ANESTHESIOLOGY™2014
OCTOBER 11-15 | NEW ORLEANS, LA

Session: L037
Session: L151

Postoperative Ulnar Neuropathy – Could I Have Done Something to Prevent It?

Sanjay Bhananker, M.D., F.R.C.A.
Harborview Medical Center, Seattle, WA

Disclosures: This presenter has no financial relationships with commercial interests

Stem Case and Key Questions Content

A 55 year old male was scheduled to undergo an elective arterio-venous fistula creation in the right arm for hemodialysis. His past medical history was notable for insulin dependent diabetes mellitus for 25 years, renal failure requiring alternate day hemodialysis for the last 12 years. He takes insulin, low dose aspirin, iron and vitamins and has no known allergies.

He had 2 previous surgeries for creation of A-V fistula (one in each arm). The first of these procedures was done under a regional anesthetic (patient describes very painful needles placed in the neck) and remembers a lot of discomfort and pain. On the second occasion, the patient had a general anesthetic and experienced severe postoperative nausea and vomiting for 3 days.

The remainder of his history and physical examination were unremarkable. His hematocrit was 33%, K+ 4.6 meq/L, fasting blood glucose level 148 mg/dL, and INR was 1.5. The rest of the coagulation profile as well as the EKG were within normal limits.

The patient expressed a preference to have the procedure done under a regional block performed while he was deeply sedated or briefly anesthetized, to avoid the severe nausea from the general anesthetic and to avoid the discomfort of multiple needle pokes in the neck.

Key Questions:

1. What additional preoperative information would you like to have before proceeding with the anesthetic?

2. What are the advantages and disadvantages of a regional anesthetic technique in this patient?

3. Does low dose aspirin intake and/or INR of 1.5 concern you for performing the block? What are the implications of intraoperative and postoperative anticoagulation? How deeply can we sedate the patient before performing the block?

4. What risks of regional anesthesia do you discuss with the patient?

A supraclavicular brachial plexus block was performed under ultrasound guidance and using a nerve stimulator, with 30 mls of 0.5% bupivacaine with 1:200,000 epinephrine, under sedation with 4 mg midazolam and 100 mcg fentanyl. The patient experienced paresthesiae along the medial side of the forearm during the localization of plexus. Twitch in response to nerve

stimulation was obtained with 0.4 mV before the local anesthetic solution was injected. The surgical procedure was scheduled for 90 minutes. The brachial artery was anastomosed with the cephalic vein at the elbow. The right arm was abducted to approximately 90o and a folded towel under the elbow was used by the surgeon for surgical positioning. However, technical difficulty because of prior thrombosis in the venous system in the right arm led to a prolonged surgical time. Three hours after the block, the patient began complaining of discomfort in the arm and could not remain still on the operating table. The surgeon requested conversion to a general anesthetic. The conduct of the general anesthetic was uneventful. The total duration of surgery was 4 hrs and 40 min. The patient received 5000 u IV heparin bolus followed by 1000 u/hr heparin infusion at the surgeons request. The patient received IV ondansetron and dexamethasone for prophylaxis of PONV. He was happy with his overall perioperative experience when the anesthesiologist visited him that afternoon. He had experienced mild nausea, but had resumed oral intake and insulin earlier that afternoon.

Key Questions:
5. What postoperative examination and documentation would you like to do at this visit?
The patients A-V fistula is working well on the 3rd postoperative day. However, the patient complained to the surgeon that he had numbness in the little finger and ring finger in the right arm. The surgeon requested your input as he felt that this complication was related to the regional anesthetic. He informed the patient accordingly and made a note to this effect in the patient's progress note.

Key Questions:
6. What additional examination, and/or tests would you like to perform? What is the differential diagnosis of postoperative ulnar nerve dysfunction?

7. How would you explain the causation of the ulnar neuropathy to the patient? What is the most likely course of the neuropathy? How would you like to follow up the patient?

8. What if the patient had developed ulnar neuropathy in the left arm? How would you then explain the complication to the patient?

Neurology consultation was obtained and nerve conduction studies were undertaken in bilateral ulnar nerves. There was impaired conduction in both the ulnar nerves (Right > Left). The patient was asked to follow up with neurology at 2 weeks and his symptoms remained unchanged at this time. He was scheduled for further follow up visit 1 month later and subsequently every other month. One year later, the patient still has persistent paresthesiae in his left arm.

