中文題目：肝惡性腫瘤患者接受肝動脈栓塞化學療法後以腦部 Lipiodol 栓塞為表現症狀之病例報告

英文題目：Cerebral Lipiodol Embolism in Hepatocellular Carcinoma Patients after Treated with Transarterial Chemoembolization**:** A Case Report

作　　者：林祐霆[1]，許樹湖[1,2]

服務單位：[1] 中國醫藥大學附設醫院內科部，[2] 中國醫藥大學附設醫院內科部消化系

Background

For unresectable hepatocellular carcinoma (HCC), transcatheter arterial chemoembolization (TACE) has widely been used, which is associated with reported complications including acute hepatic failure, hepatic infarction, extrahepatic embolization, etc. [1, 2], among which cerebral lipiodol embolism (CLE) is rarely documented. We herein report a case of CLE after TACE in HCC.

Case report

This 54-year-old male, with a history of hepatitis C related cirrhosis, unresectable HCC over right hepatic lobe s/p TACE for 4 times, and HCC rupture s/p received TAE (Fig.1.). He was admitted via OPD for scheduled 5th TACE. By proceeding medical record, the first 4 times of TACE were smoothly completed without complication. His ADL is totally independent and laboratory examinations before the 5th TACE were within normal limit. TACE was then performed. After superselective catheterization of branch of gastroepiploic and splenic arteries with microcatheter, 10 mL of lipiodol mixed with adriamycin 20 mg was injected. Then, Gelfoam cubes mixed with antibiotics was injected for proximal embolization. The process was smoothly completed (Fig.2.).

However, seizure attack was noted around 12 hours later after the treatment. No compromising vital signs were recorded but poor consciousness, nystagmus, and dilated pupils. After being treated with Lorazepam, he received brain CT examination, revealing abnormal hyperdense lesions in the cerebellum, bilateral thalami, and bilateral cerebral cortex. Diffuse cerebral lipiodol embolism was suspected (Fig.3.). Poor consciousness and intermittent seizures had been being noted in the following days under fluid hydration and anti-epilepsy medications use. EEG was suggestive of diffuse epileptogenicity in right temporal region. Lumbar puncture was performed but no evident infection. Brain MRI was arranged, demonstrating cerebral lipiodol embolism with acute to subacute infarction in the cerebellum, bilateral thalami, and cerebrum (Fig.4). Regaining more clear consciousness level was noted in the following hospitalization, but the patient presented blindness of both eyes and needed assistance while sitting up due to lower extremity weakness. For blindness study, visual evoked potential study was done and was suggestive of functional perturbation of bilateral prechiasmatic optic pathway. Visual field defect was also confirmed. He was then discharge under stable vital signs, poor visual function, sluggish speaking and weakness of both lower extremities.

Discussion

Cerebral lipiodol embolism rarely occurs in HCC after TACE. The symptoms, by reviewing previously published case reports, of CLE are nonspecific, including change of mental status, visual disturbance, headache, seizure, and hemiparetic weakness. Death or vegetative status is the worst clinical outcome. One research article indicated CLE patients are usually aged around 60 years, male predominant, have multiple times of TAE/TACE, and treated with over 20 mL of lipiodol to right liver lobe [3]. Though CLE is not as common as other complications after TACE, it is reasonable to recognize CLE via patient's history, clinical manifestation and course, and imaging results such as diffuse hyperdensity lesions on non-contrast head CT or hyperintense on brain MRI.

Several mechanisms were proposed [2, 4, 5, 6]. Cerebral lipiodol embolism may be attributable to an intracardiac right-to left shunt via a patent foramen ovale or intrapulmonary arteriovenous shunt. On the other aspect, patients having advanced hepatic diseases are more likely to have a pulmonary arteriovenous shunt. Shunt from the inferior phrenic artery to the pulmonary vasculature also a causable route in pulmonary lipiodol embolism. It can be inferred that shunting between systemic and pulmonary vasculatures would expedite the extravasation of lipiodol into the systemic circulation and the risk of CLE is escalated accordingly. No or undetectable patent foramen ovale due to too small on a routine echocardiography is showed in this patient. The patient has advanced hepatic disease, no hepatic vessels supplying the tumor, and potential shunt that is unveiled during TACE or chest CT may be inferable.

