RADIOLOGICAL STAGING IN THORACIC ONCOLOGY

Dr. G. McGann – Cheltenham General Hospital.

The advent of multi-slice computed tomography in the early part of this decade ensured that CT remains the mainstay of cancer staging in the thorax, despite promising developments firstly in MRI and more recently in positron emission tomography, particularly when fused with CT images.

The 'tumour, node, metastasis' system, with some newer subdivisions, attempts to define those patients who might be suitable for radical surgery or radical radiotherapy from the majority (about 75% of those presenting with lung cancer) who will only be suitable for palliative measures.

Areas of difficulty for all forms of imaging lies in distinguishing the thoracic malignant mass from associated lung collapse, predicting unresectability, tumours contacting major vessels or the pericardium, distinguishing reactive from malignant lymphadenopathy and in assigning significance to the increasing number of tiny nodules found on modern CT scanning.

PET-CT shows promise in increasing the relatively poor sensitivity of CT for malignant node identification but cannot operate effectively without combining with mediastinoscopy and node sampling.

CT is also effective in monitoring response to treatment in attempted downstaging pre surgery, diagnosing recurrent tumour and identifying the complications of treatment. CT screening for lung cancer has yet to show a convincing long-term mortality benefit.

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SURGICAL STAGING IN THORACIC ONCOLOGY

Mr. E. Bishay – Birmingham Heartlands Hospital.

Rational treatment decisions and ultimate prognosis of patients with lung cancer depend largely on the stage of disease at the time of diagnosis.
Surgical or invasive staging techniques will always be required as long as radiological and less invasive techniques are not 100% sensitive or specific in the pre-operative identification of patients most likely to benefit from pulmonary resection. This applies to all three components of TNM staging and no individual should be denied the chance of curative resection based on radiological or clinical findings alone.

The routine use of positron emission tomography (PET) scanning has redefined the indications for invasive staging of the mediastinum for the detection of N2 nodal involvement. Invasive staging procedures can be omitted in patients with small peripheral tumours and negative mediastinal PET images. However, in case of central tumours, PET hilar N1 disease, low fluorodeoxyglucose uptake of the primary tumour and lymph nodes $\geq$ 16 mm on CT scan, invasive staging remains indicated. PET positive mediastinal findings should always be cyto-histologically confirmed. The use of PET scans as applied to the M stage often requires cyto-histologically confirmation of distant sites of involvement.

Trans-bronchial needle aspiration (TBNA), ultrasound-guided bronchoscopy with fine needle aspiration (EBUS-FNA) and endoscopic esophageal ultrasound-guided fine needle aspiration (EUS-FNA) are new techniques that provide cyto-histological diagnosis and are minimally invasive. Their specificity is high but the negative predictive value is low. Because of this, if they yield negative results, an invasive surgical technique is indicated. They are particularly useful in restaging the mediastinum following neoadjuvant down-staging.

Bronchoscopy
Indications: Endo-bronchial T status of central tumours.
Rule out synchronous tumours in other parts of the bronchial tree.
Vocal cord involvement signifies T4.

Mediastinoscopy
Indications: Access to stations 1, 2, 4, and 7.
Ideally 2R, 2L, 4R, 4L, and 7 nodal stations should all be explored with a lesser but yet acceptable standard of 4R, 4L, and 7 to reduce the likelihood of false negative results. Also used to evaluate proximal main bronchi and medially based right tumours.
Sensitivity = 81% (TP/TP+FN), Specificity = 100 (TN/TN+FP), NPV (TN/TN+FN) =91%.
Complications: Morbidity 1-3%. Mortality 0.08%.

Mediastinotomy (Chamberlain incision)
Used alone or in combination with Mediastinoscopy.
Indications: Evaluation of left nodal stations 5 & 6, central tumours close to A-P window, appropriate T4 (Pericardial involvement) staging on right.
Sensitivity 80-85% (TP/TP+FN), Specificity =100 (TN/TN+FP)

Video Assisted Thoracic Surgery
Indications: T status in large tumours where CT can not accurately differentiate between contact and invasion. Assessment of pleural effusions and involvement (T4) including cytological confirmation.
Access to stations 5 and 6 on left and other N2 nodes not amenable to Mediastinoscopy (8, and 9).

