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Case report

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Local anesthetic resistance in a pregnant patient with lumbosacral plexopathy

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Abstract

Background: We report a case of a patient with apparent resistance to local anesthetics. While similar cases of failure of regional anesthetics are often attributed to technical failure, the overall clinical presentation and history of this patient suggests a true resistance to local anesthetics.

Case Presentation: This patient presented for elective cesarean section and the decision for regional anesthesia was made. While attempting to place an epidural, the patient failed to achieve adequate skin analgesia despite multiple attempts at local infiltration. When a spinal was ultimately placed, sensory or motor blockade was not obtained despite no evidence of technical problems with technique. Further questioning revealed multiple prior episodes of local anesthetic failure in this patient.

Conclusions: While the failure rate of spinal anesthesia has been shown range from 4–13% and is often attributed to technical failure, elements of this particular case suggest a true resistance to local anesthetics.

Background

Reports of resistance to local anesthetics are frequently attributed to common etiologies such as failure of technique, failure of medication or other similar explanations. As a result, true local anesthetic resistance is difficult to diagnose and reports may be greeted with skepticism. However, since local anesthetics work via the sodium channel, it is theoretically possible that mutations in this channel might lead to differing responses to these medications.

We report a case of a patient who presented for elective cesarean section secondary to worsening femoral neuropathy. While attempting to place an epidural, the patient failed to achieve adequate skin analgesia. When a spinal was ultimately placed, sensory or motor blockade was not obtained despite evidence of appropriate spinal location. While the failure rate of spinal anesthesia has been shown range from 4–13% [1,2], the overall clinical presentation and history of this patient suggests a true resistance to local anesthetics when taken as a whole.

Case presentation

A thirty-four year old female presented for cesarean section due to worsening symptoms of lumbosacral plexopathy. The patient reported an approximately six-week history of intermittent right lateral thigh numbness, which progressed to right lower extremity numbness and weakness prior to admission.

The patient reported a similar episode with her pregnancy eighteen months prior. At that time, she was counseled to

have a cesarean section. Her symptoms resolved completely following delivery. On initial questioning, the patient stated that her prior cesarean section was done under general anesthesia without any anesthetic complications. This prior surgery was performed at another institution and no written records were available for review. Many anesthesiologists choose to avoid regional anesthesia in cases of existing neurological deficits primarily due to concerns about medicolegal liability. It was assumed that this was the reason that the patient had her previous cesarean section performed under general anesthesia and no more details concerning the choice of anesthetic were explored.

The obstetricians had previously consulted neurology when the patient first developed symptoms earlier in her pregnancy. On admission for cesarean section, the neurologists were again consulted for recommendations concerning the patient's care and method of delivery. Their initial recommendation was to perform cesarean section under general anesthesia. We presented the patient with a detailed discussion concerning the risks and benefits of regional anesthesia versus general anesthesia. In addition, we discussed with the neurologists our belief that peripheral neuropathy is not an absolute contraindication to regional anesthesia. It was agreed that the avoidance of general anesthesia provided benefits to the patient that outweigh the theoretical risks of regional anesthesia with peripheral neurological symptoms. The patient agreed that a combined spinal-epidural would be performed; general anesthesia as a backup was planned if the regional anesthetic should fail.

The patient was taken to the operating room and routine monitors applied. The patient did not appear overly anxious and was cooperative and coherent. She was placed in the sitting position for epidural insertion. After sterile skin preparation, three milliliters of 1% lidocaine from the epidural kit was infiltrated. After allowing time for the anesthetic to take effect, an attempt was made to insert a 17-gauge Touhy needle into the skin. The patient complained immediately of pain, indicating inadequate skin analgesia. Again, the patient was not anxious or uncooperative and gave a clear, reasonable account of pain. An additional 3 milliliters of 1% lidocaine from a second vial of lidocaine, not supplied in the kit, was infiltrated at the same site. Again, the patient did not obtain skin analgesia and complained of pain on insertion of the Touhy needle.

