

Case Report

Cardiopulmonary Arrest Following Cervical Epidural Injection

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Epidural steroid injection is a common treatment for the management of pain in a wide variety of patients. It is generally well tolerated and perceived to have few side effects, with a low risk of serious complications. Only a handful of reports exist that describe life-threatening complications such as subdural hematoma, respiratory depression, vasovagal response, and pneumocephalus. This is a case report of a 67-year-old woman with a relatively unremarkable past medical history, other than rheumatoid arthritis, osteoarthritis, and hypertension, who suffered from chronic neck pain treated with cervical epidural steroid injection at the C6-C7 level. She went into immediate cardiopulmonary arrest following the injection. She was brought to the emergency department by ambulance and resuscitated, and was found to have pneumocephalus. Ultimately, she made a relatively full recovery over the following weeks.

Cardiopulmonary arrest is a rare but potentially deadly side effect of epidural steroid injection. To the best of our knowledge, this is the first report of such an arrest following a steroid injection in the cervical spinal region. There are several possible mechanisms for the immediate arrest, including cardioacceleratory center blockade, severe vasovagal response, iatrogenic pneumocephalus, and involvement of the phrenic nerve followed by apnea. Our conclusion in this case is that the most likely scenario was injection of the C6-C7 level led to a blockade of the cardiac accelerator fibers located just below in the T1-T4 spinal level, causing a sympathetic blockade and profound bradycardia, leading to cardiopulmonary arrest.

Key words: Cardiopulmonary arrest, cervical epidural steroid injection, pneumocephalus, cardiac accelerator, vasovagal, chronic neck pain.

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Cervical epidural steroid injection is a modality commonly used to manage pain in a wide variety of patients, and has been shown to be well tolerated with a low risk of complications (1-3). Minor complications exist such as pain at the injection site, increased radicular pain, and lightheadedness,

as well as headache, insomnia, vasovagal reactions, and facial flushing (4-5). Isolated cases of respiratory depression, subdural hematoma, and pneumocephalus exist in the literature (6-8). Furthermore, cases of cardiac arrest following spinal and epidural anesthesia have been reported (9-11), but to our knowledge there

has not been a case report documenting complete cardiopulmonary arrest following a cervical epidural steroid injection. This is significant because of both the level at which the injection took place as well as the seemingly more benign nature of an epidural steroid injection compared to an epidural for major surgery. We report a case in which this procedure resulted in the immediate cardiopulmonary arrest of a patient and its possible implications.

CASE REPORT

A 67-year-old woman with a history of rheumatoid arthritis, osteoarthritis, and hypertension had been evaluated in an outpatient surgery center where the decision was made to give a steroid injection at the level of C6-C7. She was premedicated with 2 mg of intravenous midazolam as well as 100 μ g intravenous fentanyl; we note that the patient already had a 100 μ g fentanyl transdermal patch. Other medications she was taking prior to the procedure include methotrexate, etanercept, prednisone, and hydrocodone/acetaminophen, all related to her rheumatoid arthritis. She was placed in a seated position with her neck flexed, and her skin was prepped and draped in a sterile fashion. She was injected with 3 mL of 1% lidocaine for local anesthetic before a 20-gauge Weiss needle was inserted. Loss of resistance with air confirmed position and under fluo-

roscopy, 2 mL of iohexol was injected for visualization. She was then injected with a steroid formulation of 12 mg betamethasone in 1% lidocaine, a total volume of 4 mL. Approximately 5 seconds after the injection of steroid, she went into cardiopulmonary arrest. Her heart rate went down to 20 beats per minute and the staff were unable to palpate the pulse. She received one mg epinephrine intravenously, flumazenil, and naloxone; 2 minutes of cardiopulmonary resuscitation was performed. A pulse was regained and the patient was subsequently brought by ambulance to the emergency department.

Between cardiopulmonary arrest and transfer to the emergency department, approximately 30 minutes elapsed. The patient was found to have foamy secretions coming from the mouth and a Glasgow Coma Scale of 6. She was spontaneously breathing and had palpable distal pulses, but was unresponsive. Further assessment showed that she was hypertensive and hyperthermic with a rectal temperature of 38°C. While no electrocardiogram (ECG) was available en route, the initial ECG in the emergency department showed sinus rhythm with p-pulmonale and no ST or T wave changes, although there were multiple premature ventricular complexes. The patient was started on metoprolol to manage blood pressure, and was intubated for airway protection. She was also started on the Arctic Sun 5000 Temperature Management System (Medivance, Inc., Louisville, CO) to induce therapeutic hypothermia within the 6-hour window per protocol. It was assumed her hypertension and hyperthermia were related to acute brain injury secondary to cerebral hypoperfusion. An initial complete blood cell count (CBC) showed mild anemia with a hemoglobin of 10.5 and an initial complete metabolic panel did not show any major electrolyte abnormalities other than an anion gap of 15.4, which was attributed to the hypoxia. An arterial blood gas test showed respiratory alkalosis and a pH of 7.52, pCO₂ of 33, pO₂ of 227, and HCO₃ of 27 on FiO₂ of 100%.

