Coronary artery fistula and bronchiectasis: case report and literature review

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Abstract: Coronary artery fistula (CAF) is characterized by abnormal communication between the main coronary artery and the cardiac chambers, pulmonary arteries, vena cava, and systemic arteries and veins, and is a rare cardiac malformation. Bronchiectasis involves remodeling and persistent dilatation of the bronchial airways. It can either be localized to one lung segment or diffused throughout the whole lung. The incidence of bronchiectasis has increased to 566 per 100,000 over the last decade. There is a 33% possibility that CAF and lung-related disease will co-occur. However, the merging mechanism of these two diseases has not been explored. We report a case of a 61-year-old female with bronchiectasis who was admitted to our hospital with dyspnea and ankle edema. The patient was diagnosed with coronary artery-pulmonary artery fistula by coronary angiography (CAG), which was performed to rule out the possibility of coronary artery stenosis. Using the keywords "coronary artery fistula" and "bronchiectasis" to search the inpatient system of our hospital, PubMed, and Web of Science, we found that these two diseases often occur due to abnormal cardiovascular and pulmonary airway connection. Previous studies also support that CAF can co-occur with bronchiectasis. Based on these findings, we explain the possible mechanism of this co-occurrence with the aim of improving diagnostic techniques and therapeutic methods.

Keywords: Coronary artery fistula (CAF); bronchiectasis; chest pain; case report

Introduction

Coronary artery fistula (CAF) was first described in 1865 (1). CAF is characterized by abnormal communication between the main trunk or branch of the coronary artery and the heart cavity, pulmonary artery, coronary sinus, and vena cava. CAF is a common congenital cardiac malformation, but it is rarely acquired from trauma or infection (2). The clinical manifestation of CAF depends on shunt location and size. Fifty percent of patients with CAF have no clinical symptoms; thus, CAF is only observed upon physical examination when symptoms of myocardial ischemia are identified due to a coronary artery shunt, accompanied by typical chest angina, dyspnea, and limitations in daily activity (3).

Bronchiectasis was first described by Laennec in 1819 and defined as irreversible expansion and wall thickening of the bronchus. Bronchiectasis involves unrecoverable abnormal expansion of the local bronchus and destruction of elastic and muscle tissue in the bronchial wall, leading to abnormal and persistent bronchial expansion. Meanwhile, expanded bronchial arteries form pseudo-hemangiomas composed of destroyed blood vessel elastic fibers and uneven blood vessel thickness, causing rupture and bleeding. The clinical manifestations of bronchiectasis include cough, purulent sputum, and recurrent hemoptysis (4,5).

There is a 33% possibility that CAF and lung-related disease will co-occur (6). However, the merging mechanism
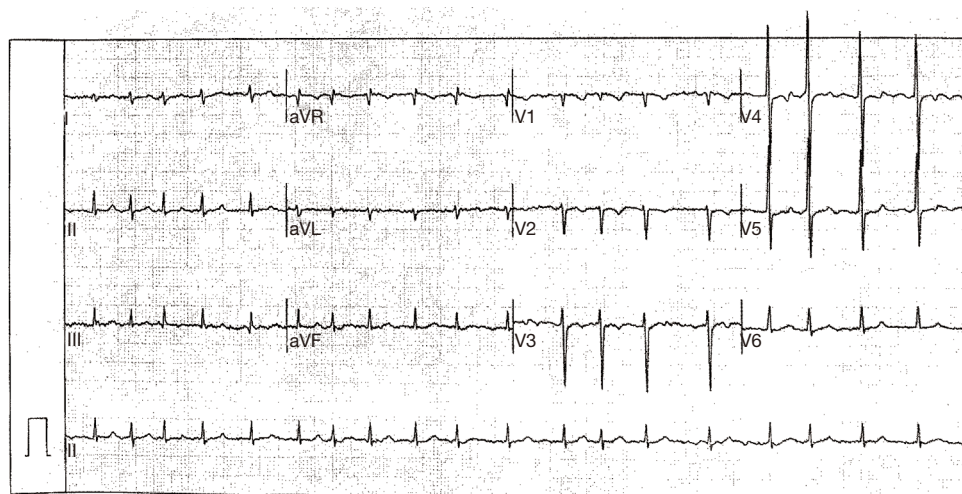
Figure 1 Electrocardiograph on admission showing atrial fibrillation with a rapid atrioventricular rate.

