



tissue plasminogen activator.

Careful induction by mask with sevoflurane up to maximum 5% was performed. A 22 gauge intravenous line was placed and fentanyl and propofol were given. After tracheal intubation, anesthesia was maintained with sevoflurane with a minimal alveolar concentration of 1.3. Dexamethasone and ondansetron were administered as anti-emetics. Antibiotic prophylaxis consisted of cefazolin and only acetaminophen was given at the end of surgery. The patient was monitored during surgery with ECG, pulse oximetry, capnography and a non-invasive blood pressure. All vital parameters stayed stable during the whole length of the procedure. The procedure consisted of an ultrasound guided puncture of a right iliac lymph node and injection of Lipiodol. After injection, there was drainage in the cisterna chyli and the thoracic duct. The thoracic duct however seemed obstructed. Probably due to earlier cardio-thoracic surgeries. Drainage of Lipiodol occurred through collaterals to the hilus, neck and axillar lymphatic vessels. After saturating the lymphatic vessels, the procedure was stopped. The patient was extubated in the operating room after completion of the surgery and admitted to the Post Anesthesia Care Unit (PACU).

Approximately one hour after admission on the PACU, the patient became acutely unresponsive and had a generalized tonic-clonic seizure. The seizure lasted only 30 seconds and faded spontaneously before any benzodiazepines were given. A head Computed Tomography (CT) scan showed countless hyper densities throughout the brain. When readmitted to the PACU, a new seizure occurred, lasting longer than the first one. Because the patient was desaturating as well, the trachea was intubated with 80 mg of propofol and 30 mg of rocuronium. An arterial and central venous line were placed due to hemodynamic instability and noradrenaline was infused. Levetiracetam was given intravenously and the patient was transferred to the pediatric intensive care unit. At the intensive care unit, the patient developed a Systemic Inflammatory Response Syndrome (SIRS) and multi-organ failure with Lipiodol embolism in the kidneys, liver and lungs. Inhaled NO and hemodynamic support was started. 21 days later, the patient was extubated and listed for heart transplantation. Control CT scan showed improvement of the hyper densities, but no complete remission. Altered neurological status persisted.

## DISCUSSION

Percutaneous interventional lymphatic embolization is performed with increasing frequency. It significantly improves symptoms in patients with plastic bronchitis. Cerebral Lipiodol embolization after lymphatic duct embolization is a rare complications and the incidence is unknown because only symptomatic patients undergo neuroimaging. The use of an oil-based contrast agent in children with right-to-left shunting poses a risk for systemic embolization. To minimize this risk, systemic to-pulmonary venous collateral embolization and temporary fenestration occlusion in these patients are performed [1]. There are multiple case reports describing cerebral Lipiodol embolization after trans arterial chemoembolization for hepatocellular carcinoma [5,6]. Less cases are published

after lymphatic embolization for chylothorax [7]. The underlying mechanism for how Lipiodol reaches the brain remains unclear. It involves hepatopulmonary, pulmonary arteriovenous or right-to-left intracardiac shunt. In a case report from Kirschen et al., cerebral embolization occurred due to direct shunting between abnormal lymphatic channels and the pulmonary venous circulation [8]. In a case series of Dori et al. [2], a similar case to ours was described. Lipiodol embolism occurred in a post TCPC patient with a complete occlusion of the thoracic duct and no known right-to-left shunts. Numerous lymphovenous connections were seen between lymphatic collaterals and the pulmonary veins, which is possibly the source of the shunting. In our case, a transoesophageal echocardiography was performed in advance and after the embolization which excluded a patent foramen ovale or any right-to-left shunt. Our hypothesis is that the thoracic duct was absent or occluded due to previous cardio-thoracic surgery. A fistula was probably formed from the thoracic duct to the left atrium. Treatment consisted of mechanical ventilation due to failing oxygenation during the second seizure. Baseline oxygen saturation was 92% in this patient with a Fontan circulation. Only supportive treatment can be given. Optimization of the TCPC pathway and cardiovascular circulation should be considered as an important part of the treatment as well. Previous reports describe that the onset of symptoms occurs less than six hours from exposure in 85% of cases [4]. The amount of contrast that reaches the brain may also influence the occurrence. The risk increases when more than 20 mL of Lipiodol is used. The recommended maximum dose of ethiodized oil is 0.25 mL.kg-1. Poor neurological outcome has been reported in previous cases up to 20%.

## CONCLUSION

Cerebral Lipiodol embolization is a rare but severe complication after lymphatic interventional radiography. It should be considered when neurological symptoms occurs in the postoperative period. Prolonged PACU admission is recommended. Neuroimaging should be performed without any delay. Supportive care is the mainstay treatment for cerebral Lipiodol embolization. Cardiac catheterization could be performed prior to the procedure in patients with congenital heart disease to exclude abnormal shunting.

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## FINANCIAL DISCLOSURES

None.

## CONFLICTS OF INTEREST

Tanay Shukla, Sofian Bouneb and Johan Vandommele have no conflicts of interest to declare.

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