

Transient Paraplegia Following Alcohol Celiac Plexus Block

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Background and Objectives. A pancreatic cancer patient developing transient paraplegia compatible with spinal cord ischemia, following alcohol celiac plexus neurolysis, is described. **Methods.** A 58-year-old man with metastatic pancreatic cancer underwent celiac (deep splanchnic) alcohol neurolysis for management of severe epigastric and midback pain. In spite of apparently adequate needle position, he developed transient paraplegia consistent with anterior spinal artery syndrome. **Results.** The clinical findings suggest ischemia of the anterior spinal cord with complete motor and sensory paralysis to a T8 spinal cord level resulting from an anterior spinal artery syndrome. **Conclusions.** The cause of the limited bilateral transient paralysis following celiac plexus block in this patient may involve ischemia of the spinal cord associated with reversible arterial spasm following the injection of ethanol solution. *Reg Anesth 1995; 20: 352-355.*

Key words: celiac block, ethanol, complication, splanchnic block, ethanol, complication, paraplegia.

Celiac plexus block neurolysis is a useful analgesic technique for patients with upper abdominal cancer. Although neurologic complications are infrequent, celiac plexus neurolysis has produced paraplegia.^{1,2} It is hypothesized that paraplegia may result from neurolytic drug-induced spasm of lumbar segmental arteries that perfuse the spinal cord, and there are data demonstrating that canine lumbar arteries undergo contraction when exposed to low concentrations of alcohol.³ We present a case of transient paraplegia, compatible with spinal cord ischemia, following alcohol celiac plexus neurolysis in a patient with pancreatic cancer.

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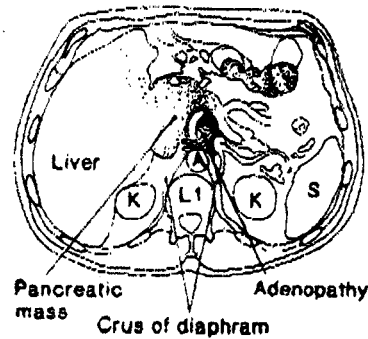
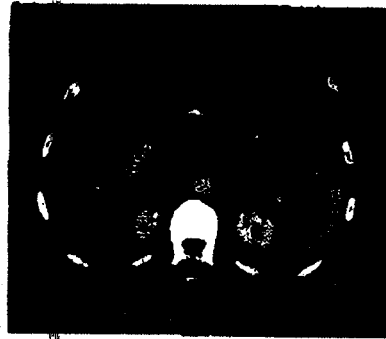
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Case Report

A 58-year-old man with metastatic pancreatic cancer was referred for evaluation and management of severe epigastric and midback pain. Over 10 months, the patient's pain had progressively increased in both constancy and intensity, and hydrocodone (2.5 mg, 12 to 16 tablets per day) no longer provided satisfactory analgesia. The patient underwent a lumbar spinal fusion at 26 years of age, had a 60 pack-year history of tobacco use, and during the year prior to referral, was treated for hypertension with atenolol and amlodipine.

A recent computed tomographic imaging of the patient's abdomen showed a large soft tissue mass in the region of the head of the pancreas and additional masses in the anterior and posterior aspects of the liver. Also noted at the time of scan were enlarged para-aortic lymph nodes, believed to be pancreatic metastases (Fig. 1). The referring physicians believed this patient's tumor was unlikely to

Fig. 1. Computed tomographic cross-section of patient's abdomen (left) and interpretive line drawing (right) at the level of the first lumbar vertebrae, highlighting the patient's pancreatic mass and para-aortic adenopathy. A, aorta; K, kidney; LI, first lumbar vertebrae; S, spleen.



be responsive to surgical, radiotherapeutic, or chemotherapeutic techniques. Thus, after a discussion of risks and benefits, the patient elected to proceed with the neurolytic celiac plexus block.

The patient underwent a retrocural celiac plexus block after being placed in a prone position, with a pillow placed beneath his abdomen to reduce lumbar lordosis. After aseptic skin preparation, 22-gauge needles were inserted bilaterally by the senior pain consultant (D.L.B.), 1 centimeter caudad to each twelfth rib, at a point 7.5 centimeters lateral to the midline of the back, angled in an oblique plane along a line joining the needle insertion site to the junction of the T12 and L1 spinous processes. In the cross-section plane, the needles were angled 45° from the coronal plane. The needles initially contacted the lateral vertebral body and subsequently were redirected to "walk off" the vertebral body anterolaterally. On final placement, the left-sided needle clearly evinced transmission of aortic pulsation, whereas the right-sided needle was inserted an additional 1 centimeter, compared to the left-sided needle.

