Case Report

Paraplegia after celiac plexus neurolysis in a patient with pancreatic cancer
- A case report and literature review -

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A 65-year-old male patient underwent C-arm fluoroscopy-guided bilateral celiac plexus neurolysis to relieve peritoneal seeding-related pain associated with pancreatic cancer. Following confirmation of spreading, and no intravascular injection of contrast media, 7.5 ml of 0.25% chirocaine was injected in each side. The pain subsided after the block, with no motor or sensory deficits. Subsequently, celiac plexus neurolysis with 99.8% alcohol was performed using a posterolateral approach under fluoroscopic guidance. The patient was instructed to maintain a prone position for 2 hours while the procedure was performed. Approximately 4 hours later, the patient experienced paralysis of both lower extremities and hypoesthesia. Emergent magnetic resonance imaging of the thoracic and lumbar spine revealed gray matter signal change in the cord and conus medullaris at the T10–L1 level, and decreased perfusion at the T11–T12 vertebral bodies, suggesting spinal cord infarction. The patient remained paraplegic until his death 24 days later.

Keywords: Celiac plexus; Infarction; Pancreatic neoplasms; Paraplegia; Spinal cord.

Celiac plexus neurolysis (CPN) is an ablative procedure of the celiac plexus, which aims to destroy afferent pain-transmitting fibers from the abdominal viscera. CPN is a relatively safe procedure; it is associated with several relatively common and mostly transient complications, such as local pain (96%), hypotension (10%), and diarrhea (44%) [1]. However, severe complications of CPN can include neurologic injuries such as paraplegia and anal and bladder sphincter dysfunction. Herein, we report a case of paraplegia by acute spinal cord infarction after CPN with alcohol.

CASE REPORT

A 65-year-old male patient (height, 166 cm; weight, 69 kg) was referred from the Department of Oncology for the management of epigastric pain caused by pancreatic cancer with peritoneal seeding. The patient had previously been treated for hypertension and diabetes. While undergoing 6 cycles of chemotherapy, he received dalteparin (10,000 IU) as treatment for pulmonary thromboembolism with deep vein thrombosis. For pain control, a 100 μg/h fentanyl patch was applied to the skin, and an average of 4 mg/h of morphine was administered intravenously. However, the patient complained of continuous pain (visual analog scale score 5/10; 0 = no pain, 10 = the most severe pain imaginable), so a C-arm fluoroscopic-guided CPN was performed. Before the procedure, the patient was fully informed of its potential complications.

The patient lay in the prone position while undergoing the C-arm fluoroscopic-guided bilateral CPN at the L1 level using

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a posterolateral approach. A 21-gauge, 14-cm needle (Hakko, Japan) was inserted for the retrocrural approach of the celiac plexus block and placed at the posterior aorta in front of the vertebral body (Fig. 1). The proper location of the needle was confirmed by real-time imaging using a contrast agent, and no blood was aspirated when the needle was placed. Following confirmation of spreading near the aorta and no intravascular injection of contrast media (Fig. 2), 2–3 ml of contrast media was injected in each side. In each site, 0.5% chirocaine (7.5 ml) was injected, and the optimal spreading of local anesthetics was confirmed by C-arm fluoroscopy. Approximately 15 minutes after the block, the patient's pain subsided, and no motor or sensory deficits were observed. Subsequently, 99.8% alcohol (7 ml) was injected in each needle for additional neurolysis. After the procedure, the patient was instructed to maintain a prone position for 2 hours. Approximately 4 hours later, the patient noted paralysis of both his lower extremities, as well as hypoesthesia. A neurological examination also showed a decrease in sensory levels below the L1 dermatome. Thus, the patient was referred to a neurologist due to complete bilateral lower extremity and anal sphincter paralysis. Using emergent magnetic resonance imaging of the thoracic and lumbar spine, it was determined that there was a gray matter signal change in the cord and conus medullaris at the T10–L1 level, and decreased perfusion at the T11–T12 vertebral bodies, suggesting a spinal cord infarction (Fig. 3). Prednisolone was injected with 1 g q every 24 hours for 3 days, followed by 60 mg of prednisolone for 1 week which was then tapered down to 10 mg for 1 week. High-dose steroids had no effect on our patient. The patient had complete pain relief following the procedure despite paraplegia. After 20 days, the amount of ascites had increased, and hypotension was aggravated. However, the patient and caregiver did not want any further treatment. A "Do Not Resuscitate" order was issued, and the patient died 4 days later.