The epidural needle was never inserted past the subcutaneous tissue. The decision was made at this time to perform a single-shot spinal. It was felt that this single needle stick would be preferable to continued attempts at local infiltration followed by epidural placement. The patient remained in the sitting position. A 24-gauge, 90 millimeter Sprotte spinal needle via an 18-gauge 1.25 inch introducer needle was inserted at level L3-4 with mild patient discomfort. The needle was directed slightly cephalad with the eyelet of the needle pointing cephalad. Free flow of cerebrospinal fluid without aspiration was obtained on first attempt. A syringe containing 1.2 cc of 0.75% bupivacaine with 50 micrograms of fentanyl and 0.25 milligrams of additive free morphine was attached to the spinal needle, easy aspiration of fluid with swirling of syringe contents was performed, and the medication was easily injected. The bupivacaine used was from a vial of local anesthetic not included in the epidural tray. Clear cerebrospinal fluid was aspirated in a volume of approximately 2 milliliters without difficulty prior to the injection of local anesthetic. An additional volume of cerebrospinal fluid of approximately 0.5 milliliters was aspirated and then reinjected at the end of the injection of intrathecal local anesthetic.

The patient was placed supine almost immediately. After five minutes, testing for sensory level was performed with an alcohol swab (for temperature) and light touch. The patient did not have any sensory level at this time. After an additional three minutes, testing for sensory level was again performed with an alcohol swab and light touch. Pin-prick testing was also performed including the lateral ankle (S1 dermatome) with the patient reporting no sensory changes. The patient had no signs of motor blockade.

Ten minutes after injection, the patient was asked if she felt any difference compared to before the spinal anesthetic was performed and the patient noted warmth in her feet and buttocks. The patient reported no sensory level or motor block. After twenty minutes, there were still no signs of sensory or motor blockade. The decision was made to proceed with general anesthesia. At this point the patient stated that the same sequence of events (inability to numb her skin, failed regional block and general anesthesia) had occurred with her previous cesarean section. The patient received an uneventful general anesthetic. At the conclusion of surgery the patient was examined again for evidence of sensory level or motor blockade. None was evident.

On questioning, the patient described repeated failures of local anesthetics associated with skin infiltration for placement of intravenous lines, including the intravenous line that had been placed preoperatively. She also stated that she was unable to obtain analgesia for dental procedures. Reportedly, her dentist had attempted to use three different types of local anesthetics without success. She did not recall the names of the medications used.

Discussion

Many anesthesiologists choose to avoid regional anesthesia in cases of existing neurological deficits primarily due to concerns about medicolegal liability. As mentioned, it was assumed that this was the reason that the patient had her previous cesarean section performed under general anesthesia. No medical records were available to provide any additional details. In this case, it was felt the benefits of avoiding general anesthesia outweighed the risks of regional anesthesia and the technique of combined spinal-epidural was chosen after discussion with the patient.

Possible causes for failed spinal anesthetics include technical errors, medication failure, and abnormal distribution of local anesthetics [1]. There have been reported cases of failed spinal with confirmed subarachnoid injection. Wiskopf reported failure of a spinal catheter where position was confirmed both radiographically and with the ability to aspirate cerebrospinal fluid freely [5]. His patient did not obtain a sensory or motor block. However, the patient had analgesia to the skin when tetracaine was injected subcutaneously. Bevacqua and Cleary reported failure to produce anesthesia with hyperbaric lidocaine (total of 125 mg) given in two separate doses through a subarachnoid catheter [6]. A subsequent dose of hyperbaric bupivacaine produced an effective spinal anesthetic and subcutaneous infiltration with lidocaine produced effective analgesia. Neither patient reported a history of ineffective local anesthetics. Interestingly, skin analgesia was easily obtained in these patients. Drasner and Rigler suggest that, in cases of truly "failed spinals," maldistribution of local anesthetic in the subarachnoid space may be the cause of failure [7]. In these cases, medication collects in a limited sacral distribution and produces a block in an area that may not be tested. This phenomenon may be a result of trabeculae in the subarachnoid space.

