Bronchial Arteries: Anatomy, Function, Hypertrophy, and Anomalies

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The two main sources of blood supply to the lungs and their supporting structures are the pulmonary and bronchial arteries. The bronchial arteries account for 1% of the cardiac output but can be recruited to provide additional systemic circulation to the lungs in various acquired and congenital thoracic disorders. An understanding of bronchial artery anatomy and function is important in the identification of bronchial artery dilatation and anomalies and the formulation of an appropriate differential diagnosis. Visualization of dilated bronchial arteries at imaging should alert the radiologist to obstructive disorders that affect the pulmonary circulation and prompt the exclusion of diseases that produce or are associated with pulmonary artery obstruction, including chronic infectious and/or inflammatory processes, chronic thromboembolic disease, and congenital anomalies of the thorax (eg, proximal interruption of the pulmonary artery). Conotruncal abnormalities, such as pulmonary atresia with ventricular septal defect, are associated with systemic pulmonary supply provided by aortic branches known as major aortopulmonary collaterals, which originate in the region of the bronchial arteries. Bronchial artery malformation is a rare left-to-right or left-to-left shunt characterized by an anomalous connection between a bronchial artery and a pulmonary artery or a pulmonary vein, respectively. Bronchial artery interventions can be used successfully in the treatment of hemoptysis, with a low risk of adverse events. Multidetector computed tomography helps provide a vascular road map for the interventional radiologist before bronchial artery embolization.

Introduction

The lungs are supplied by two separate vascular systems consisting of the pulmonary and bronchial arteries. The pulmonary arteries carry deoxygenated blood at low pressure. They supply 99% of the blood flow to the lungs and participate in gas exchange at the alveolar capillary membrane. The bronchial arteries carry oxygenated blood to the lungs at a pressure six times that of the pulmonary arteries. The bronchial arteries provide nourishment to the supporting structures of the lungs, including the pulmonary arteries, but generally do not participate in gas exchange (1,2). They are connected to the pulmonary arteries through several microvascular anastomoses at the level of the alveoli and respiratory bronchioles (2).
The bronchial circulation and other collateral vessels (eg, intercostal, internal mammary, and inferior phrenic arteries) respond to chronic pulmonary ischemia and decreased pulmonary blood flow with hypertrophy or enlargement in an effort to maintain blood flow to the affected lung and participate in gas exchange through systemic-pulmonary arterial anastomoses that develop beyond the pulmonary artery obstruction.

Abnormal dilated bronchial arteries are large (>2 mm in diameter) and often have a tortuous mediastinal course. They manifest at multidetector CT or MR imaging as nodular or linear enhancing structures that are best depicted on multidetector or three-dimensional volumetric images, given their undulating course.

Visualization of dilated bronchial arteries at imaging should alert the radiologist to obstructive disorders affecting the pulmonary circulation and prompt the exclusion of related disorders, such as chronic infectious and/or inflammatory processes, chronic thromboembolic disease, and congenital cardiovascular anomalies of the thorax.

Bronchial artery dilatation in patients with thromboembolic disease suggests chronic rather than acute disease in otherwise equivocal cases.

For reasons that are not completely understood, bronchial artery dilatation uncommonly occurs in patients with primary pulmonary hypertension and is a useful feature in differentiating this disease from chronic thromboembolic disease.

In various scenarios that involve pulmonary artery compromise (eg, vasculitis and chronic pulmonary thromboembolic disease), the bronchial arteries and their anastomotic connections may dilate, which allows a greater percentage of the cardiac output to flow through the bronchial artery system (3,4). Visualization of dilated bronchial arteries at imaging should prompt the exclusion of various associated disorders.

This article reviews the normal anatomy, variant anatomy, and function of the bronchial arteries. We also discuss the imaging appearances of dilated bronchial arteries in various disease states and the role of bronchial arteries in hemoptysis, with a specific focus on bronchial artery embolization. Finally, we review emerging concepts regarding the physiology and function of bronchial arteries, potential applications to the management of coronary artery disease, and the postulated role of bronchial arteries in the development of chronic rejection after lung transplantation.

**Bronchial Artery Origins**

The bronchial arteries usually originate from the proximal descending thoracic aorta (Fig 1a–1c). They are termed orthotopic when they originate between the superior endplate of the T5 vertebral body and the inferior endplate of the T6 vertebral body. A proposed landmark for orthotopic bronchial arteries at angiography is 1 cm above or below the level of the left main bronchus as it crosses the descending thoracic aorta (5).

