

Case Report



Spinal Cord Infarction After Transarterial Chemoembolization for Hepatocellular Carcinoma

Sang-Geun Lee , Sung Min Cho , Kum Whang , Yeon gyu Jang , Jongyeon Kim , and Jongwook Choi

Department of Neurosurgery, Wonju Severance Christian Hospital, Yonsei University Wonju College of Medicine, Wonju, Korea



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Address for correspondence:

Sung Min Cho

Department of Neurosurgery, Wonju Severance Christian Hospital, Yonsei University Wonju College of Medicine, 20 Ilsan-ro, Wonju 26426, Korea.
Email: ns1287@daum.net

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ORCID iDs

Sang-Geun Lee <https://orcid.org/0000-0003-3627-7426>
Sung Min Cho <https://orcid.org/0000-0002-7593-4815>
Kum Whang <https://orcid.org/0000-0002-3701-847X>
Yeon gyu Jang <https://orcid.org/0000-0002-4434-5540>
Jongyeon Kim <https://orcid.org/0000-0002-9407-0127>
Jongwook Choi <https://orcid.org/0000-0003-2593-3870>

Conflict of Interest

The authors have no financial conflicts of interest.

ABSTRACT

Transarterial chemoembolization (TACE) is an effective treatment for unresectable hepatocellular carcinoma (HCC). It is considered relatively safe. However, fatal complications such as pulmonary edema and liver abscesses can occur. Spinal infarction due to local embolism of the central nervous system after TACE is a very rare, but fatal complication. Here, we report a case of spinal cord infarction after TACE for ruptured HCC. Paraplegia occurred at the T10 sensory level 6 hours after the procedure. The patient received steroid megadose therapy but died 5 days later due to exacerbation of metabolic acidosis and blood loss. This case demonstrates the need for a comprehensive and extensive study of arterial blood flow prior to angiography.

Keywords: Spinal cord ischemia; Embolization; Hepatocellular carcinoma

INTRODUCTION

Hepatocellular carcinoma (HCC) is a global disease that frequently invades adjacent organs. There are treatment plans such as liver resection, parotid liver transplantation, radiofrequency ablation, and microwave resection. However, there are alternative treatments that are not subject to surgical intervention. Currently, transarterial chemoembolization (TACE) is widely used for treatment as an interventional therapy used in the treatment of mid-stage hepatocellular carcinoma. After the arteries are selected, chemotherapy is injected and sutured with an embolus. As a result, tumor growth is slowed and the patient's survival rate is improved. However, some post-TACE patients experience multiple complications, including postembolic syndrome characterized by fever, elevated liver enzymes, and right upper quadrant pain, as well as extrahepatic injury such as acute cholecystitis, pulmonary embolism, liver abscess, and bile duct damage, acute pancreatitis. Although the onset of embolism is known to be rare because the spinal cord forms multiple feeding artery anastomoses, spinal infarction after TACE can also occur very rarely. We present a case in which a patient developed paraplegia after receiving TACE.

CASE REPORT

A patient of age 58, male with past history of type 2 diabetes mellitus, hypertension and cardiac angina. Over a period of 4 months the patient lost about 30 kg of weight. Poor appetite and both lower extremity edema was noticed, and he complained of dyspnea then hepatic mass was confirmed during the examination. Followed magnetic resonance imaging (MRI) study showed ruptured infiltrative HCC entire right hemi-liver, with tumor thrombosis formation of main portal vein and right portal vein branches, and also lung metastases. On admission he did not complain of neurological signs or symptoms. Along right hepatic artery and right T9 intercostal artery, which were considered the tumor feeding arteries based on angiography, TACE was performed by interventional radiologist using adriamycin 30 mg + Lipiodol 20 cc mixed injection (**FIGURE 1**). Artery of adamkiewicz was observed in the right T9 intercostal artery angiogram (**FIGURE 2**) and catheter advance was attempted, but entry was not possible due to the tortuous vascular architecture and narrows vessel diameter, so