Key Questions:
9. How do you manage postoperative sensory deficits? Does the management change if there are motor deficits?

10. What is the normal course of postoperative ulnar neuropathy?

11. What lessons have we learned (with regards to postoperative ulnar nerve damage) from the ASA closed claims project?

12. What is the doctrine of res ipsa loquitur and how can it be applied to this case?

Model Discussion Content
Perioperative peripheral nerve injury could mar a successful surgical procedure, handicap a patient with a severe functional disability and leave the anesthesiologist facing a long and unpleasant litigation. It is a significant source of morbidity for patients and the second most frequent cause for professional liability for anesthesiologists, accounting for 16% of claims in the ASA closed claims project database.1

Incidence and etiology of postoperative peripheral neuropathy: The incidence of postoperative peripheral nerve dysfunction is estimated at 0.1 to 0.15%, or 1 in 1,000 to 1,500 anesthetics. The etiology of perioperative nerve damage is largely unknown. Stretching and/or compression with malpositioning of the patient, direct trauma from needles or instruments, and chemical toxicity of injected local anesthetics or vasoconstrictors may be implicated in nerve damage.2,3 In a recent retrospective study of over 380,000 anesthetics, Welch et al noted that hypertension, tobacco use, and diabetes mellitus were significantly associated with perioperative peripheral nerve injuries.4 General and epidural anesthesia were associated with nerve injuries. Significant associations were also found with the following surgical specialties: Neurosurgery, cardiac surgery, general surgery, and orthopedic surgery.

Predisposing factors: Ulnar nerve is the most common site of postoperative peripheral nerve damage and ulnar neuropathy accounted for 28% of claims for anesthesia-related nerve injuries in the ASA closed claims database.1 Development of ulnar neuropathy has been documented not only in surgical patients, but also medical inpatients and outpatients and irrespective of whether general anesthesia, regional anesthesia or sedation/ monitored anesthesia care is administered. Male gender, extremes of body habitus, and prolonged hospitalization have been shown to be associated with the development of ulnar neuropathy5. Increased susceptibility of the male gender is believed to be secondary to the internal compression of the ulnar nerve and its blood supply by the coronoid tubercle of the ulna, which is at least 50% larger in men as compared to women. Also, men have less adipose tissue overlying the ulnar nerve at the elbow in comparison to females.

External compression on the ulnar nerve in the cubital tunnel is regarded as one of the most important causes of anesthesia-related ulnar neuropathy. External pressure by noncompliant objects or improper arm positioning may trap the ulnar nerve within the rigid bony canal of the superficial condylar groove at the elbow and render it ischemic. However, use of arm padding to avoid pressure on the ulnar nerve does not always prevent the development of ulnar neuropathy. External compression can also occur in supine patients if the elbows are flexed and the hands rested on the chest or abdomen.

The ulnar nerve is more exposed to external pressure sources and compressive injury when the forearm is pronated. It has been shown that supination of the forearm produces the least amount of pressure over the ulnar groove, pronation produces the most, and a neutral forearm position in an intermediate value. Up to 50 % of male patients who experience pressure on their nerve sufficient to impair the electrophysiologic function, do not perceive any symptoms. 6 It is speculated that postoperative male surgical patients, who are often sedated or taking opiates,

may fail to perceive, recognize, and respond to potentially damaging pressure over the ulnar nerve.

In patients who develop postoperative ulnar dysfunction, there is a high incidence of contralateral ulnar nerve conduction dysfunction, suggesting that many patients have a subclinical ulnar neuropathy that becomes symptomatic as a result of the many maneuvers and manipulations during the perioperative period. It is also hypothesized that perioperative neuropathy in ulnar nerve and brachial plexus may develop as a result of double-crush syndrome, a phenomenon in which two relatively minor nerve lesions, which by themselves are subclinical, can combine to produce a significant lesion.

Regional Anesthesia and Nerve Damage: The incidence of nerve damage following brachial plexus block has been reported to vary from 1.9 per 10,000 regional blocks to as high as 2 percent. 8The etiology of nerve injury following a regional block is generally presumed to be traumatic, toxic, ischemic or a combination of these factors. Formation of perineural edema, inflammation, and microhematoma around the nerve may account for the 2-3 week delay sometimes seen, from performing a regional block to the onset of neurological symptoms. Tissue reaction or scar formation in response to mechanical or chemical trauma may be responsible for the cases with delayed onset of neurological dysfunction.