DISCUSSION

A considerable number of cases of acute paraplegia after CPN have been reported for some time although it is rare. Paraplegia may occur when the nerve-destroying agent enters the subarachnoid or subdural space and directly impairs the spinal cord or somatosensory nerve [2]. Many authors report that damage to the Adamkiewicz artery by the needle or
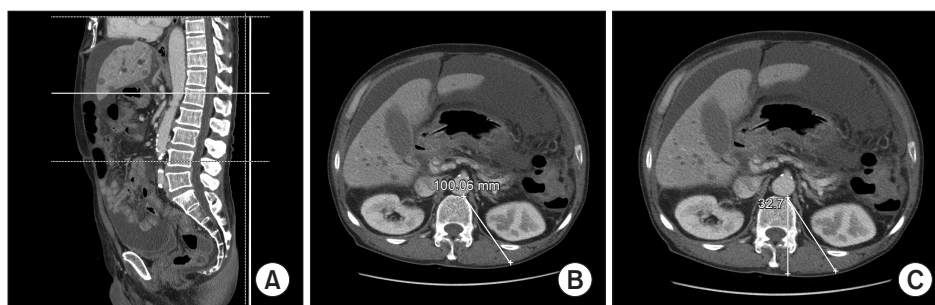

Fig. 1. The position of the transverse computed tomography (CT) image at the L1 level (A), needle depth (B), and the angle of needle insertion to the target point (C). We assessed the enhanced CT transverse images and selected the level without vessels connecting to the aorta for needle insertion.
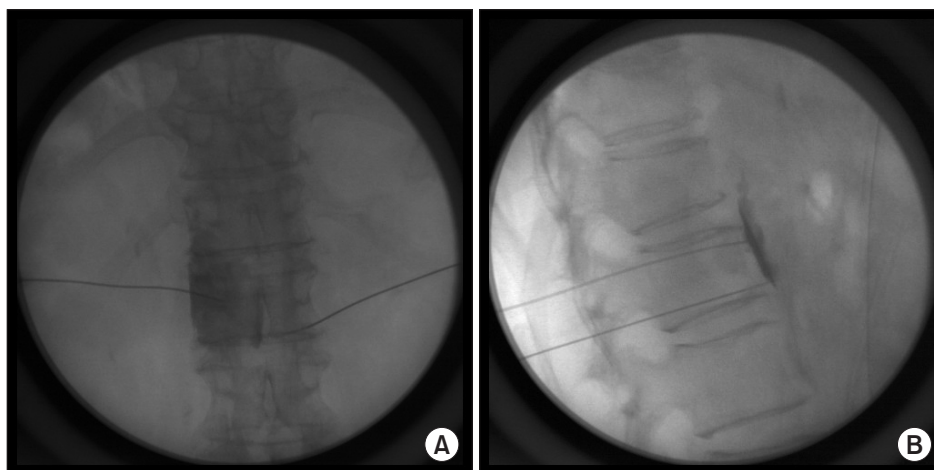