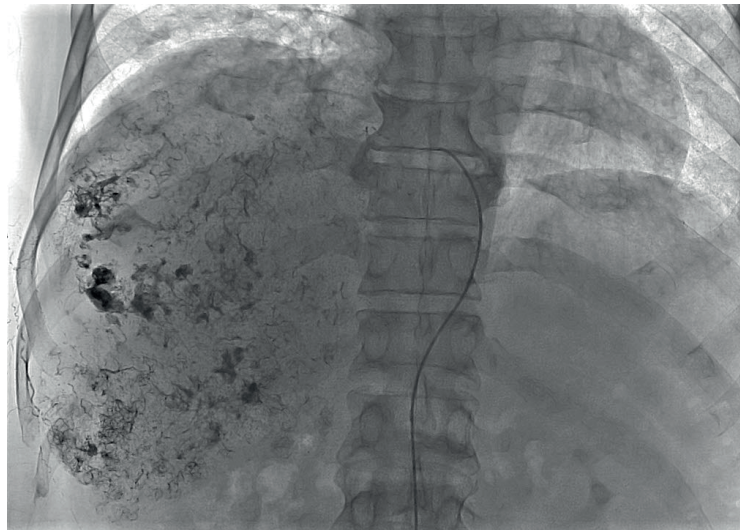


FIGURE 1. Angiograms obtained after super-selective embolization.

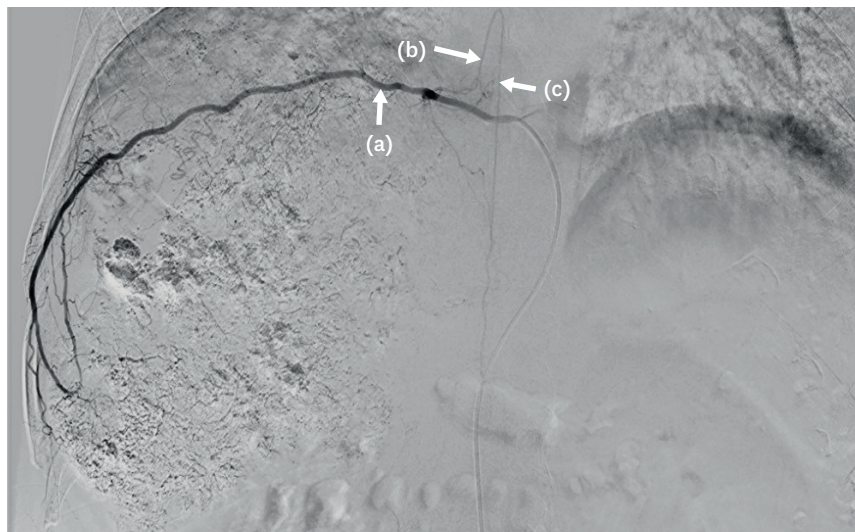


FIGURE 2. Angiograms obtained after right T9 intercostal artery selection. Right T9 Intercostal artery (a), artery of adamkiewicz (b) and anterior spinal artery (c).

the procedure was proceeded carefully. The patient was paraplegic and was loss of somatic sensation with the loss of proprioception below the T10 dermatome after 6 hours of the procedure. A thorough neurological examination of Medical Research Council (MRC) score of 0 for Lower extremity while the upper extremity showed an MRC score of 5. Deep tendon reflex and anal sphincter tone was absent and Babinski reflex present. MRI of the Spine (FIGURES 3 & 4), after 12 hours of TACE, review showed a spinal cord infarction on T4-L1 level. To prevent the progression of cord injury immediate megadose corticosteroid therapy was pursued, after the diagnoses of spinal cord infarction, with 1800mg methylprednisolone loading dose for 15 minutes, followed by 7,450 mg methylprednisolone maintain dose for 23 hours, and then 10mg methylprednisolone per day for 2 days. However, 2 days later, metabolic acidosis and hypovolemic shock was caused by continuous blood loss following HCC rupture after the onset and did not improve even with continuous sodium bicarbonate fluid infusion and transfusion. To correct acidosis and uremic syndrome, continuous renal replacement therapy was started, but after 3 days, the patient was expired.



FIGURE 3. T2-weighted sagittal magnetic resonance imaging of the spine showing increased intramedullary signal intensity at the T4 to L1 level.

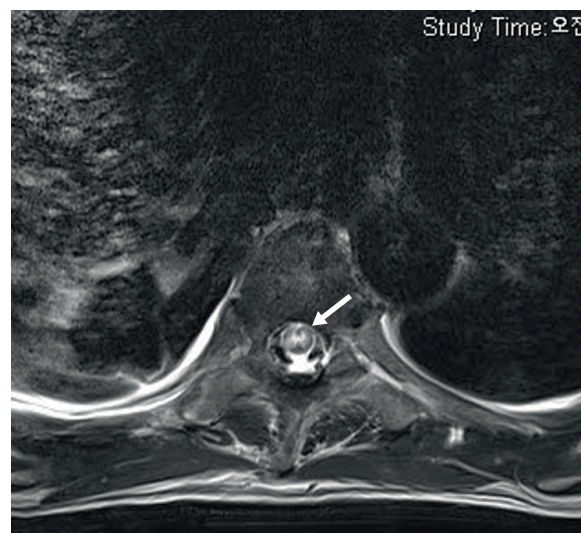


FIGURE 4. T2-weighted axial magnetic resonance imaging of the thoracic spine at the T10 level showing increased signal intensity of the spinal cord (arrow).

