Non-Neoplastic Lung Pathology Roadshow

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Normal Lung (1)

This 62 year old man died 2 days after an aortic valve replacement and internal mammary artery graft to the left anterior descending coronary artery. He had a 25 pack year history of cigarette smoking and also a history of alcohol abuse. The lung in this case is normal, with a uniform gray-tan appearance and spongy consistency. Note the absence of emphysematous destruction or consolidation. Pigmentation is minimal.
• Coal worker’s pneumoconiosis with progressive massive fibrosis (2)

• This patient was a Pennsylvania coal miner for 35 years who died at age 80. The lungs are heavily pigmented secondary to accumulation of massive amounts of coal dust. This appearance has given rise to the name “black lung disease”
Images 6 and 87 are identical
Icterus (3)
This 32 year old man had a history of alcohol abuse associated with delirium tremens, alcoholic hepatitis, and hepatic encephalopathy. The patient’s course was complicated by massive hematemesis and severe generalized peritonitis. At autopsy, the patient was markedly jaundiced secondary to his hepatic failure. The lungs are green because of post-mortem oxidation of bilirubin to biliverdin.
Cirrhosis
• Chronic passive congestion, rheumatic heart disease (4)
• This 63 year old man had a long history of rheumatic heart disease which required aortic valve replacement in 1975 and mitral valve replacement in 1988. The patient died in January of 1990 from complications of his rheumatic heart disease.
• On gross inspection, the lungs have a red-orange appearance secondary to chronic passive congestion. There are also multiple small foci of emphysematous destruction. Microscopically, the alveolar spaces contain numerous alveolar macrophages with hemosiderin granules within their cytoplasm
• **Calcified granulomas (old TB) (5)**

• This 59 year old man died from large cell carcinoma of the right upper lobe which had been treated with radiation therapy. The patient had a 35 pack year history of cigarette smoking. The immediate cause of death was massive Hemoptysis from an abscess in the right upper lobe.

• An incidental finding was the presence of multiple calcified granulomas of the right and left upper lobes, consistent with the patient’s history of tuberculosis. The patient had been treated with INH and ethambutol for one year. Notice the multiple chalky white, rock-hard, calcified granulomas present in the upper lung zones. The remainder of the lung is relatively normal.
• **Acute and organizing bronchopneumonia (6)**

• This 76 year old man was admitted for evaluation of agitation and paranoid behavior. During the hospitalization, the patient became febrile and was found to have left mid and lower lung zone opacities on chest x-ray. The patient died in spite of treatment with IV Kefzol.

• The gross specimen shows marked consolidation of much of the left lower lobe. The consolidated areas are firm and have a gray-tan appearance which is more pale than the surrounding lung tissue. Also, there is a mild amount of emphysematous destruction in the upper lobes. *Streptococcus pneumoniae* was cultured from both lungs post-mortem.
• **Invasive Aspergillosis (7)**

  This 59 year old man had a history of chronic lymphocytic leukemia, complicated by autoimmune hemolytic anemia, for which he underwent splenectomy I 1991. The patient was treated with a variety of chemotherapeutic and immunosuppressive agents, which resulted in bone marrow suppression. In May 1993, chest x-ray revealed two rounded opacities in the upper lung zones. The patient was treated with amphotericin B for possible fungal infection, but developed GI bleeding and expired on 5/28/93.

  The gross specimen shows a spherical, tan to cream colored sharply circumscribed mass in the upper lung abutting the pleura. The lesion is firm and has pale margins. Microscopic examination showed fungal hyphae consistent with aspergillus invading pulmonary blood vessels, resulting in thrombosis and infarction of the adjacent lung.
• **Tuberculosis, opportunistic (AIDS)** (8)

• This 38 year old homosexual male developed chronic diarrhea with parasitic infection identified but responded poorly to treatment. Later he developed skin lesions diagnosed as Kaposi’s sarcoma. These led to suspicion of the AIDS which was confirmed by antibody testing. Treatment was ineffective. About 1 year later, after losing 80 pound, he developed fever, dyspnea, and pulmonary infiltrate by x-ray. Treatment was declined. At autopsy each lung weighed 750 grams and had nodular consistency. The sharply defined lesions were tuberculosis. He also had pneumocystis bronchopneumonia and cytomegalovirus infection.
• Pneumocystis Pneumonia (AIDS) (9)
• This 31 year old man had two risk factors for AIDS: IV drug abuse and homosexuality. He had positive HIV tests in 1987 and was treated with AZT. He had several opportunistic infections and recently developed progressive weakness, fatigue and non-productive cough. Chest x-rays showed bilateral patchy infiltrates and pneumocystis organisms were seen in sputum. Usual therapy for this was ineffective. At autopsy each lung weighed over 1300 grams and was consolidated. This section is almost completely involved by confluent bronchopneumonia.
AIDS pneumocystis pneumonia, ARDS (10)

This 44 year old homosexual man was diagnosed in September of 1988 with the acquired immunodeficiency syndrome, and was subsequently treated with AZT. The patient developed pneumocystis pneumonia which was treated with Pentamidine. He progressed to respiratory failure requiring mechanical ventilation and oxygen therapy. The patient died in September of 1989, approximately one year from the initial diagnosis.

