Radiological Management of Hemoptysis: A Comprehensive Review of Diagnostic Imaging and Bronchial Arterial Embolization

Joo-Young Chun · Robert Morgan · Anna-Maria Belli

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Abstract Hemoptysis can be a life-threatening respiratory emergency and indicates potentially serious underlying intrathoracic disease. Large-volume hemoptysis carries significant mortality and warrants urgent investigation and intervention. Initial assessment by chest radiography, bronchoscopy, and computed tomography (CT) is useful in localizing the bleeding site and identifying the underlying cause. Multidetector CT angiography is a relatively new imaging technique that allows delineation of abnormal bronchial and nonbronchial arteries using reformatted images in multiple projections, which can be used to guide therapeutic arterial embolization procedures. Bronchial artery embolization (BAE) is now considered to be the most effective procedure for the management of massive and recurrent hemoptysis, either as a first-line therapy or as an adjunct to elective surgery. It is a safe technique in the hands of an experienced operator with knowledge of bronchial artery anatomy and the potential pitfalls of the procedure. Recurrent bleeding is not uncommon, especially if there is progression of the underlying disease process. Prompt repeat embolization is advised in patients with recurrent hemoptysis in order to identify nonbronchial systemic and pulmonary arterial sources of bleeding. This article reviews the pathophysiology and causes of hemoptysis, diagnostic imaging and therapeutic options, and technique and outcomes of BAE.

Keywords Hemoptysis · Multidetector computed tomography angiography · Bronchial artery · Embolization

Introduction

Hemoptysis can be a life-threatening respiratory emergency that requires prompt investigation and management. It is a relatively common presenting symptom in clinical practice [1] and can signify potentially serious underlying thoracic disease. Despite advances in modern-day medicine, large-volume hemoptysis still poses a diagnostic and therapeutic challenge.

Conservative management of massive hemoptysis has a 50–100% mortality rate [2, 3]. Until two decades ago, surgery was regarded as the treatment of choice for hemoptysis. However, a large proportion of patients are not suitable candidates for surgery due to pre-existing comorbidities and poor respiratory reserve, and mortality rates of up to 40% have been reported following emergency surgery [4]. Bronchial artery embolization (BAE) is a minimally invasive alternative, which is now considered to be the most effective nonsurgical treatment in the management of massive and recurrent hemoptysis. It plays an important role in primary therapy and in stabilizing patients prior to elective surgery.

This article reviews the pathophysiology and causes of hemoptysis, initial investigations including diagnostic imaging, options for management, systemic and pulmonary artery anatomy, bronchial artery angiography and embolization technique, and complications associated with BAE.

Pathophysiology and Causes of Hemoptysis

The lungs have a dual arterial supply composed of the pulmonary and bronchial arterial systems. The pulmonary arteries account for 99% of the arterial supply and are responsible for gaseous exchange. The bronchial arteries
make up the remaining 1% and supply nutrient branches to
the bronchi, vasa vasorum to the pulmonary arteries and
veins, and smaller bronchopulmonary branches to the lung
parenchyma [5, 6]. The two systems are connected by
numerous anastomoses between bronchial and pulmonary
arteries at the level of the bronchi and the pulmonary
lobules [5]. This communication produces a physiological
right-to-left shunt that accounts for approximately 5% of
the total cardiac output [6].

In conditions where the pulmonary circulation is com-
promised, such as hypoxic vasoconstriction, intravascular
thrombosis, and vasculitis, the bronchial arteries proliferate
and enlarge to gradually replace the pulmonary circulation
[6, 7]. Chronic inflammation of the lungs is also associated
with enlargement of the bronchial arteries as a result of
abnormal enhanced communication with the pulmonary
arterioles [8, 9]. Inflammatory processes release angiogenic
growth factors, which promote neovascularization and
recruitment of collateral supply from adjacent systemic
vessels [10]. These new vessels, which are usually thin-
walled and fragile, are exposed to increased systemic
arterial pressures and are prone to rupture into the airways,
resulting in hemoptysis.