of these two diseases has not been explored. This article reported a 61-year-old female admitted to hospital due to chest pain, dyspnea, and edema in both lower limbs. The patient was diagnosed with coronary artery-pulmonary artery fistula with bronchiectasis. Moreover, we briefly reviewed patients with co-existing CAF and bronchiectasis from 2011 to 2018 using data from our hospital's inpatient system. In addition, we summarized the number of cases and types of CAF, as well as the history and location of bronchiectasis, by searching combined case reports in PubMed and Web of Science. We performed a literature review of these cases to explore the possible co-occurrence mechanism of CAF and bronchiectasis. Our observations could aid clinical diagnosis and treatment of patients with co-existing CAF and bronchiectasis.

We present the following article in accordance with the CARE reporting checklist (available at http://dx.doi.org/10.21037/apm-20-2608).

Methods

We searched the inpatient system of the Second Xiangya Hospital of Central South University from January 2011 to December 2018. One hundred eighty-three patients were diagnosed with CAF. Among them, 5 patients were also diagnosed with co-existing bronchiectasis. In addition, 6,434 patients were diagnosed with bronchiectasis. Among them, 5 patients were also diagnosed with co-existing CAF. The 5 selected and matched patients from the two cohorts (a total of 5 patients with co-existing CAF and bronchiectasis)

were included in the study.

We also searched PubMed and Web of Science using the keywords "coronary artery fistula" and "bronchiectasis," the study languages were limited to English and Chinese, and only studies on diagnosis and treatment were included. A total of 14 studies published between 1999 and 2019 were retrieved. A total of 16 patients with co-existing CAF and bronchiectasis were identified.

Case presentation

A 61-year-old female patient was admitted to the Department of Cardiology of the Second Xiangya Hospital of Central South University on December 26, 2018, with symptoms of chest pain, dyspnea, and lower-limb edema for 2 years. These symptoms were induced and aggravated by walking and dressing, and were accompanied by paroxysmal nocturnal dyspnea in the recent 2 months. The patient had bronchiectasis for 10 years and massive hemoptysis 8 years ago, for which she did not receive treatment. Laboratory tests showed an N-terminal pro-brain natriuretic peptide concentration of 1,688.49 pg/mL.

On admission, electrocardiography showed atrial fibrillation with a rapid ventricular rate (*Figure 1*). Echocardiographic findings showed left atrial enlargement (44 mm), decreased left ventricular systolic function (ejection fraction: 50%, fractional shortening: 26%), arrhythmia and tachycardia, a small amount of pericardial effusion, and no signs of abnormal wall motion. Chest radiography and chest computerized tomography revealed pulmonary
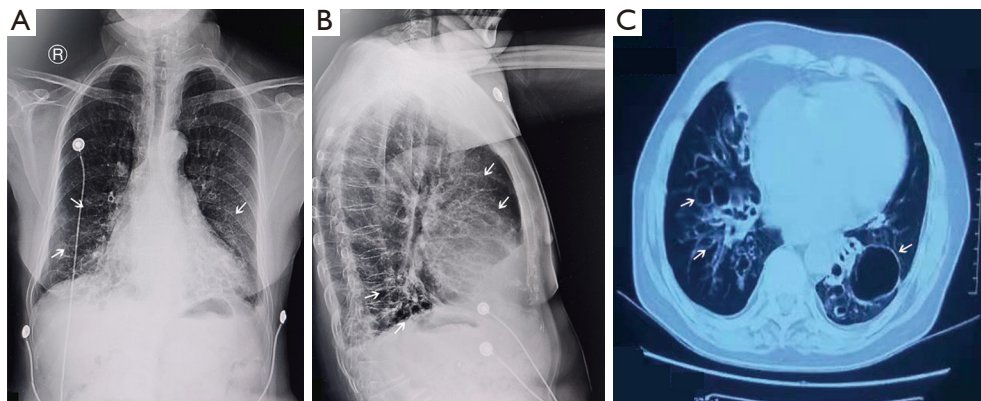
Figure 2 Pulmonary radiological images on admission. Bronchiectatic changes in the bilateral lower lobes were identified by chest radiography (A,B) and computed tomography (C). Solid arrow: location of bronchiectasis.

