Background:

Endoscopic treatments with the embolic or adhesive materials for esophageal varices in cirrhotic patients can cause cerebrovascular complications.

This mechanism is explained by the presence of porto-pulmonary venous anastomosis (PPVA). PPVA, one of portosystemic shunts associated with portal hypertension in cirrhotic patient, are collaterals between esophageal anastomosis (PPVA). PPVA, one of portosystemic shunts associated with complications.

Patients & Methods:

A 60-year-old man with liver cirrhosis was admitted to our hospital for endoscopic prophylaxis with argon plasma coagulation to prevent recurrent hemorrhage from esophageal varices. He underwent initial endoscopic variceal ligation for bleeding from raptured esophageal varices 2 years ago. He had subsequently undergone additional endoscopic variceal ligation 3 times, thus the majority of his esophageal varices diminished.

He received endoscopic argon plasma coagulation in a left lateral position. Argon plasma coagulation was applied in the lower esophagus to coagulate circumferentially. It took approximately 30 minutes to the end. Although the entry of gas bubbles into subepithelial vessels was clearly observed and gas bubbles were flowing toward the oral side slowly, no harmful phenomenon was noted on careful monitoring of the blood pressure, blood oxygen saturation, and heart rate.

Results & discussion:

He recovered to be conscious uneventfully after returning to the ward, however he suddenly develop left hemiplegia with right conjugate deviation 7 hours after the end of the procedure. Meanwhile, left-sided tonic-clonic convulsion occurred and subsequently transformed to generalized seizure. Because his convulsion was refractory to the intravenous diazepam due to status epileptic, he began to receive mechanical ventilation with endotracheal intubation under the administration of fosphenytoin, levetiracetam, and propofol.

Despite head computed tomography revealed no apparent abnormal findings including pneumocephalus, diffusion weighted head magnetic resonance imaging showed a globally hyperintense signal lesion spreading in the territory of right middle cerebral artery. There was a record of atrial fibrillation, and a patent foramen ovale was also absent on transthoracic echocardiography. His neurological impairment finally remained severe, and he passed away on 30 days hospitalization.

The most likely etiology of cerebral infarction in this case is considered that argon gas bubbles penetrated into esophageal varices during endoscopic argon plasma coagulation therapy delivered to the middle cerebral artery through porto-pulmonary venous anastomosis. Retrospectively, three-dimensional computed tomography scan at the portal venous phase taken 2 years ago revealed that his esophageal varix was directly communicating with the left atrium.

Conclusion & perspectives:

We present previously unreported case of cerebral embolism as a complication of endoscopic argon plasma coagulation for esophageal varices.

First, confirming porto-pulmonary venous anastomosis on 3D-computed tomography can be useful for preventing side effect of endoscopic treatment with argon plasma coagulation. Secondary, treatment of argon gas embolization is challenging due to extremely low solubility.

Table 1: Gas solubility in water in 298K, 101kPa