Cerebral Arterial Air Embolism After Endobronchial Electrocautery: A Case Report and Review of the Literature

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Case report

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Abstract

Background: Endobronchial electrocautery is a common and safe therapeutic endoscopic treatment for malignant airway obstruction. Cerebral arterial air embolism (CAAE) is a rare but potentially fatal complication of endobronchial electrocautery.

Case presentation: We present the first case of cerebral arterial air embolism after endobronchial electrocautery. A 56-year-old male with a pulmonary tumor in the right upper lobe received a repeated endobronchial electrocautery. During the procedure, he experienced unresponsive, hypoxemic, bradycardic and developed tetraplegia. The brain computer tomography showed several cerebral arterial air emboli with low-density spots in the right frontal lobe. He received hyperbaric oxygen therapy with almost full recovery except resident left-sided weakness.

Conclusions: General physicians should realize that CAAE can be a possible complication of endobronchial electrocautery. Some measures could be used to prevent this devastating complication: avoiding positive pressure, lowering ventilatory pressures if possible, avoiding advancing the bronchoscope to occlude the bronchus, and using the non-contact technique etc.

Introduction

Therapeutic endoscopic treatment may be considered to be used in malignant airway obstruction, such as endobronchial electrocautery, argon plasma coagulation (APC), thermal laser. Electrocautery is a common and safe bronchoscopy technique. The main complications of electrocautery include bleeding and airway fire. Cerebral arterial air embolism is an extremely rare but potentially fatal complication of bronchoscopy. Azzola et al. [1] reported that a frequency of < 0.02% of cerebral air embolism happened after bronchoscopy in their institution. Although cerebral arterial air embolism has been showed as a complication of APC and thermal laser [2], it has never been reported in electrocautery. Herein, we described a patient who developed cerebral arterial air embolism (CAAE) after endobronchial electrocautery.

Case Presentation

A 56-year-old male, never smoker, was transferred to our hospital. Approximately 16 years prior, he had been diagnosed with lung cancer and received lung surgery and chemotherapy. Three years ago, he developed a cough and underwent multiple endobronchial electrocautery therapy in another hospital. With the diagnosis of the recurrence of right lung endobronchial adenocarcinoma, the patient this time was admitted for repeated bronchoscopic thermal ablation procedure to relieve airway obstruction. Bronchoscopy revealed an endobronchial tumor emanating from the posterior segment of the right upper lobe (RUL) bronchus and causing completely obstruction of the bronchus (Fig. 1a). The patient was in supine position without intravenous sedation. The tumor was debulk using endobronchial electrocautery and blunt dissection of devitalized tissues with tooth forceps follows. (Fig. 1b, c). The setting of electrocautery cut mode was at 40W intensity and the coagulation mode was at 30 W intensity for duration of 3 s.

During the procedure the patient showed no cough and no obvious bleeding. At the end of the operation, he suddenly became unresponsive, hypoxemic (SpO₂ 88%) and bradycardic (heart rate 58 /min). High flow oxygen administration with mask and fluid resuscitation were quickly performed. His heart rate and SpO₂ recovered quickly and the consciousness gradually improved 30minutes later. However, his abnormal neurological

symptoms became obvious, including slurred speech, right gaze, paralysis in both extremities. The muscle strength in left limbs was 0/5 while 2/5 in right limbs. His left Babinski sign was positive. Hypoglycemia was excluded. Emergency brain computerized tomography (CT) revealed the presence of cerebral gas embolisms with several millimeter-sized, rounded and low-density spots in the right frontal lobe (Fig. 2).

Hyperbaric oxygen therapy (HBO₂) was initiated within 2 hours after bronchoscopy. He was transferred to the intensive care unit. That night, he received another hyperbaric oxygen therapy once again. After this treatment, the patient experienced generalized tonic-clonic seizures which were aborted using phenytoin, phenobarbitone and sodium valproate. A repeated brain CT done 24 h after bronchoscopy showed no signs of air embolism (Fig. 3). The patient received intensive rehabilitation. His mental status subsequently improved but he still had a mild left-sided hemiparesis with muscle strength 3/5 in left upper limb and power 4/5 in left lower limb.

Discussion And Conclusions

CAAE is a rare but potentially fatal complication of both diagnostic and therapeutic bronchoscopy and often iatrogenic[3]. Knowledge about the etiology of CAAE is limited and case reports therefore are important sources to get information[4]. To our knowledge only four cases of CAAE after thermal ablation have been reported[5–8]. The profiles of the reported patients, the examination procedures undertaken, the treatments, and the outcomes are shown in Table 1. This is the first reported case in the literature of CAAE developing after endobronchial electrocautery.

