Signa Vitae

REVIEW

Airway management in vascular central airway obstruction: a literature review

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Abstract

Vascular central airway obstruction (CAO) is a rare cause of upper airway obstruction in adults. CAO occurs below the level where it is invisible in a laryngoscope. Doctors therefore should pay attention to the possibilities of vascular CAO when attempting to prevent and resolve catastrophic complications from upper airway obstruction such as cardiorespiratory collapse and hemoptysis, which requires a thoughtful preoperative planning of airway management before starting a surgical reconstruction.

Keywords

Aneurysm; Aortic arch; Pulmonary artery; Difficult airway; Vascular rings

1. Introduction

Airway obstruction causes airway flow limitation [1, 2]. Upper airway is defined as the conduit between the nose or mouth and the carina [3]. Obstruction anywhere in this conductive airway may result in asphyxia and associated lethal complications [4, 5]. Central airway obstruction (CAO) is caused by the obstructive lesions involving the area from the trachea to the mainstem bronchi [6] where it is not visible by a laryngoscope during emergency airway management. Therefore, medical care for CAO requires knowledge and skills; in additional to that, one needs to deal with upper airway obstruction above the vocal cords [7, 8].

Among the benign cause of upper airway obstruction [8–12], vascular anomaly is relatively uncommon. The vascular lesions compress or obstruct the airway involving the trachea or mainstem bronchi, potentially causing vascular CAO [13, 14], and because of congenital anomalies, it is more recognized in children [15–17] than in adults [18]. The low incidence of vascular CAO in adults may lead to a low threshold of suspicion for the diseases when dealing with patients having signs of upper airway obstruction and cause unexpected potentially lethal complications not only from airway compression such as

respiratory insufficiency [19], but also from vascular-related complications, for example, massive hemoptysis [20], aortic dissection, and aortic rupture [21–23].

The rarity and high mortality of vascular CAO have got the attention of the doctors and have extensively been studied in the literature; however, to our best knowledge, it has been a decade since the last review focusing on vascular CAO in adults [18, 24, 25], and only some of them focus on airway management in vascular CAO [26]. Therefore, we start a review for the challenges in airway management among adult patients with vascular CAO.

2. Material and methods

2.1 Criteria for the inclusion of studies in the systematic review

This literature review included the publications of vascular CAO in adults who are equal or above 18 years of age, published in English until June 20, 2021. Only full-text articles were analyzed, as abstracts are not sufficient for a detailed analysis. The inclusion and exclusion criteria for literature sources are shown in Table 1.

T A B L E 1. Inclusion and exclusion criteria for literature sources.

Variable	Inclusion criteria	Exclusion criteria
Age	≥18 years of age	<18 years of age
Study population	Humans	Animals
Topic investigated	Vascular CAO	Topics out of the scope of vascular CAO: lesions without vascular involvement, lesions without airway obstruction, or airway obstruction without central airway involvement.
Language	English	Other languages
Period	Published from January 1, 1972 to June 20, 2021	Published before 1972