infection and bronchiectasis in both lower lobes and a small amount of pleural effusion with pleural thickening (*Figure 2*). Pulmonary function tests showed severe mixed ventilatory dysfunction.

After admission, the patient was administered symptomatic and supportive treatment. Quinamycin tablets were given for anti-infection. The symptoms of chest distress, dyspnea, and edema slightly improved after 1 week. N-terminal pro-brain natriuretic peptide concentration decreased to 792.00 pg/mL. The patient underwent coronary angiography (CAG) to confirm coronary artery stenosis. CAG revealed an unusually dilated, tortuous, anomalous vessel, CAF, arising from the proximal left circumflex (LCX) to the right. The CAF had three branches to supply the right lung (*Figure 3A,B,C*, Supplementary material of *Videos 1,2*). Considering that fistula and atrial fibrillation with a rapid ventricular rate further led to acute heart failure development in this patient, treatment aimed to control ventricular rate and eliminate the etiology of increased left ventricular preload caused by the CAF. There was no distal blood flow of the CAF after transcatheter coil embolization (*Figure 3D* and Supplementary material of *Video 3*). Chest pain and dyspnea subsided over a 6-month follow-up period.

All procedures performed in studies involving human participants were in accordance with the ethical standards of the institutional and/or national research committee(s) and with the Helsinki Declaration (as revised in 2013). Written informed consent was obtained from the patient for publication of this case report and any accompanying images.

Hospitalized patients with CAF and bronchiectasis from 2011 to 2018

The keywords "coronary artery fistula" and "bronchiectasis" were used to search the inpatient system of the Second Xiangya Hospital of Central South University from January 2011 to December 2018. We summarized 183 patients with CAF and 6,434 patients with bronchiectasis. One female and four males were selected with an average age of 62.5 years. Patients were admitted to hospital with chest tightness, chest pain, or dyspnea. Inpatient departments included respiratory departments (2/5) and cardiovascular departments (3/5). CAF was diagnosed by CAG (3/5), computed tomography (1/5), or color Doppler ultrasonography (1/5). It is worth noting that one case of CAF co-occurred with congenital heart disease. The heart murmur was not consistently affected by shunt site or size. Most patients had a history of bronchiectasis for many years. Patients had bronchial expansion and infection in the left lower lobe of the lung with pleural effusion (2/5) or pleural thickening (1/5). Pulmonary function tests indicated different extents of ventilatory dysfunction. Conservative treatment (3/5) with drugs or elective surgery (2/5) was often adopted considering presence of severe pulmonary disease or old age (*Table 1*).

