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Argon Plasma Coagulation: Elucidation of the Mechanism of Gas Embolism

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Argon plasma coagulation · Bronchoscopy · Gas embolism

Abstract

Background: Argon plasma coagulation (APC) is a tool used in the management of tracheobronchial obstruction or bleeding. Complications include gas embolism which can cause devastating effects including hemodynamic instability, cardiac arrest, and stroke. Multiple theories as to how gas embolism occurs with APC have been postulated; however, none have identified the exact mechanism. ***Objectives:*** To identify the mechanism by which APC causes gas embolism in the tracheobronchial tree. ***Methods:*** Using an explanted porcine tracheobronchial tree with lung parenchyma, the APC catheter was applied through noncontact and direct contact to the endobronchial airway mucosa via flexible bronchoscopy. This was done at multiple gas flow settings and pulse durations. Visual changes in the mucosa were photographed, videoed, and described. ***Results:*** Gross evidence of submucosal gas transfer occurred when the APC catheter was in direct contact with the mucosa at all gas flow settings in all applications, despite using shorter pulse durations. Whenever the catheter was not in contact with the mucosa, there was no transfer of gas at any gas flow setting or pulse duration. ***Conclusions:*** Direct mucosal contact with the APC probe leads to submucosal gas deposition and is a likely mechanism for gas entry into the intravascular space. In reported cases of APC-associated gas embolism, presence of a vascularized endobronchial tumor may have increased the risk of gas tracking into the intravascular space. Care should be taken when applying APC during brisk bleeding or limited vision, as inadvertent mucosal contact may occur and could increase the risk of gas embolism.

Introduction

Argon plasma coagulation (APC) is a modality commonly used by interventional pulmonologists in the management of tracheobronchial obstruction or airway bleeding. It is a thermal ablative technique that utilizes the ionization of argon gas to coagulate and debride tissue by denaturing proteins and causing subsequent desiccation or vaporization. Once the area of interest is desiccated and the tissue impedance increases, the plasma moves to a more hydrated area, which is particularly useful when

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trying to achieve hemostasis [1]. This property also limits its depth of penetration to 2–3 mm, unlike other thermal ablation tools such as laser therapy. By transporting the current from the tip of the catheter to the tissue, APC performs faster than conventional electrocautery and provides a uniform and superficial coagulation. It also produces less smoke than electrocautery. In addition, the density of the argon gas jet moves the blood away, improving the coagulative effect. As a noncontact probe, there is less charring, and the tip of the tool maintains its efficacy [2].

Complications from APC are uncommon and include airway fire, as with laser and electrocautery, and airway perforation, although the latter is much less likely given the shallow depth of penetration. Though infrequent, gas embolism can occur and may have devastating effects including hemodynamic instability, cardiac arrest, and stroke. The effects produced by the gas embolism depend upon whether it involves the systemic or the pulmonary circulation. Animal models have shown that APC ablation in the trachea was associated with gas in the right atria, likely via the systemic veins, whereas application in the major bronchi demonstrated gas bubbles in the left atria, presumably through the pulmonary venous system [3]. Various mechanisms for gas emboli entering the circulation have been postulated, such as the creation of bronchopulmonary venous fistulas during tissue coagulation or high pressures after bronchial recanalization forcing gas into vascular tributaries, though further studies were recommended [4, 5]. The purpose of this study is to describe a possible mechanism behind gas embolism during APC in an explanted porcine model.

Materials and Methods

Using an explanted porcine tracheobronchial tree with lung parenchyma, the single-use axial probe was connected to a dual unit of electrocautery and APC (Erbe VIO 200D; Tuebingen, Germany). It was applied sequentially in both noncontact and direct contact fashion to the endobronchial airway mucosa in the proximal, mid, and distal trachea via the flexible catheter passed through a video bronchoscope (T-180; Olympus America Inc., Center Valley, PA, USA).

The experiment was performed at 40 watts (W) with 3 different gas flow settings: 1.1, 1.6, and 2.0 L/min, which are a representative sample of various gas flows within the manufacturer's recommended range [6]. The test was additionally performed at 2 different pulse durations, one shorter (1–2 s) and one longer (4–5 s). All maneuvers were performed in triplicate and replicated by 2 different interventional pulmonologists (E.F. and S.F.B.). During the bronchoscopy, visual changes in the mucosa were photographed, captured on video, and described at each setting.