CAO, central airway obstruction.
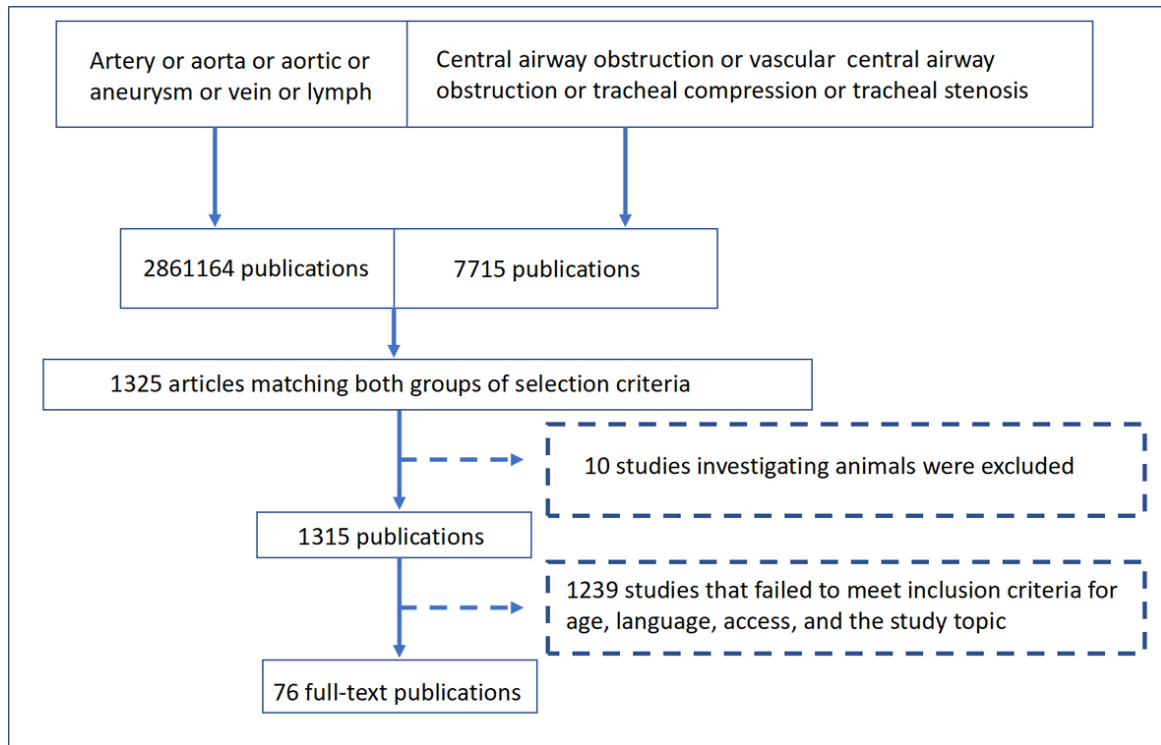

F I G U R E 1. A flow chart of study search and selection for systematic review in the study.

2.2 Strategy of search for publications

Search for publication was performed in the PubMed database. Fig. 1 depicts the key words for search and the detailed process of study identification and selection.

2.3 Data systematization and analysis

After a detailed search for publications, a total of 76 full-text publications meeting all inclusion criteria were identified. This literature review included 76 studies, and their analysis was performed based on the set aim and objectives. The following data were extracted from the publications analyzed: general information (year and gender) and characteristics of the cases in the studies (symptoms, signs, types and causes of vascular lesions, experience of management, and results).

3. Etiologies of vascular CAO

Vascular CAO in adults can be congenital or acquired. According to the mechanisms leading to airway obstruction, it can be classified into extrinsic and intrinsic vascular CAO. Vascular lesions that externally compress the central airways cause extrinsic CAO [18], whereas vascular lesions leading to intraluminal airway obstruction cause intrinsic vascular CAO [27].

Table 2 shows congenital extrinsic vascular CAO caused by central vascular anomalies or congenital heart diseases. Congenital vascular anomalies may be symptomatic soon after birth [28, 29], or become respiratory insufficiency in childhood [30–32], or symptoms may appear in adulthood [33, 34]. Therefore, the frequency of congenital vascular anomalies that present in adults is different from those that occur in children [18, 25, 35, 36]. In spite of its rarity, vascular rings, such as left aortic arch with aberrant right subclavian artery [36–38], right aortic arch with aberrant left subclavian artery [38–

T A B L E 2. Etiologies of congenital extrinsic vascular CAO.