The gross specimen shows marked consolidation of the lung throughout with a gray-tan appearance of the consolidated areas which appear to be more pale than the less involved lung. The lung parenchyma is firm and the lungs were heavy (combined weight of more than 2500 grams). The changes observed are due both to the infestation of the lung by pneumocystis organisms as well as the toxic effects of oxygen.
AIDS, PCP, Cryptococcosis (11)

This 29 year old homosexual man was the first case of acquired immunodeficiency syndrome diagnosed at Duke University Medical Center. The patient presented with Pneumocystis pneumonia and Kaposi’s sarcoma of the skin. Despite treatment with IV Septra, the patient developed progressive respiratory failure and died two weeks after admission.

The patient’s lungs are diffusely consolidated and very heavy, each weighing more than 100 grams. Microscopically, the lungs showed both *Pneumocystis pneumonia* as well as diffuse involvement of the pulmonary interstitium with *Cryptococcus neoformans*. This latter finding is best observed in the hilar lymph nodes, which are enlarged and have a mucoid appearance. Microscopically, these lymph nodes were virtually replaced by Cryptococcus organisms with few intervening lymphocytes. Kaposi’s sarcoma was identified in the skin, small intestine, right adrenal gland, gallbladder, and esophagus.
• Saccular Bronchiectasis (Post Obstruction)  (12)

• This 50 year old patient had a 25 year history of repeated pulmonary infections and episodes of pneumothorax.

• The severe Bronchiectasis readily the frequent infections. This probably was acquired as a result of a partial bronchial obstruction, but unfortunately we did not obtain the history of a specific episode, as for example the aspiration of a peanut in childhood.
• **Cystic fibrosis** (13)

This 32 year old male underwent bilateral lung transplantation for respiratory failure secondary to cystic fibrosis. End stage pulmonary disease from cystic fibrosis is one of the more common indications currently for bilateral lung transplantation. Both lungs have to be transplanted in these patients, because the infected secretions in the native lung would be a threat to the patient while receiving immunosuppressive therapy for the transplantation.

The lung appears to be small, and the most striking feature is the presence of numerous ecstatic bronchi filled with yellowish purulent secretions. Patients with cystic fibrosis have a homozygous deficiency of the cystic fibrosis transmembrane chloride conductance regulator (CFTCR), which changes the composition of bronchial mucous secretions, rendering their clearance more difficult and making the patient more susceptible to bacterial colonization of the secretions.
• **Diffuse panbronchiolitis (14)**

  This 38 year old black male had a long history of progressive shortness of breath and obstructive lung disease which began when the patient was 13 years of age. Sputum specimens repeatedly grew Pseudomonas aeruginosa. However, the patient had normal sweat chloride levels on several occasions, no evidence of pancreatic insufficiency, and is married with children. Chest x-rays and computed tomography of the thorax showed numerous peripheral 2 to 3 mm. nodules and Bronchiectasis. The patient underwent bilateral lung transplantation in March, 1993. Subsequently, the patient has had some deterioration in pulmonary function and development of new nodules in the periphery of the transplanted lungs.

  The most striking feature of the gross specimen is the presence of numerous yellow nodules throughout the lung parenchyma, especially noticeable in the upper lobes. In addition, there are bronchiectatic changes, most apparent in the mid and lower lung zones. These changes are typical for diffuse panbronchiolitis, which occurs primarily in individuals of Oriental origin. The yellow nodules consist of masses of foamy macrophages within the pulmonary interstitium, centered upon inflamed bronchioles.
• Traction Bronchiectasis  (15)

• This patient had sarcoid for many years. Diffuse fibrosis has produced marked contraction of the upper lobe. Intralobar bronchi are markedly dilated because of contraction of the collagen combined with rigidity of the chest wall. The bronchi eventually were weaker than the other structures.
- Centrilobular emphysema, severe (16)
- Note coarsening of alveolar tissue, especially in the upper lobe. This is clearly multifocal and normal alveolar tissue is often present in the same lobule with destroyed tissue.
- Note absence of scars in vicinity of lesions or elsewhere. This patient died of respiratory insufficiency.
• Panlobular emphysema （17）
• This is a 67-year-old man who had a long history of chronic obstructive pulmonary disease and a 100 pack year history of cigarette smoking. The patient had also been diagnosed with squamous cell carcinoma of the right upper lobe. Note the uniform destruction of lung parenchyma involving all areas of the lung in a more or less uniform manner.
• **Paracicatricial emphysema (18)**

This patient was 61 years old age and had undiagnosed nodular pulmonary infiltrates. At autopsy, the pulmonary lesions were shown to be tuberculosis in nature. The patient had no past history of treatment for tuberculosis.