Literature review of PubMed and Web of Science from 1999 to 2019

We used the keywords "coronary artery fistula" and "bronchiectasis" to search PubMed and Web of Science.
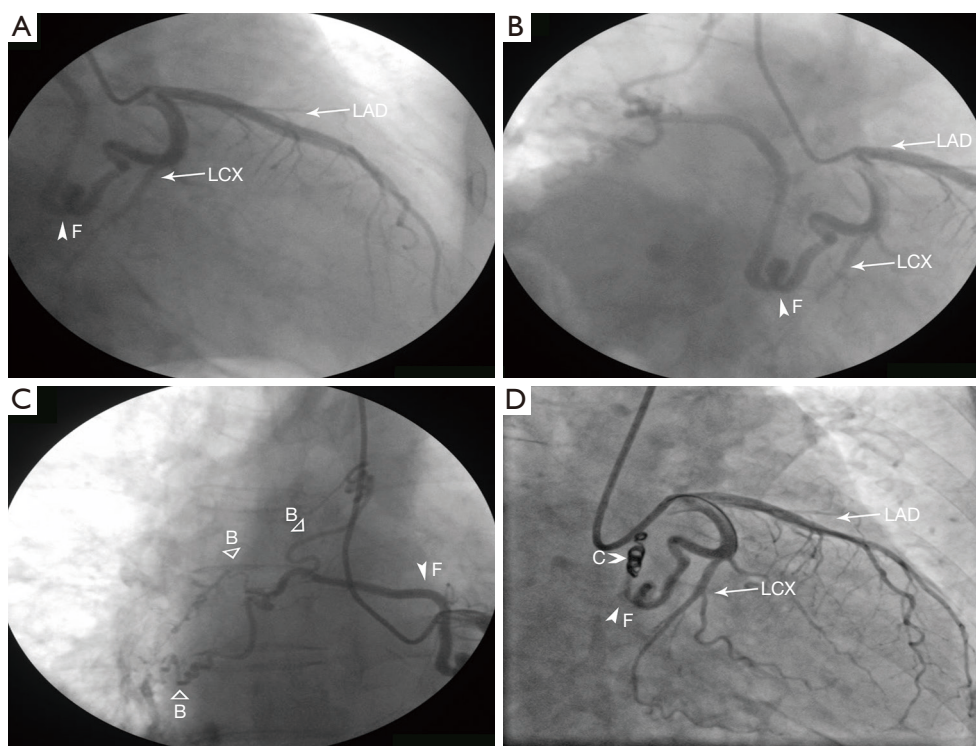
Figure 3 Coronary angiography images of CAF. The unusually dilated, tortuous, anomalous CAF is observed from the proximal LCX to the right (A,B). The CAF gave off three branches to supply the right lung (C). The CAF was closed without distal blood flow by transcatheter coil embolization (D). Short solid arrow with "F": location of CAF; short hollow arrow with "B": branches of CAF; short solid arrow with "C": location of coil embolization. CAF, coronary artery fistula; LCX, left circumflex artery; LAD, left anterior descending artery.

Fourteen studies were identified between 1999 and 2019, including studies on a total of 16 patients who completed diagnosis and treatment. The average age of patients was 61.8 years. Two patients with cystic fibrosis were <40 years of age. Patients with CAF combined with bronchiectasis had clinical symptoms, such as chest pain (11/16) and/ or dyspnea (5/16). Three patients had symptoms of hemoptysis. All patients were diagnosed with CAF using CAG. Bronchiectasis was located at the lower lobe of the left lung (9/16). Patients underwent interventional therapy for CAF or surgery for complications (12/16). Conservative treatment or medical treatment (4/16) was adopted when patients were intolerable to surgery. Dyspnea and other symptoms improved after follow up (*Table 2*) (7-20).

Discussion

At present, no large-scale clinical investigations or animal experiments have investigated the mechanism of CAF combined with bronchiectasis; thus, further research

is needed. The detection rate of CAF using CAG is 0.1–0.2% (21), and CAF accounts for 0.27–0.4% of all congenital heart disease cases (22,23). We report a unique case of coronary artery–pulmonary artery fistula with bronchiectasis. This case helps us to understand the abnormal hemodynamics of CAF and provides a platform for further understanding its etiology and pathogenesis.