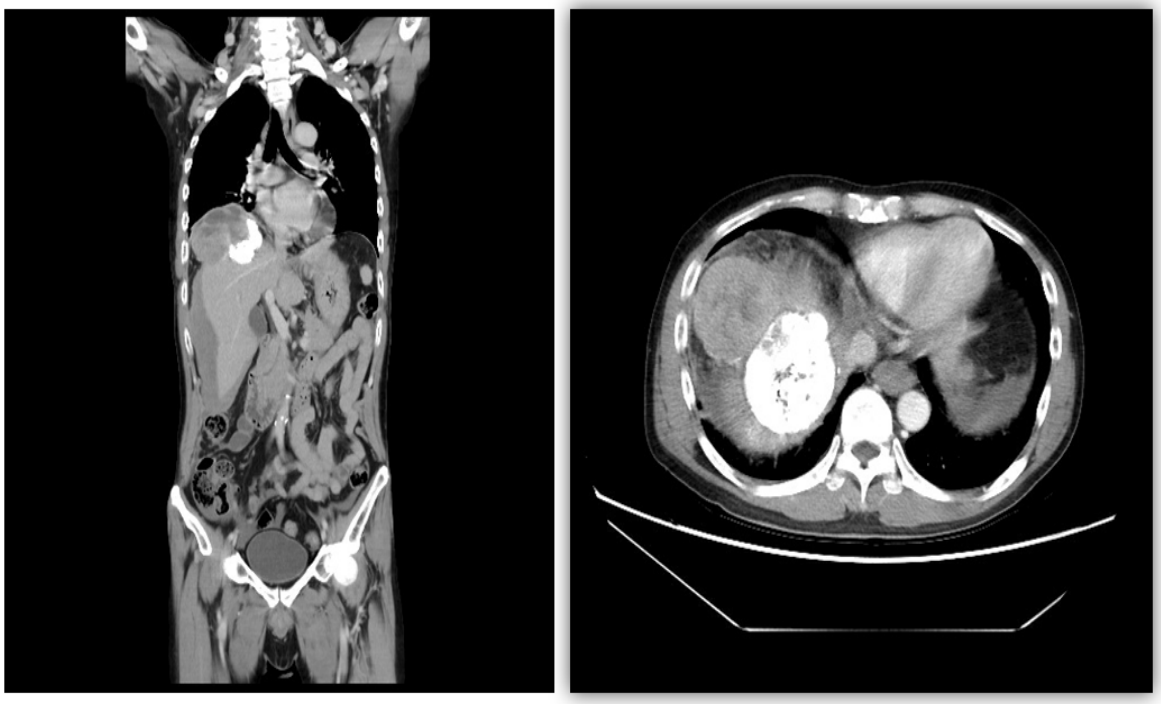

Fig.1. Abdominal computed tomography (CT) scan two months before 5th TACE show status of HCC post TACE, viable tumors in the dome region, and intraabdominal hematoma.

