

Conus Medullaris/Cauda Equina Syndrome Following a Repeated Bupivacaine 1% Spinal Anesthesia- Analysis of a Case with Review of the Literature

Claudio Melloni^{1*}, Rita Golfieri² and Francesco Staffa³

¹Consultant Anesthesiologist, Via Fossolo 28, 40138 Bologna, Italy

²Head of the Department of Radiology, Sant'Orsola-Malpighi Hospital, Bologna, Italy

³Head of the Department for surgery of the peripheral Nervous system, Azienda of Ravenna, Lugo and Neurochirurgia Ospedale di Lugo, via dante 10, Lugo di Romagna (RA), Italy

*Corresponding author: Claudio Melloni, Consultant Anesthesiologist, Via Fossolo 28, 40138 Bologna, Italy, Tel: 0039-051-390048; E-mail: melloniclaudio@libero.it

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Abstract

In the morning a healthy 40 years old female had a successful spinal anesthetic with bupivacaine 1% 10 mg for hemorrhoidectomy followed by a second injection at midnight for severe pain. The second traumatic injection caused a conus medullaris/cauda equina lesion discovered by MRI 45 days later. The contribution of hyperbaric bupivacaine dosage and pencil point needle is discussed.

Keywords: Anesthesia; Spinal; Bupivacaine; Polyradiculopathy

Introduction

Damage to the conus medullaris and cauda equina has frequently been reported in the literature with an abundance of medicolegal implications [1]. But only a handful of cases have been attributed to direct needle trauma during spinal/epidural anesthesia. The vast majority of these are caused by hematoma compression around the conus and cauda region in anticoagulated patients [2].

We present a case, in litigation for malpractice, of direct trauma to the conus medullaris/cauda equina region caused during attempted repeated spinal analgesia injections for severe postoperative pain.

The Case

Patient, a 40 year old female in good health, ASA 1, weight 80 kg, height 162 cm, lab tests, EKG and chest X-rays within normal limits, was scheduled for hemorrhoidectomy in a private hospital. Spinal anesthesia was easily administered in the sitting position with 10 mg (1 ml) hyperbaric 1% bupivacaine (a concentration readily available in Italy) injected through a 25 g Whitacre needle in the L3-L4 interspace. Vital signs remained stable. Hemorrhoidectomy (Milligan Morgan) was completed in 30 min and the patient sent to the floor about 14 hours. During the following hours, she complained of increasing pain in the perianal area: with ketorolac 30 mg IM she obtained partial relief; later an administration of 100 mg of tramadol was followed by strange sensations around the lips and by agitation, without substantial analgesia. These sensations were labeled allergic and all other rescue medication was suspended. By midnight patient was restless and in severe pain. The on call anesthesiologist repeated the lumbar puncture, starting with the patient on the left side. Spinal tap was difficult and bloody. During the puncture, patient complained of a stab in the spine similar to an electric shock. Her left leg began to shake. The anesthesiologist changed patient posture from the left lateral decubitus to the sitting position and injected 10 mg of bupivacaine 1% through a Whitacre needle 25 g obtaining immediate pain relief and cessation of

the myoclonic jerks of the left leg. Patient fell into deep sleep but awoke 2 hours later complaining of pain radiating down the entire left leg with jerks and a burning sensation from the heel up to the gluteal region. Paresthesia with fasciculation and tremor of the entire left leg were described. During the following days ambulation even with a walker was very difficult. The complaint of pain and occurrence of Paresthesia and foot edema were entered in the nursing chart. Urine was collected through a urinary catheter left in situ for 6 days. Defecation, also difficult, was helped by enemas and stool softeners. The day following the operation, a neurological consultation found pain, functional impotence of the left leg with difficult motion, difficulties in the flexo-extension movements of the left foot, Paresthesia, and Achilles and plantar reflexes depressed. Hyperalgesia was noted along the entire left extremity. The neurologist diagnosed a left L4-L5 and L5-S1 radiculopathy and prescribed prednisolone, ketorolac, gabapentin, and diazepam. An urgent RMN done that same afternoon was unremarkable, but the MR included only segments starting from L3 and going down. Physiatric consultation prescribed active and passive exercises. Patient was discharged from hospital on the 14th day, diuresis and defecation normal, walking aided by a crutch.