The definition of massive hemoptysis varies in the lit-
erature from 100 to 1,000 ml over a period of 24 h, but the
most widely used figure is expectoration of 300–600 ml of
blood in 24 h [4, 8, 9, 11]. It is estimated that 400 ml of
blood in the alveolar space is sufficient to inhibit gaseous
exchange significantly [12] and the cause of death is usu-
ally asphyxiation rather than exsanguination [2].

There are multiple causes of hemoptysis (Table 1).
Bronchogenic carcinoma, chronic inflammatory lung dis-

cases due to bronchiectasis, and aspergilloma within a
chronic sarcoid or tuberculous cavity account for most
cases in the Western world, while active tuberculosis (TB)
continues to be the leading cause of hemoptysis worldwide
[9, 12, 13]. In some cases a cause cannot be found and is
termed idiopathic or cryptogenic hemoptysis. It is a diag-
nosis of exclusion and is reported to be responsible for
3–42% of hemoptysis episodes, particularly in smokers
[13–15].

### Diagnosis of Hemoptysis

Initial evaluation of patients with hemoptysis should aim to
locate the source of bleeding and to identify the underlying
cause. Standard diagnostic studies include sputum exami-
nation, bronchoscopy, chest radiography, and chest com-
puted tomography (CT). Sputum should be tested for the
presence of bacteria, especially mycobacterium and fun-
gus, and for malignant cells, especially in smokers over
40 years of age [12].

<table>
<thead>
<tr>
<th>Table 1 Causes of hemoptysis</th>
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<tr>
<td><strong>Pulmonary diseases</strong></td>
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<tr>
<td>Tuberculosis</td>
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<td>Aspergilloma</td>
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<td>Pneumonia</td>
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<td>Bronchiectasis</td>
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<td>Lung malignancy</td>
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<td>Chronic obstructive airways disease</td>
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<td>Cystic fibrosis</td>
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<td>Sarcoidosis</td>
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<td>Vasculitis: Behçet’s disease, Wegener’s granulomatosis</td>
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<td><strong>Cardiovascular diseases</strong></td>
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<td>Pulmonary artery arteriovenous malformation/aneurysm</td>
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<td>Pulmonary embolism</td>
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<td>Bronchial artery aneurysm</td>
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<td>Thoracic aortic aneurysm rupture/dissection</td>
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<td>Aortobronchial fistula</td>
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<td><strong>Others</strong></td>
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<tr>
<td>Coagulopathy</td>
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<td>Iatrogenic: anticoagulation, Swan-Ganz catheters</td>
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<td><strong>Trauma</strong></td>
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Bronchoscopy is the primary method for diagnosis and
localization of hemoptysis for chest physicians [12, 16].
Rigid bronchoscopy is recommended in cases of massive
hemoptysis because of its ability to maintain airway
patency, although flexible bronchoscopy is used more
widely, as it can be performed at the patient’s bedside
without the use of general anaesthetic and a vasoactive
drug can be instilled directly into the bleeding source [1,
12]. However, the overall diagnostic accuracy of bron-
choscopy in localizing the site of bleeding is <50% [11,
17] and it is less useful in identifying the underlying cause.
Bronchoscopy has further disadvantages during active
hemoptysis because the airways are filled with blood,
making evaluation of the distal airways difficult, and
endobronchial therapies are ineffective in most cases [11,
12]. The ideal time for bronchoscopy is controversial but
the consensus is to perform urgent bronchoscopy in
patients with massive hemoptysis. A report from the
American College of Chest Physicians stated that 64% of
clinicians favoured early diagnostic bronchoscopy within
the first 24 h and 79% used flexible bronchoscopy [1].