Bronchial arteries that originate elsewhere in the aorta or from other vasculature are termed ectopic (6–9). In a CT angiographic study that evaluated hemoptysis, 64% of patients had orthotopic bronchial arteries, and the remaining 36% had at least one ectopic bronchial artery, most commonly originating from the undersurface of the aortic arch (Fig 1d) (7). Other reports indicate a range of ectopic bronchial arteries in 8.3%–56% of all patients, depending on the method of examination (ie, autopsy or angiography) (7,10,11). Potential ectopic sites of origin include the inferior aortic arch, distal descending thoracic aorta, subclavian artery, brachiocephalic trunk, thyrocervical trunk, and internal mammary artery and even a coronary artery (Figs 2, 3) (7,12). Bronchial arteries that originate from a coronary artery may be inconsequential or may cause myocardial infarction or angina due to a coronary steal phenomenon (13).

Left bronchial arteries most commonly arise directly from the aorta. Right bronchial arteries occasionally originate directly from the aorta but more commonly share their origin with another artery, typically an intercostal artery (14). The most common configuration is a shared origin of the right bronchial artery and an intercostal artery (52% of cases at CT angiography), a finding known as a common ICBAT. The ICBAT usually originates from the anteromedial or posteromedial thoracic aortic wall and courses superiorly, giving rise to one or more intercostal arteries before abruptly coursing inferiorly as the bronchial artery (Fig 1d) (2,14–16). Occasionally, the right ICBAT has a conical triangular infundibulum at its origin. It is important not to mistake this anatomic variant for aortic disease (eg, a penetrating aortic ulcer or traumatic aortic injury). Unlike in aortic disease, the wall of the infundibulum is smooth, and the ICBAT originates directly from its apex (Fig 1e) (17). Less commonly, the left and right bronchial arteries share a common origin (32% of right and 36% of left bronchial arteries at CT angiography), a finding known as the common trunk of both bronchial arteries. The common trunk of both bronchial arteries usually originates from the anterior to lateral thoracic aortic wall (Fig 1f) (14).

An early autopsy study of 150 cadavers described variations in bronchial artery anatomy and divided the cases into nine types (Fig 4) (15). The most common pattern, type 1 (40.6% of cases), consisted of one right bronchial artery arising from a common ICBAT and two left bronchial arteries. Type 2 was the second
Figure 1. Normal orthotopic and ectopic bronchial arteries. (a) Axial computed tomographic (CT) angiograms show an orthotopic right bronchial artery (arrow) originating from the anteromedial thoracic aortic wall and an orthotopic left bronchial artery (arrowhead) originating from the anterior thoracic aortic wall. (b) Axial CT angiograms show normal bronchial arteries (arrows) as small nodular or linear enhancing mediastinal structures with a small diameter (<2 mm). (c) Three-dimensional volumetric reformatted CT image shows the mediastinal course of a right bronchial artery (arrow). (d) Coronal CT angiogram shows a right bronchial artery (white arrow) arising with an upper intercostal artery from a common intercostal-bronchial artery trunk (ICBAT) (black arrow). Note the ectopic left bronchial artery (arrowhead) arising from the undersurface of the aortic arch. (e) Oblique axial (left) and coronal (right) CT angiograms show an infundibulum (arrowheads) at the origin of a right ICBAT (arrows). This finding is a normal anatomic variant and should not be confused with aortic disease. (f) Axial CT angiogram shows a common origin of the left bronchial artery (arrow) and right ICBAT (arrowhead). (g) Axial contrast-enhanced magnetic resonance (MR) images show a normal right ICBAT (white arrow) that gives rise to an intercostal artery (black arrow) and a right bronchial artery (arrowhead).
most common pattern (21.3%), consisting of a single bronchial artery on each side, with the right bronchial artery originating from an IC-BAT. Type 3 was the third most common pattern (20.6%), consisting of two bronchial arteries on each side, with one of the right bronchial arteries originating from an ICBAT. Types 4–9 were other variations of bronchial arteries, with up to four arteries on each side (Table 1). Although type 1 was the most common anatomic pattern in the original study (15), other authors have reported type 3 to be the most common variant (18,19).

Orthotopic right bronchial arteries most commonly arise from the anterior to medial descending thoracic aorta, whereas orthotopic left bronchial arteries usually arise from the anterior to lateral descending thoracic aorta (14). In the mediastinum, orthotopic right bronchial arteries usually run to the right of the esophagus, whereas orthotopic left bronchial arteries run to the left. Both left and right bronchial arteries usually travel behind the trachea and main-stem bronchi before entering the lung via the hila (14).