Etiologies	References
Congenital vascular anomalies	
Vascular rings	[35]
- Double aortic arch	[44–54]
- Right-sided double aortic arch and descending aorta	[55]
- Double aortic arch with Kommerell's diverticulum	[63]
- Left aortic arch	
with right ligamentum arteriosum and right descending aorta	[33]
with aberrant right subclavian artery	[36]
with aberrant right subclavian artery and Kommerell's diverticulum	[38]
- Right aortic arch	[64]
with aberrant left subclavian artery and left ligamentum arteriosum	[36, 39, 43]
with aberrant left subclavian artery and Kommerell's diverticulum	[38, 40–43]
- Pulmonary artery sling	[56–58]
Congenital aortic arch anomaly	
- Kommerell's diverticulum	[21, 22, 59–61]
- Cervical aortic arch aneurysm	[62]
Congenital innominate artery anomaly	
- Anomalous innominate artery	[65]
Congenital heart diseases	
Pulmonary atresia and major aortopulmonary collateral arteries in Tetralogy of Fallot	[32]
Atrial septal defect	[66]

CAO, central airway obstruction.

43], double aortic arch [44–54], right-sided double aortic arch coexistence with a right-sided descending thoracic aorta [55], and pulmonary artery sling [56–58]; aneurysms arising from congenital aortic arch anomaly— Kommerell's diverticulum [21, 22, 59–61] and cervical aortic arch [62]; Kommerell's diverticulum in combination with vascular rings [63]; right-sided aortic arch [64]; and anomalous innominate artery [65] can cause vascular CAO in adults. Also, congenital heart diseases that cause aneurysmal artery dilation, for example, atrial septal defect accompanied with dilated right pulmonary artery [66], and pulmonary atresia with aneurysmally dilated aortopulmonary collateral arteries in Tetralogy of Fallot [32] may obstruct the tracheobronchial tree in adults when the extraluminal pressure arising by the dilated arteries exceeds the durability of the airway.

Table 3 shows acquired extrinsic vascular CAO caused by arterial aneurysm of central vascular system or extremely thoracic anatomy. Arterial aneurysms of the innominate artery [20, 26, 67–71], ascending aorta [71–74], aortic arch [73–78], descending thoracic aorta [74–76, 79–84], and common carotid artery [85–87] that compress the central airway are secondary to atherosclerotic degeneration [20, 68, 69, 73–76, 82–84], heritable connective tissue disease [77–80], infection process [67, 81, 85, 86, 88], or aortic dissection [87, 89]. Pulmonary artery aneurysm [90, 91] and pulmonary artery dilation [92–94] are risk factors of extrinsic vascular CAO, which usually occur below the carina. Besides, extremely abnormal thoracic anatomy has the potential to cause vascular tracheobronchial compression syndrome [95, 96]. Severe kyphoscoliosis in Marfan syndrome causes trachea compression by the innominate artery anteriorly and thoracic spine posteriorly [96], which may be the result of defects in extracellular matrix composition, leading to the compromise of the structural integrity of the trachea, artery, and thoracic skeleton.

Intrinsic vascular CAO is the intraluminal obstruction of the airway caused by primary and secondary abnormalities of the central airway vasculature, such as bronchial artery aneurysm [27], bronchial varices [97], bronchial arteriovenous malformation [98, 99], and pulmonary artery aneurysm [100], pseudoaneurysm [101], mycotic aneurysm [102], and pulmonary varices [103] (Table 4). The central airway vasculature is a double circulatory system that involves bronchial circulation (a part of systemic circulation) and pulmonary circulation [27]. The bronchial arteries are connected to the pulmonary arteries through microvascular anastomoses at the level of the alveoli and respiratory bronchioles [104]. Therefore, extrinsic and intrinsic vascular CAO may exist together, for example, endobronchial varices associated with bronchial artery aneurysm [105]. Pulmonary atresia in Tetralogy of Fallot, leading to aneurysmatic dilation of aortopulmonary collateral arteries, that externally compresses trachea may be accompanied with hypertrophied bronchial collaterals resulting in intraluminal airway obstruction [32].

T A B L E 3. Mechanisms of acquired extrinsic vascular CAO in adults.