Thoracotomy
Indications: Final and yet important staging tool ideally when all other attempts have been exhausted. During resection, thorough intra-operative staging is essential for prognosis and need for adjuvant therapy.

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Introduction

ROLE OF CHEMOTHERAPY AND BIOLOGICAL AGENTS IN THE TREATMENT OF NON-SMALL CELL LUNG CANCER (NSCLC)

Dr. D. Farrugia - 3 Counties Cancer Network.

The non-surgical management of NSCLC has three major components: chemotherapy, radiotherapy and more recently biological therapies. Chemotherapy has established a role in the treatment of all stages except very early resectable disease (T1-2N0M0). Pre-operative chemotherapy in resectable tumours downstages disease and is associated with equivalent survival to post-operative chemotherapy. It is still considered experimental (Gilligan). Post-operative chemotherapy has conclusively been shown to confer an overall survival benefit of 5% at 5 years and this is now standard of care for node positive disease (Hotta).

In advanced disease (stages IIIB and IV), chemotherapy offers palliation of symptoms, improvement in quality of life and prolongation of survival. Platinum based treatments using combinations of Cisplatin or Carboplatin and another agent remain the first line treatments of choice (Schiller). Histological and biological parameters may, in the future, guide the selection of chemotherapy and in individual patients. Evidence is emerging the multi-targeted antifolate Pemetrexed has greater efficacy in non-squamous lung tumours (Scagliotti). Docetaxel and Pemetrexed have modest activity in the second line setting in patients with good performance status (Hanna). The role of maintenance chemotherapy, although not a new concept, is still being explored using the newer cytotoxic agents such as Pemetrexed (Belani).

We are now entering the era of biological therapies. Gefitinib (Iressa) and erlotinib (Tarceva) were the first small molecule oral tyrosine kinase inhibitors to enter clinical practice with the latter now licensed for use as a $2^{nd}/3^{rd}$ line agent. However combination of these agents with first line chemotherapy failed to improve outcome (Giaccone, Herbst). Recent data suggested that the degree of tumour EGFR expression may influence efficacy of these agents in selected populations (Fukuoka). The anti-VEGF monoclonal antibody Bevacizumab did achieve a survival improvement when combined to 1^{st} line chemotherapy, but was associated with an excess of fatal haemoptysis in squamous tumours and is therefore currently licensed only on non-squamous tumours (Sandler). Other biological therapies in clinical testing include monoclonal antibodies towards EGFR such as Cetuximab and small molecule tyrosine kinase inhibitors such as Sunitinib, Sorafenib and Vandetanib.

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FUNDAMENTALS AND BASIC PRINCIPLES OF LUNG CANCER SURGERY

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1.Obtain accurate preoperative staging
Non-invasive – clinical examination; CTPET
Invasive - EBUS/EUS-FNA.Mediastinoscopy/VAT

2.Assess fitness for resection
Fitness for GA : cardiovascular status
Fitness for lung resection : respiratory reserve (spirometry, gas transfer,lung volumes,ABG); exercise testing; right heart function.

3.Obtain preresection histology
Percutaneous needle biopsy
Intraoperative frozen section analysis
? Diagnostic/therapeutic segmentectomy

4.Achieve R0 resection
The primary tumour should be resected with margins which are free of malignant cells on microscopic examination. There is no place for tumour debulking. Intraoperative frozen section analysis of resection margins should be used for confirmation.
The highest removed mediastinal node must be negative.

5.Obtain accurate pN stage
Systematic nodal dissection – all lymph node tissue from at least 3 N2 stations.

6.Perform minimum required anatomical resection
Consider segmentectomy vs lobectomy ; sleeve lobectomy vs pneumonectomy

7.Non-functioning lung can be resected with therapeutic benefit
Reduction in hyperinflation by resecting non-functioning (emphysematous) lung tissue may lead to a compensatory improvement in postoperative lung function.