When the patient was deemed stable, computed tomography (CT) scans of the cervical spine and brain without contrast medium were obtained, revealing pneumocephalus. Air was seen in the subarachnoid space from C3 to C5 anteriorly, C3 down to C7 posteriorly, in the prepontine cistern, subarachnoid space, and bilaterally in the frontal convexity (Figs. 1-3). There was no evidence of subarachnoid hemorrhage. A repeat noncontrast medium CT of the brain the following day did not identify any pneumocephalus, and a magnetic

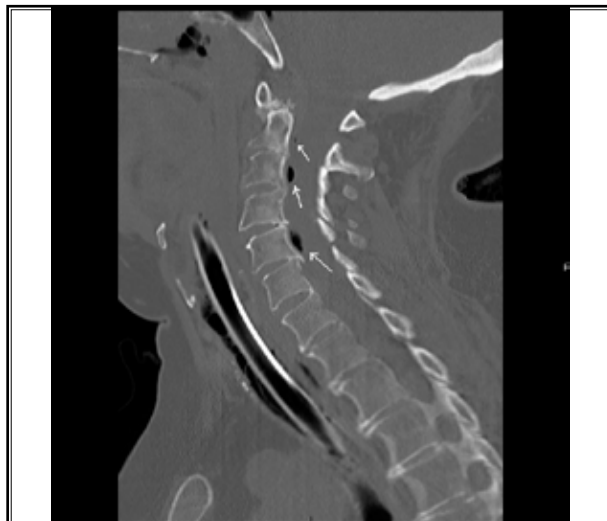


Fig. 1. CT of neck without contrast medium obtained initially upon hospitalization on day 1. Air droplets are seen in the cervical cord (white arrows), most likely in the subarachnoid space. Degenerative changes are seen in the vertebrae.



resonance image (MRI) of the brain performed 2 days later showed no acute or subacute infarct. Within 24 hours of admission, the patient was spontaneously moving her upper extremities. An electroencephalogram (EEG) on the second day of her hospitalization did not show any evidence of epileptic waves. After

48 hours, she was opening her eyes on command. She was extubated approximately 72 hours after being admitted.

At this point, the patient began a steady road to recovery while at the same time being worked up for neurologic versus cardiologic causes of the arrest. Neu-

rologically, an MRI was performed on day 4 with and without contrast medium. It showed no evidence of an acute or subacute infarct, although there was some evidence of an old infarct in the deep periventricular white matter. A repeat EEG on day 5 was normal, awake yet drowsy with some bradycardia. As part of her cardiac workup, the patient was monitored on telemetry for the remainder of her stay in the hospital. A nuclear Lexiscan stress test was completed on day 8. It showed a normal Lexiscan ECG, while the Cardiolite stress perfusion imaging showed a minimal focal photopenic area in the anterior lateral wall of the left ventricle. Because of these findings, the patient was to have a left heart catheterization. The patient refused the test at the time, continued to improve, and was discharged after a 14-day hospital stay. She eventually followed up as an outpatient for a cardiac catheterization, which showed patent coronary arteries. She continued to do fine without further issues and mental improvement.

Discussion

Epidural injection is a widely used modality for the management of spinal pain, and has been shown to be effective for the treatment of many etiologies of pain including radiculopathy, spinal stenosis, and disc herniations (2,12). With any procedure, there are some associated risks, but an extensive literature search could not find a previous case report of cardiopulmonary arrest with a cervical epidural steroid injection. We propose several possible mechanisms responsible for this patient's rapid deterioration, including blockade of the cardiac accelerator fibers, vasovagal response, pneumocephalus, and phrenic nerve involvement with apnea.

The cardioacceleratory center is located at the T1-T4 spinal levels and consists of cardiac accelerator fibers that leave the spinal cord at these levels (13). It has been postulated that a high thoracic or cervical blockade can lead to decreased sympathetic tone. One study showed 2 important concepts that could help explain our patient's condition, the first being that high thoracic epidural anesthesia can lead to diminished sympathetic outflow, which in turn could lead to bradycardia. Secondly, they show that the sympathetic blockade extends below the area of the segmental sensory block (14). In our case, we believe that an injection into the lower cervical area of C6-C7 led to sympathetic blockade of the cardiac accelerator fibers just below this spinal segment. Chan and Welch (15) also considered cardioaccelerator blockade in a case of cardiac arrest in a patient receiving thoracic epidural anesthesia. Another

study by Goertz et al (16) shows that during thoracic epidural anesthesia, there is a markedly reduced cardioacceleration in response to a decrease in arterial pressure, while preserving the slowing in response to increased arterial pressure. We suggest that a likely scenario in our patient is that the injection caused a sympathetic blockade of these cardiac accelerator fibers, which led to a rapidly induced, profound bradycardia followed by cardiopulmonary arrest. We note that the hypertension that was found upon arrival to the emergency department was most likely related to acute brain injury, and while we would expect profound hypotension in the setting of a severe sympathetic blockade, no vital signs were obtained at the time of the collapse.