Table 1. Submucosal gas deposition events observed after tracheal APC application

APC variable	Degree of submucosal gas transfer
Contact (all 40 W)[a]	
1.1 L/min flow, 1- to 2-s pulse	b
1.1 L/min flow, 4- to 5-s pulse	c
1.6 L/min flow, 1- to 2-s pulse	b
1.6 L/min flow, 4- to 5-s pulse	c
2.0 L/min flow, 1- to 2-s pulse	b
2.0 L/min flow, 4- to 5-s pulse	c
Noncontact (all 40 W)	
1.1 L/min flow, 1- to 2-s pulse	None
1.1 L/min flow, 4- to 5-s pulse	None
1.6 L/min flow, 1- to 2-s pulse	None
1.6 L/min flow, 4- to 5-s pulse	None
2.0 L/min flow, 1- to 2-s pulse	None
2.0 L/min flow, 4- to 5-s pulse	None

APC, argon plasma coagulation. [a] The lack of precise visual discrimination may be related to the short burst application of APC. [b] Small amount of gas deposition. [c] Large amount of gas deposition.

Results

When the APC catheter was in direct contact with the mucosa, there was gross evidence of submucosal gas transfer occurring at all gas flow settings (as shown in Fig. 1) in all applications (36 events in 36 applications, 100%). These results were reproducible in different areas of the tracheobronchial tree and consistently reproduced with a different operator. The submucosal transfer of gas was evident despite the use of short, 1- to 2-s pulses, though to a lesser degree based on visual observation. The lack of precise visual discrimination may be related to the short burst application of APC. Decreasing flow rate did not have any effect on gas transfer.

Whenever the catheter was not in direct contact with the mucosa, the submucosal transfer of gas was not observed at any gas flow setting in any area of the tracheobronchial tree (0 events in 36 applications, 0%). There was no gas transfer despite the use of longer, 4- to 5-s pulses (as shown in Table 1). Videos of the APC catheter application in both contact and noncontact fashion were recorded (as shown in see online suppl. Videos 1 and 2; see www.karger.com/doi/10.1159/000512687 for all online suppl. material).

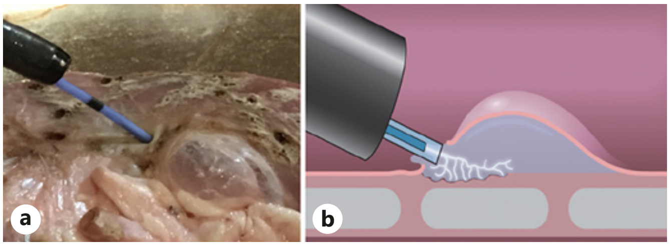

Fig. 1. Mechanism of gas embolism formation after APC application. **a** APC catheter in direct mucosal contact causing gross submucosal gas deposition. **b** Illustration of the mechanism by which the APC catheter in direct mucosal contact results in gas embolism. APC, argon plasma coagulation.

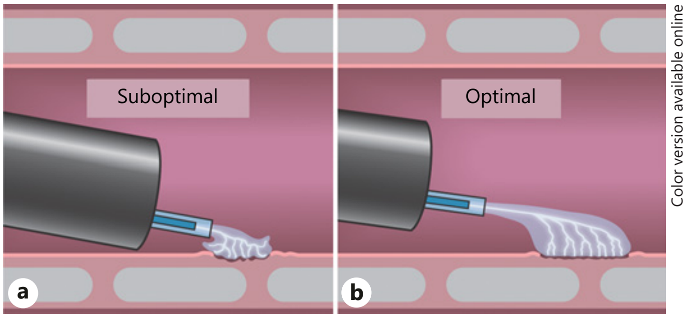

Fig. 2. Suboptimal versus optimal APC catheter positioning. **a** Illustration depicting suboptimal (direct mucosal contact) APC catheter positioning leading to submucosal gas deposition. **b** Illustration depicting optimal (noncontact) APC catheter positioning preventing mucosal gas penetration. APC, argon plasma coagulation.

Table 2. Summary of cases of gas embolism associated with bronchoscopic APC application [4, 12–16]

Series	Cases	Lesion type	Complications	Bleeding	Endobronchial lesion	Death
Goldman et al. [12]	1	Malignant	Cardiac arrest	Unknown	Yes	Yes
Osseiran et al. [13]	1	Malignant	Cerebral gas embolism, stroke	Unknown	Yes	No
Reddy et al. [4]	3	Malignant	Cardiac arrest (3/3)	Yes, 3/3	Yes, 3/3	Yes, 2/3
Shaw et al. [14]	1	Malignant	Cerebral gas embolism, stroke	Yes, ~100 mL	Yes	Yes
Kanchustambham et al. [15]	1	Malignant	Cerebral gas embolism, stroke	Yes	Yes	No
Mathew and Sriaroon [16]	1	Benign (sarcoidosis)	Cerebral gas embolism, stroke	Yes	Bronchial web	No

APC, argon plasma coagulation

Discussion

APC is a bronchoscopic ablative technique commonly used for airway recanalization in both benign and malignant conditions and for control of bleeding. It is considered a safe procedure with a low complication rate – ranging from 0% in a retrospective case series of 70 procedures to 3.7% in a review of 482 interventions [7, 8]. A rare complication is gas embolism; both fatal and nonfatal consequences have been reported with the use of APC during bronchoscopy. Clinically, gas embolism can manifest as right ventricular outflow obstruction if a significant amount of gas is introduced through the systemic venous system and into the right atrium. In addition, gas embolism can manifest as myocardial infarction or stroke, which can occur either by introduction of gas directly into the pulmonary venous system and subsequently the left atrium or by movement of gas in the right heart through a right-to-left intracardiac defect. Using a sheep model, Feller-Kopman et al. [3] demonstrated that thermal ablation in the trachea resulted in gas more often in the right atrium versus ablation in the bronchi, which produced gas in the left atrium. Emboli were also more common with higher gas flow rates and longer pulse durations [3].