EMG done 38 and 73 days later demonstrated L5-S1 radiculopathy with major damage at S1. The last examination 6 months later showed improvement in the L5 area.

MRI 45 days later at a private institution (sagittal and axial) demonstrated, at the L1-L2 interspace, a small paramedian fluid cavity (liquor), length 17 mm, width 1.5/2 mm, localized at the origin of the CE, boundaries regular. The content was not modified after gadolinium. The radiologist attributed the cavity to a liquor sac.

Patient continued to have difficulties walking, and reported dyspareunia and pain along the left leg. Consultations with another neurologist and a neurosurgeon led to the recommendation of analgesics against the allodynia and a confirmation of the L5-S1 lesions, concluding with a diagnosis of cauda equina syndrome (CES). Due to the severity of pain after 8 months patient required insertion of a medullary stimulator which resulted in good pain relief.

Unfortunately the electrodes were prone to dislodgement requiring patient to be re-operated twice for wires repositioning.

At the specialist anesthesiologist consultation ordered by the court in March 2013, 19 months after the spinal anesthetic, patient, even with a left foot arch support, walked with difficulty especially ascending the stairs. Quality of sleep depends upon the correct functioning of the medullary stimulator. She has been unable to resume her work at the sewing/knitting machine because she cannot remain seated longer than 1.5-2 hours. The left leg is susceptible to easy bruising, especially if she lies on that side. Dyspareunia improved significantly, particularly when the medullary stimulator is functioning. Diuresis is normal; defecation is helped with laxatives. The entire left side is hypotrophic. Flexo-extension of the hip and knee are almost impossible and bending provokes sharp pain. Pain remains constant (in absence of the stimulator) along the entire left leg, especially posteriorly. Hypo/dysesthesia occurs along the posterior aspects of thigh and leg including the left gluteal site. Patellar reflex is diminished, absent Achilles and plantar. The left big toe cannot dorsiflexion and the foot toes cannot stretch.

After careful re-examination of the MRI images, (Figure 1) at the level of L1, practically from the inferior aspect of the T12 intervertebral disk to the upper plate of the L1 intervertebral disk, a hyperdense area is visible, length 22 mm, width almost 2 mm, accompanied with a delamination of the dura inferiorly. The area is syrinx like and could be attributed to a scar of the conum medullaris (CM), This would support a final diagnosis of an anatomical malacia of the conus resulting in symptoms of a syndrome combining cauda equina syndrome (CES) and conus medullaris syndrome (CMS).



Figure 1: MRI image of scar.

Discussion

The risk of serious neurological damage following spinal/epidural anesthesia varies widely, between 1/10.000-1/100.000, depending on

the size of the sample and the modality of data collection. Prospective studies tend to higher figures: Auroy et al. [3] found 5 CES over 40640 regional, Phillips [4], 2/10440 anesthesia while retrospective studies present lower incidences: Horlocker et al. [5] found no CES over 4767 anesthetics, Moore [6] 1/11574, DRipps and Vandam [7] 0/10098.

Isolated case reports cannot elucidate the real frequency of major neurological damage following neuraxial anesthesia [8,9], but actual occurrence is certainly much higher than reported because the medico-legal claims mention many cases which fail to appear in the scientific literature. For instance, Morisot [10] from the Département d'Anesthésie-Réanimation, Hospital Port Royal, Paris reported between 1983-89, the cases of 10 patients suffering with severe neurological damage which occurred in 28 regional anesthetics, spinal, epidural, caudal, and were filed with leading French insurance companies.

The ASA closed claims [11] quote 670 neurological lesions, 105 localized at the lumbosacral roots and 84 to the medulla, noting a percent increase in the medullary damage from 8% between 1980-1984 to 27% from 1990-1999. The vast majority of medullary lesions (result in paraplegia/quadruplegia. 93% of lumbosacral nerve root damage was attributed to spinal anesthesia (37 cases) or peridural (25 cases). The most frequently associated factors were paresthesias occurring during the insertion of the needle: (24 cases) or the local anesthetic injection (8 cases) or the repeated attempts to obtain the blockade (14 cases). A recent survey from UK [12] estimates a frequency of major neurological damage at around 0.7-1.8/100.000 regional anesthetics, but the data reported do not point directly to the CES incidence.