Mechanism	References
Systemic artery system	
Atherosclerotic degeneration	
Aortic aneurysm of ascending aortic	[71–74]
Aortic aneurysm of aortic arch	[73–76]
Aortic aneurysm of thoracic aorta	[74–76, 82–84]
Aneurysm of innominate artery	[20, 26, 68–70]
Aneurysms of the ascending aorta and innominate artery	[71]
Ruptured thoracic aortic aneurysm	[23]
Heritable connective tissue disease	
Aortic aneurysm of thoracic aorta in Marfan's disease	[77–79]
Aortic aneurysm of thoracic aorta and innominate artery in Marfan disease	[79]
Aortic aneurysm of thoracic aorta in heritable connective tissue disease	[80]
Aortic dissection	
Dissected internal carotid artery	[87]
Dissected descending aorta	[89]
Infection process	
Mycotic common carotid aneurysm (Staphylococcus aureus)	[85, 86]
Mycotic descending aortic aneurysm (Staphylococcus aureus)	[81]
Syphilitic aortic arch aneurysms	[88]
Syphilitic innominate aneurysms	[67]
Pulmonary artery system	
Pulmonary artery aneurysm	[90, 91]
Pulmonary artery pseudoaneurysm	[91]
Pulmonary artery dilation	[92–94]
Extremely abnormal thoracic anatomy	[95]
Severe kyphoscoliosis resulting in trachea compressed by the innominate artery anteriorly and thoracic spine posteriorly in Marfan's syndrome	[96]

CAO, central airway obstruction.

4. Clinical manifestations of vascular CAO

Trachea and extrapulmonary bronchi are composed of hyaline cartilage that is resistant to external compression. When intrinsic wall weakness of the airway [86] or high external pressure that over the air column can stand, extrinsic CAO occurs [82]. Therefore, some patients present with asymptomatic vascular CAO [47–49, 56], and the others commonly demonstrate chest discomfort, including chronic cough, recurrent bronchopulmonary infections, stridor, hoarseness, and wheezing [27, 40, 53, 76]. Chronic compression of the airway may lead to laryngotracheal stenosis, laryngomalacia, and tracheobronchomalacia [12, 25, 106], which are related to the critical events of cyanosis, hypoxemia, acute respiratory failure, or fetal hemoptysis [19, 20, 32, 62, 74]. Besides, life-threatening events, such as difficult trachea intubation [56], persistent hypercapnia after trachea intubation [26], and extubation failure [59, 79], may unexpectedly occur, which challenge doctors during emergent airway management in vascular CAO.

Patients with vascular CAO may present with asthma-like symptoms but have poor response to the standard therapy of asthma [33, 39, 41, 52, 58, 94]. In this situation, pulmonary function is helpful to differential diagnosis. Flow-volume loop demonstrating a fixed obstruction pattern with flattening of expiratory portion indicates dynamic intrathoracic obstruction of CAO [1, 8, 40, 96]. Obstructive pattern showing insignificant bronchodilator response is beneficial to exclude the possibilities of asthma [107].

Intrinsic vascular CAO commonly occurs below the lower part of trachea and mainstem bronchi, so they are commonly presented as cryptogenic hemoptysis or an accidental finding under the bronchoscope [27]. Dynamic flexible bronchoscopy (DFB) is helpful for the pathological evaluation of the airway in patients with CAO. Each endobronchial vascular lesion has a distinct pattern of bronchosopic image [27]. DFB may find bronchial artery malformation presenting a non-pulsatile submucosal bulging with dilated mucosal and engorged vessels running on the surface of protrusion [99, 105]; pulmonary

T A B L E 4. Etiologies of intrinsic vascular CAO.