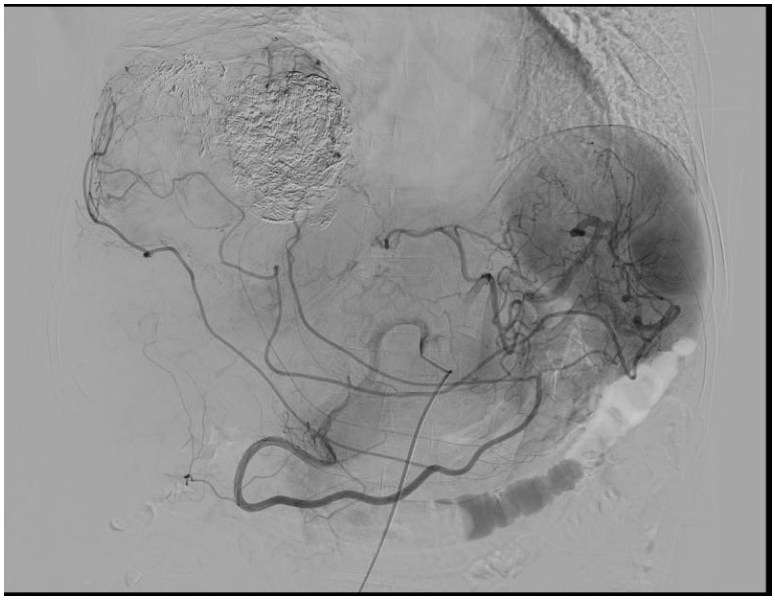

Fig.2 Angiogram show HCC in right hepatic lobes, with blood supply from branch of gastroepiploic and splenic arteries

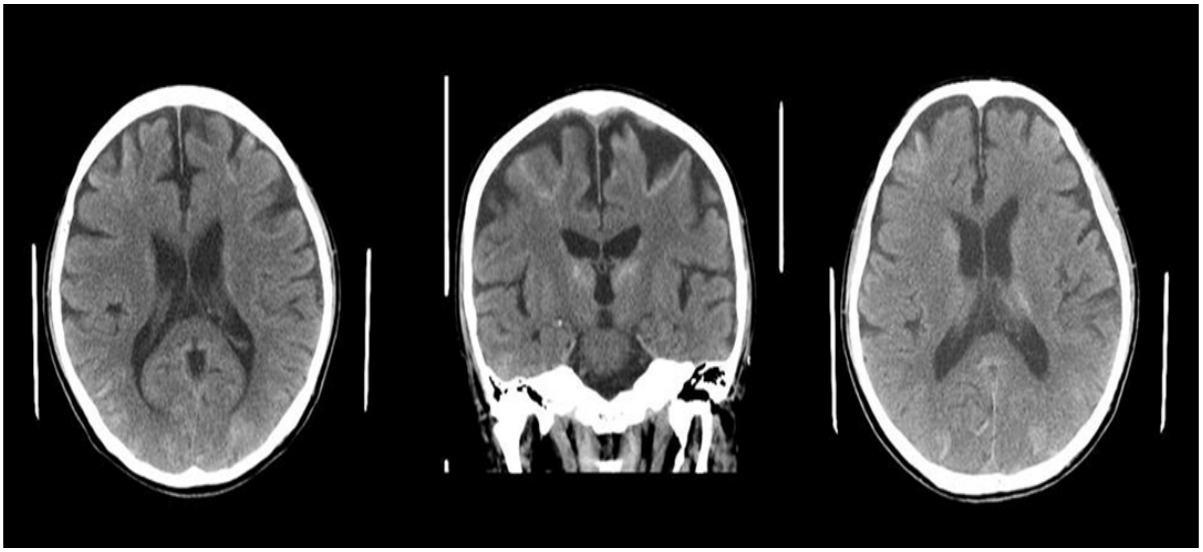

Fig.3. Non-contrast-enhanced CT scan obtained 12 hours after TACE show hyperdense lesions in the cerebellum, bilateral thalami, and bilateral cerebral cortex.