• The specimen shows many focal area of scarring, some with central caseation. Emphysematous tissue destruction of obviously related to the scars. The infection is many years old and the patient’s own immune mechanisms were responsible for the healing which occurred.
• **Localized emphysema**  (19)
  - This 54 year old man present in August, 1998 with a 5 month history of dysphagia, a 35 pound weight loss, excessive oral secretions with drooling, and a chronic productive cough. He was found to have squamous cell carcinoma of the pharynx which was treated with palliative radiation therapy. The patient died of complications from aspiration pneumonia. He had a history of alcohol abuse for 35 pack year history of cigarette smoking.
  - An incidental finding at autopsy is the presence of localized emphysema in the apical area of the right upper lobe. Only traces of emphysema can be identified in the remaining portions of the lung. Also note the presence of a few small pale areas of consolidation in the right lower lobe, secondary to this patient’s aspiration.
• Interstitial fibrosis (Honeycomb lung) (20)
• This 62 year old man had had “mixed connective tissue disease” with the first manifestation being scleroderma in 1977. Subsequently, he had diagnoses of discoid lupus, Raynaud’s phenomenon, polymyositis and progressive interstitial pulmonary fibrosis
• **Clinical History:** 53 y/o BF with oxygen-dependent Sarcoidosis.(21)

• **Gross Findings:** There is diffuse parenchyma fibrosis accompanied by emphysematous airspaces. The upper lobe exhibits an ill-defined area of consolidation/cavitation.

• **Diagnosis:** Stage IV Sarcoidosis with pulmonary fibrosis and emphysema; aspergilloma of the left upper lobe.
• **Eosinophilic granuloma (Histiocytosis X)** (22)

• This patient was a 22 year old man who suddenly developed shortness of breath about 1 year before he died. He was found to have a spontaneous pneumothorax, as well as diffuse pulmonary infiltrations. A lung biopsy showed eosinophilic granuloma. His lung re-expanded but during the next year, despite treatment, the infiltrates increased in extent and he had several more episodes of pneumothorax, during one of which he died.

• The specimen shows extensive destruction and fibrosis of the lung with marked contraction. Active inflammatory lesions involve the little remaining lung tissue at the base.
Lymphangioleiomyomatosis (23)

This 45 year old woman presented with a history of insidious onset of shortness of breath. In addition, she developed chylosous pleural effusions. Chest radiographs showed increased interstitial markings with preserved lung volumes, and computed tomography of the thorax showed numerous thin walled cysts throughout both lungs. The patient was treated initially with estrogen and progesterone, but her shortness of breath and respiratory insufficiency progressed. Bilateral heterotopic lung transplantation was performed in November, 1992.

This section of one of the patient’s native lungs shows a spongy appearance, with numerous small cysts throughout the lung parenchyma. Microscopically, proliferation of smooth muscle cells in the walls of these cysts is identified, as well as in other areas of the lung associated with pulmonary lymphatics. This disorder occurs almost exclusively in women of reproductive age and is also associated in percentage of cases with tuberous sclerosis.
• Radiation pneumonitis (24)

This 73 year old man was found to have Merkel cell carcinoma of the right posterior auricular skin in 1986. The tumor was excised and treated with radiation therapy, but the patient developed metastases within cervical lymph nodes. Four years later, metastases were identified within Mediastinal lymph nodes, and the patient received additional chemotherapy as well as radiation to the mediastinum. The patient developed bronchopneumonia and dies in July, 1993.

