Pulmonary Embolism
CTPA & beyond

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Outline

Pulmonary embolism
- Typical Findings acute vs. chronic PE
- Subsegmental emboli
- Risk stratification

...... and beyond
- Pitfalls
- Nonthrombotic emboli
Imaging Findings in PE
Arterial occlusion due to large filling defect ⇝ the artery may be *enlarged*

Partial filling defect surrounded by contrast material, forming *acute* angles with the vascular wall

Peripheral wedge shaped infarcts (hampton hump)

*Diagnostic Criteria for Acute PE*

*Wittram C et al. Radiographics 2004*
Diagnostic Criteria:

Acute vs. Chronic PE

“occlusion and enlargement”....

“occlusion and caliber reduction”....
Diagnostic Criteria: Acute vs. Chronic PE

- Acute angles....
- Obtuse angle
Diagnostic Criteria:

**Acute** vs. **Chronic** PE

- railway track sign....
- small recanalized artery
Complete filling defects in arteries that are *smaller* than adjacent perfused arteries

Peripheral crescent-shaped intraluminal defects with an *obtuse* angle to the vessel wall

Irregular small lumen and thickened walls (recanalization)

*Wall-adherent* filling defects with calcifications

*Webs* or flaps

Mosaic perfusion

Hypertrophic *bronchial* arteries

Signs of pulmonary *hypertension*

*Wittram C et al. Radiographics 2004*
Diagnostic Criteria for Chronic PE

- Bronchial arteries
- Mosaic perfusion
- Webs

Mosaic perfusion
Subsegmental Emboli
When a test is too good:
how CTPA find emboli that do not need to be found

Explosion in use of CT imaging
14-fold increase of CTPA
from 0.3-4/1000 from 2001 to 2008

Evidence of overdiagnosis
80% increase of PE diagnosis with CTPA
from 62 to 112/100,000 US adults

but
age adjusted mortality from PE did not change
(12.3 vs 11.9/1000,000)
and age adjusted case fatality decreased

Wiener RS et al. BMJ 2013;347
Incidental PE

One year survival of treated and untreated patients

- Retrospective single center study, consecutive 1966 CTs
- PE in 117 pat, 63 had a malignancy (52%)
- 58 (48%) unsuspected, 38 (2/3rd) not reported
- 49 were treated therapeutically, 21 prophylactic, 26 no treatment
- More bleeding complications (0.037) in treated group

Positive predictors of early death:
- systemic thrombolyse, high PE severity major hemorrhage,
- new onset cardiac and renal failure

Neg predictors of early death:
- false negative report, no therapeutic antocoagulation therapy

Do we need to treat them all?

Guerney JW: one of the normal functions of the lung is to remove small emboli, which if allowed to enter the arterial circulation would have disastrous consequences. Such PE would be completely asymptomatic. Thus many small PE may be a normal part of existence and unrecognized until identified by CT angiography or autopsy.

It appears to be safe to withhold treatment of SSE if:
- pulmonary-respiratory reserve is good
- no evidence of DVT on serial testing
- risk for PE transient and no longer present
- no history of central venous catheter or atrium fibrillation

Risk Stratification
Clot Burden for Risk Stratification

Semiquantitative indices
Mastora, Qanadli, Miller, Bankier index

Automatic volumetric quantification of clot burden using a modified seeded region – growing algorithm

Semi-quant. and quant. measures show a high correlation
Greater clot burden correlated with RV dilatation.
RV dilatation correlated with short term mortality,
while clot burden alone did not correlate with short term mortality.

Furlan AF et al. Radiology 2012;265: 283
CT Signs of Right Heart Dysfunction

**RV/LV ratio**
- maximum transverse diameter (minor axis)
- on different levels (mostly the image showing the AV valves)
- to be measured at inner surface of free wall and septum

**PA/Ao ratio**
- on the image at which the right PA is in contiguity
- with the main PA

**Interventricular septum morphology**
- normal = convex towards RV ➞ flattened ➞ bowed
more CT Signs of Right Heart Dysfunction

Diameter of the sup vena cava, azygos vein measured on the level where the azygos vein enters the VCS

CM reflux
from central to peripheral hepatic veins (tricuspid regurgitation)
intrahepatic part of the inferior vena cava
(caveat with flow > 3ml/′)
Assessment of RV Dysfunction

V. azygos  Hepatic reflux  Septal Bowing

Images taken from Henzler T et al. J of Cardiovasc Comp Tomography 2011;5: 3
Points of Discussion

Clot burden
- no correlation with short term mortality,
- effect on long term mortality not yet known
- study population strongly influences statistics
- clot burden does not correlate with perfusion index

RV/LV ratio
- cut-off: (> 1, > 1, > 1.1, > 1.5)
- subjective versus objective assessment
- axial vs. 4 chamber view vs. volumetric assessment
- interval increase?

Henzler T et al. J of Cardiovasc CT 2011;5: 3
Assessment of RV Dysfunction

Objective = Subjective assessment

4 chamber, volumetric assessment, ECG triggering: no advantage

Images taken from Henzler T et al. J of Cardiovasc Comp Tomography 2011;5: 3
Dual Energy CT for Assessment of PE Severity

Images taken from Chae et al. AJR 2010;194: 604
Dual Energy CT for Assessment of PE Severity

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Dual Energy CT for Assessment of PE Severity

Good Correlation with RV/LV ratio and obstruction score (Qanadli)

*Chae et al. AJR 2010;194: 604*

Good Correlation with RV/LV ratio, obstruction score (Mastora) and adverse clinical outcome (IC treatment /death)

*Apfalterer et al. EJR 2012;81: 3592*

Good Correlation with RV/LV ratio, obstruction score (Mastora) PaO2 and troponin

*Thieme SF et al. ER 2012;22: 269*
How to avoid pitfalls....
NO PE but Lymph Node
Embolus or Lymph node? MPR
Artery or vein ?