Anatomy at Cross-sectional Imaging
Normal bronchial arteries are small, measuring less than 2 mm at their origin and 0.5 mm distally as they enter the bronchopulmonary segment (1,8). Normal bronchial arteries manifest at multidetector CT as small nodular or linear enhancing structures and are best depicted on...
multiplanar or three-dimensional volumetric images, given their undulating course (Fig 1) (9,14,20). As previously stated, the right bronchial arteries run to the right of the esophagus, and the left bronchial arteries run to the left. Bronchial arteries are best depicted at multidetector CT performed with high-volume (eg, 80–100 mL of standard nonionic intravenous contrast agent) and high-flow (eg, 3–4 mL/sec injection rate) contrast agent administration. Images should be acquired during the phase of maximal descending thoracic aortic enhancement. This is determined by using bolus tracking software or a test bolus technique. Motion-free images are often possible with use of cardiac gating or high-pitch acquisition. Images are ideally reviewed using thin sections (<1 mm). MR imaging is increasingly being used to evaluate the thorax, and normal bronchial arteries can be identified on nonenhanced and contrast-enhanced MR images (Fig 1g) and have an appearance similar to that of bronchial arteries at multidetector CT.

**Bronchial Artery Function**

The bronchial arteries normally receive only about 1% of the total cardiac output and help maintain airway and lung function (21). They provide systemic blood supply to the trachea, bronchi, bronchial branches, esophagus, and visceral pleura. They also supply blood to the vasa vasorum of the thoracic aorta and pulmonary arteries as well as to the nerves, pulmonary veins, and lymph nodes in the thorax (8,22). Because of their small diameter, the bronchial arteries are a high-resistance and low-capacitance or low-distensible circulation (6). They undergo smooth muscle wall hypertrophy or dilate to direct more oxygenated blood to ischemic lung tissue in patients with systemic hypoxemia, alveolar hypoxia, pulmonary infarction, and various inflammatory disorders that affect the thorax (2,22).

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**Figure 4.** Drawing shows the three most common variations in bronchial artery anatomy. Type 1 has one right bronchial artery and two left bronchial arteries, type 2 has one right bronchial artery and one left bronchial artery, and type 3 has two right bronchial arteries and two left bronchial arteries.

**Table 1: Bronchial Artery Classification in 150 Cadavers**

<table>
<thead>
<tr>
<th>Type</th>
<th>Occurrence (%)</th>
<th>No. of Left Bronchial Arteries</th>
<th>No. of Right Bronchial Arteries</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>40.6</td>
<td>2</td>
<td>1</td>
</tr>
<tr>
<td>2</td>
<td>21.3</td>
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<td>4</td>
<td>9.7</td>
<td>1</td>
<td>2</td>
</tr>
<tr>
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<td>3</td>
<td>1</td>
</tr>
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<td>8</td>
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<td>1</td>
</tr>
<tr>
<td>9</td>
<td>0.6</td>
<td>1</td>
<td>4</td>
</tr>
</tbody>
</table>

Source.—Reference 15.

Note.—In the study, 66.8% of right bronchial arteries arose from a common ICBAT.
Hypertrophy and Dilatation
The bronchial arteries play an important role in diseases that affect the airways and lung parenchyma. The bronchial circulation and other collateral vessels (eg, the intercostal, internal mammary, and inferior phrenic arteries) respond to chronic pulmonary ischemia and decreased pulmonary blood flow through hypertrophy or enlargement in an effort to maintain blood flow to the affected lung and participate in gas exchange through systemic-pulmonary arterial anastomoses that develop beyond the pulmonary artery obstruction (6,23–26). The total systemic cardiac output to the bronchial arteries may increase from 1% to 18%–30% in certain disease states, such as chronic thromboembolic disease (27,28).

Abnormal dilated bronchial arteries are large (>2 mm in diameter) and often have a tortuous mediastinal course (23). They manifest at multidetector CT or MR imaging as nodular and linear enhancing structures that are best depicted on multiplanar or three-dimensional volumetric images, given their undulating course (Fig 5) (9,14,20,22). An important pitfall in imaging interpretation is to mistake abnormal bronchial arteries for lymph nodes, veins, endobronchial lesions, broncholiths, or esophageal enhancement (29,30). Visualization of dilated bronchial arteries at imaging should alert the radiologist to obstructive disorders affecting the pulmonary circulation and prompt the exclusion of related disorders, such as chronic infectious and/or inflammatory processes, chronic thromboembolic disease, and congenital cardiovascular anomalies of the thorax. The causes of bronchial artery dilatation can be divided into five categories; the imaging manifestations of the various diseases are discussed in the subsequent sections.

### Congenital Pulmonary Artery Obstruction or Anomalies

#### Tetralogy of Fallot
Tetralogy of Fallot is the most common cyanotic congenital heart disease. The four classic findings related to incomplete development of the pulmonary infundibulum are (a) pulmonic outflow tract stenosis or pulmonic valve atresia, (b) overriding aorta, (c) right ventricular hypertrophy, and (d) ventricular septal defect (31).

Affected patients frequently have maldeveloped or underdeveloped central pulmonary arteries, with branch vessel occlusion or stenosis (31).