Fig. 2. The fluoroscopic anteroposterior (A) and lateral (B) view show the contrast medium spreading vertically over the 1st lumbar prevertebral space.
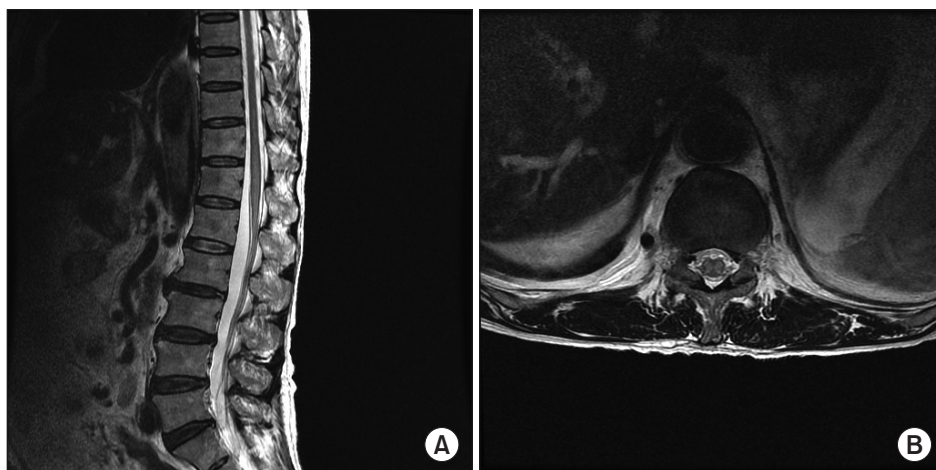
Fig. 3. T2-weighted sagittal (A) and transverse (B) magnetic resonance images show increased signal intensity in the spinal cord from the 10th thoracic to 1st lumbar level, suggesting the possibility of ischemic myelopathy.

drug-induced vasospasm may result in spinal cord ischemia which can cause paraplegia. In this case, damage to the Adamkiewicz artery may not be the cause of paraplegia as there was no paraplegia or weakness for over 15 minutes after needle insertion before the injection of alcohol. We hypothesized that the cause of paraplegia may be alcohol spreading to the spinal cord or vasospasm due to the alcohol. In a case series of 2,730 CPN procedures, major complications of neurological deficits were limited to 4 cases (< 0.2%) [3]. All 4 cases had permanent paraplegia, while 3 of the 4 cases had loss of anal and bladder sphincter function as well. Alcohol (50–100%) was used in all 4 cases.

Previously published articles on paraplegia after CPN are summarized in Table 1. In this case, the patient noted lower extremity paralysis with decreased sensory perception 4 hours after CPN. According to previous case reports, paraplegia can occur within 10–15 minutes of CPN [4–6]. In other cases, it was detected 1 or 3 hours later [7,8]. However, paraplegia can gradually develop over 2 days [9]. In some cases, it was difficult to detect the exact time of occurrence of paraplegia as the patient was under sedation [10,11]. Paralysis following a blind CPN was first reported by Galizia and Lahiri [4] in 1974. The blind procedure was performed, and the patient was symptomatic immediately after the injection of phenol on the left side suggesting a direct injury to the spinal cord or nerve rather than an arterial injury. With the exception of Galizia's article, most cases of paraplegia were reported with the use of alcohol. It is difficult to say whether paraplegia has a higher prevalence rate when using alcohol, rather than phenol, in CPN due to the lack of relative investigation. Although both alcohol and phenol are widely used for

neurolysis, literature is scant on the relative effectiveness, duration of benefit, and complication profile of the two agents. No difference in pain outcomes was observed when comparing alcohol- and phenol-based splanchnic nerve neurolysis [12]. In a pilot study of phenol-based endoscopic ultrasound-guided CPN (EUS-CPN), phenol and ethanol had similar pain-relieving effects. Phenol, however, may be superior to ethanol in EUS-CPN procedures when comparing the incidence of inebriation and burning pain [13]. In an experiment conducted in dogs, the contractions of the segmental lumbar arteries caused by ethanol or phenol did not appear to be mediated through adrenergic, opioid, muscarinic, or serotonin receptors or sodium channels. The addition of procaine eliminated the sustained response of the vessel to ethanol-induced contraction, while this was not the case for phenol-induced contractions. It is possible that a concentration of phenol or alcohol leading to a contraction of these vessels could be achieved clinically [14]. They used 1%, 3%, 6%, 7%, 8%, 9% and 12% phenol and 3%, 6%, 10%, 25%, 50%, 75% and 90% ethanol. A high concentration of phenol, such as 8%, 9%, 12%, and low concentration of ethanol, such as 3% and 6%, produced contractile responses. Therefore, a lower concentration of phenol and higher concentration of alcohol may be safe and limit vasospasms.

