Journal of the Neurological Sciences 376 (2017) 93–96

Contents lists available at ScienceDirect

Journal of the Neurological Sciences

journal homepage: www.elsevier.com/locate/jns

Clinical Short Communication

Systemic air embolism after endoscopy without vessel injury – A summary of reported cases

Peter Voigt [a,b,*], Stefan Schob [b], Sebastian Gottschling [c], Thomas Kahn [a], Alexey Surov [a]

[a] Department of Diagnostic and Interventional Radiology, Leipzig University Hospital, Liebigstr. 20, 04103 Leipzig, Germany
[b] Division of Neuroradiology, Leipzig University Hospital, Liebigstr. 20, 04103 Leipzig, Germany
[c] Department of Radiology, Leipzig Heart Center, Struempellstr. 39, 04289 Leipzig, Germany

ARTICLE INFO

Article history:
Received 29 August 2016
Received in revised form 27 January 2017
Accepted 8 March 2017
Available online 09 March 2017

Keywords:
Systemic air embolism
Endoscopy
Paradoxical embolism
Patent foramen ovale

ABSTRACT

Introduction: Systemic air embolism (SAE) is a rare but serious complication following endoscopic procedures. It may occur with or without direct vessel injury. The aim of this work is to review cases of SAE following endoscopy without proven vessel injury.
Methods: In this systematic review PubMed database was screened for SAE following endoscopy from 1990 to 2015. Only cases without proven major vessel injury were included in the analysis. Including one case of SAE after colonoscopy from our hospital the analysis comprised 40 cases.
Results: 60% of patients underwent ERCP, 33% gastroscopy and the remaining 7% other endoscopic procedures. Among patients suffering from SAE the majority had cerebral embolism (73%). In 46% of documented echocardiography a patent foramen ovale (PFO) has been confirmed as mechanism of paradoxical air embolism. Therapeutic approaches comprised most frequently hyperbaric oxygenation. In 35% of cases advanced life support was necessary whereas only 55% of patients survived SAE in total.
Conclusion: SAE is a serious complication of endoscopic procedures with high morbidity and mortality. In patients with present PFO high awareness should be paid to informed consent for the risk of SAE, especially stroke. Cautiousness with sedation is necessary in those patients not to delay clinical recognition of neurological SAE symptoms.

1. Introduction

Systemic air embolism (SAE) is an iatrogenic event which may lead to significant morbidity and mortality. SAE can be defined as air entering the systemic circulation originating from the site of intervention. It is documented within a whole variety of procedures like neurosurgery in sitting position, venous surgery, positive pressure ventilation, cardiac or laparoscopic surgery [1]. Nevertheless SAE is also a rare but serious adverse event in endoscopic procedures and has been observed for example in gastroscopy, colonoscopy and – even more frequently – in endoscopic retrograde cholangiopancreatography (ERCP). To cause venous air embolism local air penetration from the gastric or bowel lumen or the biliary system into the mucosa and further into the portal venous system has to take place. Weakening of the mucosal barrier as for example due to ulcerations might probably increase the risk of venous air embolism. In upper gastrointestinal tract penetrating duodenocaval fistulas [2] or vessel exposure to air in gastric ulcer [3] have been described. Furthermore entering of air to esophageal venous channels through tiny mucosal tears after endoscopic dilatation were proposed [4]. During colonoscopy venous air embolization can happen by entering of pressurized air to rectal varices [5]. In ERCP mechanical alteration of the bile duct wall by the endoscope, the development of biliovenous shunts as well as spontaneous transgression of air from bile ducts have been discussed as possible explanations [6]. Furthermore intramural dissection of air into the portal venous system via injured duodenal vein radicles is possible following ERCP and sphincterotomy [7]. In general, direct injury to any low pressure vessel is able to cause air embolism [8]. However, it is not crucial to cause venous air embolism as various mechanisms were proposed on how air may pass the mucosal barrier.

The aim of this work is to review cases from literature who suffered from systemic air embolism in absence of artificial vessel injury.

2. Material and methods

2.1. Data acquisition and patients

In our department we identified one patient with fatal systemic air embolism after colonoscopy (Fig. 1). Furthermore, PubMed database was screened for systemic air embolism from 1990 to 2015 by using

* Corresponding author at: Department of Diagnostic and Interventional Radiology, Leipzig University Hospital, Liebigstr. 20, 04103 Leipzig, Germany.
E-mail address: peter.voigt@medizin.uni-leipzig.de (P. Voigt).

http://dx.doi.org/10.1016/j.jns.2017.03.009
0022-510X/

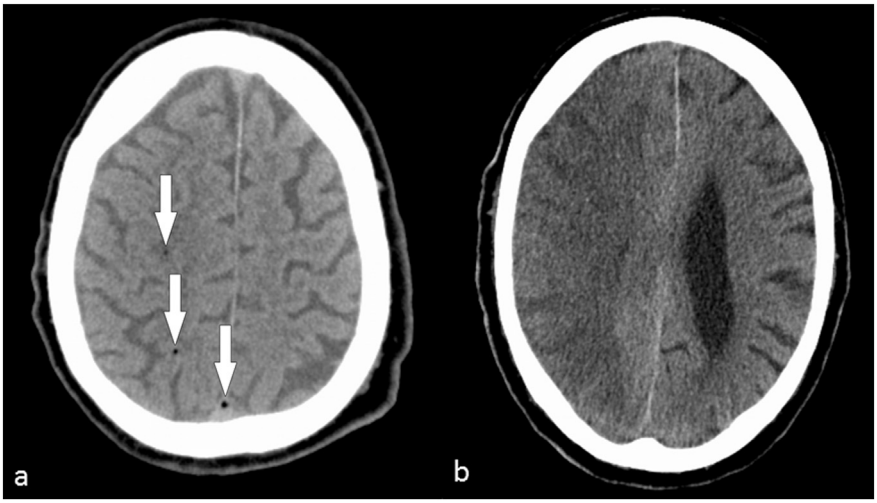

Fig. 1. Fatal cerebral gas embolism in our patient following colonoscopy. a) disseminated gas bubbles in cerebral end arteries and within the superior sagittal sinus. b) subacute infarction accompanied by postischemic edema in the territory of the right MCA.

