Interventional management options for patients in the pulmonary critical care setting

Poster No.: C-2350
Congress: ECR 2011
Type: Educational Exhibit
Keywords: Interventional non-vascular, Interventional vascular, Lung, Catheter arteriography, Catheter venography, Fluoroscopy, Embolisation, Thrombolysis, Stents, Arteriovenous malformations, Embolism / Thrombosis, Hemorrhage
DOI: 10.1594/ecr2011/C-2350

Any information contained in this pdf file is automatically generated from digital material submitted to EPOS by third parties in the form of scientific presentations. References to any names, marks, products, or services of third parties or hypertext links to third-party sites or information are provided solely as a convenience to you and do not in any way constitute or imply ECR's endorsement, sponsorship or recommendation of the third party, information, product or service. ECR is not responsible for the content of these pages and does not make any representations regarding the content or accuracy of material in this file.

As per copyright regulations, any unauthorised use of the material or parts thereof as well as commercial reproduction or multiple distribution by any traditional or electronically based reproduction/publication method is strictly prohibited.

You agree to defend, indemnify, and hold ECR harmless from and against any and all claims, damages, costs, and expenses, including attorneys' fees, arising from or related to your use of these pages.

Please note: Links to movies, ppt slideshows and any other multimedia files are not available in the pdf version of presentations.

www.myESR.org
Learning objectives

The goal of this exhibit is to:

• Discuss bronchial artery embolization (BAE) in the setting of hemoptysis.
• Discuss various techniques for the diagnosis and management of pulmonary arteriovenous malformations (PAVM).
• Discuss interventional management of SVC syndrome.
• Discuss pulmonary artery thrombectomy and thrombolysis for the treatment of pulmonary embolism (PE).

Background

• The contribution of Interventional Radiology (IR) to the management of patients in the pulmonary critical care setting has received growing attention in recent years.
• The increasing complexity and the needs of these patients as well as the diverse options available in modern IR has evolved.
• IR plays a more significant role in pulmonary critical care performing a variety of therapeutic image-guided procedures.

Imaging findings OR Procedure details

Bronchial Artery Embolization

Bronchial Arterial Anatomy

• The bronchial arteries provide a much smaller portion of the total pulmonary blood flow compared to pulmonary arteries.
• They are generally important in diseases that cause their enlargement such as cystic fibrosis and chronic lung disease.
• In such cases, erosion of these dilated bronchial arteries by the disease process will produce hemoptysis that is often life threatening.
• Bronchial artery anatomy is very variable.
• Conventional origins: T5-T6 level, anterolateral aspect of descending aorta.
• Classified into four types.
• Type 1: Two left and one right bronchial artery. This is the most common pattern. The left bronchial arteries usually arise from the anterolateral
surface of the thoracic aorta below the ligamentum arteriosum, while the right bronchial artery usually arises from a common trunk with an intercostals artery (intercostal-bronchial trunk).

- **Type 2**: Bilateral single bronchial arteries.
- **Type 3**: Two left and two right bronchial arteries.
- **Type 4**: Single left and two right bronchial arteries (type 4).
- **Variant origins**:
  - Aortic arch
  - Brachiocephalic artery
  - Subclavian artery
  - Internal mammary artery
  - Thyrocervical trunk
  - Costocervical trunk
  - Abdominal aorta
  - Inferior phrenic artery
  - It is important to emphasize that, in about 5% of the population, the right intercosto-bronchial trunk contributes to or arises with the artery of Adamkiewicz which supplies the anterior spinal artery, and needs careful consideration when performing bronchial artery embolization.

**Pathophysiology**

- Blood supply to the lungs:
  - Low pressure and low resistance pulmonary arteries (99%). They supply the pulmonary capillary bed.
  - High-pressure and high resistance bronchial arteries (1%). They supply the airways, visceral pleura, lymph nodes and mediastinum.
  - There are normally no anastamoses between both circulations but any pathological process that creates obstruction, compression, or destruction of the pulmonary capillary bed may induce a compensatory development of these anastamoses.
  - These anastamoses induce hyperplasia and tortuousity of the bronchial arteries which become fragile and prone to rupture in the presence of a pathological lesion, causing hemoptysis.

