

## Letters to the Editor

## Pulmonary Embolism: A Rare Complication of Neurolytic Alcohol Celiac Plexus Block

### TO THE EDITOR:

We encountered a case of a 23-year-old woman who developed symptomatic pulmonary emboli immediately following a neurolytic alcohol celiac plexus block. We believe that this was caused by an inadvertent intravascular injection of alcohol and we describe the mechanism by which this may have occurred.

This patient presented to our clinic for a repeat neurolytic alcohol celiac plexus block for treatment of chronic, severe abdominal pain due to pancreatitis. She was positioned prone on the fluoroscopy table and appropriate landmarks were identified under fluoroscopic guidance. Under sterile conditions with 1% lidocaine local anesthesia, a 22 gauge, 4.75 inch spinal needle was advanced from a slightly lateral and oblique approach percutaneously until the needle tip was approximately one centimeter anterior to the vertebral body. After negative aspiration, initial injection of Omnipaque 180 contrast into the left needle revealed abnormal contrast spread suggestive of venous uptake (Fig. 1). The needle was then advanced 2 cm further in the lateral view (Fig. 2). This approach was repeated on the contralateral side until the needle tip was approximately 2 cm anterior to the vertebral body. Appropriate needle position was confirmed with Omnipaque 180 and fluoroscopy, with no further evidence of vascular runoff on either side. A volume of 20 mL consisting of a mixture of equal parts 1% lidocaine and 0.25% bupivacaine was injected at each site in 3–4 mL aliquots over several minutes. Following this, a total volume of 30 mL of a solution containing 15 mL of 98% dehydrated alcohol and 15 mL Omnipaque 180 was divided between each spinal needle and injected incrementally over several minutes. The needles were flushed with normal saline and removed.

At the conclusion of the procedure, the patient developed acute, severe chest pain with associated nausea, vomiting, and shortness of breath. Her vital signs were initially stable; however, she was transported to the emergency department (ED) for further evaluation.

Upon arrival to the ED, she rated her chest pain as a 9 out of 10 in severity on an 11-point Likert scale.

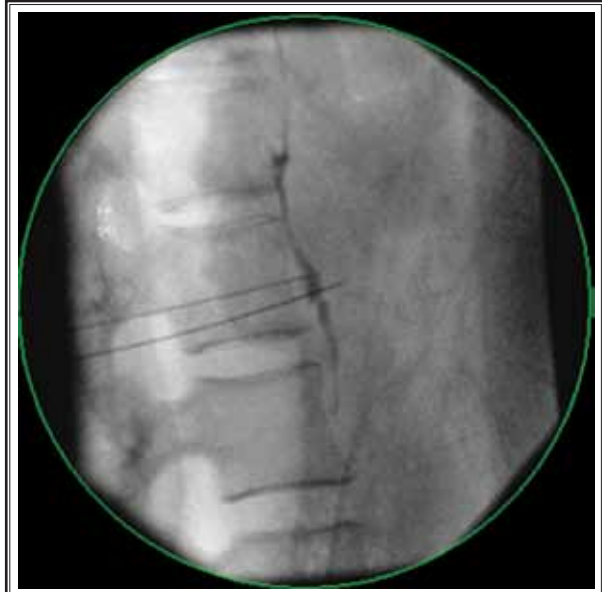


Fig. 1. Lateral view showing abnormal contrast pattern in the left-sided needle, placed just anterior to L1 vertebral body, thought to represent venous uptake.

Her initial vital signs were as follows: blood pressure of 132/99 mmHg, heart rate of 116 beats per minute, respiratory rate of 18 breaths per minute, and oxygen saturation of 98%.

Evaluation in the ED revealed elevated D-dimer at 1.05 and there was no evidence of venous thrombosis by ultrasound examination of bilateral lower extremities. Computed tomography angiogram of the chest showed non-occlusive pulmonary emboli, primarily within the segmental branches of the left lower lobe, and, to a lesser extent, the right lower lobe. A heparin infusion was initiated and the patient was admitted for further care.

Our patient developed pulmonary emboli despite having only minor risk factors and no previous history of thrombotic events. Her only potential risk factor for a thrombotic event was a hormone-releasing



Fig. 2. Lateral view after advancement of the left needle, showing normal contrast spread anterior to vertebral bodies and around the aorta.

contraceptive implant. However, thrombotic events are typically correlated with estrogen-containing contraceptives, and this patient's progestin-only implant has actually been shown to maintain proper balance between fibrinolysis and coagulation with no subsequent increase in risk for thrombosis (1). Additionally, she was noted to have a slightly elevated anticardiolipin IgM at 13 (12 is the upper limit of normal in our laboratory). However, these antibodies have been most closely linked to thrombotic events in patients with systemic lupus erythematosus (2), which was not present in this patient.

The temporal relationship of the alcohol injection with the onset of symptoms led us to believe that the ethanol was the cause of her pulmonary emboli. Intravascular ethanol is known to be associated with serious hemodynamic complications, including pulmonary hypertension, cardiovascular collapse, and death. Proposed mechanisms include direct pulmonary vasospasm, dislodgement of existing thrombi, and induction of sludge emboli via precipitation of endothelial cell and erythrocyte proteins (3-5).