Etiologies	References
Bronchial circulation	
Bronchial artery aneurysm	[27]
Bronchial varices	[97, 105]
Bronchial arteriovenous malformation	[98, 99]
Pulmonary atresia with hypertrophied bronchial collaterals in Tetralogy of Fallot	[32]
Pulmonary circulation	
Pulmonary artery aneurysm	[100]
Pulmonary artery pseudoaneurysm	[101]
Mycotic pulmonary artery aneurysm	[102]
Pulmonary varices	[103]
Extrinsic vascular CAO coexistence with intrinsic vascular CAO	
Endobronchial varices associated with bronchial artery aneurysm	[105]
Pulmonary atresia with hypertrophied bronchial collaterals and major aortopulmonary collateral arteries in Tetralogy of Fallot	[32]

CAO, central airway obstruction.

artery aneurysm or pseudoaneurysm demonstrating a pulsatile nodule or mass with smooth contour [108]; and bronchial or pulmonary varices showing dilated submucosal vessels protruding into the lumen [97, 103]. Since the coexistence of intrinsic and extrinsic vascular lesions has put patients at risk of fetal hemoptysis in together with asphyxia [32, 105], DFB can help identify intrinsic and extrinsic vascular lesions that cause CAO. A mass with variable extrinsic tracheal compression and collapse while pulsating synchronously with the arterial pulsation indicates a requirement to exclude extrinsic vascular CAO [76, 81] so additional image studies for vascular lesions like contrast CT is recommended [62, 76].

5. Airway management for vascular CAO

Surgical reconstruction is required to cure vascular CAO [8, 19, 20, 62, 71]. Antibiotics are essential for mycotic aneurysm [81, 85, 86]. However, prompt airway management for vascular CAO is critical for the success in the treatment (Table 5).

5.1 Airway management for intrinsic vascular CAO

Doctors should keep alert on CAO caused by vascular endobronchial lesions because an attempt to biopsy the lesions during bronchoscopy can result in life-threatening bleeding and should be avoided [98, 108]. Vascular embolization or surgical management is the best option for treatment [27].

5.2 Airway management for extrinsic vascular CAO

Delicate interdepartmental perioperative planning is important for patients with extrinsic vascular CAO. Signs of unexpected vascular CAO, preparation and selection of airway stenting and endobronchial tubes, anesthesia before artificial airway establishment, and diagnosis of extrinsic tracheobronchomalacia

(TBM) are vital issues that are required to be considered during airway management.

5.2.1 Signs of unexpected vascular CAO

Difficult intubation [56], persistent hypercapnia with/or without oxygen desaturation after trachea intubation [26, 75], difficulty in passing through the suction tube [75, 79], difficulty in weaning from mechanical ventilation [79], and extubation failure that presents with smooth breathing when using endotracheal tube but becoming airflow limitation soon after remove the endotracheal tube [59, 79, 86] should be signs that alert doctors to exclude the possibilities of vascular CAO. Mechanical ventilation variability is another sign of tracheal compression [26, 75, 81, 109], which explains why the patients with CAO still present with low tidal volume, hypercapnia, and arterial oxygen desaturation under the assistance of artificial positive-pressure ventilation.

5.2.2 Preparation and selection of airway stenting and endobronchial tubes

Endotracheal stenting is a therapeutic option for airway stenosis [75]. Technological improvement in endobronchial procedures has provided more options for managing complex cases [77, 82]. In extrinsic vascular CAO, the trachea may be compressed by vascular lesions with high pressure that metallic endobronchial stents rather silicon stent can stand [75, 82]; variable tracheal compression and collapse requires expandable stents [83]; and the possibility of coexistence with tracheomegaly needs a custom-made and fully covered metallic stent [77]. Expandable metallic stent placement offers a rapid, effective, and custom-made approach to restore airway patency. However, complications of airway stenting such as acute life-threatening airway closure [62, 110], reduced mucociliary clearance [110], airway inflammation and inflammation [111, 112], stent fracture and collapse [111], stent migration and fetal perforation [82, 112], and aortobronchial

T A B L E 5. Medical considerations in airway management in vascular CAO.