**Hemoptysis**

- Can be defined as the expectoration of blood or blood-stained sputum from the lungs or airways (bronchi, larynx, and trachea).
- **Massive hemoptysis**: expectoration of 300-600ml of blood in a period of 24 hours or less.
- **Moderate hemoptysis**: Three or more episodes of 100 ml or more in a week.
- **Mild hemoptysis**: Chronic or slowly increasing episodes.
  - Massive hemoptysis is a medical emergency since mortality rate can be as high as 75%.
  - The main source of hemoptysis is the bronchial artery in 90% of cases.
• The main cause of the death is due to asphyxiation because of the obstruction of the airway by the expectorant, so any amount of hemoptysis is significant if it compromises the airway and/or ventilations of both of the lungs.

Etiology

• **Most common causes of hemoptysis:**
  - Cystic fibrosis
  - Bronchiectasis
  - Tuberculosis
  - Bronchogenic carcinoma

• **Other causes include:**
  - Idiopathic
  - Invasive aspergillosis
  - Pyogenic abscesses
  - Sarcoidosis nWegner’s granulomatosis
  - Pulmonary AVM
  - Pulmonary artery pseudoaneurysm

Management of Hemoptysis

• **Mild and moderate:**
  - Clinical monitoring in most cases.

• **Massive hemoptysis:**
  - Selective intubation of the non-bleeding lung.
  - Place the patient with the bleeding side dependant.
  - Perform bronchoscopy to:
    - Detect location of bleeding.
    - Perform intrabronchial balloon tamponade, laser coagulation, thrombin injection.
    - Perform bronchial angiogram when the patients condition is stabilized.

Imaging and Workup

• **Chest X-ray:**
  - May help in showing the underlying pathology and side of the lesion. Normal in 20-30% of patients.

• **CT scan:**
  - Shows in detail the underlying pathology in lung.

• **CT angiogram:**
  - Helps locate the source of bleeding (bronchial vs. pulmonary vs. systemic arterial source).

CT Angiogram on page 14 (Fig. 1) on page 14
Technique

- Trans-femoral approach.
- Most common and convenient catheters are Mikaelson, Simmons I and shepherd’s Hook catheters. However, H1, Cobra and RC2 catheters can also be used.
- Angiograms are usually performed through hand injection of 3-10 ml of contrast material which have the advantage of adjusting the pressure and flow of contrast to avoid injury and rupture of the small distal diseased branches.
- Embolization should always be done as distally as possible.
- Careful search of and avoidance of artery of Adamkiewicz, esophageal, tracheal and pericardial branches should be performed.
- Microcatheters are preferably used for controlled delivery of embolic agent.
- Polyvinyl alcohol particles are usually used (300-500 or 500-700 um). Gelfoam pledgets (gelatin sponge) can also be used.
- Embolization is performed slowly under direct fluoroscopy to prevent reflux of the embolic materials into non-target vessels.

Angiographic Anatomy (Fig. 2) on page 15

Angiographic Findings in Hemoptysis

- Hypertrophic tortuous bronchial arteries >3mm in diameter
- Parenchymal hypervascularity
- Bronchial neovascularity
- Bronchial artery pseudoaneurysms
- Bronchial artery to pulmonary artery shunting
- Active extravasation (very rare)

Angiographic Signs (Fig. 3) on page 15

Case 1 (Fig. 4) on page 16

Case 2 (Fig. 5) on page 16

Case 3 (Fig. 6) on page 17

Case 4 (Fig. 7) on page 18

Complications and Prognosis

- Complication such as spinal cord infarction, pulmonary infarction, esophageal and tracheal necrosis are rare.
• Recurrence is common due to persistence of disease in most patients or due to revascularization from systemic arterial supply.
• Bronchial artery embolization may be repeated as needed to control future bleed. Surgical therapy is considered if bronchial artery embolization fails.
• In some patients, bronchial artery embolization acts as a bridge to more definitive surgery to remove the underlying lung segment/pathology.