One of the most important malpractice defense firms of the UK (MPS, Medical Protection Society) [13] reports 54 cases of major neurological damages, 72% surgical and 28% non-surgical. From January 1, 2003 to December 31, 2007 MPS received 63 citations for SCE, 46 from the UK, but none was attributed to spinal anesthesia. This would indicate that the incidence of serious damage from spinal anesthesia could be very low. However since no data about the total number of spinal/epidural blocks done within the same period of time is reported, actual damage remains unknown.

Data from Finland and Sweden on closed claims [14] gave conflicting results, ranging from 1 CES, 5 paraplegia, 6 radiculopathies in over 550.000 cases to higher estimates [15], since in a sample of 1,700,000 operations, 1,260,000 spinal and 450,000 peridural, in the period 1990-1999 127 neurological complications were found, with 32 CES. Drugs blamed were lidocaine 5% (8 cases), bupivacaine 0.5% (11 cases-6 hyperbaric, 5 isobaric), and mepivacaine (1 case). According to these data the more serious neurological squeals are estimated between 1/20000-30000 following spinal, 1/25000 following obstetric epidural, and 1/3600 following non obstetric analgesia. These statistics are impressive; however they are retrospective and evaluated data were obtained voluntarily from participating hospitals (85% response rate from all institutions contacted).

A contemporary estimate of risk based upon Medline search and high quality publications [16] estimates CES incidence after spinal at 0.11/10000.

A more recent report from Switzerland [17] includes claims collected from 1987 through the end of 2009; in 171 cases, 54% were caused by regional anesthesia (9 cases (5% of total)) resulted in paraplegia, 7(4% of total) in CE. Of note is that in 12 cases the first injection failed. The complication occurred following the second

administration- 6 during brachial plexus blockade, 4 during peridural, and 2 during spinal.

The attribution of CES to the repeated administration of bupivacaine is described in 2 cases by Kubina et al. [18]. The first patient received spinal bupivacaine 0.5% 3.6 ml, followed by general anesthesia, while the second received a combined spinal/epidural anesthesia: bupivacaine 0.5% 3.5 ml for surgery followed by a continuous epidural infusion for 42 hours. with bupivacaine 0.25%.

Persisting neurological symptoms have also been described by Chabbouh et al. [9] and Rohm et al. [19] casting serious doubts about the safety of the repeated administration of the higher concentrations of bupivacaine.

In fact we suggest that the dosages of bupivacaine administered in this case were too high. For perianal surgery, blockade of the S2-S5 roots is sufficient to establish a saddle block. A consistent block is obtained with a 0.4% bupivacaine concentration [20]. Correct dosage varies from 1.5 [21] to 7.5 mg of the 0.5% concentration, while 10 mg obtains a T10 level.

In a dose finding study [22] the authors have randomized hyperbaric bupivacaine 2-5 mg; the dose of 3 mg was found to be sufficient in 90% of cases. Therefore we cannot exclude that with the second administration of 10 mg of hyperbaric bupivacaine the CSF levels could have resulted in localized neurotoxicity due to high CSF levels since bupivacaine 1% was administered twice in the period of 11 hours.

Moreover such concentration is probably directly neurotoxic since the 0.75% solution was already capable of neurotoxicity (CES) in sheep [23]. The neurotoxicity of bupivacaine has been shown in many papers [24-26] and damage has not been limited to nervous tissue, since bupivacaine, and other local anesthetics, have also been implicated in chondrotoxicity [27]. The damage done to bovine chondrocytes culture is proportional to the dosage/concentration and time of exposure [28]. In neuroblastoma cell lines amide and ester local anesthetics determine the same lesions at the higher concentrations [29]. Concern over the danger of the higher concentrations of local anesthetic is probably at the basis of the FDA ban against the higher concentrations of bupivacaine - while it is still marketed in Italy, Greece and Germany.

Takenami et al. [30] demonstrated that the damage from bupivacaine involves preferentially the posterior roots and the posterior white matter, suggesting that the neuro-functional impairment is prevalently affecting the sensory system, sparing the motor systems. If these observations are extended to our case, the prevailing damage to the sensory system could explain the severity of pain (allodynia) experienced by this patient, at the same time explaining the relatively minor motor impairment.