Elicitation of paresthesiae during performance of a nerve block has often been used to indicate an optimal needle position in relation to the nerve. However, paresthesiae should also be used as a warning to indicate a possible needle trauma to the nerve. The use of a nerve stimulator allows for precise needle placement without eliciting paresthesia. Multiple injection technique, using a nerve stimulator to locate individual nerves is reported to have a higher success rate than a single injection technique. Short bevel needles are less likely to impale a nerve, but could produce more damage than long bevel needles if they do impale a nerve.

In theory, mechanical trauma from an indwelling catheter, or neurotoxicity as a result of sustained exposure to local anesthetic agents can increase the risk of nerve injury when a continuous peripheral nerve blockade is undertaken. However, studies have shown that the risk of neurologic complications with continuous axillary blocks is similar to that of single-dose techniques.

Though many patients may experience transient paresthesias, dysesthesias and pain not related to surgery in early postoperative course (up to 2 weeks), very few patients (0.2%) experience long term neurological complications from peripheral neural blockade. Peripheral nerve blocks are often performed under deep sedation, or even under general anesthesia, especially in pediatric patients. In a survey of American Society of Regional Anesthesia, 42% respondents reported that they performed brachial plexus blocks in anesthetized patients.14 The use of a nerve stimulator cannot replace the patients ability to report paresthesia and pain due to needle trauma or intraneural injection. Direct intraneural injection of local anesthetics questionably damages the nerves and intraneural pressures as high as 700 mm Hg may be generated during such injection. Also, stimulation with a peripheral nerve stimulator may not evoke any motor response even when the needle is close enough to the nerve to evoke paresthesiae.

Anticoagulation and Regional Anesthesia: Data on neurological injury following peripheral

nerve blocks in patients receiving anticoagulation therapy is scanty and is in the form of isolated case reports. The consensus statements on neuraxial anesthesia and systemic anticoagulation published by the American Society of Regional Anesthesia could be applied to any regional anesthetic technique.15 However, close monitoring of anticoagulated patients undergoing peripheral nerve blocks, for early signs of neural compression such as pain, weakness and numbness may help in early detection and timely intervention to prevent neurological sequelae from compression due to hematoma.

Ultrasound Guidance for Regional Anesthesia: Ultrasound guidance for performing peripheral nerve blocks is becoming popular worldwide. Animal studies have shown that ultrasound may prove useful to detect intraneural injection, whereas a motor response above 0.5 mA may not exclude intraneural needle placement. However, Bigeleisen, Sala-Blanch and Liu have shown that puncturing of the peripheral nerves and apparent intraneural injection during axillary plexus block does not lead to a neurologic injury. 10,11,12 Perlas et al noted that paresthesia was found to be 38.2% sensitive and motor response was 74.5% sensitive for detection of needle-to-nerve contact as detected by ulrasound. 8Deliberate intraneural (extra-fascicular) injections are becoming common in clinical practice and these have thus far been reported to improve success rates.

ASA Practice Advisory Recommendations: The American Society of Anesthesiologists (ASA) task force on the prevention of perioperative peripheral neuropathies drew up a practice advisory that focused on perioperative positioning of patient, use of protective padding, and avoidance of contact with hard surfaces or supports that may apply direct pressure on susceptible peripheral nerves.13 The current recommendations to prevent postoperative neuropathy include a preoperative screening to detect any subclinical neuropathy, preoperative history and physical examination directed at defining the comfortable range of stretching and movement at different joints, meticulous attention to avoiding intraoperative compression of superficial nerves, padding of the extremities and points at which nerves may get compressed, measures aimed at reducing the stretching of the nerves, periodic intraoperative checking for optimal positioning of the extremities and, not performing regional blocks under general anesthesia.

Clinical Presentation of Ulnar neuropathy: Symptoms of ulnar nerve injury after anesthesia manifest in most cases within 48 hours, but may occasionally be delayed up to 3 days or more. The symptoms can be minor, ranging from hypo or hyperesthesia of the medial 1.5 fingers with intermittent tingling to major complete sensorimotor deficit. Confounding factors such as postoperative pain, immobility, effects of analgesics and other medications, application of casts, bandaging should be considered as contributing or causative factors and ruled out. The severity and duration of symptoms are dependent on the severity of the injury.

