SUPPURATIVE AND INFECTIOUS LUNG DISEASE THAT MAY REQUIRE SURGERY

TYPES:
1. HYDATID CYST OF THE LUNG
2. LUNG ABSCESS
3. EMPYEMA
4. BRONCHIECTASIS
5. TUBERCULOSIS
6. NECROTISING/DESTRUCTIVE PNEUMONIA - ACUTE
   - CHRONIC

PULMONARY HYDATID CYST

Echinococcosis or hydatid disease is caused by larvae, which are the metacestode stage of the tapeworm Echinococcus. Four species are recognised and belong to the family Taeniidae. The vast majority of infestations in humans are caused by E. granulosus. E. granulosus causes cystic echinococcosis, the pastoral form, which has a worldwide distribution and is concentrated in sheep-raising areas. Humans are exposed less frequently to E. multilocularis, which causes alveolar echinococcosis, because E. multilocularis infestation usually occurs in colder areas and is associated with animals in wild ecosystems, especially foxes. E. vogeli and E. oligarthrus are rare species and cause polycystic echinococcosis.

Parasite Biology

The fully developed cysts are composed of three layers. The outer layer, or pericyst, is composed of inflamed fibrous tissue derived from the host; the exocyst is an acellular laminated membrane; and the innermost layer, or endocyst, is the germinative layer of the parasite and gives rise to brood capsules (secondary cysts), which bud internally. Protoscolices are produced within the brood capsules and take approximately a year to develop after infection.

Organ involvement

Following ingestion of E. granulosus eggs, the metacestode cyst can infest any organ (primary echinococcosis). Secondary echinococcosis results from the spread of the metacestodes from the primary sites. Of patients with cystic echinococcus, 85–90% show single organ involvement and w70% harbour a solitary cyst.
The liver is the most common site of cyst formation, followed by the lung in 10–30% of cases and other sites (usually the spleen, kidney, orbit, heart, brain and bone) in 10% of cases.

In children, the lungs may be the commonest site of cyst formation. Of patients with lung cysts, 20–40% also have liver cysts. Pulmonary hydatid disease affects the right lung in 60% of cases, 30% exhibit multiple pulmonary cysts, 20% bilateral cysts and 60% are located in the lower lobes.

Pulmonary echinococcosis can follow intrathoracic rupture of a cyst of the liver, but most patients with pulmonary hydatid disease do not show liver involvement. Within the chest, echinococcosis can primarily involve the pleural cavity, mediastinum and chest wall.

**Clinical features**

Pulmonary cysts typically increase in diameter at a rate of 1–5 cm/year. The initial phase of primary infection is asymptomatic and may remain so for many years. Hydatid disease is seen in subjects of any age and sex, although it is more common in those aged 20–40 yrs. Most intact lung cysts are discovered incidentally on chest radiographs. Occasionally, an un-ruptured cyst results in cough, haemoptysis or chest pain. Subsequent clinical features of *E. granulosus* infection depend upon the cyst site and size. Small cysts may remain asymptomatic indefinitely, but cysts may enlarge to about 20 cm in diameter and cause symptoms by compressing adjacent structures.

Symptomatic hydatid disease of the lung, however, more often follows rupture of the cyst. The cyst may rupture spontaneously or as a result of trauma or secondary infection. In a contained rupture, only the endocyst is torn and the contents of the cyst are contained by the pericyst. In a communicating rupture, the contents of the cyst escape into the tracheobronchial tree through bronchioles that have been incorporated into the pericyst. Direct rupture into the pleura follows tearing of both the endocyst and the pericyst, with discharge of the contents of the cyst directly into the pleural cavity. Rupture may be associated with the sudden onset of cough and fever. If the contents of the cyst are expelled into the airway, expectoration of a clear salty or peppery tasting fluid containing fragments of hydatid membrane and scolecites may occur.

Symptoms of hydatid disease can result from the release of antigenic material and secondary immunological reactions that develop following cyst rupture. Fever and acute hypersensitivity reactions ranging from urticaria and wheezing to life-threatening anaphylaxis may be the principal manifestations. Although allergic episodes may develop after cyst rupture, fatal anaphylaxis is uncommon.

Calcification, which usually requires 5–10 years for development, occurs quite commonly with hepatic cysts but rarely with pulmonary cysts.
Diagnosis:
In the majority of cases, a combination of imaging and serological methods usually yields the diagnosis of cystic echinococcosis. A patient who has lung cysts should be investigated for associated liver cysts.

Imaging:
1) Plain chest radiograph PA & lateral views: Typical chest radiographic appearances of uncomplicated pulmonary hydatid disease are one or more homogeneous round or oval masses with smooth borders surrounded by normal lung tissue. Large cysts can shift the mediastinum, induce a pleural reaction or cause atelectasis of adjacent parenchyma. Calcification of pulmonary cysts is rare. On chest radiography, calcification of hepatic cysts may be evident.

2) Computerised tomography (CT) scan with contrast may demonstrate a thin enhancing rim if the cyst is intact. CT scanning can elucidate the cystic nature of the lung mass and provide accurate localisation for planning of surgical treatment of complicated cysts.

3) Laboratory and special investigations
Laboratory. In pulmonary cystic echinococcosis, routine laboratory tests do not show specific results. Less than 15% of cases exhibit eosinophilia, which generally occurs only if there is leakage of antigenic materia.

Treatment:
Surgical treatment. for patients who are able to undergo surgery, it is considered the treatment of choice since the parasite can be completely removed and the patient cured. The surgical options for lung cysts include cystectomy, pericystectomy and capitonnage by controlled evacuation technique. Lobectomy is for large cysts causing destruction of more than 75% of the lobe.