8.Reduce functional deficit
Consider muscle-sparing thoracotomy ; video assisted lung resection or preferably thoracoscopic resection. Evidence for improved recovery.

9.Return patient to preoperative activity
Early extubation.Epidural anaesthesia. Early mobilization. Portable drainage devices.

10. Postoperative follow-up
5 year program to treat immediate complications, detect tumour progression, early metachronous tumours

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PALLIATION IN THORACIC ONCOLOGY: ENDOBRONCHIAL TREATMENT

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Patients with severe central airway obstruction from malignant causes have disabling symptoms of dyspnea, respiratory distress, and obstructive pneumonia. For many of these patients, in the absence of intervention, the airway pathology may be the direct cause of distressing death from suffocation. A large number of patients with symptomatic and life threatening airway pathology are not candidates for definitive surgical correction because of the extent of the disease or comorbidity. The endoscopic techniques are minimally invasive and can provide significant palliation. Although the long-term outlook in these cases is often dismal, the temporary or permanent relief of airway obstruction provides significant palliation with improvement in quality of life and, in few cases increase in survival.

The malignant causes of airway obstruction include direct extension from adjacent tumour – bronchogenic (30 -50%), oesophageal or thyroid, or metastasis from renal, breast or thyroid tumours. Primary tumours of central airways are rare. Extrinsic compression from hilar, mediastinal tumours or bulky lymphadenopathy counts for rest of the causes.

The management of central airway obstruction is individualized depending on the underlying cause. Endoluminal lesions are managed by coreout of tumour, thermal laser vaporization, photodynamic therapy, brachytherapy, cryotherapy, and electrocautery. However, these therapeutic bronchoscopic techniques provide transient benefit and have to be consolidated with either chemotherapy or radiotherapy.

Airway stents are a valuable adjunct to these techniques and can provide prolonged palliation from rapidly recurrent endoluminal tumour, extrinsically compressing mass or managing trachea-oesophageal fistulae. An ideal stent should be easy to insert or remove, have sufficient strength, flexible to mimic natural airway, promotes clearance of secretions, biologically inert and does not migrate. None of the various stents available (metallic, non-metallic, covered, uncovered) satisfies all these conditions. The silicone stents are inexpensive, less reactive with minimal granulation, solid and hence no tissue ingrowth and can be repositioned and removed easily. However, these are difficult to insert, have reduced inner diameter and can migrate. The metal stents on the other hand are easy to insert, larger internal:external diameter ratio, conforms to distortions and curves and gets incorporated into airway helping in mucociliary clearance. The disadvantages include difficulty in removal and uncontrolled expansion and erosion into adjacent structures.

Any attempt to manage central airway obstruction should involve team approach involving experienced anaesthetist, surgeon, nursing staff and physiotherapist for optimal results and avoidance of complications.

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STAGE SPECIFIC LUNG CANCER SURGERY

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There have been changes to the staging system with reclassification of T stage on basis of tumour size, other nodules and the presence of pleural effusion.

Stage I (tumour <7cm, N0)

"Lobectomy is the gold standard treatment". This statement may be challenged in cases of stage Ia cancer or in patients with limited pulmonary function. In these cases an anatomical segmentectomy with lymph node dissection is an acceptable alternative.

Stage II (any T size N1, Tumour > 7cm, chest wall, mediastinal pleura N0)

Pneumonectomy is a disease in itself
Extended lung resections i.e bronchoplastic and angioplastic sleeve resections should be employed to avoid pneumonectomy.

T3 N0/1
Chest wall invasion is not a contraindication to resection. En-bloc rib resection and reconstruction is the treatment of choice.
Satellite nodules in the same lobe as the primary (now T3) should be resected.

Stage IIIB : T4 N0/1
Local invasion – in rare circumstances T4N0 tumours can be primarily resected with SVC / L atrial wall or vertebra.
Ipsilateral nodules – the 7th revision downstaged ipsilateral nodules in a separate lobe to the primary from M1 to T4. In these rare circumstances sublobar resection is preferable to pneumonectomy.