Physical examination should include a comprehensive assessment of sensory and motor function. The findings should be meticulously documented. A neurologist consultation is advisable if motor weakness is present. The possibility of a nerve compression (hematoma) should be excluded early in the workup. Ultrasonography of the elbow region and/or magnetic resonance imaging (MRI) may be indicated if hematoma is suspected. The possibility of a vascular injury should be kept in mind and assessment of both arterial (ischemia) and venous (thrombosis) circulation should be performed.

Management of postoperative ulnar neuropathy: Most sensory deficits resolve completely within 4-6 weeks. Patients should be reassured, and regularly followed up in this period. However, disability secondary to severe nerve damage may last years, and very rarely may be permanent. The occurrence of painful neuropathic pain is usual after axonotmesis and neurotmesis. The occurrence of early or late complex regional pain syndrome (CRPS) type II is possible.

Nerve conduction studies (NCSs) are a useful tool to evaluate the functional integrity of peripheral nerves. Abnormal recordings can be seen early (within the first 2-3 days) after a nerve lesion occurs. NCSs enable focal nerve damage to be localized in patients with a mononeuropathy. Localized peripheral nerve damage is characterized by reduced or changed abnormality in amplitude. Conduction velocity may also be slowed. Combined with electromyography (EMG), NCSs can determine whether a nerve injury is complete or incomplete and therefore provides information about the prognosis and the likely course of recovery. In the case of a complete lesion, motor units cannot be activated volitionally in a distal muscle. Stimulation of the nerve proximal to the lesion does not elicit a response in muscles supplied by branches arising distal to a complete injury. However, electrical stimulation distal to the site of a complete nerve transection continues to elicit a distal response. In the case of a partial lesion, a smaller response can be elicited.

Lessons learned from ASA Closed Claims Project: The ASA Closed Claims Committee reported that 15% of 1541 claims in 1990 and 16% of 4,183claims in 1999 were for anesthesia-related nerve injury. The 1990s has seen a relative decrease in the percentage of ulnar nerve injury as a proportion of the total nerve injury claims and an increase in the proportion of claims for spinal cord injury. Overall, the most frequent site of injury was the ulnar nerve responsible for 28% of claims for nerve damage, followed by brachial plexus (20%). The injuries were bilateral in 14% of ulnar injuries and in 12% of brachial plexus injuries. Men predominated in ulnar nerve injury claims (75%). The median age of patients with ulnar nerve (50 yr) was significantly higher than the median age of nonnerve damage claimants (39 yr) and the youngest patient was 20 years. The closed claims analysis did not find any relation between obesity and the occurrence of ulnar neuropathy.

Eighty five percent of the ulnar nerve injuries were associated with general anesthesia. The mechanism of injury was explicitly stated in the claim file in only 10 (9%) of 113 ulnar nerve injuries These included preoperative trauma (4), intraoperative trauma (1), the surgical procedure (1), the use of crutches (1), and the performance of an axillary block (3). Paresthesias were not present during the performance of the block in any of these cases. In another four claims, the ulnar nerve injury was present before surgery. The onset of symptoms of injury was noted immediately postoperatively in 21% of ulnar nerve claims and was delayed from 128 days postoperatively (median 3 days) in 62%. Padding to the elbows was present in 27% of all ulnar nerve injuries. Also, there were eight claims in which ulnar neuropathy occurred in patients who were sedated and underwent spinal, epidural, or local anesthesia for lower-body surgical procedures in whom, the signs and symptoms of ulnar neuropathy became apparent 14 days after surgery.

The anesthesia care was significantly more often judged as appropriate in ulnar nerve damage claims (73%) as compared to nonnerve damage claims (42%). Payment was made in half of the

ulnar nerve injury claims in which care was appropriate. Payment was even made in half of the ulnar nerve damage claims in which the patient was awake or sedated during regional anesthesia and the surgery was performed on the lower body. The lack of apparent mechanism poses a problem for the defense of these nerve injury claims because the presumption is often made by the patient, the patients attorney, and by consulting specialists that something must have been performed incorrectly by the anesthesiologist during the perioperative period (res ipsa loquitur).