### Diagnostic Imaging

Chest radiography is readily available and may help lat-
eralize the bleeding and diagnose underlying parenchymal
Management of Hemoptysis

The management of hemoptysis should include initial resuscitation and supportive measures, such as monitoring of cardiorespiratory parameters, correction of hypoxia, stabilization of blood pressure, and transfusion with blood products as necessary. In the case of massive hemoptysis, most clinicians favor management in an intensive care setting with early endotracheal intubation [1]. Endobronchial therapy such as infusion of cold saline solution, use of balloon catheters, or instillation of epinephrine has been reported to be unreliable and of limited use [1, 16].

Until the 1970s, surgery was regarded as the treatment of choice for hemoptysis once the bleeding site was localized by bronchoscopy. Documented mortality rates following surgical intervention vary between 7.1 and 18.2% but increase to 40% in the emergency setting [4, 28]. Surgery during an episode of acute hemorrhage carries a high risk of operative bleeding, asphyxia, bronchopleural fistula, and respiratory failure [29]. In addition, a large proportion of patients with hemoptysis are not suitable candidates for surgery due to pre-existing comorbidities and poor respiratory reserve. However, surgery remains the procedure of choice in the treatment of massive hemoptysis caused by iatrogenic pulmonary artery rupture, chest trauma, and aspergilloma resistant to other therapeutic options [12, 30].

BAE has become a well-established vascular interventional technique in the management of massive and recurrent hemoptysis [4, 31–39]. The mode of action is the occlusion of systemic arterial inflow to the fragile vessels within inflammatory tissue, reducing the perfusion pressure and the likelihood of further bleeding [9]. Interest in systemic pulmonary circulation in the 1960s led to visualization of the bronchial arteries by nonselective thoracic aortography [40, 41]. In 1964, the first selective bronchial artery catheterization and angiography was performed by Viamonte [42]. The technique was slow to take off in the subsequent decade due to reports of spinal cord ischemia, which was caused by inadvertent occlusion of the spinal arteries that may arise from bronchial or intercostal arteries, exacerbated by large-diameter catheters in use at the time and ionic contrast media [43–45]. These reports prompted further evaluation of the arterial supply to the spinal cord and improvements in angiographic techniques. The first successful BAE for hemoptysis was performed by Remy et al. in 1974 [46]. Since then, many authors have demonstrated the efficacy and safety of this procedure in controlling hemoptysis [31, 35–39, 47–50], and a survey by the American College of Chest Physicians showed that a higher proportion of chest physicians favored interventional radiology over either conservative or surgical management [1].

Bronchial Artery Anatomy

The bronchial arteries have a variable anatomy in terms of origin and branching distribution. It is important that the
operator is familiar with these variants prior to undertaking BAE. Most commonly, they arise from the thoracic aorta at a level between the superior margin of T5 and the inferior margin of T6 in approximately 70–83.3% [51, 52]. Cauldwell et al. [51] described the four most common bronchial branching patterns based on a study of 150 adult cadavers, as illustrated in Fig. 1. Type 1 has one right bronchial artery arising from an intercostobronchial trunk (ICBT) and two left bronchial arteries (40.6%). Type 2 has one right from an ICBT and one left (21.3%), type 3 has two from the right (one from an ICBT) and two from the left (20.6%), and type 4 has two from the right (one from an ICBT) and one from the left (9.7%). The most constant vessel is the right ICBT, present in 88.7% (Fig. 2). This vessel usually arises from the right posterolateral aspect of the thoracic aorta, whereas the individual left and right bronchial arteries arise from the anterolateral aspect. The right and left bronchial arteries can also arise from a common trunk.

Bronchial arteries that originate outside the T5–T6 vertebral levels of the thoracic aorta are considered to be anomalous or ectopic and their prevalence ranges from 16.7 to 30% [51, 52]. They have been reported to originate from the aortic arch, brachiocephalic artery, subclavian artery, internal mammary artery, thyrocervical trunk, costocervical trunk, inferior phrenic artery, or abdominal aorta [52]. All these variants can be distinguished from nonbronchial systemic arteries because they extend along the course of the major bronchi. The nonbronchial systemic arteries enter the lung parenchyma through the inferior pulmonary ligament or the adherent pleura, and their course is not parallel to that of the bronchi [53].