Until 2010, most cases of paraplegia after CPN were due to a percutaneous approach, with the exception of 2 cases of injection in the surgical field. After 2012, all 4 cases of paraplegia were from EUS-CPN. This may be due to the increased use of EUS-CPN, and the inability to confirm the intravascular injection during EUS-CPN. As shown in Table 1, 14 (73.68%) of the 19 cases were permanent paraplegia, while

Table 1. A Brief Summary of Published Articles

Study	Year	Indication	Route	Paraplegia	Neurolytic agent	Other detail information
Galizia [4]	1974	Cancer	PCN	Paraplegia	Phenol 6%	The Blind procedure, symptom was occurred immediately after the injection of phenol on the left side
Leung [19]	1983	Chronic pancreatitis	PCN	Monoplegia only	Alcohol 75%	Left side monoplegia only
Woodham [7]	1989	Cancer	PCN	Paraplegia	Alcohol 90%	Alcohol was injected on the left only
van Dongen [6]	1991	Cancer	PCN	Paraplegia	Alcohol 48%	Under sedation
Jabbal [8]	1992	Cancer	PCN	Reversible paraplegia	Alcohol 50%	Right side dominant paraplegia
Na [2]	1993	Cancer	PCN	Reversible paraplegia	Alcohol 50%	30 min after, burning sensation was occurred, slowly progression of paraplegia
Davies [3]	1993	Chronic pancreatitis	PCN	Paraplegia	Alcohol 66%	Seven previous coeliac plexus blocks
			PCN	Paraplegia	Alcohol 50%	Under general anaesthesia, patient moved with injection
		Chronic pancreatitis	PCN	Paraplegia	Alcohol 90%	Under sedation
		Chronic pancreatitis	PCN	Paraplegia	Alcohol 100%	Under general anaesthesia
Wong [5]	1995	Cancer	PCN	Reversible paraplegia	Alcohol	Transient paraplegia with anterior spinal artery syndrome
Kinoshita H	1996	Cancer	OP direct	Paraplegia	Alcohol	Performed by open anterior approach under direct vision
Jun Hayakawa	1997	Cancer	Op direct	Paraplegia	Alcohol 99.5%	Performed by open anterior approach under direct vision
Kumar [18]	2001	Cancer	PCN	Reversible paraplegia	Alcohol	Left side dominant paraplegia magnetic resonance imaging and myelography were normal Dorsal column pathways was spared
Jeon [9]	2010	Cancer	PCN	Reversible paraplegia	Alcohol 99%	Left side blood aspiration
Fujii [10]	2012	Cancer	EUS	Paraplegia	Alcohol 99%	Under general anaesthesia anterior spinal cord infarct
Mittal [15]	2012	Cancer	EUS	Paraplegia	Alcohol 99%	Postprocedural computed tomography showed that neurolytic/contrast agents were distributed on both sides of the celiac trunk
Minaga [11]	2016	Cancer	EUS	Paraplegia	Alcohol 100%	Under sedation
Ibrahim	2017	Cancer	EUS	Paraplegia	Alcohol 100%	Anterior spinal cord infarct, embolic occlusion of Adamkiewicz arteries are detected

PCN: pancreatic cystic neoplasms, Op: operation, EUS: endoscopic ultrasound.

5 (26.32%) cases were reversible. Reversible lower extremity paralysis was first reported by Jabbal and Hunton [8] in 1992, where they assumed that the cause of paralysis was damage to the artery. However, no correlation between the position of the needle tip and the artery was found in this case due to the lack of a contrast agent.

The anatomy of the blood supply to the spinal cord is variable, and the major anterior radicular artery of Adamkiewicz is the largest of the anterior medullary feeders for the supply of the lower two-thirds of the spinal cord. In approximately 80% of cases, this artery appears on the left side between the T7 and L4 vertebral levels with a predilection for the T9 to T11

levels. Occasionally, this artery can be closely related to the celiac ganglion. The relative blood supply to the cord from the radicular arteries and the artery of Adamkiewicz varies considerably and depends on the degree of collateral circulation. Our patient's underlying diseases, such as hypertension and diabetes, would have caused poor collateral blood perfusion. Therefore, the spinal cord infarction was likely caused by weakening of the collaterals which could not maintain sufficient spinal cord perfusion once the major radicular artery was damaged during CPN. The mechanism of acute spinal cord ischemia after vascular puncture may involve vasospasm due to the high alcohol content (50–100%) and high