Pulmonary Arteriovenous Malformations (PAVM)

Definition

• PAVMs are abnormal fistulous communication between pulmonary arteries and veins that bypass the normal capillaries.
• They are high-flow, low-pressure shunts, consisting of a single feeding artery connecting via an aneurysmal sac to a draining vein. The aneurysmal connection is referred to as an aneurysmal sac.

Epidemiology

• **Incidence**: Rare; 3 cases of 15,000 autopsies
• **Gender**: Twice more common in women in the adult population.
• **Age**: Usually present in 4th to 6th decades of life.
• Only 10-15% of PAVM are identified in infancy & childhood.
• PAVMs have a close relationship with hereditary hemorrhagic telangiectasia (HHT).
• Approximately 60-90% of PAVMs are associated with hereditary hemorrhagic telangiectasia (HHT), also known as Rendu-Osler-Weber (ROW) syndrome.
• 15-30% of patients with HHT have PAVM.

Hereditary Hemorrhagic Telangiectasia

• Autosomal dominant disease characterized by telangiectasia of the skin and mucous membranes as well as AVMs and aneurysms of the viscera like brain, lung, bowel and liver.
• Usually presents with epistaxis (>90%), telangiectatic stigmata of the skin and mucous membranes (50-80%) or PAVMs (15-30%).
• Diagnosis of HHT is based on **Curacao** criteria:
• **Epistaxis**: Spontaneous, recurrent nosebleeds.
• **Telangiectases**: Multiple at characteristic sites (lips, oral cavity, fingers, nose).
• **Visceral lesions**: Such as gastrointestinal telangiectasia (with or without bleeding), PAVMs, hepatic AVM, cerebral AVM, spinal AVM.
• **Family History**: First-degree relative with HHT according to these criteria. The HHT diagnosis is classified as definite if 3 criteria are present, possible or suspected if 2 criteria are present, and unlikely if fewer than 2 criteria are present.

**Pathology**

• **Location**:
  • 53-70% in Lower lobes (60-95% in HHT).
  • 75% Unilateral (Bilateral in HHT)
  • 36% Multiple (35-65% in HHT)
  • 81% Involve pleura.
  • 19% Subpleural

• **Size**: Typically 1-5 cm (occasionally may be > 10 cm)

• **Histopathology**: Thinned walled vascular channels lined by endothelial, with scant connective tissue stroma.

• **Classification**:
  • Simple (80-90%): Single feeding segmental artery & single draining vein.
  • Complex (10-20%): 2 or > feeding arteries or draining veins.

**Diagnosis of PAVMs**

• Clinical Presentation
• Symptoms
• Signs Pulmonary Function Tests
• Shunt Fraction Measurement
• Contrast Echocardiography
• Chest Radiography
• Computerized Tomography (CT)
• Pulmonary Angiography

**Clinical Presentation**

• Symptoms and signs are mainly due to:
• Shunting of deoxygenated blood through PAVM.
• Loss of filtration function of the lung.
• Clinical presentation is usually in the 4th-6th decade.
• The most common presentation is epistaxis related to HHT seen in 50% of patients at the age of 20, and 95% of patients at the age of 45.
• Other clinical presentations include; dyspnea, digital clubbing and cyanosis, hemoptysis, skin and mucous membrane telangiectases (face, mouth, chest and upper extremity) GI bleed, anemia, Murmur and bruit over the site of PAVM.
Pulmonary Function Tests

- Most patients have a PaO2 < 80mmHg, and SaO2 < 97% on room air.
- Most patients have orthodeoxia with an average decrease of 6% in SaO2 on standing.
- Orthodeoxia: Decrease in PaO2 or SaO2 when going from recumbent to upright position.

Shunt Fraction Measurement

- Determined by arterial blood gas measurement using 100% oxygen method.
- PaO2 and SaO2 are measured after breathing 100% O2 for 15-20 minutes.
- Normal: <5%
- PAVM patients: >5%, Ranges from 10-40%

Contrast Echocardiography

- Technique: Injection of 5 to 10 ml of indocyanine green or saline (agitated with a small amount of air) into a peripheral vein while simultaneously imaging the right and left atria with 2-D mode.
- Both liquids contain microbubbles which are visualized as contrast.
- In normal patients, the contrast is visualized only in the right atrium and then dissipates when trapped in the pulmonary circulation.
- In intra-cardiac shunts, contrast appears in the left atrium in <1 sec.
- In intra-pulmonary shunts, contrast appears in the left atrium after a delay of 2 to 5 sec (3 to 8 cardiac cycles) which is the time required for the contrast to traverse the pulmonary vasculature.
- The finding of intrapulmonary shunt by contrast echocardiography warrants additional evaluation for PAVM, usually with standard pulmonary angiography.