The possible mechanisms responsible for the development of CAAE during the therapeutic bronchoscopy include formation of broncho-vascular fistula, occlusion of bronchus by the bronchoscopy and paradoxical embolization[3, 4, 8, 9]. A broncho-vascular fistula was formed because of inflammation or heat coagulation and mechanical destruction of the tumor and adjacent tissue[7, 10]. In this case, electrocautery ablation of the tumor accompanied by mechanical debridement of the bronchus might result in the formation of the broncho-vascular fistula[1, 11]. The gas could enter the circulation through fistula. Gas embolism can easily enter a broncho-vascular fistula if the internal airway pressure goes up with the bronchoscopy process or when the patient is accepting positive pressure ventilation, coughing or taking a deep breath during the procedure [7, 12–15]. Paradoxical embolization occurs when the gas migrates from the venous circulation into the arterial system via an intracardiac shunt (patent foramen ovale)[16]. Actually, the air can be eliminated through diffusion into the alveoli, but if the capacity of the air exceeds 50 mL, it is easily for it to enter the pulmonary veins [16, 17]. In an animal study, Feller-Kopman et al. [10, 13]found that bronchoscopic thermal ablation with gas flow was associated with the occurrence of gas embolism in a dose-dependent fashion. What they found was consistent with the fact that high flow of coolant gas can lead to vascular air embolism during the use of contact probe[10]. Other possible risk factors for CAAE include patients with chronic obstructive pulmonary disease, a cavity inside the mass, lesions in the upper lobe, left lateral position, semi-recumbent position and bleeding[1, 7, 18].

Iatrogenic CAAE can be caused by a broad range of procedures, such as central venous catheter, cardiopulmonary bypass, bronchoscopy, lung biopsy, Nd-YAG laser resection of tumor etc.[4, 17]. Early in 1979, CAAE is suggested as a complication of transbronchoscopic lung biopsy[12]. This means CAAE should be considered as a possible cause of neurological symptoms after any procedure with a possibility of CAAE. Clinicians should keep in mind the possibility of CAAE after endobronchial electrocautery.

The symptoms of CAAE develop suddenly and are the same as those of cerebral hemorrhage or thromboembolism, including hemiparesis, tetraplegia, seizures and coma with cardiovascular collapse. In some reported cases, the diagnosis of CAAE should be suspected when there was protracted recovery from sedation[8]. In this case, the patient was noted to loss of consciousness. The symptoms of CAAE could be fatal or asymptomatic which indicates that it is important to bear the condition in mind even without symptoms[4]. Because asymptomatic CAAE exists, the actual incidence is higher than previously reported.

Imaging of the brain is useful to visualize air bubble. A CT was done within 2 hours of the symptom onset in this case and air bubbles were showed in the right frontal lobe. Sometimes CT may not reveal the presence of air in the brain as bubbles may be reabsorbed quickly if there is a delay in imaging or using inadaptable window settings on noncontrast CT[19]. And when the air bubble diameter is less than 1.3 cm, it may not be detected by CT[15]. Kanchustambham et al. [8] reported that no air bubbles could be visualized on the CT images and the diagnosis was made by exclusion. A repeated CT after 24 hours showed no signs of air bubbles in this case. Hence, CAAE cannot be ruled out through normal imaging. The acute infarct can be confirmed by MRI which is more time-consuming than CT and might delay the therapy. Transesophageal echocardiography and precordial doppler are used to detect intravenous and intracardiac air embolism, and end-tidal carbon dioxide may be an early sign of air embolism[9, 13]. As a result, clinical evaluation is still preferred for the assessment of CAAE[8].

Hyperbaric oxygen therapy is recommended as the most beneficial treatment of CAAE[9, 16–18, 20]. The mechanisms of hyperbaric oxygen therapy for CAAE include: reduced air volume, increased diffusion gradient out of the bubbles, reduction in cerebral edema, decreased endothelial damage, promotion of restoration of distal blood flow[3, 6, 16, 20]. In some hospitals, HBO₂ is not available due to the lack of hyperbaric chamber or unable to transport patients [1, 7, 15, 18]. The time to start HBO₂ treatment determines the outcome of the patients. It showed better outcome for patients treated with HBO₂ within five to seven hours from the symptom onset[3, 4, 20]. However, there were several reports of good outcomes in patients with delayed onset of treatment from 40 hours to multiple days[4, 6, 21]. Our patient received the first HBO₂ treatment within 3 hours and the second time HBO₂ treatment within 24 hours and no signs of air bubbles were showed in a repeated CT. The mortality in CAAE patients without HBO₂ treatment was 93% while 7% with HBO₂ treatment [21]. In conclusion HBO₂ should be initiated as soon as possible after the diagnosis of CAAE to reach the best effect. When there is no available of HBO₂, normobaric oxygen (NBO₂) can be administered as the most frequent non-HBO₂ treatment[8, 18].