volume (20–50 ml) needle injury, causing acute thrombosis or the propagation of alcohol along segmental arteries leading to multiple artery spasms [15]. Preliminary data regarding radiofrequency ablation of splanchnic nerves are encouraging [16] and this method does not require neurolytic agents, such as alcohol and phenol. Even though further study is required to evaluate the safety of radiofrequency ablation of splanchnic nerves; theoretically, it could be considered an alternative choice to replace CPN with alcohol [16]. Furthermore, we hypothesize that eliminating the use of alcohol or phenol by splanchnic radiofrequency ablation could help prevent spinal cord ischemia due to vasoconstriction and damage caused by the direct injection of the drug into the subarachnoid or subdural space.

Paraplegia may occur when alcohol spreads to the subarachnoid or subdural space and directly damages the spinal cord or somatosensory nerve. If the spinal cord or somatosensory nerve is directly damaged, the patient may experience an abnormal sensation at the time of the procedure, or an immediate loss of muscle strength. In this case, the patient did not complain of burning pain or other symptoms after the alcohol injection. After this injection, the spreading within the anterior vertebral body area was limited as evidenced by C-arm fluoroscopy. Therefore, direct injection or spreading of alcohol to the epidural, subarachnoid, or subdural space may not have occurred in this case. We hypothesized that the position change after CPN could have caused alcohol to spread to another site or nerve. Therefore, we instructed the patient to maintain the prone position for 2 hours, eliminating the possibility of spreading the alcohol to other sites, such as the nerve root, subarachnoid, or subdural space. Some physicians instruct patients to maintain a prone position for over 2 hours after CPN [17]. However, we did not find studies examining the post-CPN position and maintaining time.

Of the 19 cases of paraplegia following CPN, 18 (94.74%) were bilateral paraplegia while only 1 (5.26%) case was monoplegia. Kumar et al. [18] reported left leg-dominant paraplegia, while Jabbal and Hunton [8] reported right side-dominant paraplegia. Leung et al. [19] reported left side monoplegia only. Jeon et al. [9] reported that blood was aspirated during the initial insertion of the left needle which may have caused spinal cord ischemia by puncturing the arterial structure. As spinal edema developed, progressive neurological deficits were noted, and the patient was thought to have progressively improved with the spinal edema. In a case described by Woodham and Hanna [7], the spread of the dye did not conform to the classical picture. They injected 90% alcohol (30 ml) on the left side, and 0.25% bupivacaine (20 ml) on the right. As described above, the Adamkiewicz artery is likely located on the left side of the aorta, therefore, the possibility of spinal cord ischemia may be high when CPN is performed on the left side. As mentioned above, Jeon et al. [9] reported that blood was aspirated during the initial insertion of the left needle suggesting a need to be more careful when performing injections on the left side for CPN. The use of real-time radiography and digital subtraction angiography may help identify vascular injury more quickly. However, there were two cases of paraplegia of the lower extremities despite visualization of the CPN in the operation field. While the abdomen was open for the operation, the alcohol was injected posterior to the aorta at the level of the celiac plexus. Thus, even if the procedure is performed with caution, complications may occur due to anatomical variations in blood vessel location.

Although these fatal complications may occur after CPN, there is no doubt that CPN can reduce the side effects of opioids, as well as pain. This procedure also helps improve the quality of life of patients with terminal cancer. However, this procedure should be performed carefully in patients with benign disease like chronic pancreatitis due to the risk of serious complications. Furthermore, the use of radiofrequency ablation, rather than alcohol or phenol injection, should be further investigated for its potential to reduce spinal cord ischemia due to vasospasm and damage caused by direct injection of the alcohol into the subarachnoid or subdural space. Paraplegia has been considered a rare adverse effect of CPN, but is a very serious complication; therefore, pain physicians should always be aware of the risk of potentially serious consequences.

CONFLICTS OF INTEREST

No potential conflict of interest relevant to this article was reported.

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