Stage IV
Oligometastatic disease – in rare cases where a stage I/II tumour presents with an isolated brain or adrenal metastasis initial metastasectomy can be justified if subsequent complete pulmonary resection is possible.

Stage IIIA (N2 disease)
N2 disease represents both a spectrum of disease and the interface between surgical and non-surgical treatment of lung cancer

Evidence from the staging revision suggests that single zone N2 disease has a similar prognosis to multizone N1 disease and therefore arguably should be treated primarily by surgery

Evidence from Intergroup 0139/EORTC 08941 trials suggests that multizone or unresectable N2 disease should be treated primarily by chemoradiotherapy. There may be a role for surgery if N2 is downstaged to N0 and lobectomy is possible but pneumonectomy is avoidable.

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BULLOUS DISEASE: PRIMARY PNEUMOTHORAX

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Pneumothorax is air in the pleural space. Primary pneumothorax occurs in otherwise healthy patients without any lung disease with an incidence of up to 28/100,000 per year for males. There is a strong association with smoking and despite no underlying lung disease sub pleural blebs and bullae are likely to play a role and are present in 90% of patients at surgery and 80% of patients on CT scan,

It is not felt that physical activity causes a pneumothorax. Most cases are symptomatic but nearly half the patients wait more than 2 days with symptoms before seeking medical attention. Clinical history and physical examination usually suggest the presence of a pneumothorax but are not reliable indicators of pneumothorax size. Diagnosis is normally established by plain chest X-ray. Size of the pneumothorax is divided into "small" or "large" depending on the presence of a visible rim of <2cm or >2cm between the lung margin and the chest wall (2cm approximates to a 50% pneumothorax).

Patients with a small pneumothorax and minimal symptoms do not require hospital admission but are discharged with appropriate advice. Symptomatic patients who require admission require active intervention, observation alone is inappropriate. Supplemental high flow oxygen should be given. Simple aspiration is recommended as first line treatment for all primary pneumothoraces requiring intervention (A). If this fails and <2.5L has been aspirated a second aspiration should be considered. If this fails or >2.5L aspirated at the first attempt an intercostal drain should be inserted. Pneumothoraces which fail to respond within 48 hours should be referred to a respiratory physician(C). Chemical pleurodesis should not be considered after a first time uncomplicated primary pneumothorax. Chemical pleurodesis with tetracycline can be recommended for recurrent primary pneumothorax when surgery is not an option.

With the statistical and perceived risk of recurrence the accepted indications for surgery are:

- Second ipsilateral pneumothorax
- First complicated pneumothorax
- Bilateral spontaneous pneumothorax
- Persistent air leak
- Spontaneous haemothorax
- Professions at risk

Surgical treatment is based on two principles, firstly resection of the cause of the air leak to remove the underlying defect and secondly create pleural symphysis.

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SECONDARY PNEUMOTHORAX

Mr. M. Kalkat – Birmingham Heartlands Hosptial

A pneumothorax may be spontaneous or secondary to traumatic, diagnostic or a therapeutic procedure. A spontaneous pneumothorax can be "primary", occurring without known aetiology or clinical evidence of an underlying disease or "secondary" to a disease process that predisposes to pneumothorax.
In 20% of patients with spontaneous pneumothorax the event is related to underlying localized or generalized pulmonary disease process. The annual incidence of secondary pneumothorax is 6.3 per 100,000 population in males and 2.0 per 100,000 population in females.