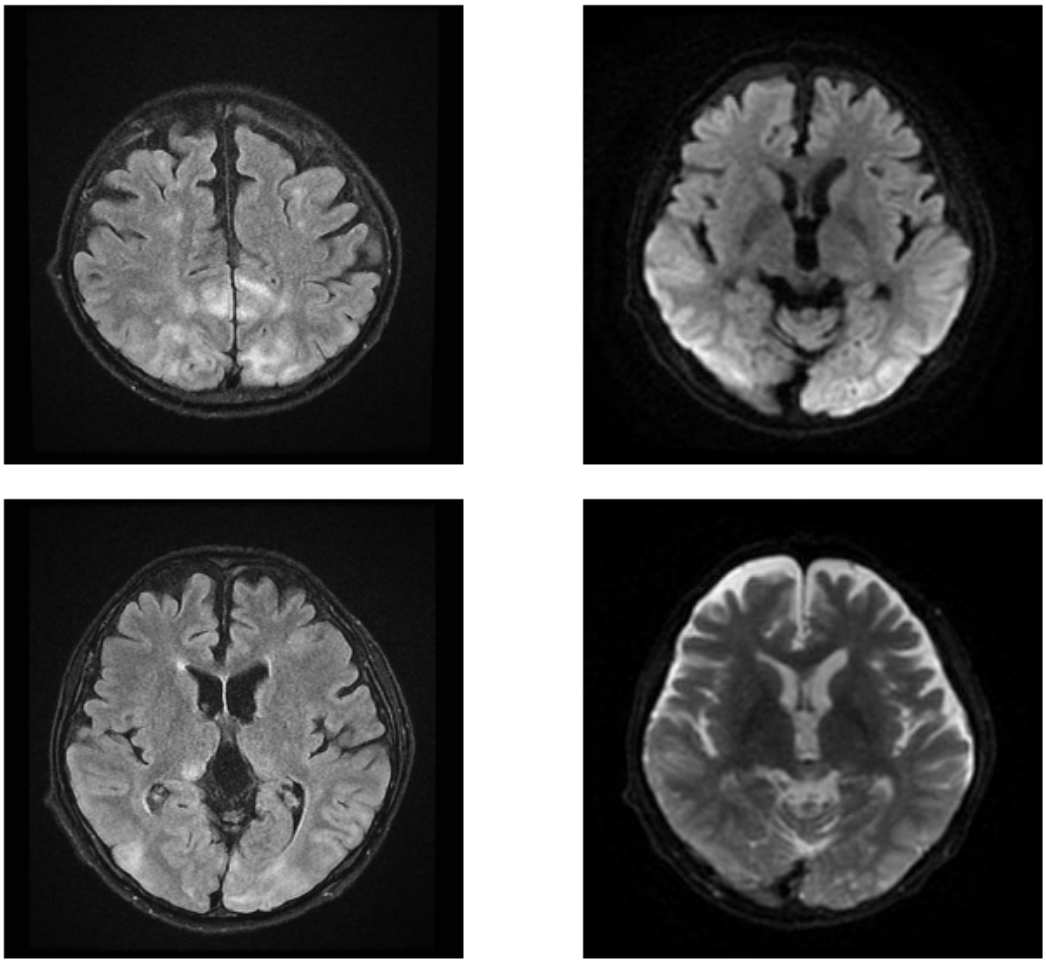

Fig.4. Nuclear Magnetic Resonance Imaging obtained 5 days after TACE show multifocal abnormal hyperintense lesions in the gray matter of cerebellum, bilateral thalami, and cerebrum on T2WI and diffusion-weighted imaging (DWI). Focal lesion in the right occipital lobe was with low apparent diffusion coefficient (ADC) value. CLE with acute to subacute infarction in the cerebellum, bilateral thalami, and cerebrum were recorded.

Reference:

1.Takao H, Makita K, Doi I, Watanabe T. Cerebral lipiodol embolism after transcatheter arterial chemoembolization of hepatocellular carcinoma. J Comput Assist Tomogr 2005; 29:680-682

2.Matsumoto K, Nojiri J, Takase Y, Egashira Y, Azama S, Kato A, Kitahara K, Miyazaki K, Kudo S. Cerebral lipiodol embolism: a complication of transcatheter arterial chemoembolization for hepatocellular carcinoma. Cardiovasc Intervent Radiol 2007; 30: 512-514

3. Chu H-J, Lee C-W, Yeh S-J, Tsai L-K, Tang S-C, Jeng J-S Cerebral Lipiodol Embolism in Hepatocellular Carcinoma Patients Treated with Transarterial Embolization/Chemoembolization. PLoS ONE 2015 10(6): e0129367

4. Chung JW, Park JH, Im JG, et al. (1993) Pulmonary oil embolism after transcatheter oily chemoembolization of hepatocellular carcinoma. Radiology 187:689–693

5. Krowka MJ, Cortese DA (1994) Hepatopulmonary syndrome: Current concepts in diagnostic and therapeutic considerations. Chest 105:1528–1537
6. Bánsághi Z. Cerebral iodized lipid embolization via a pulmonary arteriovenous shunt: rare complication of transcatheter arterial embolization for hepatocellular carcinoma. World journal of surgical oncology. 2013; 11:122.