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Fatal cerebral venous air embolism during Endoscopic Retrograde Cholangiopancreatography – Case Report and review of the literature

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Abstract

Iatrogenic systemic air embolism during Endoscopic Retrograde Cholangiopancreatography (ERCP) is a rare but potentially fatal complication. We present the case of a patient that showed a persistent comatose state after ERCP. Radiological imaging was consistent with venous cerebral air embolism followed by venous congestion with fatal brain swelling. Only 5 cases of fatal intracranial air embolism during ERCP have been reported in the literature before. Awareness of this potentially fatal complication and knowledge of adequate therapeutic measures is necessary to improve the outcome in those patients.

Keywords: ERCP, air embolism, cerebral, systemic, fatal, radiological imaging

Introduction:

Endoscopic retrograde cholangio-pancreaticography (ERCP) is a routine minimal invasive technique for diagnostic and therapeutic purposes. According to a multi center study from 1998 the rate of major complications was 1.38 % in diagnostic and 5.4% in therapeutic ERCPs; fatal complications occurred in 0.21% and 0.49% respectively¹, being even lower in centers with high case volumes. Major complications include pancreatitis, cholangitis, duodenal perforation, hemorrhage and the exceedingly rare event of systemic air embolism.

According to our own research of the literature and a review article from 2010² only 10 cases of fatal air embolism during ERCP have been reported by now. These include systemic embolism of the heart and lung as well as 5 cases with embolism of the intracerebral veins.

In the case described here diagnosis of intracranial air embolism could be made from post interventional computed tomography (CT) scan and was confirmed in autptic examination.

Case report:

A 58-year-old female patient with increased cholestase parameters, dilation of the intrahepatic bile ducts and a lesion suspected for a cholangiocellular carcinoma located near the hilus of the liver received two times ERCP for unsuccessful papillotomy. For a third ERCP the patient was transferred to a specialized center. Bilateral biopsy of the hepatic ducts and stenting of the ductus hepaticus communis was performed. During ERCP a temporary abnormal breathing pattern and decrease of oxygen saturation to 89% was observed. After completion of the procedure the patient did not awake when anesthesia with Disoprivan was stopped and therefore was transferred to the emergency room.

Cranial CT scan revealed massive venous air embolism with venous congestion and brain swelling (Figure 1a-d). Radiologic imaging of the abdomen showed aerobily and obstruction of the left portal vein due to the mass near the hilus, but no signs of air embolism within the heart or lung, however subsegmental pulmonary embolism could not be ruled out.

Due to the infaust prognosis the patient received palliative comfort therapy only, with consent of her relatives. The patient died 1 day after ERCP. Autoptic macroscopical and histological examination of the body showed Echinococcosis of the liver with nodular multicystic tumor-like lesions in liver segments IVa and IVb, peripheral pulmonary embolism and pneumonia probably due to aspiration. Autopsy of the brain revealed multiple acute bilateral supra- and infratentorial infarctions with generalized edema and concomitant mass effect including midline shift and right uncal herniation.

Discussion:

Fatal complications of ERCP are exceedingly rare and occur more often in therapeutical than pure diagnostic interventions. Only 5 cases of fatal cerebral air embolism have been described in the literature before. Air embolism can arise when the integrity of the bile ducts and accompanying vessels is disrupted by mechanical damage of these structures or preexistent fistulas are laid open. Autoptic examination in the case described showed changes suspicious for a small (2 mm x 3 mm) triangular defect of the wall of the right hepatic duct. The vessels in this region showed perivascular hemorrhage. These findings indicate the possibility that iatrogenic air inclusions of the bile duct entered the liver veins and vena portae. Potentially embolic air could have directly reached the lung via the right atrium and pulmonary arteries and could have reached the brain via the right atrium and retrogradely the vena cava superior and vena jugularis interna leading to venous infarctions. Another possible way would be dissemination of air via portocaval collaterals. The post interventional cranial CT scan shows evidence for intracranial venous, but not arterial embolism and definite arterial infarction is not mentioned in the neuropathology report. Such paradox arterial embolism can occur when the patient has a cardiac septal defect, most likely a persistent foramen ovale (PFO) which was observed at autopsy of the patient here. A formal possibility is also transpulmonal passage of air. However both effects require additional pulmonary hypertension to induce paradox embolism, which may explain, why in the case reported solely or at least predominantly venous infarctions occurred.

The initial signs of systemic air embolism can be subtle and unspecific. Therefore immediate symptomatic treatment and further diagnostic has to be performed if suspicion arises.

Beside auscultation and transthoracic echocardiography, CT scans can provide most helpful information in such cases. With this imaging modality macroscopic air emboly can often be identified within vessels of the organs affected. Measurement of hounsfield units (HU) helps to distinguish air inclusions (mostly HU <500) from fat (HU about 100). Furthermore secondary signs of embolism can be visualized, e.g. infarction pneumonia in the lung and hypodense areas and swelling of the brain due to edema.

Regarding a rapid onset of neurological symptoms in cases of intracranial air embolism, the main differential diagnoses include intracranial hemorrhage and infarctions of other etiology, while tumors or infections which may also present with hypodense areas and brain swelling in CT scans are unlikely from the clinical course. Rarely prolonged unconsciousness post anesthesia can be caused by a non-convulsive epileptic state, but which is not accompanied by imaging findings mentioned above.

If subtle signs of systemic embolism occur during ERCP the intervention has to be stopped and the patient should receive high flow oxygen and volume expansion. Furthermore head down position to prevent air from reaching the brain and left lateral position to trap air in the right heart is recommended (ref). For treatment of confirmed systemic embolism hyperbaric oxygenation is the therapy of choice (ref: blanc p et al). In cases of air trapping in the right atrium aspiration of air can be achieved e.g. with the help of a Bunegin-Albin multiorificed catheter (ref).

For prophylaxis of severe complications in ERCP risk factors like....have to be taken into account.

If insufflation of gases different from air, e.g. CO₂ in ERCP could be helpful to reduce the risk of embolism is not known yet (ref). However further improvement of technical equipment may reduce the already low rate of complications.

Conclusion:

Our case illustrates one of the rare examples of intracranial venous air embolism during interventional ERCP. The initial symptoms of systemic embolism can be very subtle, but afford immediate therapeutic intervention and prompt diagnostic examination with adequate modalities. Being aware of air embolism as a potentially fatal complication and knowledge of the necessary medical approaches will improve the outcome of these patients.

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