Parenchymal Lung Diseases

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The tracheobronchial airway tree: the trachea, primary bronchi, intrapulmonary bronchi, terminal bronchioles, and air passages of the pulmonary acinus.
The alveolar-capillary membrane. This diagram of an electron micrograph shows the way the alveolar and capillary cells on one side of the alveolar septum fuse to form an ultrathin layer which offers little barrier to diffusion. The other side of the septum is thicker and provides physical support. RBC, red blood cell.
Structure of lung parenchyma: Respiratory compartment

- Contact area between air and blood = surface area of a tennis court, 30,000 acini and 300 million alveoli.
- Tissue barrier between air and blood is extremely thin to allow saturation of hemoglobin in erythrocytes within 1 sec.
- Tissue barrier contains capillaries lined with complete endothelium.
- Alveoli are densely packed and lined by epithelium.
- Interstitium contains few cells – fibroblasts with contractile filaments.
Cells of the lung parenchyma

- 75% of all lung cells are contained in the parenchyma.
- The air: blood barrier consists of 25% epithelial cells, 25% endothelial cells, 35% interstitial cells and 15% collagen fibres.
- Alveolar epithelium consists of lining cells (type 1), secretory cells (type 2 pneumocytes) and brush cells.
Function of parenchymal cells

★ Type 1: tight junctions, cannot multiply (mitosis)
★ Endothelial cells: leaky junctions with exchange of H₂O, solutes and molecules between capillaries and interstitium.
★ Type 2: synthesis, storage and secretion of surfactant
★ Injury to type 1 cells: Type 2 cells undergo transformation into type 1 cells (2-5 days).
★ Catastrophic injury: repair mechanism too slow and respiratory failure occurs which require ICU and mechanical ventilation and supplemental O₂.
Parenchymal Defense system

1st defense: alveolar macrophages
2nd defense: histiocytes, plasma cells, lymphocytes, leucocytes (rare in healthy lungs), mast cells.
3rd defense: Lymph nodes
Etiology of parenchymal diseases

- Inhalation Route:
  1. bacteria, viruses, fungi, protozoa
  2. Organic materials: bird prot, wood dust
  3. Anorganic substances: asbestos, silica
  4. Gastric contents
  5. Water, blood, toxic gases
  6. Idiopathic diseases ? inhalation
Etiology of parenchymal diseases

★ Vascular route:
1. Vasculitis: Wegeners, SLE, Churg Strauss
2. Increased vascular permeability: ARDS
3. Increased Starling forces: pulm oedema
4. Pulmonary embolism
5. Septicaemia: multiple lung abscesses
6. Primary Pulmonary Hypertension
Pathogenesis

- Alveolitis with desquamation of epithelial cells, inflammatory cell infiltrates, hialine membranes, necrosis.
- Involvement of interstitium with fibroblast proliferation and granuloma formation, entrapment or occlusion of lymphatics
- Obliteration/occlusion of capillary bed and necrosis
- Honey combing
Alveolar diseases

★ Alveoli filled by:
1. Pus: pneumonia, lung abscess
2. Water: pulm oedema, ARDS
4. Cells: Malignant, T4 lymphocytes (sarcoidosis), neutrophils (IPF), eosinophils (E pneumonia)
5. Phospholipoprot: surfactant (Pulm alveolar proteinosis)
Pathogenesis of gram-negative pneumonia. (From: concise handbook of respiratory diseases, ed 2, Easton, 1985, Appleton & Lange.)
Interstitial diseases

- Unknown etiology: Idiopathic pulmonary fibrosis, sarcoidosis, collagen vascular diseases
- Known etiology: asbestosis, silicosis, hypersensitivity pneumonitis
Physical signs of alveolar disease

- Consolidation:
  1. dullness on percussion,
  2. increased vocal sounds on palpation and auscultation
  3. Bronchial breathing
  4. Aegophony and E to A (auscultation)
  5. Whispering pectoriloquy
Physical signs 2

★ Crepitations:
1. Alveolar disease: fine, early inspiration.
2. Interstitial disease: coarse, high tone, late inspiration – velcro type

★ Cough:
1. Productive: pneumonia
2. Non-productive: interstitial disease
**Diagnosis**

★ Chest Xray:
1. lobar veiling with air bronchograms – pneumonia
2. Diffuse alveolar veiling – pulm oedema-ARDS, aspiration
3. Interstitial veiling: linear: IPF, asbestosis
   nodular: miliary TB, sarcoidosis, silicosis
4. Focal decreased vascularity: pulm embolus, vasculitis with occlusion
5. Breakdown: cavitation: lung abscess
Laboratory investigations

- Sputum MCS, ZN+ culture
- Blood culture
- FBC
- Serology: ANF, RF, ACE, chlamydia, mycoplasma
- LFT+E
- Renal function
Special Investigations

- CT Scan
- Bronchoscopy
- Open lung biopsy
Complications

- Respiratory failure
- Cor pulmonale
- Extra pulmonary manifestations: SIADH, meningism, arthralgia, septicaemia and DIC
Management

- Depending on etiology:
  - Infection: Abs
  - ARDS: Mechanical ventilation and $O_2$
  - IPF, sarcoidosis: Steroids
  - Cor pulmonale
  - Respiratory failure: Type 1 (hypoxaemic)-$O_2$
  - Type 2 (Hypercarbic + hypoxaemia)-mechanical ventilation + $O_2$. 