The various causes of secondary pneumothorax are

1. Airway disease
   Bullous disease, COPD, Asthma, Cyst, Pneumatocele, Cystic fibrosis
2. Interstitial disease
   Idiopathic pulmonary fibrosis, Eosinophilic granuloma, Sarcoidosis, Tuberous sclerosis, Connective-tissue disease (Rheumatoid arthritis -causes pyopneumothorax, Ankylosing spondylitis, Polymyositis and dermatomyositis, Scleroderma, Marfan's syndrome, Ehlers–Danlos syndrome)
3. Infections
   Anaerobic, Staphylococcal, Gram –ve pneumonias, Lung abscesses, Tuberculosis, Nocardiosis, PNP (6% of patients with AIDS)
4. Neoplasms
   Primary and Metastasis (sarcomas in particular)
5. Oesphageal perforation
6. Traumatic
7. Iatrogenic
   Mechanical ventilation 3-4%, Thoracentesis, pleural biopsy, CVP line insertion, Nasogastric tube, endoscopic procedure, laparoscopic surgery, inappropriately manage drains
8. Catamenial pneumothorax
9. Lymphangiomyomatosis (LAM)
10. Inhalation and IV drug use – marijuana and cocaine

Most common cause of secondary pneumothorax is COPD. The second peak of pneumothorax occurs between ages of 45 and 65 years of age and is usually secondary to underlying lung pathology. Sign and symptoms often resemble the underlying disease and hence it may go unnoticed. The secondary pneumothorax can precipitate respiratory failure as the patients are older and usually have compromised lung function. Pneumothorax should always be excluded in the case of decompensated COPD or cystic fibrosis. The mortality rate in patients with severe COPD reaches 16 – 17%.

Treatment:
The aim of treatment remains alleviation of symptoms, treat underlying cause, recognize complications and prevent recurrences. Most of the patients with secondary pneumothorax are dealt non-surgically, as compared to primary pneumothorax. The treatment is individualized depending on the underlying cause.

TUBERCULOSIS

Mr. S. Barnard – Freeman Hospital – Newcastle upon Tyne.

Background

Tuberculosis is one of the oldest diseases known to man, with skeletal evidence appearing about 8000 years ago, and written descriptions of the infection from over 2500 years ago. Its first appearance coincides with mans transition from hunter gatherer to domesticator of animals, and links through *Mycobacterium Bovis* have been postulated on that basis, but gene analysis has cast doubt on that hypothesis. It is *Mycobacterium Tuberculosis* that causes the greatest burden of disease in the past and at present. It is a disease of poverty.

Epidemiology

Worldwide the incidence of tuberculosis is 8.8 million a year (141/100,000) with about 1.6 million deaths. Africa has the highest incidence with 343/100,000. The number of cases in the UK is much lower, with 8113 cases in 2006, an incidence a magnitude lower (14/100,000) than the worldwide incidence. The situation now seen in Africa was similar to that in this country in 1880, when 11% of all deaths were caused by tuberculosis with an incidence of the disease in that year of 308/100,000. Currently in the UK, at least 40% of the cases are from the London area, with the Brent area having an incidence 8 times that of the UK as a whole (114/100,000)

The disease has declined through the latter half of the 20th century but increased since 1987 to remain a major world health problem. Some of this is due to the AIDS epidemic, and in subSaharan Africa, 31% of new adult TB cases were in patients with preexisting HIV (2002 figures).

Risk factors for the disease include overcrowding (including prisons), immunosuppression, certain ethnic groups and alcoholism, but HIV has become the strongest risk factor for the disease. Being a contagious disease, contact with a known case of TB is a strong risk factor: it is estimated that a patient with active TB will go on to infect 20 patients.

Molecular studies on strains of *Mycobacterium Tuberculosis,* suggest that, in high incidence areas, exogenous reinfection is a significant contributor to disease, in addition to reactivation of latent disease.

Microbiology

Gram positive curved rods, but stain poorly with this method. Non sporing and aerobic. They are acid fast: retaining arylmethane dye even when treated with acid. There are 62 lineages within the MTB group, e.g .the Beijing subtype, of interest particularly to community epidemiologists. The infection is usually passed on via droplets.

Pathology

Primary infection occurs in the alveoli, with macrophages ingesting the bacilli. The macrophages undergo transformation to multinucleated giant cells and in the majority (90%) of primary infections, healing takes place. In 5-10% the infection persists and can spread to the lung (producing miliary tuberculosis) and other organs including bone. Even if healing takes place the disease may remain dormant and a reactivation may occur if the balance of tubercle growth against destruction by cell mediated immunity is altered e.g. by immunosuppression.