Following needle placement and negative aspiration, 10 mL 1.5% lidocaine was injected via each needle as a test dose. After 10 minutes of observation, the patient retained complete sensory and motor function of his lower extremities to focused examination, while reporting resolution of his abdominal pain. Following an additional 4 minutes, the neurolytic solution, consisting of 25 mL absolute alcohol and 25 mL 0.25% bupivacaine (Sensorcaine, Astra USA, Westborough, MA) was injected. Twenty-five milliliters of this solution was injected in 5-mL increments via each needle over a 3-minute interval, with frequent incremental negative aspirations. Five minutes after the completion of the injection, the patient reported a heaviness in both legs, initially in the left more than the right. Then, during the next 5 minutes, the patient developed

paralysis of both legs. He reported no abdominal pain, and neurologic examination revealed a T8 bilateral sensory level to pinprick, and no muscle strength in his lower extremity, lower abdominal, or truncal muscles (approximately the level of his midrectus, or T8). Vibration and proprioceptive senses were not evaluated at this time. The patient was emergently transferred to an angiographic suite, where neuroradiologists and neurosurgeons were consulted; they planned to visualize his aorta radiographically and to consider infusing a vasodilator if spasm of a major thoracolumbar artery was identified. Approximately 80 minutes after completion of the neurolytic block, the patient was in the neuroradiology suite and his dense paraplegia began to fade. It resolved over approximately a 10-minute period, and nearly 90 minutes after injection of the neurolytic solution, sensorimotor function had returned to near normal levels. With the improvement in symptoms, angiography was deferred. The patient was observed overnight in the neurologic intensive care unit, while a spinal cord trauma protocol was initiated. Approximately 90 minutes after the neurolytic injection, the patient received 30 mg/kg of methylprednisolone intravenously as a loading dose, and approximately 5.4 mg/kg/h for the next 19 hours while he was in the hospital. On follow-up examination at 2 days and 3 weeks after the neurolytic block, the patient continued to report epigastric and midback pain relief and complete recovery of neurologic function.

Discussion

The relevant issue in this patient's experience with a neurolytic alcohol celiac plexus block was that bilateral motor paralysis and loss of sensation to a T8 level developed within 22 minutes, and completely resolved within 90 minutes of the block, with prolonged pain relief as evidence of cor-

rect needle placement. To review, after a negative needle aspiration, the injection of the local anesthetic test with 10 mL 1.5% lidocaine bilaterally, prior to injecting ethanol, was neurologically inconsequential, and the needles were unremoved until after the neurolytic block was completed. Thus, direct injection of a neurolytic ethanol solution into the subarachnoid or epidural spaces was considered extremely unlikely because of limited spread of paralysis up to the midthorax, a completely transient paralytic effect, and extremely rapid resolution of paraplegia. Also, the patient reported excellent epigastric pain relief for weeks after the celiac plexus block, suggesting correct placement of the injectate.

The clinical findings suggest ischemia of the anterior spinal cord with complete motor and sensory paralysis to a T8 spinal cord level, resulting from an anterior spinal artery syndrome, a condition first discussed by Spiller in 1909.⁹ The vascular supply of the spinal cord was originally described by Adamkiewicz in 1882, and while there is great variability among the radicular arteries, the largest is typically called the artery of Adamkiewicz (Fig. 2). The takeoff of the artery of Adamkiewicz from the segmental artery occurs in 80% of cases on the left side at any level between T7 and L4 vertebrae, most often between T9 and T11.¹⁰ The relative importance of this artery depends on the collateral vascular supply, which is most easily estimated by the number of anterior

radicular arteries. The greater the number of other anterior radicular arteries, the less critical the condition with dysfunction of the artery of Adamkiewicz. The anterior spinal artery, which is formed by the joining of the left and right anterior radicular arteries, supplies the major portion of the cord including the anterior horns, the central gray matter, the lateral horns, and the anterior and lateral spinal tracts.^{11,12} A pair of posterior radicular arteries supply nutrient branches to the dorsal white matter and dorsal portion of the posterior horns.