The congenital CAF is related to the vascular sinusoid cavity formed by cardiac endothelial cells during the embryonic period. The sinusoid cavity between the normally developed myocardial trabeculae gradually shrinks into small tubes as an individual grows, forming a regular coronary vascular system. Coronary arteries grow from the aortic root and are connected to the capillary network on the cardiac surface (2). With some developmental abnormalities, the sinusoid cavity persists and does not shrink during the growth, forming abnormal branches between coronary arteries and the cardiac cavity (24). Moreover, some cases of CAF are acquired from surgical trauma, coronary artery bypass grafting, CAG,

Table 1 Hospitalization patients with coronary artery fistula and bronchiectasis [2011–2018]

Items	1	2	3	4	5
Age/gender	62/F	76/M	68/M	53/M	55/M
Complaint	Cough/expectoration/shortness of breath	Cough/expectoration/CP/dyspnea	CP/shortness of breath	Palpitation/CP/shortness of breath	CP/shortness of breath
Smoke history	None	50 y	None	20 y	None
Clinical departments	Respiratory department	Respiratory department	Cardiovascular department	Cardiovascular department	Cardiovascular surgery
Diagnostic methods	CAG	Echocardiography	CT	CAG	CAG
Types of fistula	Multiple/LCA-RA and RCA-PA	Single/LCA-LA	Single/LCA-RA	Single/LCA-PA	Single/LCA-PA
Cardiac murmurs	None	Aortic systolic murmur/mitral systolic murmur	Arrhythmia/P2 hyperactivity/aortic diastolic murmur	None	Mitral diastolic murmur
History and location of bronchiectasis	30 y/ULL and LLL/pleural thickening	25 y/LLL/bullae in right upper lung and pleural effusion	Cystic columnar dilatation in the ULL and LLL	10 y/LLL	1 y/LLL/pleural effusion
Pulmonary function	Severe OVD	Moderate OVD	Mild OVD	Moderate RVD	Mild OVD
Complications	Pulmonary tuberculosis	COPD/anemia/AF/esopha-gectomy	HBP/CAD/PFO/AF/hyperlipidemia/pulmonary nodules	Kidney stone/cholecystitis/chronic hepatitis B	RHD
Therapy	Elective operation	Conservative treatment	Elective operation	Conservative treatment	Elective operation

F, female; M, male; CP, chest pain; CAG, coronary arteriography; CT, computerized tomography; LCA, left coronary artery; RA, right atrium; LA, left atrium; PA, pulmonary artery; ULL, upper left lung; LLL, lower left lung; OVD, obstructive ventilatory dysfunction; RVD, restrictive ventilatory dysfunction; COPD, chronic obstructive pulmonary disease; CAD, coronary artery disease; PFO, patent foramen ovale; AF, atrial fibrillation; RHD, rheumatic heart disease.

Table 2 Literature review of PubMed and Web of Science (1999–2019)

Reference	Age/gender	Symptoms	Types of fistula	Location of bronchiectasis	Associated disorders	Diagnosis	Management
Ponangi 2017 (7)	34/M	CP/CF	Multiple/BA	WL	CF/bronchial infection	CAG/MDCT	Lung transplant
Choi 2017 (8)	71/M	Dizziness/dysphonia	Single/LCX to BA	WL	AF	MDCT/TTE/CAG	–
Massi 2015 (9)	75/F	Hemoptysis	Single/RCA-BA	LLL	HBP/PVD/anemia/left carotid endarterectomy	CAG	Fistula embolism
Said 2014 (6)	74/M	CP	Single/RCA-RBA	MLL/LLL	HBP/COPD	CAG/MDCT	PCI
Eryilma 2013 (10)	64/M	Dyspnea	Single/LCX-RBA	LLL	CAD/saccular aneurysm	CAG/MDCT	Conservative treatment
Parida 2013 (11)	75/M	CP/dyspnea	–	LL	HBP	TTE/CAG/CT	PTE covered tent
Nacer 2012 (12)	81/M	CP/dyspnea	Single/RCA-RBA	LLL	COPD/	TTE/CAG	CABG/surgical ligation
Ybarra 2012 (13)	62M	CP/dyspnea	Single/RCA-RBA	WL	DM/HBP/interstitial fibrosis	CT/MI/TTE/MDCT/CAG	PTE coils
Yoon 2012 (14)	68/F	Hemoptysis	Two/RCA-RBA /LCX-RBA	LL	Tuberculosis	CAG	PTE gel foam /PVA particles
Lee 2012 (15)	57/M	Massive hemoptysis/ dyspnea/CP	Single/RCA-RBA	LLL	DM	MDCT/TTE/CAG	Surgery/left lower lung lobectomy/removal of CBF fistula
Kim 2011 (16)	35/M	CP	Two/RCA-LBA /LCX-LBA	WL	–	MDCT/MPI/CAG	PTE coils
Bury 2010 (17)	62/F	CP	Two/LCX-LBA /LCX-PA	WL	–	MRI/MDCT/CAG	Conservative treatment
Khalpey 2009 (18)	29/M	CF	Single/LCX-RCA	WL	CF/inflammatory cellulitis	CAG	Lung transplant
Kang 2008 (19)	67/F	CP	Single/LCX-LBA	LLL	HBP	MPI/MDCT/CAG	Conservative treatment
Jarry 1999 (20)	67/M	CP	Single/LCX-LBA	LLL	HBP/hyperlipemia/F	MPI/CAG	PTE coils
	68/M	CP	Single/LCX-RBA	LLL	Mastectomy and AF	CT/CAG	Conservative treatment