Bronchial arteries are the most common source of bleeding in hemoptysis [11]. They appear as enhancing nodular or linear structures within the mediastinum and around the central airway in contrast-enhanced axial CT images. Abnormal bronchial arteries are most commonly found in the retrotracheal and retroesophageal areas, as well as the posterior wall of the main bronchus and aortopulmonary window [20, 21]. Bronchial arteries with a diameter $>2$ mm are considered to be abnormal and are candidates for embolotherapy.

The vascular supply of the thoracic spinal cord is an important consideration during angiography. The anterior portion of the cord is supplied by the anterior spinal artery which originates from branches of the vertebral arteries. It receives blood from anterior medullary branches, which usually arise from intercostal and lumbar arteries [54]. In the thoracic region, the anterior spinal artery is usually supplied by a single anterior medullary artery but the largest anterior medullary branch, also known as the artery of Adamkiewicz, can arise at any level between T5 and L4 [52]. Although it is uncommon, the anterior medullary...
artery can originate from the intercostal branch of the right ICBT and has a characteristic ‘hairpin’ configuration on angiography (Fig. 3). Embolization of the anterior medullary artery has been associated with transverse myelitis and should be avoided [43, 45]. When the source of bleeding is seen in a vessel with a visible spinal branch, safe embolization can be achieved by advancing a microcatheter beyond the origin of the spinal artery and injecting the embolic material with care to prevent reflux into the spinal branch [9].

Assessment of Nonbronchial Systemic Arteries

In addition to bronchial arteries, chronic inflammatory processes recruit collateral blood supply from nonbronchial arteries via transpleural vessels. Overlooking these systemic arteries at initial angiography may result in persistent hemoptysis after what is thought to be a technically successful BAE. These collateral vessels can arise from branches of the subclavian, axillary, and internal mammary arteries as well as infradiaphragmatic branches from the inferior phrenic, left gastric, and celiac axis (Figs. 4 and 5). Many studies have reported the importance of actively searching for nonbronchial collateral supply on initial angiography [9, 11, 21, 34, 55] and CT has an important role to play in predicting the presence of these vessels prior to BAE. On contrast-enhanced CT, they appear as arteries running a course that is not parallel to the bronchi, usually along a pleural surface (Fig. 6). Features suggestive of a
nonbronchial systemic arterial supply as a source of hemoptysis include pleural thickening of more than 3 mm adjacent to an area of pulmonary abnormality and tortuous enhancing vascular structures within hypertrophic extrapleural fat [22, 26].

**Assessment of Pulmonary Arteries**

Although the systemic arterial system is the primary source of bleeding in hemoptysis [31], bleeding may result from a pulmonary arterial source in approximately 5% of cases [33, 56]. Pulmonary angiography has been advocated in patients with early recurrent hemoptysis following systemic arterial embolization, especially in cases of chronic TB [9, 57]. One study of 306 patients [33] showed that the pulmonary artery was the source of bleeding in 93% of patients in whom immediate control of hemoptysis was not achieved. The underlying diagnosis in these patients included lung abscess, TB, and lung malignancy. Muthuswamy et al. [58] described a patient with active TB who had a normal bronchial angiogram but went on to have emergency pneumonectomy for persistent hemoptysis. The resected specimen revealed a fistula from a pulmonary artery to an adjacent bronchus, which may have been identified on pulmonary angiography had it been performed.

