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Case Presentation

A 61 year old man presented to the emergency department reportedly vomiting blood. He was unable to speak due to blood flooding his oropharynx and severe respiratory distress. The airway was secured with tracheal intubation. Gastric lavage showed no active bleeding. The patient then coughed and filled the ventilator circuit with bright red blood. Laboratory testing revealed hemoglobin of 8 mg/dl, platelet count $260 \times 10^3/\mu\text{l}$, INR of 1.1 and PTT of 35 s. Chest X-ray revealed an area suspicious for bronchiectasis in the Left Lower Lobe (LLL) and infiltrates in the Right Lower Lobe (RLL) and Right Upper Lobe (RUL) (Fig. 26.1).

The patient was transferred to intensive care. Flexible bronchoscopy showed no active bleeding but blood was seen in the superior segment of the RLL. Another episode of hemoptysis esti-

mated at 500 ml occurred. The patient was positioned with his right side down and aggressively suctioned through the endotracheal tube. Selective intubation of the left mainstem bronchus was unsuccessful because of poor visibility due to blood in the airway and endotracheal tube.

Multi-detector computed tomography with contrast demonstrated right lower lobe consolidation and associated atelectasis. There was narrowing of the right upper lobe bronchus by soft tissue density (Fig. 26.2).

Bronchial angiography was undertaken. The right and left bronchial arteries both arose from a single trunk. The left bronchial artery was relatively small with no abnormal vessels. On the right, the vessel divided into a branch to the upper lobe and a major lower lobe branch which was moderately enlarged with hyperemia in the lower lobe, associated with the area of consolidated lung. The right lower lobe bronchial artery was embolized and post embolization no flow in the right lower lobe bronchial artery was observed (Fig. 26.3).

Soon thereafter, there was another episode of massive hemoptysis, which filled the ventilator circuit. This was associated with vasopressor-dependent hypotension, worsening hypoxemia and bradycardia.

A dual lumen endotracheal tube was placed, and both right and left sides were ventilated. The patient became more stable with decreasing vasopressor need. A right lower lobectomy was performed.

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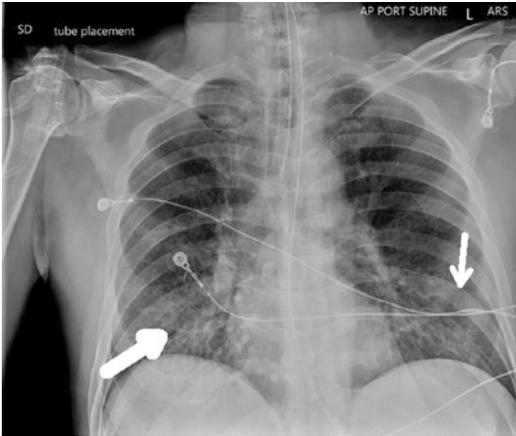


Fig. 26.1 Arrows demonstrate Right lower lobe consolidation (*wide arrow*) and Left Lower Lobe bronchiectasis (*thin arrow*)

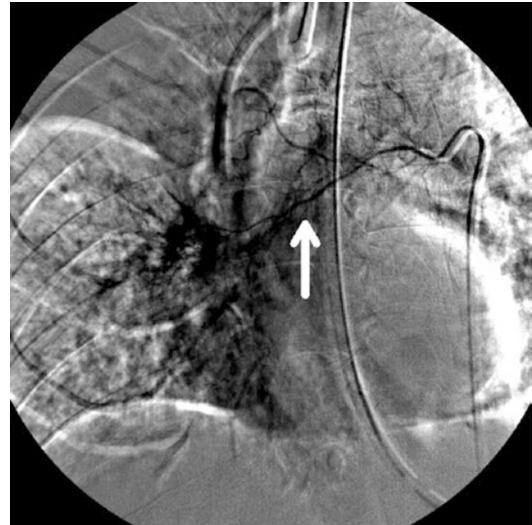


Fig. 26.3 Right Bronchial artery arteriogram demonstrates both upper lobe and lower lobe branch (*arrow*)