Vasovagal reaction could lead to severe bradycardia, although we believe this is less likely due to the patient's deterioration. Vasovagal reactions are associated with hypotension and bradycardia, as well as loss of consciousness, but rarely complete cardiopulmonary collapse. One study of vasovagal reactions in cervical epidural steroid injections showed that as many as 8% of patients receiving a translaminal injections were associated with a vasovagal reaction (17). While the same article found that vasovagal reaction is significantly higher in cervical versus lumbar injections, none of the cases (n = 249) of epidural steroid injection resulted in cardiopulmonary arrest. A literature review by Abbasi et al (18) showed an even lower rate of vasovagal complications from cervical epidural spinal injection, ranging from 0-4%; again, there was no report of any complete cardiopulmonary arrest incident similar to what was seen in our case. We were able to find one case study describing cardiac arrest resulting from vasovagal response during an epidural procedure, where Sprung et al (19) describes the case of a 54-year-old man admitted for abdominal aortic aneurysm repair receiving epidural anesthesia at the T8-9 spinal level. Their patient was monitored by ECG, and upon insertion of the epidural needle, became unresponsive with a subsequent 42 second period of asystole. This patient was given atropine and ephedrine intravenously as well as cardiopulmonary resuscitation, and regained consciousness shortly thereafter. While this isolated case certainly puts vasovagal syncope on our differential, the fact that our patient did not shortly regain consciousness, as well as the cervical level of the injection, makes us less suspicious of vasovagal response.

Pneumocephalus is a rare but not unheard of complication of any procedure involving injection into the spinal cord region (8,20). Head and neck CT scans of

our patient showed pneumocephalus, thought to be in the subarachnoid space, however no intraventricular air was seen. The issue at hand becomes whether or not the pneumocephalus itself actually caused cardiopulmonary arrest, or was just another complication associated with the case. We could only find one case in the literature of pneumocephalus associated with cardiac arrest, and this was an isolated case of a 5-year-old child undergoing posterior fossa surgery for a cystic astrocytoma (21). One possible explanation for the pneumocephalus is that it resulted from the cardiopulmonary resuscitation by a mechanism described by Imanishi et al (22), where a "pump" mechanism is postulated. By this mechanism, negative thoracic pressure created by external cardiac massage can lead to a backflow of air entering a venous line and eventually traveling to the intracranial veins. This does not explain why air was found in the subarachnoid space, nor does it explain why air was found in the brain. If the air was introduced iatrogenically, we have yet to see literature describing subarachnoid pneumocephalus as the cause of cardiac arrest in a patient, and we cannot postulate on how this could have led to arrest. One possibility is that pneumocephalus is very painful, and may have led to arrest due to a vasovagal response as described above.

Due to the nature of the spinal levels in which the injection was taking place in this case, we cannot rule out the possibility of phrenic nerve involvement leading to apnea, followed by cardiopulmonary arrest. With phrenic nerve innervation located at the C3 spinal level, any involvement of this area of the spinal cord can lead

to respiratory compromise. A case study of patients undergoing cervical epidural spinal injection at the C7-T1 interspace showed that cervical epidural anesthesia leads to a measurable reduction in pulmonary functions consistent with the spread of analgesia to the C3 dermatome (23). While it has been shown that once analgesia reaches the C3 dermatome, there is significant change in forced expiratory volume in the first second of expiration (FEV1) and forced vital capacity (FVC), no outcomes of complete pulmonary arrest have been reported. Even when there was involvement of the C3 dermatome, the change in FEV1 was reported to be no more than 85% of normal, and FVC 84% of normal after 20 minutes. The injection in our case occurred at a similar, albeit closer, distance to this C3 spinal level (C6-C7 versus C7-T1), but we still would not expect to see such a significant and devastating drop in pulmonary function.

CONCLUSION

We report a case of cardiopulmonary arrest in a patient without any previous serious comorbidities except rheumatoid arthritis after a cervical epidural spinal injection. We believe that the most likely scenario was that the respiratory arrest was caused by severe sympathetic blockade of the cardiac accelerator fibers, leading to severe bradycardia and apnea. While clearly this is a rare outcome in what has become a common and well tolerated procedure, we believe it is important to be aware of the possibility of cardiac and/or pulmonary arrest, even in healthy patients and patients who have tolerated the procedure before.