The most common cause of bleeding from the pulmonary circulation is Rasmussen’s aneurysm, which is a pseudoaneurysm due to erosion of a peripheral pulmonary artery by chronic inflammation, such as chronic cavitary TB. These can be identified on contrast-enhanced CT images as avidly enhancing nodules within walls of tuberculous cavities [59]. Their incidence varies between 4 and 11% in the literature [56, 57, 60]. In a series of 1114 autopsies of patients with chronic pulmonary TB, 45 cases (4%) of such aneurysms were found [60]. In 38 of these cases, aneurysm rupture was the immediate cause of death. A prospective study of 72 patients presenting with hemoptysis was carried out to determine the incidence of pulmonary arterial source of hemorrhage [56]. These patients underwent both bronchial and pulmonary angiography, which revealed pulmonary artery pseudoaneurysms in five cases (6.9%). Three of these patients had cavitary TB, one of which was complicated by an aspergilloma. This patient was treated with embolization of branches of the right pulmonary artery without further rebleeding.

A recent study observed that 8 (10.5%) in a series of 76 patients undergoing bronchial angiography for hemoptysis had visible pulmonary artery pseudoaneurysms [57]. Five of these patients had a history of TB, three with cavitary disease and two with active TB. Although the majority of pseudoaneurysms were easily detected on bronchial and nonbronchial arterial angiograms due to bronchial-pulmonary arterial shunt and complete reversal of flow in pulmonary artery branches, they were only seen on subsequent pulmonary angiography in two of the eight patients. Both patients initially underwent BAE and continued to rebleed until pulmonary angiography was performed and the underlying pseudoaneurysm successfully embolized. The conclusion from all these studies is that pulmonary angiography should be performed in any patient who continues to bleed or has early recurrent bleeding, after what is believed to have been adequate BAE or in the presence of normal bronchial arteries.

More uncommonly, hemoptysis may occur following rupture of a pulmonary AVM, which is an abnormal communication between pulmonary arterial and pulmonary venous circulations resulting in a right-to-left shunt. The majority are congenital and occur in patients with hereditary hemorrhagic telangiectasia [61]. Embolization of the feeding pulmonary artery with stainless-steel coils and detachable balloons has demonstrated high success rates, with permanent involution in the majority of treated cases [62–64].

**Bronchial Artery Embolization: Angiographic Technique**

BAE should be carried out in a dedicated vascular interventional suite equipped with digital subtraction technology, by experienced interventional radiologists familiar with embolization techniques. Rapid acquisition and review of images with good contrast resolution are
necessary during embolization procedures. Prior to embolization, a preliminary descending thoracic aortogram is carried out in order to demonstrate bronchial artery anatomy and to identify other systemic collateral vessels (Fig. 7). The majority of abnormal, hypertrophied bronchial arteries are visualised on this initial flush aortogram [9, 11, 65].

Selective catheterization of bronchial arteries should be attempted even in cases with an apparent normal aortogram, as bleeding may occur from normal diameter vessels [9]. Although Cobra catheters are most commonly used, a variety of shaped catheters should be readily available for optimal selective arterial catheterization. These may include Simmons, Shepherd’s hook, Headhunter, Sidewinder, and Sos-Omni catheters. Coaxial microcatheters allow superselective catheterization in cases where a secure catheter position cannot be achieved with a conventional catheter. This is of particular importance when catheterizing the bronchial branch of the right ICBT, in order to avoid occlusion of the intercostal branch that can occasionally give off the anterior medullary artery [9, 11].

Opacification of bronchial arteries during selective angiography is achieved by hand injection of nonionic contrast medium. The rate and volume of injection are dependent on the size of the bronchial artery and concurrent acquisition images. Angiographic findings in hemoptysis include hypertrophic and tortuous bronchial arteries, areas of hypervascularity and neovascularity, shunting of blood into pulmonary artery or vein, and bronchial artery aneurysm (Figs. 8, 9, 10). Although extravasation of contrast medium is a specific sign of active bronchial bleeding, it is an uncommon finding, with a reported prevalence ranging between 3.6 and 10.7% [17, 36]. It is important to note that although bronchial circulation is the source of hemoptysis in the majority of cases, bleeding may occur from nonbronchial systemic arteries as well as the pulmonary arteries [11]. Therefore, if
no abnormal bronchial arteries are identified, nonbronchial systemic arteries should be scrutinized including the intercostal, subclavian, and inferior phrenic arteries, depending on the known site of pulmonary disease.