M, male; F, female; CP, chest pain; CF, cystic fibrosis; BA, bronchial artery; LCX, left circumflex coronary artery; RCA, right coronary artery; RBA, right bronchial artery; LBA, left bronchial artery; PA, pulmonary artery; WL, whole lung; LLL, left lower lung; AF, atrial fibrillation; PVD, peripheral vascular disease; COPD, chronic obstructive pulmonary disease; CAD, coronary artery disease; HBP, high blood pressure; DM, diabetes mellitus; CAG, coronary arteriography; MDCT, multidetector computerized tomography; TTE, transthoracic echocardiogram; MPI, myocardial perfusion imaging; MRI, magnetic resonance imaging; PCI, percutaneous coronary intervention; PTE, percutaneous transcatheter embolization; CABG, coronary artery bypass grafting; PVA, polyvinyl alcohol particles; CBF, coronary-bronchial fistula.

atherosclerosis, acute myocardial infarction, or permanent pacemaker implantation (25).

Some scholars have suggested that the complications of CAF and bronchiectasis may be related to pressure differences between heart and lung. Coronary arteries usually originate from the aorta and contain high-pressure blood flow, providing blood to the heart, in both the systolic and diastolic states. When there is a blood pressure gradient between coronary artery and the pulmonary arterial circulation, fistula flow occurs from high pressure to low pressure. The "coronary blood theft" phenomenon of the myocardium in patients with CAF occurs when coronary artery pressure increases, such as in aortic valve stenosis, or when pulmonary artery pressure decreases, such as in pulmonary atresia, Takayasu's arteritis, or chronic obstructive pulmonary disease (COPD) (26-30). Persistent infection and inflammation dilate bronchial arteries (31), leading to decreased resistance between the coronary circulation and the pulmonary circulation. When there is a pressure difference or when the body's demand or compensatory demand increases, the connection between the coronary artery and the pulmonary artery or bronchial artery reopens, forming a fistula (32). Most patients with COPD present with pulmonary hypertension combined with bronchiectasis, resulting in a low blood oxygen concentration. The body needs more lobes to integrate when breathing or exercising. Active peptides, including endothelin-1, in blood vessels are mostly secreted by endothelial cells, smooth muscle cells, and airway endothelial cells, and they exert strong vasocontraction (33). Endothelin-1 may stimulate initially closed non-functional outer pericardial coronary arteries and build functional connections with other organs, forming a functional pathway.