Intrinsic vascular CAO
Avoid endobronchial biopsy
Extrinsic vascular CAO
Before surgical reconstruction
Multidisciplinary preoperative discussion
- Anticipated difficult intubation
- Anticipated difficult ventilation
- At risk of cardiorespiratory embarrassment after induction anesthesia
Preparation and selection of airway stenting and endobronchial tubes
- Intubation with small caliber endobronchial tubes using bronchoscope
- Airway stenting as bridge therapy before curative treatment or palliative therapy
Anesthesia before artificial airway establishment
- Avoid muscle relaxants
- Mechanical ventilation preparation
- Percutaneous cardiopulmonary support preparation
Diagnosis of extrinsic TBM
- Awake DFB with a thinner scope
- Airway stenting as bridge therapy before curative treatment
During surgical reconstruction
Check residual airway stenosis and/or TBM
Careful interpretation the DFB findings under artificial ventilation
After surgical reconstruction
Airway stenting as bridge therapy before curative treatment
Signs of unexpected CAO and TBM before, during, and after surgical reconstruction
Difficult intubation
Hypercapnia after trachea intubation
Difficulty in passing through the suction tube
Difficulty in weaning from mechanical ventilation
Extubation failure
Airway stenting as palliative therapy before, during, and after surgical reconstruction
Patients with major risk during the curative surgery
Patients fail in the curative surgery

CAO, central airway obstruction; DFB, dynamic flexible bronchoscopy; TBM, tracheobronchomalacia.

fistula [84, 89, 113] have made a clinical dilemma in the applications of airway stenting for benign CAO, which demands careful justification.

Tracheal necrosis may occur after metallic stenting to restore the airway patency compressed by high pressure of aortic aneurysm because of the pressure opposing the airway walls by the stent and aneurysm sac, which leads to the complications, aortobronchial fistula and massive hemoptysis [84]. Hence, airway stenting is recommended as a palliative therapy or bridge therapy before curative treatment for extrinsic vascular CAO [8, 114] among those who fail in endovascular repair [82, 83], those who require temporary symptom relief of residual airway stenosis after major surgery [89, 115], or those with major risks during the corrective surgery [33, 75, 83]. In case airway stenting is not suitable for the patient, a small caliber endobronchial tube using bronchoscope may be an alternative device to bypass the airway stenosis [78, 116] because severe hypercapnia may occur during intubation above the stenotic area with endotracheal tube in its usual size [26, 117].

5.2.3 Anesthesia before artificial airway establishment

Extrinsic CAO caused by huge aneurysm may present as a mediastinal mass [26, 72, 83, 116]. Patients with tracheal compression by a mediastinal mass or aneurysm can pose special problems for the anesthesiologist [118, 119]. Tracheal compression is associated with the incidence perioperative cardiorespiratory collapse [72]. Those with perioperative tracheal compression greater than 50% diminution of tracheal cross-sectional area are at risk of respiratory complications during anesthesia [120]. Without sufficient preparation, some patients with aortic aneurysm get sudden difficult mask ventilation, CO_2 retention, oxygen desaturation, and bradycardia resulting from acute progression of tracheal stenosis when they loss spontaneous breathing during general anesthesia [72, 78]. Therefore, careful preoperative assessment and preparation in anesthesia before artificial airway establishment is essential in extrinsic vascular CAO.

Spontaneous breathing should be maintained before establishing an artificial airway beyond the trachea compression in extrinsic vascular CAO [78]. Patients who stop the spontaneous breathing after the induction of general anesthesia lose negative intrathoracic pressure during inspiration, moving the

extraluminal airway pressure toward to exceed the intraluminal airway pressure and predisposing the airway collapse [72]. Therefore, awake fiberoptic intubation with local anesthesia is preferred for artificial airway establishment, and muscle relaxants should be avoided to prevent ventilatory failure during general anesthesia [116].

Blood pressure management is paramount for extrinsic vascular CAO. Not only does hypertension pressure increase the risk of rupture of vascular abnormalities, but also hypertension elevates the pressure of vascular compression of the airway that increases the risk of airway occlusion [116], increases the ventilation pressures, as well as decreases the tidal volume and minute ventilation [81]. Induced hypotension is a therapeutic strategy in this situation [116].