Chest Radiography in PAVM (Fig. 8) on page 19

- Abnormal in 98% of patients.
- Round or oval mass of uniform density.
- Frequently lobulated but sharply defined.
- More commonly in the lower lobes.

Computerized Tomography in PAVM (Fig. 9) on page 20

- Nodular or rounded enhancing density of variable size, with both afferent and efferent vessels.
- Multi-detector capability reduces breath holding time.
- CT is more sensitive (98%) than pulmonary angiography (60%) in detecting PAVM.
- Pulmonary angiography is better in determining the angioarchitecture of individual PAVM than CT.
• CT may show small or thrombosed PAVMs which may be missed by pulmonary angiography.
• Pulmonary venous varices may occasionally be mistaken for PAVM on CT, and can be differentiated on CT by absence of afferent artery, or by performing pulmonary angiography.

**Pulmonary Angiography in PAVM (Fig. 10) on page 21**

• Pulmonary angiography is no longer used to primarily diagnose PAVM, and it is more commonly performed during transcatheter embolotherapy.
• Baseline right heart & pulmonary artery pressure should be measured.
• Super-selective angiograms can accurately define angioarchitecture of PAVMs (simple or complex) and show early venous drainage.
• PAVMs are supplied by pulmonary artery in 95% of cases.
• Type and size of the occluding device to be used for embolotherapy can be determined.

**Complications of PAVM (Fig. 11) on page 21**

• Migraine headache (43%)
• Transient ischemic attack (37%)
• Cerebrovascular accident (18%)
• Brain abscess (9%)
• Seizure (8%)
• Hypoxemia/orthodeoxia
• Hemothorax
• Life-threatening hemoptysis
• Pulmonary hypertension
• Congestive heart failure
• Polycythemia
• Anemia
• Infectious endocarditis

**Treatment of PAVMs**

• **Indications for treatment:**
  • Symptomatic PAVM.
  • Progressive enlargement.
  • Paradoxical embolization.
  • PAVM >2cm.
  • Feeding artery >3mm.
• **Modalities of treatment:**
  • Surgery: ligation, segmentectomy, lobectomy or pneumonectomy.
  • Percutaneous Transcatheter embolotherapy.

**Coil embolization for PAVM (Fig. 12) on page 22**
• **Advantages of coils:**
  - Low cost compared to other embolic devices (although detachable coils are more expensive).
  - Easy to deploy through diagnostic catheters or microcatheters.
• **Disadvantages of coils:**
  - Paradoxical coil embolization (2%-4%). Minimized by using anchoring technique (see figure).
  - Multiple coils must be used in most cases.
  - Increased number of angiographic runs.
  - Artifacts on follow up CT chest hindering evaluation of recanalization.

**Hydrocoils**

- Hydrocoils are fabricated from a platinum metallic core surrounded by a layer of hydrogel polymer.
- When the coil comes in contact with blood, the hydrogel polymer expands several times of its original diameter.
- This increases volumetric filling of the arterial lumen allowing less dependence on clot formation.
- Theoretically this reduces the chance of recanalization
- Their cost is higher compared to other embolic agents.

**Amplatzer Vascular Plug (AVP) treatment of PAVM (Fig. 13) on page 23**

- **Advantages of AVP device:**
  - AVP can be more precisely placed within the feeding artery.
  - Its position can be easily verified with a test injection through the guiding catheter or sheath.
  - The device can be repositioned or retrieved before release if its position was unsatisfactory.
  - There is no risk of device migration if adequate oversizing was achieved (30-50%).
  - Occlusion time is short compared to coils (average 3-4 minutes).
  - Only one device is required in most cases.
  - No significant artifacts on CT making assessment of recanalization feasible by CT chest.
- **Disadvantages of AVP device:**
  - Should be delivered through a sheath (4-7F) or guiding catheter (5-8F).
  - Relatively stiff delivery cable makes delivery slightly difficult in tortuous anatomy.