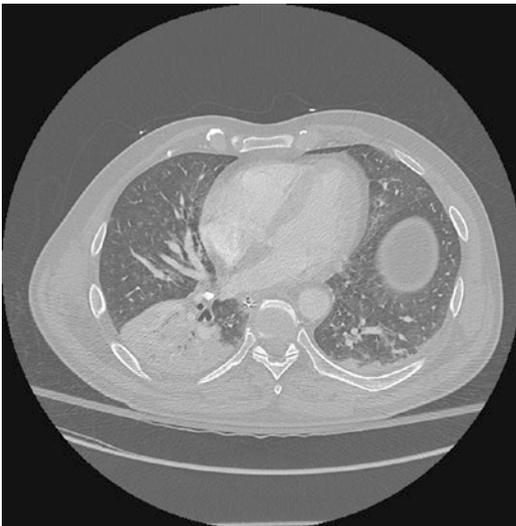


Fig. 26.2 Computed tomography image demonstrates dense right lower lobe infiltrate with broncholithiasis

likely reflects the lack of a standard definition of massive hemoptysis as well as the variability in underlying etiology [3–5].

A scoring system to identify risk for mortality was derived from a 14 year experience of 1,087 patients admitted for hemoptysis, 717 of whom were admitted to the Intensive Care Unit (ICU), and 10% of whom demonstrated massive hemoptysis with a mean cumulative volume of >200 ml. Risk factors include chronic alcoholism, ≥ 2 chest x-ray quadrants involved, pulmonary artery involvement, cancer, aspergillus infection or mycetoma, and initial mechanical ventilation. Two or more risk factors demonstrated a higher mortality [2]. (These risk factors are noted in the clinical algorithm, Fig. 26.4).

Principles of Management

Epidemiology

Massive hemoptysis occurs in about 5–15% of all admissions for hemoptysis [1, 2]. Mortality was 7% in a single center retrospective review of 1,087 patients [2] but has been reported as high as 75% when the volume of hemoptysis exceeds 600 ml in 24 h. The wide range of mortality

Sources of Hemorrhage

Hemoptysis, including massive hemoptysis, usually originates from pathology that involves the high pressure bronchial arterial supply (90%) [6, 7]. The bronchial artery or arteries originate from the descending thoracic aorta (DTA) at thoracic vertebral level T3–8 most often T5–6. While the most common pattern is a right and left bronchial artery arising from the DTA, variability is extensive, and commonly involves a right bronchial