REFERENCES

1. Derby R, Lee SH, Kim BJ, Chen Y, Seo KS. Complications following cervical epidural steroid injections by expert interventionalists in 2003. *Pain Physician* 2004; 7:445-449.
2. McGrath JM, Schaefer MP, Malkamaki DM. Incidence and characteristics of complications from epidural steroid injections. *Pain Med* 2011; 12:726-731.
3. Johnson BA, Schellhas KP, Pollei SR. Epidurography and therapeutic epidural injections: Technical considerations and experience with 5334 cases. *Am J Neuro-radiol* 1999; 20:697-705.
4. Huston CW, Slipman CW, Garvin C. Complications and side effects of cervical and lumbosacral selective nerve root injections. *Arch Phys Med Rehabil* 2005; 86:277-283.
5. Botwin KP, Castellanos R, Rao S, Hanna AF, Torres-Ramos FM, Gruber RD, Bouchlas CG, Fuoco GS. Complications of fluoroscopically guided interlaminar cervical epidural injections. *Arch Phys Med Rehabil* 2003; 84:627-633.
6. Bose B. Quadriplegia following cervical epidural steroid injections: Case report and review of the literature. *Spine J* 2005; 5:558-563.
7. Reitman CA, Watters W. Subdural hematoma after cervical epidural steroid injection. *Spine (Phila Pa 1976)* 2002; 27:E174-176.
8. Simopoulos T, Peeters-Asdourian C. Pneumocephalus after cervical epidural steroid injection. *Anesth Analg* 2001; 92:1576-1577.
9. Liguori GA, Sharrock NE. Asystole and severe bradycardia during epidural anesthesia in orthopedic patients. *Anesthesiology* 1997; 86:250-257.
10. Limongi JAG, Lins RS. Cardiopulmonary arrest in spinal anesthesia. *Rev Bras Anesthesiol* 2011; 61:110-120.
11. Geffin B, Shapiro L. Sinus bradycardia

- and asystole during spinal and epidural anesthesia: A report of 13 cases. *J Clin Anesth* 1998; 10:278-285.
12. Abdi S, Datta S, Trescot AM, Schulz DM, Adlaka R, Atluri S, Smith HS, Manchikanti L. Epidural steroids in the management of chronic spinal pain: A systematic review. *Pain Physician* 2007; 10:185-212.
 13. Cousins MJ, Bridenbaugh PO, Carr DB, Horlocker TT, Veering BT. Epidural Neural Blockade. In: Veering BT, Cousins MJ (ed) *Cousin's and Bridenbaugh's Neural Blockade in Clinical Anesthesia and Pain Medicine, 4th edition*. Lippincott, Williams & Wilkins, Philadelphia, 2009, pp 243-244.
 14. Hopf HB, Weißbach B, Peters J. High thoracic segmental epidural anesthesia diminishes sympathetic outflow to the legs, despite restriction of sensory blockade to the upper thorax. *Anesthesiology* 1990; 73:882-889.
 15. Chan KK, Welch KJ. Cardiac arrest during segmental thoracic epidural anesthesia. *Anesthesiology* 1997; 86:503-505.
 16. Goertz A, Seeling W. Baroreflex control of heart rate during high thoracic epidural anesthesia. *Anaesthesia* 1992; 47:984-987.
 17. Trentman TL, Rosenfeld DM, Seamans DP, Hentz JG, Stanek JP. Vasovagal reactions and other complications of cervical vs. lumbar translaminar epidural steroid injections. *Pain Pract* 2009; 9:59-64.
 18. Abbasi A, Malhotra G, Malanga G, Elovic E, Kahn S. Complications of interlaminar cervical epidural steroid injections. *Spine (Phila Pa 1976)* 2007; 32:2144-151.
 19. Sprung J, Abdelmalak B, Schoenwald PK. Vasovagal cardiac arrest during the insertion of an epidural catheter and before the administration of epidural medication. *Anesth Analg* 1998; 86:1263-1265.
 20. González-Carrasco FJ, Aguilar JL, Llubíá C, Nogués S, VidalLópez F. Pneumocephalus after accidental dural puncture during epidural anesthesia. *Reg Anesth* 1993; 18:193-195.
 21. Somasundaram T, Frost EA, Singh T, Shulman K. Cardiac arrest associated with tension pneumocephalus. *Anesthesiology* 1982; 56:73-75.
 22. Imanishi M, Nishimura A, Tabuse H, Miyamoto S, Sakaki T, Iwasaki S. Intracranial gas on CT after cardiopulmonary resuscitation: 4 cases. *Neuroradiology* 1998; 40:154-157.
 23. Stevens RA, Frey K, Sheikh T, Kao TC, Mikat-Stevens M, Morales M. Time course of the effects of cervical epidural anesthesia on pulmonary function. *Reg Anesth Pain Med* 1998; 23:20-24.