There are some different opinions on the patient's position to prevent air embolism. Kanchustambham et al. [9] suggested using left lateral decubitus and Trendelenburg position. The Trendelenburg position was reported to reduce the air bubbles going to the brain[20]. However, some studies revealed that Trendelenburg position may worsen cerebral edema and patient should be placed flat and supine in cases of arterial air embolism[4, 16]

The patient suspected of diagnosed with air embolism should be transferred to intensive care unit for careful monitoring and management. Other treatments include endotracheal intubation, volume expansion and extracorporeal membrane oxygenation (ECMO)[9, 22]. Targeted temperature management (TTM) may be helpful to prevent the deterioration of cerebral function in the cases of CAAE[23]. Other organs should be examined by echocardiography, electrocardiogram or other examinations when encountering a patient with CAAE, because systemic air embolisms should be considered[24].

This is the first case of CAAE after endobronchial electrocautery. General physicians should realize that CAAE can be a possible complication of endobronchial electrocautery. Some measures could be used to prevent this devastating complication: avoiding positive pressure, lowering ventilatory pressures if possible, setting the flow to the lowest rate possible when using Nd-YAG or APC, avoiding advancing the bronchoscope to occlude the bronchus, using the non-contact technique etc.

Abbreviations

CAAE
Cerebral arterial air embolism; APC:argon plasma coagulation; RML:right middle lobe; LUL:left upper lobe; RUL:right upper lobe; N/A:not available; HBO₂:hyperbaric oxygen; NBO₂:normobaric oxygen; CT:computerized tomography; HBO₂:Hyperbaric oxygen therapy; ECMO:extracorporeal membrane oxygenation; TTM:Targeted temperature management; Nd-YAG:Neodymium-doped Yttrium Aluminium Garnet

Declarations

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Authors Contributions

Dr. He and Dr. Liu were major contributors in writing the manuscript. Prof. Gao and Prof. Wang analyzed the case.

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Availability of data and materials

All data obtained is available within the manuscript.

Ethics approval and consent to participate

Written informed consent was obtained from the patient's daughter for publication of this case report and any accompanying images. This case report has been approved by the ethics committee at Yunfu People's Hospital.

Consent for publication

Written informed consent was obtained from patient's daughter for publication of this case report and any accompanying images. A copy of the written consent is available for review.

Competing interests

The authors declare that they have no conflict of interest.

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Tables

Table 1: The profile of patients who developed CAAE after thermal ablation for endobronchial tumor					
Reference number	[5]	[6]	[7]	[8]	
Author and year	Osseiran et al.	Mimy et al.	Yasmeen et al.	Kanchustambham et al.	This case
	2008	2011	2012	2017	
Patient characteristic					
Age	64	48	88	68	56
Sex	Male	Male	Female	Male	Male
Bronchoscopy					
Procedure	APC	Thermocoagulation	APC	APC	Electrocautery
Location	RML	LUL	RUL	Bronchus intermedius	RUL
Positioning	N/A	N/A	Semi-recumbent	Semi-recumbent	Supine
Bleeding	N/A	>200mL	100mL	Middle	Little
Sedation	Midazolam	N/A	N/A	Fentanyl, Midazolam	(-)
Diagnosis, treatment and outcome of CAAE					
Air bubbles in the CT images	☒+☒	☒+☒	☒+☒	(-)	☒+☒
Echocardiography shows air	N/A	(-)	N/A	(-)	N/A
Oxygen delivery	Intubation	Intubation, HBO ₂	Intubation	NBO ₂ , HBO ₂	HBO ₂
Seizure	☒+☒	☒+☒	☒+☒	☒+☒	☒+☒
Outcome	Dead	Dead	Dead	Almost improved	Almost improved
APC, argon plasma coagulation; RML, right middle lobe; LUL, left upper lobe; RUL, right upper lobe; N/A, not available; HBO ₂ , hyperbaric oxygen; NBO ₂ , normobaric oxygen.					

Figures

Figure 1

Bronchoscopy revealed that the posterior segment of the RUL bronchus was completely obstructed by the tumor (a, arrow). Endobronchial electrocautery was used to debulk the tumor (b). Bronchoscopy showed the posterior segment after the ablation (c).

Figure 2

The cerebral CT scan following bronchoscopy showed multifocal cerebral air embolism in the right frontal lobe (arrows).

Figure 3

The repeated cerebral CT scan done 24h after bronchoscopy demonstrated no signs of air embolism.