In a more recent closed claim analysis of peripheral nerve injuries associated with RA, nerve damage was associated with 59% of peripheral nerve block claims and was evenly split between temporary and permanent injuries. The brachial plexus was the most com- mon location injured permanently, followed by median and ulnar nerves, presumably from block needle damage or intraneural injection of local anesthetic. The most common type of block associated with permanent nerve injuries was axillary, followed by interscalene and supra-clavicular blocks.

Medical malpractice suits and peripheral neuropathy: Medical negligence suits for postoperative peripheral nerve damage are not uncommon. When an anesthesiologist agrees to administer anesthesia to a patient, his/her duties to the patient include:
1. Practicing at the professed level of care
2. Obtaining an informed consent
3. Protecting confidences
4. Offering continuing treatment and
5. Seeking consultation when needed.

The patient or plaintiff must introduce evidence in each of the following categories to prove negligence on the part of the physician:
1. Duty of care owing
2. Breach of duty
3. Nature of the injury
4. Proximate causation
5. Extent of damages

The exception to this need is the doctrine of res ipsa loquitur (the thing speaks for itself). The criteria to be met to invoke res ipsa loquitur include:
1. Exclusive control (of the defendant) over the causation of injury
2. Nature of injury is such that it can only be caused by someones negligence
3. Common knowledge that this injury can only be caused by negligence
4. Absence of contributory negligence (on the part of the plaintiff).

References

1. Cheney, F.W., et al., Nerve injury associated with anesthesia: a closed claims analysis. Anesthesiology, 1999. 90(4): p. 1062-9.
2. Barrington, M.J. and G.L. Snyder, Neurologic complications of regional anesthesia. Curr Opin Anaesthesiol, 2011. 24(5): p. 554-60.
3. Horlocker, T.T., Complications of regional anesthesia and acute pain management. Anesthesiol Clin, 2011. 29(2): p. 257-78.

4. Welch, M.B., et al., Perioperative peripheral nerve injuries: a retrospective study of 380,680 cases during a 10-year period at a single institution. Anesthesiology, 2009. 111(3): p. 490-7.
5. Warner, M.A., et al., Ulnar neuropathy in surgical patients. Anesthesiology, 1999. 90(1): p. 54-9.
6. Prielipp, R.C., et al., Ulnar nerve pressure: influence of arm position and relationship to somatosensory evoked potentials. Anesthesiology, 1999. 91(2): p. 345-54.
7. Hogan, Q.H., Pathophysiology of peripheral nerve injury during regional anesthesia. Reg Anesth Pain Med, 2008. 33(5): p. 435-41.
8. Perlas, A., et al., The sensitivity of motor response to nerve stimulation and paresthesia for nerve localization as evaluated by ultrasound. Reg Anesth Pain Med, 2006. 31(5): p. 445-50.
9. Koscielniak-Nielsen, Z.J. and J.B. Dahl, Ultrasound-guided peripheral nerve blockade of the upper extremity. Curr Opin Anaesthesiol, 2012.
10. Bigeleisen, P.E., Nerve puncture and apparent intraneural injection during ultrasound-guided axillary block does not invariably result in neurologic injury. Anesthesiology, 2006. 105(4): p. 779-83.
11. Liu, S.S., et al., Incidence of unintentional intraneural injection and postoperative neurological complications with ultrasound-guided interscalene and supraclavicular nerve blocks. Anaesthesia, 2011. 66(3): p. 168-74.
12. Sala-Blanch, X., et al., No clinical or electrophysiologic evidence of nerve injury after intraneural injection during sciatic popliteal block. Anesthesiology, 2011. 115(3): p. 589-95.
13. Practice advisory for the prevention of perioperative peripheral neuropathies: an updated report by the American Society of Anesthesiologists Task Force on prevention of perioperative peripheral neuropathies. Anesthesiology, 2011. 114(4): p. 741-54.
14. Bernards, C.M., et al., Regional anesthesia in anesthetized or heavily sedated patients. Reg Anesth Pain Med, 2008. 33(5): p. 449-60.
15. Horlocker, T.T., et al., Regional anesthesia in the patient receiving antithrombotic or thrombolytic therapy: American Society of Regional Anesthesia and Pain Medicine Evidence-Based Guidelines (Third Edition). Reg Anesth Pain Med, 2010. 35(1): p. 64-101.
16. Lee, L.A., et al., Complications associated with eye blocks and peripheral nerve blocks: an american society of anesthesiologists closed claims analysis. Reg Anesth Pain Med, 2008. 33(5): p. 416-22.