In each of these reported cases, the patients responded to lidocaine given subcutaneously, despite the inability to obtain successful spinal blockade. These patients were described as having "relative resistance to lidocaine." Conversely, our patient did not obtain analgesia from subcutaneous lidocaine infiltration. Although she received only a single shot spinal, the dose was appropriate to produce an adequate spinal anesthetic for cesarean section. Failure of technique is a possible cause for a failed spinal, but cerebrospinal fluid was easily aspirated before and after injection. The patient reported warmth in her feet, which may suggest correct placement of intrathecal fentanyl. Inactive local anesthetic preparations provide another explanation for failed skin analgesia and spinal anesthesia. However, medication failure is usually an isolated incident confined to a single vial or lot of medication. In this case, lidocaine used on the skin was from two different sources, while the spinal bupivacaine was from an individual package. It is unlikely that all of these medications were defective. Although no formal testing was performed on the bupivacaine or lidocaine used, successful regional anesthetics were obtained using vials from the same lot on other patients during the same time period. Failure of subarachnoid distribution is a possible cause of the failed spinal. This explanation would not account for failure of skin infiltration during this procedure and previous dental procedures.

We cannot explain the etiology for failure in what should have been a successful spinal anesthetic and for failure to produce skin analgesia. It is possible that our patient may have an abnormality at the cellular level that makes her unresponsive to local anesthetics.

This case raises the question of possible local anesthetic receptor mutations and sodium channel abnormalities. An inappropriate receptor site might result from a genetic variation in the amino acid sequence within the sodium channel. Specifically, the sodium channel has been shown to consist of alpha, beta-1 and beta-2 subunits. The alpha subunit involves four homologous domains (I – IV) and each of these domains is made up of six transmembrane segments (S1 – S6). Local anesthetic action is believed to be due to an interaction with the sixth segment of domain four of the alpha subunit (IV-S6), involving sites of phenylalanine and tyrosine amino acid residues [8]. Genetic variations that alter this site of action might account for this and other reported cases of local anesthetic "resistance" or failure.

These findings may have been coincidental; however, we feel that this is very unlikely. The patient's similar experience with local anesthetics in the past suggests that this was a patient specific complication.

Conclusions

This is a case of a patient who failed to achieve local anesthesia on skin infiltration and failed to achieve any sensory or motor block after spinal anesthesia. This occurred in the absence of evidence of technical failure. In addition, different local anesthetics from different lots and vials were utilized during the patient's care. Lastly, on further questioning the patient relates a history of multiple failures of local anesthetic in the past. The clinical picture and history give a picture of true local anesthetic resistance. This finding of local anesthetic resistance may be due to genetic variations in the sodium channel.

Competing Interests

None declared.

Author contributions

Both authors participated in the interview, examination and care of this patient. Both authors have read and approved the final manuscript.

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Unfortunately, the patient has been lost to follow-up and can no longer be tracked; hence consent was not obtained for the publication of this report.

References

1. Munhall RS, Sukhani R, Winnie AP: **Incidence and etiology of failed spinal anesthetics in a university hospital.** *Anesth Analg* 1988, **67:**843-8.
2. Levy JH, Ioles JA, Ghia JN, Turnbull C: **A retrospective study of the incidence and causes of failed spinal anesthetics in a university hospital.** *Anesth Analg* 1985, **64:**705-710.
3. von Basedow KA: **Uber neuralgia pueperarum cruralis.** *Wochenschrift fur die gesammte heiljunde* 1938, **6:**636-639.
4. Beatty TE: **Paralysis after delivery.** *Irish J Med Science* 1938, **12:**304-306.
5. Weiskopf RB: **Unexplained failure of a continuous spinal anesthetic.** *Anesthesiology* 1970, **33:**114-116.
6. Bevacqua BK, Cleary WF: **Relative resistance to intrathecal local anesthetics.** *Anesth Analg* 1994, **78:**1024-1026.
7. Drasner K, Rigler ML: **Repeat injection after "failed spinal": at times a potentially unsafe practice [letter].** *Anesthesiology* 1991, **75:**713-714.
8. Ragsdale DS, McPhee JC, Scheuer T, Catterall WA: **Molecular determinants of state-dependent block of Na+ channels by local anesthetics.** *Science* 1994, **265(5179):**1724-1728.

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