If one considers that an anterior spinal artery syndrome may be the cause of our patient's transient neurologic deficit, the possible mechanisms include an effect on the radicular arteries by: arterial vasospasm in response to localized exposure or direct injection of alcohol; arterial vasospasm due to mechanical damage of an artery caused by a needle; arterial vasospasm in response to local anesthesia; and extraluminal arterial compression due to limited spread of neurolytic solution in a confined space.

There are reports^{13,14} that support the hypothesis that alcohol-induced vasospasm of the feeding arteries of Adamkiewicz could be a possible cause of prolonged neurologic deficit following celiac plexus block. Furthermore, there is recent evidence that suggests that ethanol or phenol exposure causes concentration-dependent contractions in vascular smooth muscle.⁵ This vascular reactivity may theoretically occur with alcohol exposure to an artery without requiring a direct intra-arterial injection. In our patient, negative needle aspiration for blood prior to injections suggest the ethanol was not injected into the arterial lumen.

The possibility of localized trauma to a major lumbar radicular feeding artery, with or without a resultant hematoma, is perhaps less likely. Case reports^{14,15} suggest that actual damage to a major medullary feeding artery or vasospasm of the artery resulting from needle probing may cause the anterior spinal artery syndrome. Our patient had only transient paralysis, suggesting no permanent damage, as might occur with a significant arterial injury. However, reversible arterial vasospasm secondary to needle placement could result in the transient neurologic deficits that our patient experienced.

Extraluminal compression of a major feeding artery(ies) within the retroperitoneal space may have also occurred, and led to the anterior spinal artery syndrome. Relevant facts include the volume of neurolytic solution placed into a confined space, with the presence of enlarged para-aortic lymph nodes perhaps further limiting a potential space. It

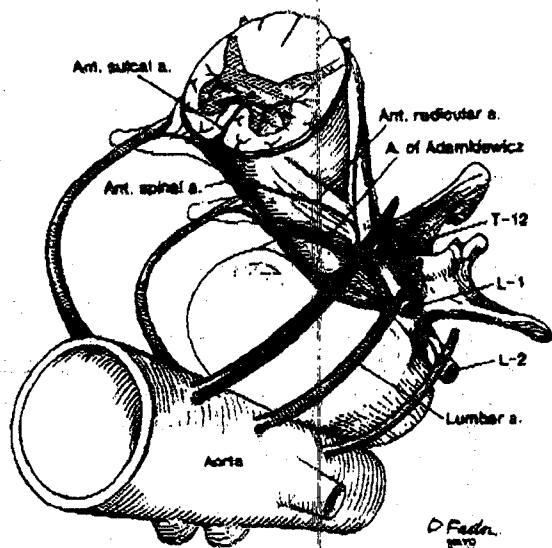


Fig. 2. An example of spinal cord arterial blood supply at the level of low thoracic and high lumbar vertebrae. The most common major feeding artery from the aorta arises on the left, and its most frequent location is vertebral level T9-T11.

is conceivable that compression of the major feeding artery(ies) could cause relative ischemia of the spinal cord, which is consistent with our patient's rapid onset of paralysis. Nevertheless, data suggest solutions injected into the retrocrural space diffuse freely to at least the midthoracic level, making extraluminal compression seem less likely.¹¹

Additionally, local anesthesia may potentially cause concentration dependent contractions that are reversible in vascular smooth muscle.¹⁴⁻¹⁶ Celiac plexus block requires the use of local anesthesia, both as a test dose confirming correct needle placement, and to provide analgesia when used concomitantly with ethanol, which is painful on injection. This vascular reactivity could be reversible and may not require direct intra-arterial injection, but simply injection of local anesthesia in close proximity to the artery.

In our case, anatomic landmarks—not radiologic guidance—were used to assist with the positioning of needles for neurolytic injection. However, it is doubtful that this potential anterior spinal artery syndrome would have been minimized with needle placement assisted by the use of radiographic imaging or more experienced operators.¹⁷ It is possible that radiocontrast dye might detect an intravascular injection, but not the placement of injectate surrounding a vascular structure. This assertion is supported by the evidence of several case reports of resultant paraplegia despite the use of radiologic guidance.¹⁷⁻¹⁹

Conclusions

The cause of the limited, bilateral, transient paralysis after celiac plexus block in this patient may involve the anterior spinal artery syndrome. We believe ischemia of the spinal cord associated with reversible arterial vasospasm of the feeding artery(ies) of Adamkiewicz after the injection of ethanol solution may have contributed to the paralysis.

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