Alternative therapy should be prepared in case that endotracheal intubation fails to maintain airway patency or respiratory demand in extrinsic vascular CAO. Therefore, mechanical ventilation or percutaneous cardiopulmonary support should be ready before induction anesthesia [26, 78]. Mechanical ventilators, including non-invasive ventilation, support positive intraluminal airway pressure and prevent airway collapse while assisting ventilation and gas exchange [59, 77, 121]. Percutaneous cardiopulmonary support is the resolution for patients who present with persistent tracheal stenosis in CT images under ventilator [118], who experience refractory respiratory hypercapnic or hypoxemic distress during anesthesia [26, 72], and who fail to have their airway patency resorted by an endotracheal tube [116] or airway stenting [82].

5.2.4 Diagnosis of extrinsic TBM

Chronic compression of the central airway by great vessels restricts cartilage growth and softens the cartilage rings, leading to airway malacia in extrinsic vascular CAO [122]. The affected airway in TBM predisposes to collapse during expiration when the negative intrathoracic pressure for inspiration is reduced, which increases positive pressure gradient between extraluminal and intraluminal airway pressures. The phenomenon is known as dynamic airway limitation [12].

Airway malacia in TBM complicates the management of extrinsic vascular CAO. TBM coexistence with extrinsic vascular CAO prolongs the duration of artificial ventilation, intensive care, and hospital stay, and also increases considerable morbidity, mortality, and the requirement of reintubation and reoperation [57, 76, 77, 79, 106]. To treat airway malacia in vascular CAO simultaneously, high index of suspicion of central airway malacia before and after surgical reconstruction is vital in vascular CAO.

The use of awake DFB is practical to identify TBM [123]. A thinner bronchoscope may be required in case of severe airway stenosis [57]. To exclude the coexistence of extrinsic TBM with extrinsic vascular CAO during DFB, a subsequent investigation (asking the patient to perform the following commands: take a deep breathing, perform a forced exhalation, or make a cough) is the maneuver to elicit the collapsibility of the airways [106, 123]. The dynamic inhalation and exhalation maneuver has been the gold standard to confirm the diagnosis of TBM.

DFB affects decision-makings in the process of surgical reconstruction for extrinsic vascular CAO. Before surgery, DFB confirms the area and extent of tracheal compression

and/or TBM where needs airway management [116]. After surgery, a repeat DFB is required to check residual tracheal narrowing and/or TBM before closing the surgical wound [76, 79]. An endobronchial tube or airway stenting is suitable as the bridge therapy for residual stenosis and/or TBM that causes respiratory discomfort after major surgery [115], for which subsequent proper management is required to cure the diseases [106].

A delay in management of the airway malacia complicates the clinical course of extrinsic vascular CAO [79, 124]. Failure in recognition of residual TBM leads to resistance during the advancement of a suction tube, unexpected desaturation, difficulty weaning from mechanical ventilation, and unexpected extubation failure soon after surgery [79]. Therefore, doctors should interpret the bronchocsopic findings carefully, especially intraoperative DFB images because the positive pressure supported by artificial ventilation may prevent dynamic airway collapse and mask the diagnosis of TBM caused by extrinsic vascular CAO [79].

6. Conclusions

Doctors should increase suspicion of extrinsic vascular CAO in patients who have difficulty in mask ventilation or intubation but presenting with normal anatomy on the glottis and upper respiratory tract. Among patients, anesthesiologists, radiologists, vascular surgeons, thoracic physicians, cardiothoracic surgeons, otolaryngologists, preoperative interdisciplinary discussions focusing on the risk of respiratory distress in patients who are expected difficult intubation or difficult ventilation and who are at risk of hemodynamic instability during induction and maintenance of anesthesia are recommended [83, 125].

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CONFLICT OF INTEREST

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