Yata et al (5) describe 2 groups of pigs that had either pre-formed sludge products without alcohol or absolute ethanol alone injected into their pulmonary

artery. The sludge solution was generated ex vivo by mixing ethanol with serum, and the ethanol was removed prior to infusion after centrifugation. Microscopic examination of the lungs of both groups demonstrated the same amorphous sludge in their pulmonary vessels, which was histologically distinct from a normal thrombus. Additionally, both groups showed the same pattern of hemodynamic changes with increased pulmonary artery pressure and reduced aortic pressure. This proposed mechanism of ethanol-induced sludge embolism provides a potential explanation for the pulmonary emboli experienced by our patient.

In addition, multiple reports exist in the literature describing cases of pulmonary embolism following IV alcohol administration for sclerotherapy (3,6).

While the definitive cause of our patient's pulmonary emboli will likely never be known, the temporal relationship to alcohol injection during the neurolytic celiac plexus block and the known effects of alcohol in the vascular system certainly lends credence to the proposal that inadvertent intravascular injection of alcohol was the most likely inciting event. It is important to consider ways to avoid this adverse event in the future.

Complications from celiac plexus blocks can be divided into 2 categories: those due to the technique itself and those due to the drugs injected (7,8). Any pain originating from visceral structures that are innervated by the celiac plexus can also be alleviated effectively by blockade of the splanchnic nerves (7). The avoidance of alcohol by using radiofrequency techniques has been shown to be effective for blockade of the splanchnic nerves and may provide an alternative to alcohol neurolysis (7,8). Additionally, the use of a blunt tip needle may reduce, but not eliminate, the occurrence of vascular puncture, and some authors advocate the use of these needles as another potential way to minimize the risk of complications (9). Furthermore, advanced radiologic imaging that better defines anatomy, as well as contrast flow, can also reduce the risk of complications (9).

Although it is an uncommon complication, we feel that alcohol induced sludging and resultant pulmonary emboli should be considered in the differential diagnosis of patients who experience cardiopulmonary symptoms following neurolytic alcohol celiac plexus block. Vigilant prevention and prompt recognition of this rare complication are important aspects of maintaining patient safety.

Emily W. Petersen, MD  
Department of Anesthesiology  
Baylor Scott and White Health  
2401 South 31st St  
Temple, TX 76508  
E-mail: EmilyPetersenMD@gmail.com

Christopher J. Burnett, MD  
Department of Anesthesiology  
Baylor Scott and White Health  
2401 South 31st St  
Temple, TX 76508  
E-mail: Christopher.Burnett@BSWHealth.org

Kelsey R. Pohler, MD  
Department of Anesthesiology  
Baylor Scott and White Health  
2401 South 31st St  
Temple, TX 76508  
E-mail: Kelsey.Pohler@BSWHealth.org

Russell K. McAllister, MD  
Department of Anesthesiology  
Baylor Scott and White Health  
2401 South 31st St  
Temple, TX 76508  
E-mail: Russell.McAllister@BSWHealth.org

---

## REFERENCES

1. Egberg N, van Beek A, Gunnervik C, Hulkko S, Hirvonen E, Larsson-Cohn U, Bennink HC. Effects on the hemostatic system and liver function in relation to implanon and norplant. A prospective randomized clinical trial. *Contraception* 1998; 58:93-98.
2. Ginsberg JS, Wells PS, Brill-Edwards P, Donovan D, Moffatt K, Johnston M, Stevens P, Hirsh J. Antiphospholipid antibodies and venous thromboembolism. *Blood* 1995; 86:3685-3691.
3. Rimon U, Garniek A, Galili G, Golan G, Bensaid P, Morag B. Ethanol sclerotherapy of peripheral venous malformations. *Eur J Radiol* 2004; 52:283-287.
4. Hammer F, Boom LM, Mathurin P, Vanwijck RR. Ethanol sclerotherapy of venous malformations: Evaluation of systemic ethanol contamination. *J Vasc Interv Radiol* 2001; 12:595-600.
5. Yata S, Hashimoto M, Kaminou T, Ohuchi Y, Sugiura K, Adachi A, Kawai T, Endo M, Takasugi S, Yamamoto S, Matsumoto K, Kodani M, Ihaya T, Takahashi M, Ito H, Ogawa T. Influence of ethanol-induced pulmonary embolism on hemodynamics in pigs. *Ann Card Anesth* 2013; 16:245-249.
6. Tachibana K, Kobayashi S, Kojima T, Kaseno S, Kemmotsu O. Pulmonary emboli in sclerotherapy for peripheral vascular malformations under general anesthesia; a report of two cases. *Masui* 2004; 53:645-649.
7. Raj P. Celiac plexus/splanchnic nerve blocks. *Techniques in Regional Anesthesia and Pain Management* 2001; 5:102-115.
8. Raj P, Thomas J, Heavner J, Racz G, Lou L, Day M, Shaw B. The development of a technique for radiofrequency lesioning of splanchnic nerves. *Current Review of Pain* 1999; 3:377-387.
9. Scanlon GC, Moeller-Bertram T, Romanowsky SM, Wallace MS. Cervical transforaminal epidural steroid injections: More dangerous than we think? *Spine* 2007; 32:1249-1256.