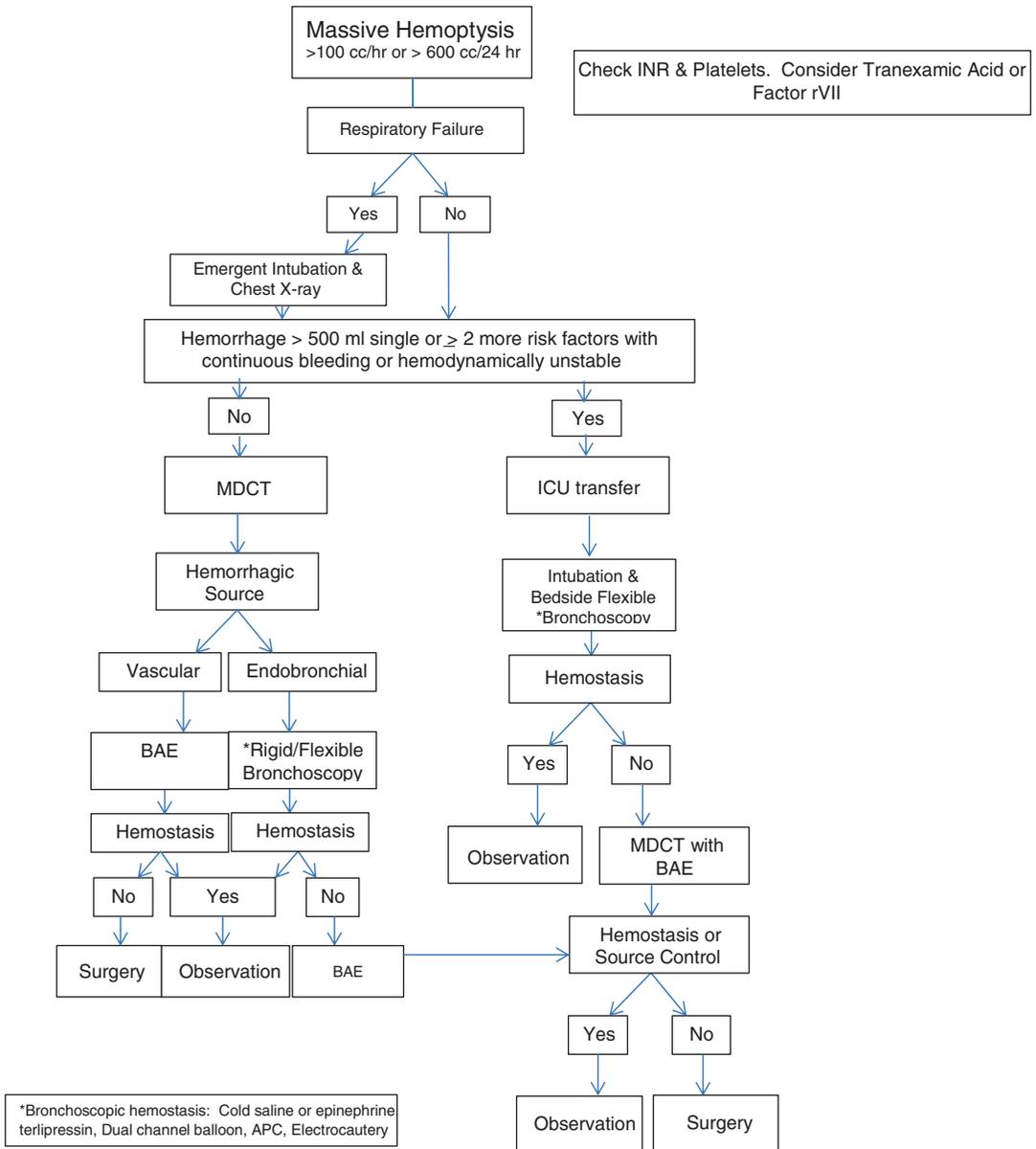


Fig. 26.4 Clinical algorithm to massive hemoptysis

artery arising from an intercostal artery (also-called intercostobronchial trunk) or 2 right bronchial arteries arising from the DTA and/or intercostobronchial trunk. Left bronchial arteries can also arise from intercostobronchial trunks [7]. The spinal artery of Adamkiewicz supplies the distal thoracic and lumbar spinal cord and originates from an intercostal artery at thoracic vertebral level T9-12 in 75% of patients.

However, it infrequently originates between T5-T8 and may be occluded during embolization of the bronchial artery resulting in spinal cord ischemia or infarction [8].

The pulmonary artery infrequently is the source of hemoptysis. Infection, malignant disease, traumatic injuries (e.g. Swan Ganz catheter balloon rupture of PA) and arteriovenous malformations are the most common causes for pulmonary arterial

bleeding [6, 9–11]. Rarely hemoptysis may involve non-bronchial or pulmonary arteries. Non-bronchopulmonary arteries may enter the lung via transpleural adhesions as a result of chronic inflammation or congenital anomalies and may anastomose with the pulmonary artery circulation [9, 10]. Other potential nonbronchial arterial sources include the aorta (ruptured aneurysm), intercostal arteries, coronary, thyrocervical, pericardiodiaphragmatic, musculophrenic, and inferior phrenic arteries. Diffuse alveolar hemorrhage syndromes are rarely a cause of massive hemoptysis.

Diseases Associated with Massive Hemoptysis

Many diseases have been associated with massive hemoptysis and vary by geographic region. Tuberculosis continues to be a major cause of massive hemoptysis world-wide. Bronchiectasis, malignancy, trauma, pneumonia, and mycetoma are the most causes of massive hemoptysis in the USA [12, 13] (Table 26.1).

The etiology for hemoptysis will direct treatment decisions. Systemic diseases such as vasculitis will require anti-inflammatory or immunosuppression whereas coagulopathies will require reversal of coagulation abnormalities [13–19]. Focal lung or bronchial conditions causing massive hemoptysis may require either bronchoscopic intervention or

bronchial artery embolization (BAE) as a first approach.