Clinical Presentation

Cough, fever and weight loss, particularly in conjunction with one of the risk factors mentioned above or a contact history with a known case of tuberculosis, is suspicious of tuberculosis infection. Sputum smear staining, the Tuberculin skin test and sometimes Interferon γ release assay are used to confirm the diagnosis.

Treatment

Standard treatment is 6 months of combination (usually quadruple) treatment with Rifampicin, Isoniazid, Pyrazinamide and Ethambutol. Compliance is a major issue in the developing world and amongst some of the high risk groups. Under very favourable conditions (compliance, no drug resistance) 90% of patients can be cured of their tuberculosis[1].

Surgery

Surgery for tuberculosis can be considered under 4 headings:

1. Diagnosis
2. Treatment to eradicate infection
3. Treatment for complications
4. Management of spaces-usually from collapse treatment in the past.

1. **Diagnosis**-This is probably most frequently performed in the UK by mediastinoscopy. Mediastinal widening noted on a chest X-ray is further investigated by CT and enlarged nodes demonstrated. There is usually a contact history or the patient is in a high risk group. In a study from Lille, 6% of isolated mediastinal lymphadenopathy was due to tuberculosis (the majority were sarcoid). Lung biopsy and pleural biopsy are occasionally used to establish a diagnosis of tuberculosis-a study from Nagano in Japan found 5% of SPN to be due to tuberculosis.

2. **Eradication**- This includes failed medical treatment of "standard" tuberculosis, eg through poor compliance and the more common indication of Multiple Drug Resistant (MDR) tuberculosis, (defined as resistance to Isoniazid and Rifampicin) which has a low incidence in the UK of 0.7% [1] (in previously untreated patients) but is higher in previously treated cases. It is a huge problem, in numeric and percentage terms, in certain parts of the world such as Eastern Europe, where there may be 30,000 new patients/year with TB, 10% of these cases have MDR TB[2]. These patients would be treated medically with second line antibiotics but a significant number come to surgery.

   The surgical principles include:
   1. There should be localised disease
   2. Fitness for surgery: preoperative sputum negativity preferred especially in MDR TB
   3. Wide resection including pneumonectomy
   4. Care and reinforcement of the bronchial stump: pneumonectomy BPF rate of up to 25%
   5. Postoperative treatment with antibiotics (second line therapy) for up to three years in the case of MDR TB.

   The surgery can be technically taxing due to fibrosis and obliteration of fissural planes[4]. In MDR TB, there are now a number of case series of lung resection, with low mortality and excellent outcomes with conversion to sputum negativity in 85-90%[6].
   Most recent surgical figures from SCTS data in 2002: there were 37 resections for tuberculosis, of which 6 were pneumonectomy, three combined with thoracoplasty.

3. **Management of spaces**-usually from collapse treatment in the past. Various materials used to effect collapse of the lung which promoted healing. Although artificial pneumothorax was popular, surgical methods such as thoracoplasty were widely used. A more straightforward operation was plombage, the insertion of foreign materials to aid collapse of the lung. Lucite balls were preferred and are made of methacrylate. However they could erode through the chest wall or into mediastinal structures and removal may be required. There are very few patients with plombage balls in situ as it fell out of favour at the beginning of the Streptomycin era around 1950[5].

Management of the residual space left from artificial pneumothorax can be difficult, should the space become infected, and if simple drainage fails to improve the situation, myoplasty or even thoracoplasty may need to be considered.

4. **Treatment of complications**-eg (i)bronchostenosis from fibrosis: dilatation and stenting may be required.

(ii)Lobectomy for Aspergillus infection, bronchiectasis
(iii)Haemoptysis-usually erosion into a bronchial artery. Embolisation can be tried.
(iv)Bronchial obstruction by nodes-can respond to steroids
(v)Pericarditis-1-4% get effusions which may require a pericardial window or even full pericardectomy: however, initial management is medical unless there is tamponade.
(vi) Empyema-for small effusions, medical treatment is indicated. Drainage, including rib resection may be required-a decortication may not be successful due to the diseased underlying lung.

