Thrombus in situ: after radiation/pneumonectomy--not a pulmonary embolism.

Radiation changes can peak at 1-2 years, looks like normal day to day stuff for radiation pneumonitis.

Valvular mass: vegetation (endocarditis), papillary fibroelastoma, thrombus

'Interstitial Lung Abnormality' with peripheral and basilar reticulation. State this on non-HRCT exams in which you are worried about ILD.

3 layers of stacked cysts to call honeycombing (call pulmonary fibrosis). 'Stacked cystic change'

CPFE: Combined pulmonary fibrosis-emphysema (see emphysema and ILD). Call it when you see moderate emphysema with a background of pulmonary fibrosis (may normalize on PFT's because of mixed pattern).

Look at membranous portion of the trachea--- if its a donut thats good inspiration, if flattened that is expiratory. Don't read an HRCT with suboptimal expiratory phase (D shaped trachea).

If considering NSIP, sparing subpleura, say need to look for autoimmune ILD.

Chronic HP can look like regular UIP… need clinical history

HALT (Hypoattenuated leaflet thickening): On TAVR leaflets, clinically can present with a mass/thrombus with elevated aortic gradients. DDx is pannus (granulation tissue on ventricular side of valve) or endocarditis (on ventricular side of valve).

ARVD/C now termed Arrhythmogenic cardiomyopathy (AC), inheritable. Review newer Padua criteria.

'Hot-Phase' of ARVD/C can present like a myocarditis (LV vs. RV vs. both predominant).

LV Dominant AC should be considered if you have subepicardial, particularly circumferential, MDE. Arrhythmogenic left ventricular cardiomyopathy (ALVC). Desmoplakin mutation.

Lymphocytic myocarditis is the most common subtype. Seen in Covid and covid-vaccine related myocarditis.

Pericardial thickness greater than 4 mm is abnormal

Combined ischemic and non-ischemic cardiomyopathies are being more recognized. Such as myocarditis causing ischemia or occurring on top of previous MI. Use function/wall motion as a helpful tool to differentiate.

Constrictive pericarditis:

Myocarditis early enhancement? Lake Louis criteria???? Most people not doing.

Blastomycosis can look just like TB (and Histo)-- cavitary mass with TIB opacities. Add it to the differential.

LungPLAN: Free and accessible.. Check out yo.

Stop tracking at 2 years for incidental pulmonary nodules, however if the patient meets criteria for continued Lung CA screening than move to that arm

Look at L1 and if HU less than 100, can call low bone density (need to do this)

Pannus usually > 145 HU and thrombus < 90 HU when looking at surgical valves

Stuck valve: when mechanical leaflet opening angle is less than 73 degrees that is limited motion or stuck

Photon counting CT: No scintillator, photons go directly in the semiconductor without any loss. Massive reduction in radiation. Every photon is counted. Progenitors is dual energy/source imaging.Better SNR, higher spatial resolution. MOre precise imaging.All data available for spectral imagin… all different Kev's…Better image quality in obese patients. Can use less contrast. Ultra high resolution is great for CCTA. 0.2 mm with less blooming.Better stent imaging.

MI on regular chest CT's: look for it… 10% of patients who presented with non-STEMI chest pain had either acute or chronic MI.Look at wall thickening, subendocardial hypoenhancement, look at non-con for lipomatous metaplasia for old infarct.

When measuring larger arteries in pre-TAVR planning.. Measure 20% into the calcified plaque to account for blooming.

Think about CTPA as a screening test vs. diagnostic test… sub-mSv for normal sized patients… need to crank MA or really sick patients
40mL contrast, 100kvp, top of arch to diaphragm (reduced Z-axis coverage), no breathing instructions (get used to read studies at end expiration leading to dependent atelectasis), acquire with thicker collimation for shorter breath holds

CTEPD (chronic thromboembolic pulmonary disease/hypertension): nearly 50% have no history of pulmonary emboli. CTEPH is 1-5% after pulmonary emboli. Can have CTEPD WITHOUT pulmonary hypertension. Unilateral defect is not typical of CTEPH.
VQ: high sens/spec (>90%), mores sensitive than CT, mismatched moderate to large defects

CTPA: supposed to be a late, second line test after VQ scan, but usually comes first without the clinical suspicion. Consider dual energy for iodine map/perfusoin snapshot to correlate with pulmonary findings.

1. Direct findings: webs/mural non-occlusive thrombi. Smaller vessels over time/retracted. bands/calcifications as well.
2. Indirect findings: mosaic perfusion, classically narrowed vessels in low attenuation areas.DDx is small airway disease with hypoxic vasoconstriction (look for mucous plugging). Subpleural scars from infarcts. Dilated bronchial arteries assoc. With chronic pulmonary emboli.
3. DDX: vasculitis, with beading (sarcoidosis), extrinsic compression of pulmonary vasculature (tumnor or fibrosing mediastinitis), pulmonary artery sarcoma (unilateral)

PUlmonary emboli

1. 3rd most common cardiovascular disease (after MI and stroke)
2. RV failure is the primary cause of death in PE
3. Central or bilateral clot burden does not significantly worsen prognosis
4. **Use RVd:LLVd > 1, mixed results but does have some type of impact on mortality (main thing to measure)**
5. Subhepatic reflux of contrast (below the pulmonary veins) nonspecific, helpful
6. Septal bowing/flattening, less reproducible, helpful

Dual energy CTPA

1. Used looking for CTEPD, not for acute symptoms
2. After equivocal patients, try looking at perfusion/iodine maps to see if there is perfusion loss
3. Optimum 60-65 kev

CT Hemoptysis

1. CTA modality of chioce
2. Peak is descending thoracic aorta (look at both pulmonary arterial and bronchial artery opacification
3. ¾ 5 cc/s, ¼ 3 cc/s.
4. Try to differentiate site of blood (ground glass) vs. aspirated blood (bilateral ground glass)