Imaging

Plain radiographs will not permit localization of bleeding site in upwards of 35 % of patients [20]. Multidetector CT angiography is capable of ascertaining the site of bleeding and/or suggesting a diagnosis in more than 80 % of patients and the yield may be higher with the use of reformatted image techniques [21]. Further, CT angiography is helpful to guide bronchial artery embolization [6, 7, 9, 20].

An Approach to Managing Life Threatening Hemoptysis (Algorithm, Fig. 26.4)

Several algorithms have been developed to provide guidance for an approach to severe or massive hemoptysis and most agree that an initial multidisciplinary approach is indicated with immediate evaluation by pulmonology, interventional radiology and thoracic surgery [22–25]. We propose the following:

The initial survey should include a simple “ABC” assessment: Airway, Blood loss, and Clinical condition.

Table 26.1 Causes of massive hemoptysis

Infectious	Mycobacterial (TB), Fungal (mycetoma), Necrotizing Bacterial Pneumonia (Klebsiella, Streptococcus, Staphylococcus, Actinomyces)
Neoplastic	Bronchogenic, Renal cell, melanoma, carcinoid, adenocystic, etc.
Vascular	Bronchial artery aneurysm, Pulmonary artery aneurysm, Cardiac and Pulmonary Malformations, Arteriovenous malformations, Airway vascular fistula, Dieulofoy’s Disease, etc.
Pulmonary	Bronchiectasis, Broncholithiasis, Diffuse alveolar hemorrhage, Cryptogenic, Inhalation: crackcocaine, etc.
Diffuse Alveolar Hemorrhage Syndromes	Granulomatosis with Polyangitis, Microscopic polyangitis, Anti-GBM (Goodpastures), Isolated pulmonary capillaritis, SLE, Eosinophilic granulomatosis with polyangitis, Antiphospholipid antibody, toxin and drug induced lung injury, IgA deposition vasculitis, etc.
Trauma	High dose brachytherapy, Pulmonary artery rupture, Biopsy and Trauma
Hematologic	Coagulopathy and Platelet disorders

Airway: Does the volume of bleeding into the airway require emergent airway management?

Blood: Has there been a single bleeding event of large volume >100 cc or is the bleeding recurrent? Is the bleeding in excess of 100 per hour for >2 h?

Clinical Condition: Is there hemodynamic instability (signs of hypoperfusion, tachycardia, or hypotension)?

The decision regarding immediate airway management should be determined. Adequate intravenous access should be established with at least two large bore IV lines. Fluid resuscitation should be initiated, if indicated. A complete blood count, testing for coagulopathies, and cross match for transfusion should be obtained. In addition, perform electrocardiogram and obtain cardiac biomarkers.

Intubation: Options are dual lumen or single lumen endotracheal tube with the intent to protect the lung on the non-bleeding side. Single lumen endotracheal intubation is preferred when airway visibility is poor (see discussion of dual lumen endotracheal tubes below).

Position: The patient should be positioned with the presumed side of bleeding dependent (lateral decubitus) to reduce the likelihood of flooding of the unaffected lung with blood. However, lateral decubitus position with the bleeding side down may worsen hypoxemia (due to increased perfusion to poorly aerated lung) [26].

Assessment

- **Active Hemorrhage and unstable:** If transport is not considered safe, the initial evaluation to localize and treat would be with rigid and/or flexible bronchoscope. This may allow therapeutic endoscopic intervention (Cold Saline/epinephrine [EPI], anti-diuretic hormone [ADH], argon plasma coagulation [APC], tamponade endobronchial blocker). (Table 26.2) [27, 30–33].
- **Active Hemorrhage and stable:** In this setting bronchoscopy is limited due to poor visibility

and proceeding to radiologic investigation maybe more informative.

- **Non-active hemorrhage and the unstable patient:** ICU setting to stabilize the patient with possible bronchoscopic approach for localization and treatment.
- **Non-active hemorrhage and the stable patient:** Multidetector Computed Tomography (MDCT) and 3D volume averaging provides the best detail for localization and selection of treatment options [11, 21, 23] (Table 26.2).