the following search words: "cerebral air embolism" (1379 articles), "brain air embolism" (688 articles), and "air embolism, endoscopy" (487 articles). Therefore, overall 2554 articles were retrieved. Only cases with air embolism after gastrointestinal endoscopies were included into the analysis. Inclusion criteria were as follows: reported systemic air embolism during or after gastrointestinal endoscopy as defined by air entering the systemic circulation (as confirmed by either imaging, necropsy or clinical suspicion by acute neurological symptom onset) regardless of specific diagnostic criteria by the different authors. Cases with proven vascular injury during gastrointestinal endoscopy and non-english publications were excluded. After a thorough analysis of the data and exclusion of cases with proven vascular injury (defined as reported iatrogenic or non-iatrogenic great vessel injury), 39 patients were identified [4–6,9–39]. Therefore, together with our patient, our analysis comprised 40 cases.

2.2. Statistical analysis

Collected data were analyzed by means of descriptive statistics (absolute and relative frequencies). Continuous variables were expressed as mean ± standard deviation (SD), and categorical variables as percentages.

3. Results

Our own patient was a 67 year old male who underwent colonoscopy in preparation for reconstructive surgical colonic interponation after previous esophageal resection due to Boerhaave's syndrom. Other comorbidities except diabetes and essential hypertension were not known. Signs of air embolism during the procedure were suspected due to seizure and left side hemiparesis and confirmed by CT (Fig. 1). Together with our patient and data from the reviewed literature we identified 40 cases. There were 17 women and 23 men (mean age 49.5 ± 18.3 years; range 8 to 87 years). 60% of patients underwent ERCP (n = 24), 33% gastroscopy (n = 13). The other 3 patients received colonoscopy (n = 1), rectoscopy (n = 1) and intraoperative endoscopy (n = 1). 11 patients had more than one endoscopic procedure, all of them were ERCPs.

In 35 cases, imaging was conducted to confirm the diagnosis of SAE (23 CT, 8 echocardiographies, 3 plain X rays, 1 coronary angiogram). CT was mainly conducted as neuroimaging when cerebral embolism was suspected clinically. In 3 remaining cases, signs SAE were observed during necropsy [21,25,28]. In two other cases, there is no documentation on how SAE was confirmed. Regarding systemic air locations cerebral embolism was most frequent (73% of cases, n = 29), further embolizations were seen cardial (45%, n = 18), pulmonary (8%, n = 3), along the aorta (8%, n = 3), within the coronary arteries, intrahepatic, intraspinal and in bone marrow (each 3%, n = 1). Air embolism in more than one organ was observed in 15 cases.

In 28 out of 40 cases echocardiography has been documented. 7 of them were transesophageal examinations. 46% of those patients (n = 13) had a confirmed patent foramen ovale (PFO) whereas it was excluded in 50% of cases (n = 14). 4/13 PFO were diagnosed by transesophageal echocardiography. One further patient had an intraatrial shunt which has not been specified further within the report. In five patients, PFO has been detected during necropsy.

The time of symptom onset was documented in 97,5% of cases (n = 39). Of those, symptoms began during or immediately after the endoscopic procedure in 84,6% (n = 33). In the 6 remaining cases, symptom onset was noted with delay due to sedation (prolonged somnolence, n = 3; seizure, n = 2; hemiparesis, n = 1). Changes of body position in relation to symptom onset were documented in 3 cases. In all of them, symptoms began immediately when changing body position to supine.

Therapeutic approaches included hyperbaric oxygenation (HBO) in 25% (n = 10), ventilation with 100% O2 in 13% (n = 5). In 35% of patients (n = 14) measures of advanced life support were necessary. Only supportive or no therapy was obtained in 21% (n = 8). The remaining patients were treated with hypothermia (n = 2) or treatment has not been documented within the case report (n = 2).

Among included patients 55% survived systemic air embolism (n = 22) whereas 45% died (n = 18).

4. Discussion

Systemic air embolism is a rare but serious adverse event in endoscopic procedures. An important mechanism of venous air embolism entering the systemic arterial circulation is a patent foramen ovale (PFO) which occurs in 30% of healthy individuals and allows right-left shunting of gas bubbles [40]. Therefore especially patients with PFO are at risk of paradoxical air embolism. In our reviewed collective almost half of the patients with documented echocardiography report had a proven PFO. However, most of the PFO have been diagnosed by transthoracic echocardiography, which has only limited sensitivity for the detection of PFO in contrast to transesophageal echocardiography. On the other hand, PFO is not a conditio sine qua non for SAE. 11 of the reviewed patients had repetitive ERCP. One might speculate that subsequent repetitive bilio-vascular microtrauma leads to an increased