Major conotruncal congenital anomalies such as tetralogy of Fallot are associated with systemic pulmonary supply provided by aortic branches known as major aortopulmonary collateral arteries (MAPCAs) or systemic-pulmonary collateral arteries (Table 2). The majority of patients with MAPCAs have pulmonary atresia with ventricular septal defect, the so-called severe form of tetralogy of Fallot (Fig 6) (32). Patients with pulmonary atresia and a closed ductus arteriosus depend on MAPCAs for pulmonary perfusion and survival. An angiographic study showed that MAPCAs likely represent dilated bronchial arteries, given their similar anatomy and branching patterns and the absence of other native bronchial arteries in affected patients (26). MAPCAs provide competing blood flow to the lungs and are frequently embolized around the time of definitive surgery (31) or are used to reconstruct the neopulmonary trunk in patients with long-segment pulmonary trunk atresia or hypoplasia (31,33).

#### Proximal Interruption of the Pulmonary Artery
Proximal interruption or agenesis of the pulmonary artery is a rare developmental disorder. The mediastinal portion of the right or left pulmonary

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**Table 2: Conotruncal Abnormalities Associated with MAPCAs**

<table>
<thead>
<tr>
<th>Abnormality</th>
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<tbody>
<tr>
<td>Pulmonary atresia with ventricular septal defect (a severe form of tetralogy of Fallot and the most common disease associated with MAPCAs)</td>
</tr>
<tr>
<td>Tetralogy of Fallot</td>
</tr>
<tr>
<td>Transposition of the great arteries</td>
</tr>
<tr>
<td>Double-outlet right ventricle</td>
</tr>
<tr>
<td>Truncus arteriosus</td>
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</table>
Figure 6. Tetralogy of Fallot. (a) Axial (left) and coronal (right) CT angiograms show large arteries (arrows) arising from the thoracic aorta to provide systemic supply to the lung. These aortic branches originate in the bronchial arteries and are known as MAPCAs. (b) Axial CT angiogram shows the characteristic findings of tetralogy of Fallot, with the aorta (Ao) overriding the interventricular septum, a ventricular septal defect (s), and right ventricular hypertrophy (arrow).

artery is atretic, and the distal pulmonary circulation is supplied by systemic collaterals, most commonly the bronchial arteries (34). An interrupted left pulmonary artery is generally associated with a right aortic arch and severe congenital heart defects, including tetralogy of Fallot. An interrupted right pulmonary artery is more common and generally is an isolated finding. Patients are almost always symptomatic and present with recurrent infections, dyspnea, and, occasionally, pulmonary hypertension (35). Hemoptysis secondary to ruptured systemic collateral blood vessels occurs in about 10% of patients (36).

CT is used to diagnose this condition and shows complete absence of the mediastinal portion of the affected pulmonary artery or a short pulmonary artery that terminates within a centimeter of its origin (Fig 7). Proximal interruption of the pulmonary artery generally occurs opposite the side of the aortic arch. The affected lung is generally hypoplastic, and the contralateral lung is hyperinflated and rotates across the midline (37). There are abundant systemic collaterals to the affected lung that manifest as dilated bronchial, subclavian, and intercostal arteries (37). Linear opacities are frequently seen at the lung periphery and likely represent transpleural vascular collaterals (38).

Anomalous Origin of the Left Coronary Artery from the Pulmonary Artery

Anomalous origin of the left coronary artery from the pulmonary artery (ALCAPA) syndrome is a rare congenital heart defect that affects one of 300,000 live births. If the condition is left untreated, over 90% of affected infants die of myocardial infarction related to coronary steal, with myocardial blood flow being diverted from the myocardium to the pulmonary trunk (39). Uncommonly, affected patients present in adulthood. These patients typically develop sufficient right coronary artery to left coronary artery collaterals as infants, which prevents immediate myocardial infarction and death (40). The most common cause of death in untreated patients who survive to adulthood is silent myocardial ischemia with resultant dysrhythmias and sudden cardiac death (40).

The imaging hallmark of ALCAPA syndrome is an anomalous origin of the left coronary artery from the pulmonary trunk, usually along its left inferolateral aspect just beyond the pulmonary valve. In adults, there is marked dilatation and tortuosity of both coronary arteries related to chronic shunting from the right coronary artery to the left coronary artery, which eventually drains into the pulmonary circulation. There usually are abundant intercoronary collaterals on the epicardial surface of the heart (40,41). Dilated bronchial arteries are seen only in adults with ALCAPA syndrome and function as a collateral systemic supply to the left coronary artery territory (Fig 8) (40,41).

Acquired Intrinsic Pulmonary Artery Obstruction

Chronic Thromboembolic Disease

The majority of pulmonary thromboemboli resolve with appropriate therapy. In about 4% of patients with symptomatic pulmonary throm-
boembolic disease, the thromboemboli incompletely resolve, causing partial or complete obstruction of the pulmonary vascular bed that leads to pulmonary hypertension and eventually to cor pulmonale (23,42). The clinical manifestations are often insidious and nonspecific, consisting of progressive dyspnea, exhaustion, and clinical deterioration that parallel the degree of right heart dysfunction. A large percentage of patients (63%) lack a history of acute thromboembolism, making the disease difficult to diagnose (43).