No localization of a culprit lesion via bronchoscopy is an indication for immediate multi-detector CT. This may indicate that embolization is needed.

If hemostasis cannot be achieved then urgent resectional surgery [34, 35] should be considered.

Evidence Contour

Rigid Versus Flexible Bronchoscopy

In massive hemoptysis, rigid bronchoscopy has several advantages: the large inner diameter and more effective suctioning capability improve clearance of blood from airways (especially in voluminous flooding); the ability to ventilate; and ancillary devices that are not available with flexible bronchoscopy e.g. laser. Disadvantages include the lack of availability and/or expertise for rigid bronchoscopy [24]. Flexible bronchoscopy has the advantages of ease of access, and wide availability of skilled individuals plus it may allow specialized techniques to control airway bleeding (i.e. APC, cryotherapy, electrocautery).

Dual Lumen Versus Unilateral Airway Intubation: Benefits and Limitations

Consideration should be given to placing a dual lumen endotracheal tube to isolate the side of hemorrhage and protect the contralateral lung from blood. Dual-lumen endotracheal tubes have various designs but all have two lumens,

Table 26.2 Bronchoscopic and systemic treatments for massive hemoptysis

Treatment	Mechanism of action	Procedure	Complications
Cold Saline	Vasoconstriction with localized thrombosis	50 cc aliquots at 4 °C	Bradycardia [22, 27]
Topical Epinephrine (Epi), terlipressin or oripressin	Epi, Terlipressin (5iu), Ornipressin (0.5 mg), vasoconstriction	1:20000 Epi in cold saline	Tachyarrhythmias [27]
Balloon Tamponade	Tamponade	Double lumen occluding balloon with saline	Post-obstructive Pneumonia
Fibrinogen –thrombin	Fibrinogen, fibronectin, factor III, plasminogen and aprotinin/thrombin		[28, 29]
Argon Plasma Coagulation	High frequency electric current with dessication of surface	Direct visualization	Gas embolism [30–32]
Tranexamic Acid	<i>Endobronchial or Systemic.</i> antifibrinolytic	Endobronchial 500–1,000 mg or oral for chronic bleed	Clotting disorders. i.e. PE [17–19]
Factor rVII	<i>Systemic:</i> coagulation in tissue damage. Activates IX then Xa then fibrin thrombin	60–90 ug/kg	CVA [14–16]

each of which can be ventilated, permitting both isolation of each lung and instillation of vasoactive agents or saline into the airway. Disadvantages: the lumens are small, potentially limiting flexible bronchoscopy and the removal of blood and secretions. During patient transport or turning, dual-lumen tubes can easily become displaced. Placement of dual lumen ET tubes can be difficult and requires considerable expertise and the ability to visualize the airway. If visualization of the airway is limited due to airway flooding then a single lumen ET tube may be directed into the main bronchus of the unaffected lung.

One Transport or Two?

The unpredictability of repeat hemorrhage heightens the need to quickly identify location and cause. In the stable patient (per Algorithm), multidetector CT with 3D volume rendering is the first choice. MDCT allows a noninvasive approach to arterial and pulmonary anatomy for potential identification of both the site and cause of hemorrhage in 77% and 73% of patients,

respectively [20]. The stable patient could transfer to radiology for diagnostic CT followed by Interventional Radiology (IR) for possible BAE if a significant vascular pathology is identified (control of hemorrhage in 66–90%). In the unstable patient, ICU transfer and immediate bedside bronchoscopy with control of the source when possible and supportive care [36] has been evaluated and is considered standard of care. ICU care limits diagnostic testing to bedside bronchoscopy and portable chest x-ray. Bronchoscopy may localize disease but frequently does not achieve hemostasis (up to 60%) and a second transport to IR for BAE is required [37].

Recurrent Bleeding; Repeat Embolization or Surgery?

Recurrent bleeding or persistent bleeding occurs in 5–44% of patients after initial BAE. For recurrent hemoptysis, a second BAE may be effective and avoid surgery; surgery for massive hemoptysis has a high risk of morbidity (25%) and mortality (7–40%) [38]. Generally, second attempt at embolization is favored if the anatomy

suggests an amenable lesion but the uniqueness of each patient and pathology requires multidisciplinary decision making [37].

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