DISCUSSION

HCC is a common cause of cancer-related death worldwide. The dominant arterial vascular supply of HCC provides a rationale for treating these cancers with TACE. Several studies have designated TACE as an important option.^{8,9)}

Although the positive characteristics of TACE are that it is less invasive and relatively safe, TACE may be associated with several side effects.⁷⁾ Fever, abdominal pain, nausea and vomiting, increased white blood cells, and elevated liver enzymes last for hours to days. Among the rare ischemic complications, neurological complications are rarely TACE-related and are usually caused by cerebral embolism. Spinal ischemia following TACE is very rare and has a fatal outcome with a prevalence of 0.3%.^{6,10)}

Spinal cord injury after TACE is usually described as either hepatic artery injury or decreased blood flow to the site of the previous TACE, which can lead to collateral recruitment. Occasionally it can result from accidental mobilization of spinal cord branches that arise from the intercostal or lumbar collateral vessels. The main blood supply to the spinal cord (**FIGURE 5**) is through a single anterior vertebral artery (AVA) and two posterior vertebral arteries (PVA). The AVA is formed by the vertebral arteries that originate in the first part of the subclavian artery.^{1,2,6)} before they connect to each other and become the basilar artery, the vertebral artery provides branches that each become the AVA.^{3,7)} The spinal arteries and PVA supply additional arteries that run down the spinal cord at each level of the vertebrae through the intervertebral foramen. These additional arteries are called segmental vertebral arteries. Infarction due to intercostal artery embolism was suspected due to anatomical structure, but additional studies were not conducted due to the patient's medical complications. In another case Kim et al.⁷⁾ reported a spinal cord infarction on T9 and T10 level that occurred 6 hours after TACE. Electromyography was followed, and central conduction defect was observed. The patient received intravenous dexamethasone injection, and motor and sensory functions recovered after 2 months.

Although there is no specific guideline regarding the lipiodol dose, it is reported that the frequency of embolic events increases when lipiodol of 0.3 mL/kg or 20 mL or more is used.^{4,5)}

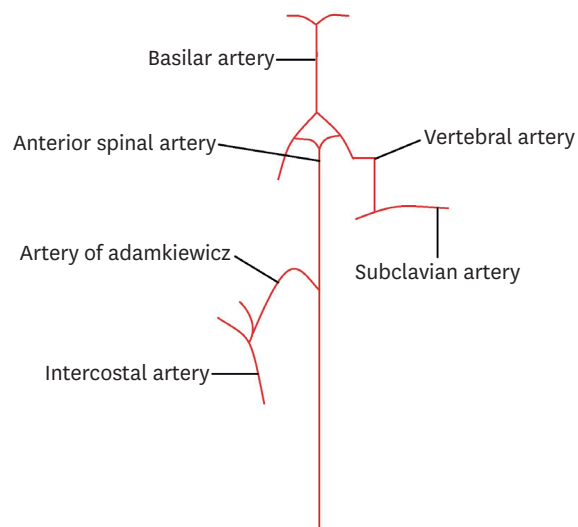


FIGURE 5. Vascular architecture of anterior spinal artery and artery of adamkiewicz (schematic illustration).

Currently, 0.1–0.3 mL/kg is suggested as an appropriate amount, and if there is a possibility of an embolic event, it is recommended to use a smaller amount of lipiodol than usual.

For the treatment of acute spinal cord injury, the National Acute Spinal Cord Injury Study (NASCIS) 2 recommends megadose methylprednisolone steroid therapy. However, while the use of high-dose steroids is currently considered a treatment option, it is not the standard of care due to questions about the reproducibility, functional significance, and risk of complications of benefits.

CONCLUSION

Through this case review, it was confirmed that TACE could cause serious neurological complications and raises the importance of its prevention. Extreme caution is required for the procedure while targeting the proper vessel and verifying its target during embolization. As for this case, when the artery of adamkiewicz and ASA are contrasted in the TACE, precise selection of artery and appropriate of approach method to the selected artery or embolization through another vessel is vital in the procedure. If appropriate selection of artery inaccessible and every other feeding artery approach is implausible, termination and another approach to treatment should be considered.

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