Chronic thromboembolic disease may manifest at CT with vascular and parenchymal abnormalities (23) (Fig 9). The vascular abnormalities include partial or complete obstruction of the pulmonary arteries, which manifests as endoluminal filling defects that are often peripheral, crescentic, calcified, or linear. Complete obstruction manifests with abrupt termination, narrowing, or obliteration of a pulmonary artery. Secondary signs of pulmonary hypertension include pulmonary trunk dilatation (>29 mm), right ventricular or right atrial enlargement or hypertrophy, pulmonary artery wall calcification, and leftward bowing of the interventricular septum (44). Parenchymal signs include lower lobe subpleural bandlike, linear, or wedge-shaped opacities that represent the sequelae of pulmonary infarction. Mosaic attenuation is often seen and manifests as sharply demarcated regions of normal lung attenuation alternating with regions of decreased lung attenuation, findings due to differences in vascular perfusion (23).

The presence of dilated bronchial arteries in patients with chronic thromboembolic pulmonary hypertension correlates with a lower mortality rate after thromboendarterectomy (3). Bronchial artery dilatation in patients with thromboembolic disease suggests chronic rather than acute disease in otherwise equivocal cases (45).

**Takayasu Arteritis**

Takayasu arteritis is an idiopathic large-vessel vasculitis that most commonly affects the aorta, the great vessels, and, less commonly, the pulmonary arteries. It is usually seen in young women, often those of Asian descent (46,47). The patient presentation varies depending on the disease activity and course. Early in the disease, patients present with nonspecific systemic symptoms and signs that include fever, night sweats, malaise, and weight loss. As the inflammation progresses to cause vascular narrowing and occlusion,
patients may present with limb claudication, renovascular hypertension, visual symptoms, absent pulses, or discrepant blood pressures (47). Imaging plays a vital role in the diagnosis of Takayasu arteritis, given its nonspecific clinical presentation. Bronchial artery dilatation usually occurs only in patients with pulmonary artery stenosis or occlusion and can help suggest the diagnosis (Fig 10). Pulmonary artery involvement is reported in 20%–80% of patients but usually is a late manifestation of the disease (46–48). Imaging findings suggestive of Takayasu arteritis include vessel wall thickening with enhancement in the acute phase of the disease and flame-shaped stenosis or occlusion of the lobar or segmental pulmonary arteries in the late phase. Unilateral occlusion of a pulmonary artery can occur in late phase disease and should be considered in the differential diagnosis of patients with unexplained unilateral pulmonary artery occlusion (46). Pulmonary artery aneurysms with or without associated vascular stenosis can also occur (47). Patients with pulmonary artery involvement usually have other great vessel involvement, which can be a helpful clue to suggest this rare diagnosis.

**Acquired Extrinsic Pulmonary Artery Obstruction**

**Fibrosing Mediastinitis**

Fibrosing mediastinitis is a benign but sometimes progressive disorder characterized by infiltrative fibrous tissue in the mediastinum. The two types are nongranulomatous and granulomatous (49). Nongranulomatous fibrosing mediastinitis usually occurs in association with autoimmune disorders or radiation therapy or as a reaction to drugs such as methysergide. In the United States, granulomatous fibrosing mediastinitis is usually an idiosyncratic reaction to *Histoplasma* antigens but can be caused by other infections such as tuberculosis, cryptococcosis, and aspergillosis (49). The granulomatous variety of fibrosing mediastinitis usually manifests as a focal mediastinal mass, often with calcification. The nongranulomatous variety often manifests with diffuse mediastinal infiltration without calcification. The initial symptoms and clinical presentation generally depend on the mediastinal structures affected by the fibrotic tissue (49). The most common mediastinal structures to be narrowed or obstructed include the airways, superior vena cava, esophagus, pulmonary arteries, and pulmonary veins (50). When fibrosing mediastinitis encases a pulmonary artery or pulmonary vein, patients may present with pulmonary hypertension and symptoms of cor pulmonale that mimic the manifestations of other causes of pulmonary hypertension, such as chronic thromboembolic disease (51).

Fibrosing mediastinitis may manifest at imaging as a focal mediastinal or hilar mass and less commonly as diffuse mediastinal infiltration. The most common imaging appearance is a focal soft-tissue mass that distorts and narrows adjacent structures (Fig 11). There often is dense or stippled calcification within the mass, and other signs of remote granulomatous infection are frequently seen, such as calcified granulomas in the lymph nodes, lungs, liver, and spleen (52). Occasionally, there is a diffuse and infiltrative noncalcified mass that involves
several mediastinal compartments. Because the diffuse form is generally idiopathic or associated with noninfectious diseases, signs of remote granulomatous infection are generally absent (52). Constriction and obstruction of the pulmonary arteries is generally unilateral (49). Ipsilateral bronchial artery dilatation is seen when fibrous tissue critically encases and narrows a pulmonary artery as a physiologic response to decreased blood flow to the lung (Fig 11) (53).

