

Pulmonary Disabilities Workshop
Summaries for answering Dr. Richie's Questions

COPD:

- 4th leading cause of death in the U.S, affecting more than 5% of U.S. population.
- Although cigarette smoking is the most common cause for development of COPD, fully 20% of COPD patients have never smoked, with the underlying etiology being underlying chronic asthma with remodeling, exposure to pollution or cooking-fire smoke, etc.
- For any given degree of severity of COPD, mortality and mortality is worse in women.
- The rate of decline in FEV1 is greater in current smokers, persons with emphysema compared to those without emphysema, and persons with some bronchodilator reversibility (Asthma-COPD overlap, now termed ACO) compared to those without reversibility.
- The typical person with COPD is just as likely to die from a cardiovascular disease as they are from a respiratory one. Cardiovascular morbidity is not confined to those with more advanced airflow obstruction but occurs across the entire spectrum of COPD disease severity. Having a diagnosis of COPD approximately doubles the mortality risk of an MI.
- The key measurements on spirometry are:
 - Forced Vital Capacity (FVC, clinically relevant when reported as FVC %predicted)
 - Forced Expiratory Volume in 1 Second (FEV1 %predicted)
 - the ratio of FEV1 to FVC (FEV1/FVC ratio – no % of predicted for this measurement)
 - Forced Expiratory Flow 25th to 75th of expiratory flow (FEF₂₅₋₇₅ %predicted) – also called Maximal Mid-Expiratory Flow (MMEF %predicted)
- Spirometry results are either:
 - normal (FEV1/FVC > 80%, FVC >80%)
 - obstructive (FEV1/FVC < 80%, with ratio <70% defining COPD)
 - restrictive (FEV1/FVC normal with both FVC and FEV1 proportionally decreased)
- Obstruction (especially COPD) is defined with FEV1/FVC ratio <70% and the severity of COPD then reflected in the percentages of FEV1 %predicted below normal.
- Restriction may be caused by suboptimal or poor effort and requires further testing with measurements of lung volumes and lung diffusion.

Asthma

- More significant morbidity and mortality is seen in:
 - Adult-onset asthma, or childhood/teen asthma persisting into adulthood
 - Women (compared to men with same severity of asthma)
 - Black persons
 - Obese persons
 - Persons with Type 1 or Type 2-low (neutrophil cascade) asthma, compared to Type-2 high (eosinophilic cascade)

- Although adult asthma patients have an increased risk of all-cause mortality, only a small number of asthma patients die directly from the asthma itself. The increased risk of death in asthma patients is mainly due to comorbidities.
- Patients with adult asthma often have their disease morph from Type-2 high (eosinophilic cascade) disease into Type-2-low (neutrophilic cascade) or Type 1 disease through “airway remodeling”. This is evident when spirometry with bronchodilators fail to revert the FEV1 %predicted back to normal.

Solitary Pulmonary Nodules (SPNs)

- Definition of SPN includes
 - Solitary and not multiple nodules
 - Nodule ≤ 3 cm (anything greater is a malignant mass until proven otherwise)
 - Surrounded by aerated lung (i.e., not associated with atelectasis or fibrosis)
- First, try and determine if the nodule has been present on prior imaging. A nodule that has not changed in \geq two years is unlikely to be malignant.
- Second, CT Lung imaging should be available.
 - Look for smooth margins (benign) versus Spiculated margins (malignant)
 - Look for calcification (excluding eccentric calcification) – benign
 - The smaller the nodule the less likely the malignancy
 - Upper lung zone nodules more likely to be malignant than lower lung zone nodules
 - Association of the nodule with “ground-glass change” is worrisome
- History of prior malignancy and smoking history are “red flags” for concern.
- Increasing age is also a major concern for malignancy.
- For SPNs > 8 mm, Positive Emission Tomography (PET) imaging is more sensitive than CT at identifying cancerous nodules – not this is NOT the case with SPNs ≤ 8 mm.
- Be aware that a positive PET nodule does not confirm malignancy – metabolically active nodules (tuberculosis, sarcoidosis, other inflammatory nodules) may appear (+).
- Any biopsy of an SPN – whether by fiberoptic bronchoscopy or by open-lung biopsy – must reveal a pathologic cause for the nodule. “Normal tissue” is not an acceptable pathologic result upon which an underwriting decision can be made.
- Underwriters should consult Fleischner Society Guidelines for Management of Incidentally Detected pulmonary Nodules for further drill-down recommendations for needed follow-up for SPNs.

Interstitial Pulmonary Diseases

- The descriptive term “interstitial” reflects the pathologic appearance that the abnormality begins in the interstitium of the lung, but the term is misleading as most of these disorders are also associated with extensive alteration of alveolar and airway architecture.

- The diffuse parenchymal lung diseases are divided into those that are associated with known causes and those that are idiopathic.
- Clinical presentation of ILD is typically because of unusual dyspnea-on-exertion and/or a nonspecific cough, an abnormal chest x-ray or CT Lung, and occasionally because of “restriction” found on spirometry (reduced FVC %predicted and FEV1 %predicted but with normal or near-normal FEV1/FVC ratio)
- Relevant studies that may help identify and quantify the significance of ILDs are:
 - Lung volume determination revealing a <90% reduction in FRC %predicted and/or TLC %predicted
 - Lung diffusion (DLCO) < 70 %predicted
 - Reduction of 6-minute walk test < 60 %predicted
 - High-resolution CT (HRCT) imaging of the lung. Reticular opacities, traction bronchiectasis, and honeycombing (clustered airspaces 3 to 10 mm in diameter) in a predominantly subpleural and basal distribution are the imaging features associated with a histopathologic pattern of usual interstitial pneumonitis (UIP)
- As an underwriter, the finding of an interstitial lung disease admitted by an applicant, or finding this diagnosis in the medical records, will require some homework – as the number of interstitial diseases and their prognosis is long and varied. The most helpful criteria at arriving at an impairment rating will be:
 - The severity of disease at its worse and activity of current disease
 - The on-going requirement for any steroids or other suppressive medication
 - And the time-course of the disease, as the longer the course and lessening of activity, generally the less the impact the disease will have on morbidity and mortality