• The gross specimen shows fibrosis and contraction of the lung parenchyma in the medial portion of the specimen. The area of fibrosis does not correspond to any anatomic distribution, and is limited to the radiation ports. Microscopically, there is dense fibrosis associated with obliterated vascular structures, changes which are typical for radiation pneumonitis. The patient was also found to have radiation esophagitis at autopsy. Two creamy colored nodules of metastatic tumor can also be identified with the region of radiation fibrosis.
• **Idiopathic inflammation (25)**

• This is a case of pulmonary alveolar proteinosis. The yellowish, firm, non-encapsulated consolidated areas are the lesions. The material is non-cellular, amorphous, gel-like protein. It can occupy so many alveoli as to cause of death from respiratory insufficiency. Here, it was an incidental finding in a patient with a head injury.
• **Diffuse pleural thickening (asbestos), CLE (26)**

• This 67 year old man was a former grain mill worker who also had a 60 pack year history of cigarette smoking. However, there was no identifiable history of asbestos exposure. The patient died from complications of coronary atherosclerosis, calcific aortic and mitral valvular disease, and diabetes mellitus. He had also been treated for squamous cell carcinoma of the larynx in 1963.

• The gross specimen shows marked thickening of the visceral pleura with virtual encasement of the lung. Note also the presence of centrilobular emphysema which is most severe in the upper lung zones. This patient also had numerous calcified parietal pleural plaques (see Specimen A-73-69).
Asbestosis (27)

This 72 year old male had a clinical history of diffuse pulmonary fibrosis, cor pulmonale, and insulin dependent diabetes mellitus. The patient had worked as an insulator for more than 30 years. The lung specimens are not well inflated. However, close examination demonstrates the presence of gray, streaky fibrous tissue at the lung periphery and in the lung bases. In some areas, this fibrosis is associated with honeycomb changes. In the upper lung zones, there are focal areas of emphysematous destruction. Parietal pleural plaques were also demonstrated bilaterally at autopsy. Microscopic examination showed numerous asbestos bodies in histologic sections
• **Asbestos pleural plaque (28)**

• This specimen of diaphragm illustrates a peculiar lesion often seen in persons exposed to asbestos. This consists of a zone of fibrotic thickening with projecting nodules having the appearance of drippings from a wax candle. These occur especially on the diaphragm but also on the parietal pleura of the chest wall.

• The lungs are heavily pigmented secondary to accumulation of massive amounts of coal dust. This appearance has given rise to the name “black lung disease”.
• **Coal Workers’ Pneumoconiosis “Black Lung Disease” (29)**

• This patient was a coal miner in Pennsylvania for 35 years, and stopped at age 55 (in 1947) probably because of age. He was reasonably well until the past few years when he noted cough, with black sputum, and increasing shortness of breath. Recently, he was found to have tuberculosis. He died at age 80.

• The specimen shows heavy pigmentation with fibrotic (silicotic) nodules in some of the lower lobe foci. The upper lobe shows dense fibrosis and cavitation due to the combination of pneumoconiosis and tuberculosis. This was the source of the black sputum.
Images 6 and 87 are identical.
• **Silicotuberculosis (30)**

This is another, more severe case of silicosis. The larger lesions in the upper lobe are called conglomerate silicosis. This is often the result of associated tuberculosis. The gray, granular center of the largest lesion is in fact tuberculous caseation and so this case is an example of tuberculosilicosis.
• **Massive saddle embolus (31)**
  
  This 72 year old man suffered a left hip fracture in November, 1985, which was treated with insertion of Moore prosthesis. The patient was to be discharged on December 12, 1985, and was being wheeled in a wheelchair to his car when he suddenly became limp and nonresponsive. Cardiopulmonary resuscitation was attempted but was unsuccessful. Arterial blood gases at the time of the Code showed an arterial PO2 of 8 and 13 mm Hg.

  • At autopsy, the cause of death was a massive saddle embolus involving the main pulmonary artery trunk and the right and left main pulmonary arteries. The thrombus material is coiled within the main pulmonary artery, interfering with blood flow and causing acute right ventricular failure. The source of such a thromboembolus is almost always the deep leg veins. Risk factors include congestive heart failure with venous stasis, hypercoagulable states, and immobilization as often follows orthopedic surgical procedures.
• **Pulmonary infarct (32)**
• The infarct is at the base of the lower lobe, laterally. The embolized vessel is seen at the hilar margin of the infarct and also nearer the hilum
• Multiple PTE secondary to anabolic steroid abuse (33)

• The 37 year old man was a world class weight lifter who presented in July, 1991 with a history of syncope. In October, 1991, the patient was brought to the Emergency room following collapse on the street. Resuscitative efforts were unsuccessful. The gross specimen shows multiple pulmonary thromboemboli with peripheral wedge-shaped infarcts in the upper and lower lobes. The lower lobe infarct is pale and appears to be undergoing organization and probably occurred at the time of the 1991 syncopal episode. Also present along with the specimen are portions of thrombus material removed from the main pulmonary artery. Anabolic steroid abuse has rarely been associated with hypercoagulable states, which in turn increase the risk of development of pulmonary thromboemboli