré syndrome and chronic inflammatory demyelinating polyradiculoneuropathy. *J Clin Immunol*. 2003;23:259–67.
5. Oguz B, Oguz KK, Cila A, Tan E. Diffuse spinal and intercostal nerve involvement in chronic inflammatory demyelinating polyradiculoneuropathy: MRI findings. *Eur Radiol*. 2003;13:230–4.
6. Kissel JT. The treatment of chronic inflammatory demyelinating polyradiculoneuropathy. *Semin Neurol*. 2003;23:169–80.
7. Katz JS, Saperstein DS. Chronic inflammatory demyelinating polyneuropathy. *Curr Treat Options Neurol*. 2003;5:357–64.
8. Hughes RA, Donofrio P, Bril V, Dalakas MC, Deng C, Hanna K, Hartung HP, Latov N, Merkies IS, van Doorn PA, ICE study group. Intravenous immune globulin (10% caprylate-chromography purified) for the treatment of chronic inflammatory demyelinating polyradiculoneuropathy (ICE study): a randomised placebo-controlled trial. *Lancet Neurol*. 2008;7:136–44.
9. Hara K, Minami K, Takamoto K, Shiraishi M, Sata T. The prolonged effect of a muscle relaxant in a patient with chronic inflammatory demyelinating polyradiculoneuropathy. *Anesth Analg*. 2000;90:224–6.
10. Feldman JM. Cardiac arrest after succinylcholine administration in a pregnant patient recovered from Guillain-Barré syndrome. *Anesthesiology*. 1990;72:942–4.
11. Puehringer FK, Kristen P, Rex C. Sugammadex reversal of rocuronium-induced neuromuscular block in Caesarean section patients: a series of seven cases. *Br J Anaesth*. 2010;105:657–60.
12. Beloiartsev A, Gableske S, Huebler M. Neuromuskulaeres Monitoring bei Patienten mit neuromuskulaeren Erkrankungen. *Anaesthesist*. 2009;58:731–44.
13. Rockel A, Wissel J, Rolfs A. Guillain-Barré syndrome in pregnancy—an indication for caesarian section? *J Perinat Med*. 1994;22:393–8.
14. Tsen LC. Neurologic complications of labor analgesia and anesthesia. *Int Anesthesiol Clin*. 2002;40:67–88.
15. Cappelen-Smith C, Lin CS, Kuwabara S, Burke D. Conduction block during and after ischaemia in chronic inflammatory demyelinating polyneuropathy. *Brain*. 2002;125:1850–8.
16. Chan LY, Tsui MH, Leung TN. Guillain-Barré syndrome in pregnancy. *Acta Obstet Gynecol Scand*. 2004;83:319–25.
17. Wiertelowski S, Magot A, Drapier S, Malinovsky JM, Pereon Y. Worsening of neurologic symptoms after epidural anesthesia for labor in a Guillain-Barré patient. *Anesth Analg*. 2004;98:825–7.
18. Steiner I, Argov Z, Cahan C, Abramsky O. Guillain-Barré syndrome after epidural anesthesia: direct nerve root damage may trigger disease. *Neurology*. 1985;35:1473–5.
19. Gautier PE, Pierre PA, Van Obbergh LJ, Van Steenberge A. Guillain-Barré syndrome after obstetrical epidural analgesia. *Reg Anesth*. 1989;14:251–2.
20. Schabel JE. Subarachnoid block for a patient with progressive chronic inflammatory demyelinating polyneuropathy. *Anesth Analg*. 2001;93:1304–6.
21. Velickovic IA, Leicht CH. Patient-controlled epidural analgesia for labor and delivery in a parturient with chronic inflammatory demyelinating polyneuropathy. *Reg Anesth Pain Med*. 2002;27:217–9.
22. Lichtenfeld P. Autonomic dysfunction in the Guillain-Barré syndrome. *Am J Med*. 1971;50:772–80.
23. Perel A, Reches A, Davidson JT. Anaesthesia in the Guillain-Barré syndrome. A case report and recommendations. *Anaesthesia*. 1977;32:257–60.