**Bronchial Artery Embolization: Embolic Material**

The aim of embolization is to reduce the perfusion pressure to fragile vessels in pathological areas of lung by occluding the systemic arterial inflow. It is important to embolize as close to the site of the abnormal bronchopulmonary anastomoses as possible, in order to prevent their recurrence from nonbronchial systemic collateral vessels. A number of embolic materials are available for BAE. It is important to avoid embolic material that can pass through abnormal bronchopulmonary anastomoses, as there is a risk of pulmonary infarction via bronchial artery-pulmonary artery shunts or systemic artery embolization via bronchial artery-pulmonary vein shunts. Bronchopulmonary anastomoses of up to 325 μm have been demonstrated in an anatomical study [5] and embolic materials less than this diameter should not be used in BAE.

Absorbable gelatin sponge is readily available and easy to use. However, it can be resorbed by the body, resulting in recanalization and recurrent bleeding [35, 66]. Polyvinyl alcohol (PVA) is a nonabsorbable particulate agent available in a variety of particulate sizes, with 350- to 500-μm-diameter particles the most frequently used worldwide [67]. More recently, gelatin cross-linked particles called tris-acryl microspheres have been used in BAE [68]. These are of a more uniform diameter than PVA particles, have a hydrophilic coating, which prevents clumping inside the catheter lumen, and are especially useful with microcatheters. Metal coils are generally avoided because they tend to occlude more proximal vessels and preclude further embolization if bleeding recurs. However, they are used to occlude pulmonary artery aneurysms and pulmonary AVMs. Other agents which have been used include glue (n-butyl-2-cyanoacrylate) [69].

**Outcomes of Bronchial Artery Embolization**

Transcatheter embolization of bronchial and nonbronchial systemic arteries is now a well-established procedure for the control of massive and recurrent hemoptysis in various pulmonary diseases [4, 31–39, 70, 71]. Immediate control of hemoptysis is achieved in 73–99% of treated patients. However, recurrent hemoptysis is not uncommon, occurring in 10–55.3% [4, 31–39, 70–72]. Table 2 compares outcomes of BAE in previous reports. There has been some improvement in the immediate success rates, probably secondary to refinement of angiographic and embolization techniques, but there has been no significant change in the overall recurrence rates since the 1970s. This is unsurprising, as BAE is essentially a palliative procedure for symptomatic control of hemoptysis, which does not address the underlying disease process.
Early recurrent bleeding, within the first weeks and months of embolization, is caused by incomplete embolization of the abnormal vessels, which may be due to the extensive nature of the underlying disease or incomplete search for all abnormal vessels. Several studies have reported rebleeding within the first month of embolization in 10%–29% of patients [33, 36–38, 70]. Late rebleeding following embolization occurs due to recanalization of previously embolized vessels or revascularization of collateral circulation secondary to progression of the underlying pulmonary disease [33, 35, 37, 50, 70]. It is therefore important to identify and embolize all vessels that may be contributing to the abnormal blood supply, including any nonbronchial systemic or pulmonary arteries. The underlying pathology should be treated if possible, in order to achieve long-term hemoptysis control.

Long-term hemoptysis control can be improved with repeat BAE, which has been shown to improve the outcome of those with recurrent bleeding by embolizing previously overlooked feeder vessels. However, if the underlying disease process continues, revascularization results in further rebleeding after a period of 2–5 years [50, 70].