We believe that the systemic collateral supply in patients with pulmonary hypertension forms both bronchial and/ or non-bronchial systemic circulations. We speculate LCX artery fistula combined with bronchiectasis in our case for the following reasons. First, the patient had severe bronchiectasis in the lower part of the lung for 10 years, bulla in the right lung (*Figure 2*), and pulmonary hypertension. This may have led to insufficient oxygen supply to the lung parenchyma, and hypoxia may have induced new blood vessel formation in the lungs. Second, bronchiectasis is prone to co-infection, and repeated inflammatory conditions may also induce angiogenesis or dilatation. Third, the closed coronary sinusoidal space in juvenile development is only a physiological closure that can functionally open and form a fistula under conditions of pathological hypoxia and inflammation. Fourth, the LCX artery had a more prolonged course that extended behind the pulmonary artery compared with the right circumflex artery and left anterior descending vessels in our case, thus increasing the chances of developing a fistula from the LCX artery. Similarly, it has the possibility of fistula formation from the coronary artery to the bronchial artery combined with bronchiectasis, as reviewed in *Table 2* in the bronchial system. We speculate that coronary artery-bronchial artery fistula may occur due to recurrent inflammation associated with bronchiectasis and secondary angiogenesis (which forms new connections) or increased vasodilation caused by local inflammation and opening of the existing latent fistula.

With advancements in diagnosis and treatment in recent years, whole-exome sequencing suggests that CAF and bronchiectasis may co-occur. In the selected cases, two young patients with cystic fibrosis were diagnosed with CAF during hospitalization due to severe bronchiectasis (7,10). Cystic fibrosis is a common cause of bronchiectasis. In cases of abnormal coronary vascular malformation, abnormal mutations in the cystic fibrosis transmembrane conductance regulator (*CFTR*) gene were identified by exon sequencing (34). *CFTR* is a common gene locus mutation abnormality in cystic fibrosis, and mice with *CFTR* mutations are susceptible to respiratory disease (35). *CFTR* induces ectopic production of vascular epidermal growth factors, which contribute to new blood vessel formation (36). We speculate that this could lead to cystic fibrosis with coronary arteries of an abnormal origin and shape. In addition, patients with bronchiectasis are accompanied by diffuse inflammation, and inflammation triggers formation of new blood vessels. Therefore, when patients with cystic fibrosis experience chest tightness and pain, they should be alerted to the possibility of the CAF "stealing blood," which leads to chest tightness and pain.

The diagnostic technology of CAF combined with bronchiectasis is also being updated. In the past, CAG was the diagnostic standard for CAF. With improvements in non-invasive examination technology, it may be possible to consider computed tomography angiography (CTA) to diagnose CAF. It was regrettable that this patient did not undergo CTA of coronary artery because she had atrial fibrillation with a rapid ventricular rate, and the vascular condition around the bronchial arteries had no chance to show clearly. Although CTA is not as timely and accurate as CAG, three-dimensional reconstructed images obtained using CTA can show anatomical structures and complex

lesions around nearby arteries, contributing to the diagnosis of CAF and identifying abnormal connections with peripheral blood vessels. CTA can also analyze whether bronchiectasis is combined with bronchial artery thickening and hypertrophy.

Treatment of patients with CAF usually involves occlusion technology. The prognosis of patients is relatively stable and safe when the treatment regime considers any comorbid symptoms. Patients with bronchiectasis often have severe respiratory infection, poor cardiopulmonary function, and obstructed ventilation, which may expand the contraindications to surgery. Taking conservative treatment with drugs could eliminate inflammation. The patient reported in this article underwent CAF closure surgery after respiratory system symptoms were controlled, and clinical symptoms, such as chest tightness and dyspnea, improved significantly. Asymptomatic patients with CAF may be misdiagnosed without cardiovascular imaging examination in other departments. Other patients with CAF are admitted to the Department of Respiratory with hemoptysis due to bronchiectasis. If patients combined with secondary and persistent hemoptysis after bronchial artery embolization, clinicians should be aware of the possibility of CAF and take suitable treatment to prevent disease progression.

In general, as a rare cardiovascular disease, CAF often co-occurs with bronchiectasis. Careful consideration should be given to patients' clinical symptoms and their general conditions during diagnosis and treatment. With the development of precision medicine and popularization of gene-sequencing technology, targeted prevention and medical treatment should be performed to prevent complications over time, such as congestive heart failure and coronary aneurysmal rupture, and to improve patient prognosis and quality of life.

Acknowledgments

Footnote

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