**Chronic or Acute Inflammation**

**Bronchiectasis, Infection, and Malignancy**

The bronchial circulation often dilates in patients with inflammatory conditions that affect the lungs and airways (21,54). It is thought that vasculitis and thrombosis of pulmonary vessels may occur in response to airway or parenchymal inflammation and infection. Angiogenic growth factors such as vascular endothelial growth factor are postulated to promote collateral supply and neovascularity, with proliferation and expansion of the bronchial arteries (55). Furthermore, an increase in the bronchopulmonary anastomoses at the precapillary and capillary levels occurs in patients with bronchiectasis due to cystic fibrosis and other conditions (6). The most common inflammatory lung and airway disorders associated with bronchial artery dilatation include tuberculosis and nontuberculous mycobacterial infections, cystic fibrosis and other causes of bronchiectasis, mycetoma, and chronic fungal infections.

Bronchiectasis usually occurs secondary to chronic or recurrent infection, aspiration, airway obstruction, or various congenital disorders such as cystic fibrosis (56). Patients with bronchiectasis present with recurrent cough, dyspnea, hemoptysis, sputum production, and recurrent infections. Hemoptysis is caused by recurrent airway inflammation that leads to bronchial artery dilatation and neovascularity (Fig 12) (1,29).
Infection by *Mycobacterium tuberculosis* is one of the most common causes of hemoptysis worldwide and occasionally requires treatment with bronchial artery embolization (8). Nontuberculous mycobacterial infection and other diseases characterized by acute and chronic inflammation may also produce hemoptysis through similar mechanisms (8,57). Saprophytic aspergillosis or mycetoma represents infection by *Aspergillus fumigatus* without tissue invasion. The *Aspergillus* organisms colonize preexisting lung cavities and cystic spaces most commonly associated with tuberculosis infection or sarcoidosis (58). Patients may be asymptomatic or may present with life-threatening hemoptysis resulting from bronchial artery hypertrophy, dilatation, and eventual rupture. The characteristic CT appearance of mycetoma is a solid round or oval gravity-dependent mass within a cavity (Fig 13). There frequently is a crescent of air separating the mass from the cavity wall, a finding termed the “air crescent” sign (58). In patients who present with hemoptysis and mycetoma, the radiologist should search for hyper- trophyed bronchial arteries to provide a vascular road map for the interventionalist before bronchial artery embolization.

Lung cancer (frequently necrotic squamous cell cancer) is the most common cause of massive
hemoptysis in adults older than 40 years. Other
cancers, including metastatic disease and endo-
bronchial carcinoid tumors, may also cause hemop-
tysis through similar mechanisms (59,60). The most
common reason for hemoptysis in patients with
cancer is tumoral neovascularity, destruction of
lung parenchyma by the tumor, and tumor angio-
invasion (60). Multidetector CT is useful in dem-
onstrating the cause and site of bleeding and iden-
tifying abnormal bronchial arteries or, rarely, active
contrast agent extravasation as the source (59).

Pulmonary Hypertension

Bronchial Arteries in Pulmonary Hypertension

Pulmonary hypertension is defined as a mean pul-
monary artery pressure greater than 25 mm Hg
at rest or greater than 30 mm Hg with exercise,
with associated increased vascular resistance (61).
A useful way of classifying pulmonary hyperten-
sion is to divide diseases into those that primarily
affect the arterial system (precapillary causes)
and those that primarily affect the venous system
(postcapillary causes) (Table 3) (61).

The most important imaging finding of pul-
monary hypertension at CT is pulmonary trunk
dilatation (>29 mm in diameter) (44). Other sug-
gestive signs include narrowing and tapering of the
peripheral pulmonary arteries, right ventricular
dilatation, right atrial dilatation, leftward bowing
of the interventricular septum, and reflux of con-
trast material into a dilated inferior vena cava and
the hepatic veins (62). Parenchymal abnormalities
that may be seen with pulmonary hypertension
include mosaic lung attenuation and linear scars
or wedge-shaped subpleural opacities related to
previous lung infarction (23).

Primary pulmonary hypertension is an idio-
pathic precapillary cause of pulmonary hyperten-
sion without an identifiable embolic source. It
most commonly affects young or middle-aged
women. Patients present with progressive dys-
pnea on exertion, fatigue, Raynaud phenomenon,
or symptoms of cor pulmonale (63). Patient sur-
vival is poor, with the majority succumbing to the
disease within 2–5 years after diagnosis (63).