A contributory causal link in neurotoxicity could be attributed to the local anesthetic maldistribution occurring with the Whitacre needle since the side port of this type of spinal needle could allow higher concentration of the LA if the port is directed caudally and the injection done at slow speed [31]. Pencil point needles could be more prone to causing nervous tissue damage since, in order to inject the local anesthetic they must be inserted into the CSF a little more deeply as the hole is a few mm from the tip and in so doing they cause damage in case of impalement of a nerve [32].

By using a short beveled needle there is less chance to encounter a nerve, but the damage, in case of contact, is more consistent with a contusion and more damaging than a clear cut [33]. Since the point of

the needle is blunt, the anesthesiologist needs greater force during its advancement with the subsequent risk of overshooting into the narrow subdural space and therefore more possibility of encountering a nerve or the conus if the insertion is too high. There is also the chance that the dura could be pushed anteriorly during the advancement before penetration [34] restricting the space and increasing the compression [35].

Anesthetists' ability to identify correctly a marked lumbar interspace is accurate less than 30% of palpations as has been shown by Broadbent et al. [36]. The level of markers ranged from one space below to four spaces above the level at which the anesthetist believed it to be. The marker was one space higher than assumed in 51% of cases. Accuracy was unaffected by patient position (sitting or lateral), although it was impaired by obesity. The location of the CM is lower than the body of L1 in at least 10% of patients [38].

These anatomical variations, together with the risk of accidentally selecting a higher interspace than intended for intrathecal injection, implies that spinal cord trauma is more likely when higher interspaces are selected. To eliminate the risk of incorrect palpation and lower termination of the cord, Broadbent et al. [36] recommend anesthesiologists strive to always select the lower interspaces, a suggestion reinforced by Reynolds [38]. The intercrystal line by palpation of the iliac crests tended to identify higher levels-the L3 or L3-4 spinal levels in 77.3% of cases and more commonly in females than in males (85.7 vs. 61.5%) and in patients with higher body mass indices. There was also a poor correlation with levels obtained through RM imaging which identifies L4 or the L4/L5 interspace [39] Better accuracy can only be obtained with the use of fluoroscopy or ultrasound but all these papers focus on the utmost attention and care in the choice of the correct interspace for spinal anesthesia.

The differential diagnosis between CES and CMS is difficult [40] and all in all not very helpful in this case, where there was no surgical option for treatment, only medical and rehabilitative [41,42].

In the case described, we believe components of both syndromes coexist, since the medullary lesion extends for some millimeters caudally and laterally, contributing to both upper motor neuron and lower motor neuron damage, with peculiar distribution in the L5-S1 regions.

Paresthesias experienced on needle insertion indicates contact with either the spinal cord or the nerve roots of the cauda equina, and has been shown to increase significantly the likelihood of subsequent neurological deficit [5]. Rajakulendran et al. [43] suggests following the golden rule of not injecting the local anesthetic in the case of the patient complaining of pain or paresthesias even if transient. What was missing in this case on the part of the anesthesiologist was a careful balance of the risk/ benefits ratio and sufficient care taken to titrate all drugs and consider all therapeutic options; being roused from sleep with a night call and time pressure resulted in a decrease in the caution and vigilance necessary for adequate consideration and intervention, all components insisted upon in a review written by highly experienced authors [44] The appearance of myoclonus is a confirmatory sign of nerve or cord needle impingement [45,46] and cannot be attributed to bupivacaine, since it appeared before the injection [47,48].

In conclusion, we believe that the initial damage was caused by a direct puncture of the conus medullaris similarly to the cases reported by Hamandi et al. [49].

Incorrect determination of the lumbar interspace level was the most likely factor leading to trauma to the spinal cord evidenced by the paresthesias and myoclonic jerks of the lower left leg: the precise mechanism of injury and the role of pencil point needle, hyperbaricity of 1% bupivacaine and total dosage remain unclear. Other possible causes include contusion of the conus, intramedullary hematoma formation from vascular bleeding followed by cicatrization [50] and neurotoxic effects of the injected agent or a combination of these.

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