**Algorithm for Management of PAVMs (Fig. 14) on page 23**

**SVC syndrome**
Causes Of SVC syndrome

- Malignant: Most common (60%)
- Metastatic
- Lung
- Lymphomas
- Mediastinal tumors (e.g. Thymoma)
- Pleural tumors (Mesothelioma)
- Primary tumors
- Leiomyosarcoma
- Angiosarcoma
- Benign:
  - Iatrogenic
  - Catheter induced. Incidence is rapidly increasing
- Fibrosing mediastinitis
- Histoplasmosis, tuberculosis and radiation therapy
- Behcet
- Due to associated fibrosing mediastinitis or thrombophlebitis

Clinical Picture

- Asymptomatic
- If partial obstruction develops gradually.
- Facial flushing and erythema.
- Swelling of the head, face and neck.
- Dilated and engorged upper limb and thoraco-abdominal cutaneous veins.
- Severe obstruction
- Difficulty in lying down and breathing
- Disturbed sleep
- Neurological signs

Imaging Features of SVC Syndrome:

Collateral Pathways (Fig. 15) on page 24

- Azygos - hemiazygos -accessory hemiazygos: (Fig. 16) on page 25
  - Obstruction Above the Azygos Arch: Antegrade flow from azygos to right atrium.
  - Obstruction below the Azygos arch: Retrograde flow toward the inferior vena cava.
- Vertebral and sub-scapular plexuses: (Fig. 17) on page 26
  - Paravertebral, intervertebral, and epidural veins via azygos and/or ascending lumber veins.
- Mediastinal, esophageal & diaphragmatic plexus:
  - Mediastinal, pericardial and pericardiophrenic veins, drain to IVC, either through inferior phrenic or transhepatic collaterals
• **Lateral thoracic & superficial thoracoabdominal plexus:**
  • Internal thoracic and lateral thoracic veins communicate with thoracoepigastric and superficial epigastric veins.
• **Abdominal venous (Fig. 18) on page 26**
  • Superficial epigastric and left portal vein (hot spot). Inferior phrenic veins and subcapsular hepatic veins
• **Systemic-pulmonary**
  • Innominate veins and superior pulmonary veins via bronchial venous plexus.

**Treatment**

• Initial management focuses on supportive care
• Head elevation
• Fluid restriction and diuretics.
• Further therapy depends on the severity of symptoms and the prognosis of the patient.
• In malignancy chemo- and radiotherapy were traditionally considered the first line of treatment.
• Endovascular treatment is now considered the first line of treatment.
• Stent placement is performed after angioplasty to improve long term patency.
• If the obstruction responds well to angioplasty stent implantation can be avoided.
• High grade stenosis/obstruction that is resistant to guide wire traversal are difficult for the endovascular approach.
• In case of existing central venous catheter crossing the stenotic segment, the lumen of the catheter can be used as guiding rail to facilitate recanalization.

**Malignant SVC obstruction** on page 27

**Benign SVC obstruction** on page 28

**Pulmonary Artery Thrombectomy and Thrombolysis**

**Acute Pulmonary Embolism (PE)**

• Incidence:
• Around 600,00 cases per year in the USA.
• Mortality rate of acute PE is 30%. Clinically
• Asymptomatic:
• In healthy patients with normal lungs and hearts.
• Cardiopulmonary collapse, elevated right heart pressures and hypoxia.
- Caused by large emboli, that lodge in the large central pulmonary artery.
- Described as massive PE or "death embolus".
- Pleuritic chest pain and tachypnea, but stable hemodynamic parameters and normal oxygenation.
- Caused by small peripheral pulmonary arteries.

**Imaging Criteria for Diagnosis**

Intraluminal filling defect that is consistent in several images/planes.

Sudden discontinuation of the artery.

Sudden change in the diameter of the artery.

Absence of normal arborization pattern of the pulmonary artery.