For reasons that are not completely under-
stood, bronchial artery dilatation uncommonly
occurs in patients with primary pulmonary
hypertension (Fig 14) and is a useful feature in
differentiating this disease from chronic thrombo-
embolic disease (24,27). In a study of 36 patients
evaluated with CT angiography (24), only 14%
two of 14) with primary pulmonary hyperten-
sion had dilated bronchial arteries, whereas
73% (16 of 22) with pulmonary hypertension
secondary to chronic thromboembolic disease
had dilated bronchial arteries. An angiographic
study (27) had similar results, with only 26% of
patients with primary pulmonary hypertension
having increased bronchial blood flow, compared
with 100% of patients with chronic thromboem-
bolic disease.

Bronchial Artery Anomalies

Bronchial arteriovenous malformation, or bron-
chial artery malformation, is a rare congenital or
acquired disorder that results in a left-to-left or
left-to-right extracardiac shunt. Acquired causes
of bronchial artery malformation are inflam-
matory or infectious lung diseases, penetrating
trauma, and tumor (64). It is characterized by
a tortuous and enlarged bronchial artery that
communicates with a pulmonary artery or
pulmonary vein (64,65). It is most common in
men, can occur at any age, and usually affects
the right lung. Patients often are asymptomatic,
but the condition may result in massive hemop-
tysis and death (66).
Bronchial artery malformation manifests at multidetector CT as a tortuous and dilated bronchial artery, often with focal aneurysms (Fig 15a). Anomalous communication with a pulmonary artery or vein may be demonstrated. Bronchial angiography is used to confirm the diagnosis and also is therapeutic in patients who require nonparticulate embolization (Fig 15b). Bronchial artery malformation may manifest at bronchoscopy as a nonpulsatile endobronchial nodule, which if inadvertently biopsied may result in life-threatening bleeding (67).

**Hemoptysis and Bronchial Artery Intervention**
Massive hemoptysis is generally defined as expectoration of greater than 300–600 mL of blood in 24 hours. The source of hemoptysis in 90% of cases that require intervention is the bronchial arteries (1,8). Conservatively treated massive hemoptysis has a high mortality rate (50%–100%), with death usually resulting from asphyxiation rather than from exsanguination (8). Most clinically significant cases of hemoptysis are secondary to entities that increase bronchial and systemic blood flow to the lungs, such as bronchiectasis, lung cancer, chronic bronchitis, cavitary infection, cavitary sarcoidosis, and bronchial artery malformation (8). The reason for hemoptysis is secondary to the following two factors that increase bronchial arterial flow:

1. Bronchial arterial flow increases in conditions that result in decreased pulmonary blood flow, such as chronic thromboembolic disease and Takayasu arteritis. The normal bronchial-pulmonary arterial anastomotic connections vasodilate to accommodate the increased flow and to direct systemic blood to the lungs. The dilated
anastomotic connections are prone to rupture because they are thin walled and under systemic blood pressures (1).

2. Bronchial arterial flow also increases in inflammatory disorders of the lung, such as bronchiectasis and tuberculosis infection. Inflammatory and obstructive conditions likely release angiogenic growth factors that promote collateral supply and neovascularity (68,69). These newly formed collateral vessels are prone to rupture because they are fragile and “leaky” (1).

Most cases of clinically significant hemoptysis are treated with bronchial artery embolization (BAE). Multidetector CT with volumetric reformatted images is useful in the pre-embolization assessment of patients with hemoptysis because it allows identification of dilated bronchial arteries and their origins as well as the disease process causing hemoptysis. The imaging examination is fast and provides a detailed vascular road map for the interventionalist before the procedure (1,9).

In a comparative study involving 200 patients, the use of multidetector CT before angiography reduced the rate of catheterization failures and the number of patients needing surgical intervention (70).

BAE for hemoptysis often begins with descending thoracic aortography for identification of the bronchial artery bleeding source (Fig 16a) (8). Angiographic manifestations of an abnormal bronchial artery include tortuosity, dilatation, areas of hyper- and/or neovascularity, aneurysms, and shunts (Fig 16b) (8). Even in cases of clinically documented hemoptysis, visualization of contrast material extravasation at angiography is rare. Identification of a bronchial artery origin of the anterior spinal artery is critical to avoid postembolization transverse myelitis (Fig 16c). The anterior spinal artery is reported to arise from the right ICBAT in about 5%-10% of cases, although the exact prevalence is probably lower (8,71). The anterior spinal artery is best identified as a thin enhancing vessel that overlies the ventral cord on multiple angiographic projections, with a characteristic “hairpin” configuration as it enters the spinal canal (9).