Pulm. window
Mucoid Impaction
Tricks for Image Evaluation

Peripheral Arteries

Windowing
Width: 2 \times \text{max. PA} - \text{HU}, \quad \text{Level: } \frac{1}{2} \text{mean PA} - \text{HU}
for lower HU and for peripheral arteries wider W and L

MPR for differentiation
flow artifacts vs. thrombi
lymphoid tissue vs. wall adherent thrombi

Overlapping thin slab MIP
2.5mm thin slab MIPs (91% versus 87%)
Nonthrombotic pulmonary embolism

Bach AG et al. Review in EJR 2013, 82; 120-141
Septic embolism

Drug addict with tricuspid valve endocarditis and multiple septic emboli
Fat Embolism (FE) Syndrome

- Mechanical occlusion by fat released from bone marrow, and triggered inflammatory reaction by fat acids
- Seen after major (lower extremity) trauma, but also after liposuction, pancreatitis, viral hepatitis.....
- Pulmonary, cerebral and cutaneous symptoms in 85%
- Usually self limiting and good outcome
- Imaging findings resemble ARDS: widespread opacification, rarely ill defined small centrilobular nodules
Fat Embolism (FE)

16 ys old boy with femur fracture, after endomedullary nailing
Amniotic Fluid Embolism

- Amniotic fluid enters blood stream through small venuous tears in the uterus during normal labor.
- Mechanical obstruction by fetal cells, lanugo meconium, and systemic reaction by metabolites causing coagulation, vasospams and decreased cardiac function.
- Risk factors: advanced maternal age, section, uterine stimulants, large fetal size, placental path.
- High mortality (21%), 1.9/1,000,000 maternities (UK).
- Imaging findings resemble diffuse edema, sometimes more nodular pattern, pleural effusion.
Hydatid Embolism

- Tapeworm parasitosis (Echinococcus granulosus)
- Embolism occurs after rupture of a hydatid cyst (cardiac or hepatic echinococcus)
- Mechanical occlusion
  no blood clots or local thrombosis
- Hemoptysis most frequent symptom
- May be clinically fulminant, cause pulm. hypertension
- Surgical removal, transplantation
Tumor embolism

- **Type I**
  - true tumor embolism
  - cellular material from a distant tumor enters the pulmonary circulation
  - DD: hematogeneous metastases invade the vascular wall

- **Type II**
  - growing tumor cone into PA

- **Type III**
  - tumors arising from the intima of the PA (sarcoma, malign fibrous histiocytoma)
Tumor embolism: Type I

Pat with chondrosarcoma suspected for having tumor cells in small centrilobular arteries.
18 months later

Type I
tree in bud sign
mildly enlarged centrilobular arteries
progressive irreversible obstruction
pulmonary hypertension
Tumor embolism: Type II
Tumor embolism: Type III

Sarcoma of the PA
Two Cases......from the RSNA 2012 Film panel

The key lies in the lung parenchyma...
Case 1

Patient with CREST
Isolated PAH in absence of fibrosis in 8-12%.

However a subgroup of patients is less responsive or refractory to treatment with vasodilators (epoprostenol = prostacyclin). Shows adverse effects with development of life-threatening pulmonary edema.
Vasculopathy in PAH

Idiopathic PAH
remodelling of precapillary pulm arteries, plexiform lesions

Pulm capillary hemangiomatosis
angioproliferation of capillaries causing extrinsic narrowing of the venous lumen

Pulm venoocclusive disease
remodeling of post-capillary septal veins and preseptal venules, occluding the lumen

PAH in SSc
intimal fibrosis of both, arterioles and venules
Role of HRCT

Pulmonary hypertension with
- GGO, in a centri-lobular distribution
- Interlobular septal lines
- Lymph adenopathy

Indicative of pulm. venoocclusive disease

HRCT pattern serves as predictor of edema under prostacyclin therapy
Case II
Cysts in Birt Hogg Dubé Syndrome

**Size**
- Any size (from 2mm to > 7cm) but usually > 2cm
  - larger cysts in lower lobes

**Shape**
- very variable:
  - round to oval, lentiform, lobulated, multisepatsted

**Distribution**
- lower lung, costophrenic angle (> 50%)

**Cysts abutting proximal aspect**
- of pulmonary arteries or veins - resemble
  - pulmonary interstitial emphysema (PIE)

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n=12 patients

Agarwal PP, Gross BH, … Kazerooni EA. AJR 2011;196: 349
n=17 patients
Birt Hogg Dubé Syndrome

Rare autosomal-dominant genodermatosis
Hamartomas of the skin
Renal tumors (renal cell carcinoma, oncocytoma, etc)
Colonic polyposis and chorioretinal disease

LUNG: thin-walled cysts ± pneumothorax

Age-dependent manifestations:
20-40 years  pneumothorax
> 40 years  renal tumors

Birt AR, Hogg GR, Dubé WJ. Arch Dermatol 1977
Thank You