Oligemic lung.

Slow flow in pulmonary artery.

Lack of venous return to pulmonary vein.

**Treatment**

- Indications for aggressive intervention:
- Arterial hypotension (systolic <90mmHg).
- Cardiogenic shock.
- Circulatory collapse requiring resuscitation.
- Echocardiographic findings indicating right ventricular afterload stress and/or pulmonary hypertension.
- Widened arterial-alveolar O2 gradient (>50mmHg)
- Diagnosis of pre-capillary pulmonary hypertension.
- Clinically severe PE with contraindication to anticoagulation.
- Systemic thrombolysis
- Catheter directed thrombolysis
- Surgical embolectomy
- Percutaneous embolectomy, fragmentation and thrombectomy
- Greenfield embolectomy device
- Balloon angioplasty
- Kensey dynamic device
- Angiojet
- Rotating mechanical devices
- Suction thrombectomy devices
- Pulmonary artery stent placement
- Thrombolysis:
- rt-PA infused at the rate of 1-2 mg/h over 12-24 hours.
- Either peripherally or centrally into pulmonary artery.
- Risk of bleeding is 25%.
- Small track created through the pulmonary artery obstruction can result in major clinical improvement.

**Pulmonary Artery Thrombectomy and Thrombolysis**

**CT Angiogram** on page 29

**Pulmonary Angiogram 1** on page 30

**Pulmonary Angiogram 2** on page 30

**Pulmonary Angiogram 3** on page 31

Images for this section:
**Fig. 1:** Axial and coronal CT angiogram images in 46 y/o female patient with bronchiectasis and hemoptysis showing enlarged, tortuous bronchial arteries.

**Fig. 2:** A. Right intercostobronchial trunk dividing into right superior intercostal and right bronchial artery showing hypervascularity. B. Common trunk giving rise to right superior intercostal, right bronchial and left bronchial arteries. Arrow pointing towards the esophageal/tracheal branches.

**Fig. 3:** A. 18 y/o male patient with cystic fibrosis and hemoptysis. Bronchial angiogram showing enlarged left bronchial artery showing hypervascularity and parenchymal staining. B. 41 y/o male patient with tuberculosis and hemoptysis. Selective right intercostobronchial angiogram showing pseudoaneurysm (arrow) and hypervascularity in the right upper lobe.
Fig. 4: A. Thoracic aortogram showing hypertrophic tortuous two right bronchial arteries in this patient with cystic fibrosis and hemoptysis. B. Selective angiogram of the superior right bronchial artery revealed significant hypervascularity and parenchymal staining. C. Post embolization angiogram following administration of 300-500um PVA microspheres shows occlusion of the superior right bronchial artery.
**Fig. 5:** Selective catheterization of the inferior right bronchial artery in the same patients showing similar angiographic findings. Post embolization angiogram following administration of 300-500 um PVA microspheres shows occlusion of the right inferior bronchial artery.
Fig. 6: Thoracic aortogram showing hypertrophic tortuous right intercostobronchial trunk in this patient with cystic fibrosis and hemoptysis.
Fig. 7: -Selective angiogram of the right intercostobronchial trunk revealed significant hypervascularity and parenchymal staining. -Post embolization angiogram following administration of 300-500um PVA microspheres shows occlusion of the right bronchial artery. Note the tracheal/esophageal branches arising from the intercostal branch.
Fig. 8: Lobulated well-defined mass with enlarged pulmonary vessel coursing towards the lesion (arrow).
**Fig. 9:** A. CT angiography of the chest showing multiple PAVMs in both lower lobes, some appear subpleural in location. B. Magnified view showing feeding artery and draining vein. C. Large PAVM.

**Fig. 10:** A. Left pulmonary angiogram showing multiple left lower lobe PAVMs. B. Selective catheterization of the feeding artery supplying the largest PAVM. C. Embolization using Amplatzer Vascular Plug II.
Fig. 11: A.&B. Axial and coronal post contrast MR brain images showing peripherally enhancing lesion in the left cerebral hemisphere consistent with an abscess. C. Pulmonary angiogram showing basal PAVM (arrow).
**Fig. 12:** Pulmonary Angiogram through microcatheter showing left lower lobe PAVM before and after coil embolization.