Adjunctive antihelmintic therapy before and after surgery appears to reduce the risk of recurrence by inactivating protoscolices and reduces the tension of the cysts for easier cyst removal. Therapy generally should begin 4 days prior to surgery and be continued for 1–3 months.

The usual dose of mebendazole is 40–50 mg/kg body weight/day, given in three divided doses after meals (maximum daily dose 6 g). Therapy is usually indicated for 3–6 months. Albendazole is given at a dosage of 10–15 mg/kg body weight/day in two divided doses and the usual dose is 800 mg daily. Therapy is most often indicated for a minimum of 3–6 months. Albendazole is preferred because it has better bioavailability.
LUNG ABSCESS

Pathology:
Lung abscess is defined as a pus-containing necrotic lesion of the lung parenchyma. The cardinal histologic change in all abscesses is suppurative destruction of the lung parenchyma within the central area of cavitation. As abscesses can be of varied sizes, the distinction between necrotizing pneumonia (or pulmonary gangrene) and lung abscess is somewhat arbitrary as these entities represent a progression along a continuum of lung infections. Generally, lung abscess is often defined as ≥ 2 cm in diameter whereas abscesses <2 cm are considered necrotizing pneumonia. Acute lung abscess is arbitrarily defined as < 4 weeks duration, whereas chronic lung abscess is > 4 weeks duration, although the clinical implications of this distinction are not clearly established.

Types:
I. Primary lung abscess  Alcoholis, Seizure disorder General anesthesia Drug abuse Esopohgeal lesions Neurological deficits (e.g.,CVA, bulbar disease) Insulin-treated diabetes mellitus
II. Secondary lung abscess Pulmonary embolism with infarction, Septic emboli with pulmonary infarction , Endobronchial obstruction by neoplasm (benign or malignant) , Obstruction by foreign body , Bronchiectasis
III. Cryptogenic lung abscess  Absence of identifiable risk factor

Bacteriology:
Usually multiple organisms, aerobes (G --ve uncommon) + anaerobes (Bacteroides, Fusobacteria, microaerophilic Strep)

Clinical Features:
Lung abscess is suspected from the history and physical exam, but requires radiographic confirmation. Clinical suspicion for lung abscess should be heightened in persons with a recognized pre-existing condition that increases risk for development of lung abscess . In primary lung abscess, symptoms of lung abscess are similar to other anaerobic lung infections, and include indolent (1–3 weeks) development of fever, cough, pleurisy, dyspnea, sweats,weight loss, and sputum production (often foul smelling). Sputum production may be absent in the absence of a communication of the abscess cavity and the airways. The production of putrid sputum occurs in approximately 49% of the cases, and is generally considered diagnostic of anaerobic infection (as aerobic bacteria are generally not capable of producing this characteristic odor).
Diagnosis:
1. CXR: Thick-walled cavity, usually in dependent site
2. Sputum for C & S, AFBs & TB Culture
3. Blood culture
4. FBC: Hb (anaemia of chronic disorders)
5. Chest CT scan is more sensitive than a routine chest radiograph, and may detect small cavities, demonstrate obstructing endobronchial lesions, or distinguish lung abscess from air–fluid levels in the pleural space.

Treatment:
1. Antibiotics: IV penicillin & oral metranidazole OR oral co-amoxiclav & metronidazole OR IV clindamycin (mean 1 week) Convert to oral when fever
2. Physiotherapy & postural drainage
3. Surgery Rarely needed as drains via bronchi
Indications:
1. Giant abscess (>5cm)
2. Bleeding
3. Failure to respond to medical treatment

Prognosis:
Good if adequately treated
Cavity closure takes a mean of 4 weeks, cure takes a mean of 8 weeks (occurs in 80%)
Relapse occurs in 10%

BRONCHIECTASIS

Definition:
Bronchiectasis is abnormal chronic irreversible dilatation of one or more bronchi due to loss of structural elements in the bronchial wall.

Causes:
I. CONGENITAL: Immotile Cilia Syndrome, Hypogammaglobulinaemia, Sequestration
II. ACQUIRED: Cystic fibrosis, Post-infective (TB, Measles, Pertussis, Pneumonia (esp. necrotising) Asthma, Inhalation injury, Bronchial obstruction (eg. FB, LN, Tumour), Fibrotic lung disease (eg. CFA, Sarcoid)

Types:
Usually generalised (occasionally localised)
Tubular/Cylindrical
Cystic/Saccular
Clinical Features:
Symptoms:
Cough (usually productive of purulent sputum++), Haemoptysis, Shortness of breath (if extensive & severe disease), Wheeze
Signs: Halitosis, Clubbing, Crackles (coarse, early insp., heard at mouth, Wheeze

Special Investigations:
1. CXR honey comb appearance
2. High Resolution Computer Tomography (HRCT)
3. Sputum (C & S; AFBs & TB culture)
4. Pulmonary Function tests
5. Blood gases
6. Sinus XRs

Treatment:
MEDICAL:
Antibiotics whenever superadded infections
Physiotherapy & postural drainage
Bronchodilators

SURGICAL:
The only curative treatment of bronchiectasis is surgical resection. Surgery indicated whenever:
1) Localized bronchiectasis to one lobe segment
2) Life threatening hemoptysis
3) Empyema
4) Parenchymal destruction and recurrent life threatening chest infections

Pulmonary Tuberculosis

Is primarily a medical disorder but surgery indicated whenever:
1) Tuberculous empyema
2) Fibrothorax
3) Lung parenchymal destruction and recurrent chest infection
4) Life threatening hemoptysis
5) Suspicion of malignancy on TB granuloma or old pulmonary scar