BAE is usually performed with the use of particles (spherical embolics or polyvinyl alcohol) with a diameter of 350–500 µm (8). Microcatheter subselection of the abnormal bronchial artery helps avoid reflux into the aorta and nontarget embolization (Fig 17). BAE is typically successful.
in the initial control of hemoptysis (73%–98% of patients), but recurrence is common (9%–55%) and may require re-embolization (8,72). It is for this reason that particles are favored over coils, because proximal embolization with coils may prevent attempts at future embolization by “closing the door” and preventing access to the bleeding vessel (73). A recent retrospective review by Woo et al (74) showed improved 1-, 3-, and 5-year hemoptysis-free survival periods in patients treated with n-butyl-2-cyanoacrylate (NBCA) compared with those treated with polyvinyl alcohol particles, a finding indicating that NBCA may be a more effective agent for BAE. Postprocedural chest pain is the most common complication after BAE and affects 24%–91% of patients. Much more significant but less frequent complications include dysphagia (0.7%–18.2%) and spinal cord ischemia (1.4%–6.5%) with transverse myelitis (8).

Emerging Clinical Applications of Bronchial Arteries
Bronchial arteries are arteriosclerosis-resistant blood vessels. Koutoulas et al (75) studied bronchial arteries in 40 patients who had undergone major thoracic procedures and found that unlike coronary arteries, the bronchial arteries were unaffected by diseases such as atherosclerosis. Although further research must be performed, it is possible that bronchial arteries may one day be used to revascularize the coronary arteries.

The main causes of death after lung transplantation are infection and chronic rejection, the latter often manifesting as bronchiolitis obliterans syndrome (76). Bronchiolitis obliterans syndrome is a fibrotic small-airways condition resulting in physiologic airflow obstruction and a reduced forced expiratory volume in 1 second from the posttransplantation baseline. Bronchiolitis obliterans syndrome manifests at high-resolution CT with mosaic lung attenuation, bronchial wall thickening, bronchiectasis, and expiratory air trapping (77). The airways derive about 50% of their blood supply from the bronchial arteries and 50% from the pulmonary arteries. Transplant surgeons usually revascularize only the pulmonary blood flow after lung transplantation. Emerging evidence links the development of bronchiolitis obliterans syndrome to airway ischemia, with some surgeons advocating bronchial artery revascularization in an attempt to avert or delay the development of bronchiolitis obiterans syndrome (76).

Conclusion
Normal bronchial arteries are often visible at multidetector CT. Abnormal bronchial arteries measure more than 2 mm in diameter and manifest as round, linear, or serpentine structures that may give rise to aneurysms. Multidetector CT is useful in the pre-embolization assessment of patients with hemoptysis to help identify abnormal bronchial arteries and the disease process causing hemoptysis. Incidental visualization of dilated bronchial arteries should prompt the radiologist to exclude various causes of pulmonary artery obstruction or parenchymal lung disease (Table 4).
Table 4: Conditions Associated with Bronchial Artery Dilatation

<table>
<thead>
<tr>
<th>Disease</th>
<th>Bronchial Artery Dilatation*</th>
<th>Key Imaging Features</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tetralogy of Fallot</td>
<td>+++</td>
<td>MAPCAs are most commonly seen in pulmonary artery with ventricular septal defect (so-called severe tetralogy of Fallot)</td>
</tr>
<tr>
<td>Proximal interruption of the pulmonary artery</td>
<td>+++</td>
<td>Recurrent infection or dyspnea, small ipsilateral lung</td>
</tr>
<tr>
<td>ALCAPA</td>
<td>+</td>
<td>Seen only in affected adults</td>
</tr>
<tr>
<td>Chronic thromboembolic disease</td>
<td>+++</td>
<td>Peripheral, crescentic, calcified, or linear pulmonary emboli; bronchial arteries supply the most severely affected lung</td>
</tr>
<tr>
<td>Takayasu arteritis</td>
<td>+</td>
<td>Patients with pulmonary artery involvement usually have great vessel involvement</td>
</tr>
<tr>
<td>Fibrosing mediastinitis</td>
<td>+</td>
<td>Soft-tissue mass with stippled or dense calcification; narrowing or obstruction of the pulmonary arteries</td>
</tr>
<tr>
<td>Lung cancer</td>
<td>++</td>
<td>Cavitary lung mass (squamous cell cancer)</td>
</tr>
<tr>
<td>Bronchiectasis</td>
<td>++</td>
<td>Abnormal bronchoarterial ratios, nontapering bronchi, bronchi within 1 cm of lung periphery; bronchial arteries usually supply the affected lung</td>
</tr>
<tr>
<td>Primary pulmonary hypertension</td>
<td>+</td>
<td>Bronchial artery dilatation is uncommon; when present, consider chronic thromboembolic disease</td>
</tr>
</tbody>
</table>

* + = low likelihood of dilatation, ++ = intermediate likelihood of dilatation, +++ = high likelihood of dilatation.

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**References**