**Fig. 13:** A. Coronal reformatted CT chest image showing right upper lobe PAVM, with tiny foci of calcification. B. Selective pulmonary angiogram shows lobulated aneurysm sac (large arrow), with draining vein (arrowhead). C. Selective pulmonary angiogram following placement of AVP II device (small arrow) in the feeding artery showing occlusion of the PAVM.
Algorithm for management of PAVMs

- Positive findings on screening of HHT family members
- Clinical Suspicion for PAVM
- Incidental finding suggesting PAVM on chest x-ray/chest CT

1. Shunt measurement by 100% O2 method
   - <5%
     - Chest X-ray +/- Chest CT
       - Normal: PAVM unlikely
       - Abnormal: Standard evaluation of chest abnormality

   - >5%
     - Contrast Echocardiography
       - Intrapulmonary Shunt
         - Intracardiac Shunt
         - Standard evaluation of intracardiac shunt

2. Chest CTA
   - No PAVM
   - PAVM
     - Consider Pulmonary Angiography to exclude telangiectatic or plexiform types of PAVM
     - Consider transcatheter embolotherapy or surgical resection

Fig. 14
Fig. 15: -Absence of SVC enhancement (*) -Intraluminal filling defect (*) -Severe SVC narrowing -Development of collateral pathways (arrows).
**Fig. 16:** CTA of the Chest showing prominent azygos arch (arrow), and chest wall collaterals (arrow head), secondary to SVC obstruction.

**Fig. 17:** CTA of the Chest, showing paravertebral (arrow), sub-scapular (arrow head) and chest wall collaterals, secondary to SVC obstruction.
Fig. 18: CTA of the abdomen showing hepatic capsular collaterals.
**Fig. 19:** A. 66 years old male Patient with lung cancer. Central venogram via right femoral vein approach, showing SVC obstruction below the azygos vein (arrow), with collateral flow through the Azygos (yellow arrow head), hemiazygos (white arrow head) and mediastinal collaterals (black arrow head). B. Same patient after placement of two overlapping stents across the obstruction.
Fig. 20: A. 60 yr old male with right forearm AV fistula and swollen painful extremity. Central venogram showing occlusion of the SVC (arrow). Note multiple chest wall collaterals. B. Post angioplasty venogram showing successful treatment of the previously seen SVC obstruction with mild residual stenosis.
Fig. 21: 26 year old male patient with acute shortness of breath following orthopedic surgery for extremity fracture. CT angiogram shows extensive bilateral pulmonary thrombo-embolism.

Fig. 22: Right pulmonary angiogram artery, showing extensive PE, with some perfusion to the right lower lobe. The right upper and middle lobes show no appreciable perfusion (arrow). Nine mg of rt-PA were slowly injected through the catheter placed in the right pulmonary artery embolus.
Fig. 23: Right pulmonary angiogram shows interval improvement and recanalization of a major segmental right upper lobe pulmonary artery (arrow). The previously seen segmental right lower lobe artery is still patent (not shown). Multiple clots filling the main pulmonary artery are still seen. It was decided to keep the patient on a 1 mg per hour drip of rt-PA through the catheter. The patient was reevaluated after 24 hours.
Fig. 24: Right pulmonary angiogram shows interval improvement, however residual filling of the main right pulmonary artery is still seen. Angiojet thrombectomy device was attempted however, it was abandoned once the patient desaturated during the procedure. Two month follow up showed significant improvement of the patient's symptoms, and hemodynamic parameters.
Conclusion

- IR plays an important role in management of pulmonary critical care (PCC) patients. With the advances in IR techniques and devices, the contribution of IR to the management of PCC patients will continue to grow.

Personal Information

Section of Vascular and Interventional Radiology, The University Of Alabama at Birmingham (UAB), 619 19th Street South, NHB H 623, Birmingham, AL 35249.
Email to: rmetcalf@uabmc.edu

References

5. James R Gossage:Pulmonary AVMs,including hereditary hemorrhagic telangiectasia: Diagnosis and Treatment.UpToDate May 14 2009.