Several studies have noted varying outcomes according to the underlying cause. Favorable outcomes have been reported in patients with active TB where concurrent management with embolization and antituberculous therapy has resulted in high immediate success rates and low recurrence rates [50, 70]. Poor outcomes have been observed in patients with aspergillosis [34, 47, 70, 73, 74], where the underlying disease process is known to be aggressive and extensive, often involving nonbronchial arteries. A recent study showed a recurrence rate of 100% in these patients, the majority of which occurred within the first 2 weeks of BAE, and a mortality rate of 50% within the first month [70]. Aspergillosis has been shown to be a statistically significant risk factor for development of recurrent hemoptysis [70, 73] and these patients should be managed aggressively with a combination of repeat embolization and elective surgery. Pulmonary malignancy has also been reported to show poor immediate and long-term outcomes and is associated with a high mortality due to the progressive nature of the disease [35, 38].

### Complications of Bronchial Artery Embolization

Bronchial arteries not only supply the bronchi and vasa vasorum of the aorta and pulmonary vessels, but also contribute to the esophagus, diaphragmatic and mediastinal visceral pleura, and spinal cord. Reported complications result from inadvertent occlusion of these branches. The most common side effects are chest pain and dysphagia, reported in 24–91% and 1–18%, respectively [36, 75, 76]. These symptoms are usually transient and are likely to represent occlusion of intercostal and esophageal vessels. The most serious complication of BAE is transverse myelitis due to spinal cord ischemia. Although the majority of cases of spinal cord damage are thought to be related to the toxic effects of nonionic contrast medium during the early years, the reported prevalence ranges between 1.4 and 6.5% [36, 37, 66, 77] in more recent studies. Superselective embolization has been shown to reduce the risk of inadvertent embolization of the anterior medullary artery by embolization of more terminal branches of the bronchial artery beyond the origin of the spinal arteries [66]. Other rare reported complications include aortic and bronchial necrosis, non-target-organ embolization causing ischemic colitis, pulmonary infarct, bronchoesophageal fistula, and transient cortical blindness [78–82].

### Conclusions

Hemoptysis can present as a life-threatening respiratory emergency and warrants urgent investigation. Initial assessment with chest radiography, bronchoscopy, and CT is useful in localizing the bleeding site and diagnosing the underlying cause. MDCTA allows rapid and detailed assessment of the lung parenchyma and thoracic vasculature. It is possible to delineate abnormal bronchial and

<table>
<thead>
<tr>
<th>Study</th>
<th>Year of study</th>
<th>No. of patients</th>
<th>Immediate control, n</th>
<th>Recurrence, n</th>
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<tr>
<td>Remy et al. [31]</td>
<td>1977</td>
<td>49</td>
<td>41 (84%)</td>
<td>14 (28.6%)</td>
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<tr>
<td>Rabkin et al. [33]</td>
<td>1987</td>
<td>306</td>
<td>278 (90.8%)</td>
<td>103 (33.7%)</td>
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<td>Hayakawa et al. [35]</td>
<td>1992</td>
<td>58</td>
<td>50 (86.2%)</td>
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<td>Ramakantan et al. [36]</td>
<td>1996</td>
<td>140</td>
<td>102 (73%)</td>
<td>38 (27.1%)</td>
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<td>Mal et al. [37]</td>
<td>1999</td>
<td>56</td>
<td>43 (77%)</td>
<td>31 (55.3%)</td>
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<td>Swanson et al. [38]</td>
<td>2002</td>
<td>54</td>
<td>51 (94%)</td>
<td>13 (24.1%)</td>
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<td>Poyani et al. [39]</td>
<td>2007</td>
<td>140</td>
<td>138 (98.5%)</td>
<td>14 (10%)</td>
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<td>Lee et al. [71]</td>
<td>2008</td>
<td>70</td>
<td>69 (99%)</td>
<td>25 (26%)</td>
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<tr>
<td>Chun et al. [70]</td>
<td>2009</td>
<td>50</td>
<td>43 (86%)</td>
<td>14 (28%)</td>
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References