Multiple theories have been postulated regarding the exact mechanism of gas embolism formation during endobronchial thermal ablation. One is that occlusion by the body of the bronchoscope of a newly recanalized bronchus with high distal pressures forces gas into the vessels exposed by the thermal ablation. Another is that the highly pressurized gas exiting the tip of the APC catheter crosses the mucosa and enters the vasculature via bronchovascular fistulae formed during tissue desiccation and coagulation. This mechanism has additionally been postulated in cases of gas embolism following laser therapy [9]. Tellides et al. [5] proposed that systemic embolism during bronchoscopic laser procedures occurred due to laser fiber air coolant used at high flow settings as well as in contact fashion and recommended using noncontact mode whenever possible. However, the precise mechanism of how embolism occurs remained unclear.

In this study, we performed APC ablation in the trachea of an explanted swine model using various flow rates and pulse durations. We visually observed submucosal gas transfer when the probe was applied in contact fashion with the mucosa; this also occurred at the lowest flow rates and the shortest pulse durations. No gas transfer was observed when the probe was applied in noncontact fashion, despite higher flow rates and longer pulse durations. This model also allowed 2 different operators to reproduce the same results.

In light of our findings and in an attempt to explain the mechanism of embolism that has been described in prior case reports, it is important to highlight the following issues: most published cases had active hemoptysis that may have provided easy access for gas bubbles to reach the vasculature. In addition, in the vast majority of reported cases of APC-associated gas embolism, there was a highly vascularized endobronchial tumor which may have increased the risk of gas tracking into the intravascular space (as shown in Table 2). It is also possible that brisk bleeding obstructed the view of the bronchoscopist, and the position of the APC probe may have been in contact with the mucosa. As we have shown in this study, direct mucosal contact with the APC probe resulted in gross deposition of gas in the submucosa, despite short pulse length and independent of flow. While this model did not allow for further evaluation of gas embolism into the heart via echocardiography, it stands to reason that submucosal gas deposition leads to gas entering the intravascular space. The obvious submucosal bubble formation, as shown in the online suppl. video, is not observed during a live case, which can be explained on the basis of instantaneous absorption of the argon gas into the blood circulation of the airway. Whether or not this produces clinically significant gas embolism likely depends on the volume and size of the gas bubbles. Regardless, emphasis should be placed on using the APC probe in a noncontact fashion, and extra caution should be maintained when applying it in settings of bleeding to ensure that contact with the mucosa is not inadvertently made (as shown in Fig. 2).

Conclusion

APC is a tool that has been used routinely in the gastrointestinal tract since the early 1990s and in the airway since the late 1990s/early 2000s [10, 11]. Generally, it is a safe procedure used for both tumor debulking and to achieve hemostasis. Gas embolism is a rare complication of bronchoscopic APC use but carries severe consequences. Ablation in the trachea and bronchi has been shown to lead to gas development in the right and left atria, respectively, though the mechanism of gas transfer into the vasculature was unclear. We have shown that direct mucosal contact with the APC probe leads to gross submucosal gas deposition and is a likely mechanism for gas entry into the intravascular space. Extreme care should be taken when applying APC during brisk bleeding or limited vision, as this increases the risk of mucosal contact and could increase the risk of air embolism occurrence. Though in this study we did not identify submucosal gas transfer when the probe was in the noncontact position, we did observe increased gas transfer at higher pulse durations when the catheter was in contact with the mucosa. We recommend that when using APC, it is done strictly in noncontact fashion with the shortest pulse duration and lowest gas flow rate in order to minimize the risk of complications.

Statement of Ethics

This study did not require IACUC approval as there were no live animals used. This study was performed solely in the previously explanted porcine lung.

Conflict of Interest Statement

Dr. Folch serves as a scientific consultant for Medtronic as well as an Education and Scientific consultant for Boston Scientific. He has a research grant from Intuitive Surgical. None of these represent conflicts of interest for the content of this manuscript. Drs. Oberg, Mehta, Majid, Keyes, and Fernandez-Bussy have no conflicts to disclose.

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Author Contributions

All authors contributed to the development of the project, data collection, and manuscript preparation. The procedures were performed by E.F. and S.F.B.

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