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Lung Abscess

Incidence/Epidemiology

Accurate statistics on the incidence of lung abscess are difficult to obtain but HES data suggests about 500-1000 cases per year or 1-2/100,000 population. The M:F ratio is 2:1.

Classification

Sometimes classified into primary or secondary.

Primary: as a consequence of necrotising infection
Following aspiration (infected oral secretions/gastric contents)
In immunosuppressed patients

Secondary bronchial obstruction including foreign body
Pulmonary infarct (<5% of infarcts become an abscess)
Lung diseases-Sarcoid, Wegener's
Bacteraemia

Lung abscess may be acute or chronic, after 6 weeks. A lung abscess is >2cm in diameter, smaller abscesses are classified as necrotizing pneumonia.

Pathophysiology

Most cases occur in the apical segments of the lower lobes or the posterior segments of the upper lobes, especially on the right. Necrotising infection (*Klebsiella, Staph aureus,* rarely *Strep. Pneumoniae,*) may account for 40% of lung abscesses. The most common cause is said to be aspiration of infected oropharyngeal secretions. Therefore, poor gingivodental hygiene is a risk factor. Impaired consciousness (general anaesthesia, high alcohol intake) and conditions that predispose to aspiration , such as oesophageal diverticula and carcinoma (the latter sometimes through tracheal fistulisation) , are also risk factors. In one study of 252 cases from Brazil, alcohol was implicated in over 70% of patients with lung abscess. In cases of aspiration, anaerobes predominate and may include *Fusobacterium, Bacteroides* and *Peptostreptococcus* species. Most are mixed.

Clinical

The onset may be insidious and over weeks. Cough, productive of a foul sputum is a suspicious symptom, particularly in the setting of the risk factors mentioned. Chest pain, malaise and weight loss may occur. Haemoptysis occurs in 15% and may be life-threatening. About 10% are complicated by empyema formation. Clinical examination may reveal finger clubbing, poor gingivodental hygiene, and rarely amphoric breath sounds. Cachexia may be evident.

Investigation

The chest X-ray shows a cavitating lesion with an air-fluid level. CT is helpful, especially in ventilated patients, where the air-fluid level is less easy to demonstrate. Bronchoscopy is advised to rule out an obstructing lesion such as a carcinoma or foreign body. Secretions and lavage specimens may give the diagnosis.

On CT the question arises: is the fluid level from an empyema or lung abscess? Empyema tends to have smooth edges and forms an obtuse angle with the chest wall rather than an acute angle in lung abscess-the adjacent lung (vasculature, bronchial markings) are more compressed in an empyema. Fine needle aspirate can produce a high yield of the responsible organisms and may be followed by percutaneous drainage in suitable cases.

Differential diagnosis

1. Cavitating carcinoma-more likely if no risk factors and statistically. Carcinomas tend to be thicker walled. Pyrexia much less common.
2. Tuberculosis
3. Infected bulla
4. Empyema-see above
5. Hydatid-eosinophilia may be present
6. Wegener's granulomatosis
7. Fungi-aspergilloma

Treatment
The mainstay is antibiotics, Clindamycin and Metronidazole, intravenously at first and continued for 6 weeks. The majority (90%) respond in the first two weeks to this treatment.
Failure of antibiotic treatment, larger abscesses, and contamination of the contralateral side should prompt consideration of percutaneous drainage or surgery. The percutaneous route is usually performed with the Seldinger technique under CT guidance.
Surgery is reserved for a small number of cases (5% or less, 29 cases in UK 2002). Indications are

(i) Failure of medical treatment

(ii) Massive haemoptysis

(iii)Inability to exclude carcinoma

Lobectomy is the operation of choice. Early control of the bronchus is recommended to prevent contamination of the contralateral lung.

Prognosis
Mortality in the preantibiotic era was 30-60%. Now it should be in the region of 5%, although higher in the immunosuppressed population.

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Aspergillosis
Mycology
Aspergillus is a genus of saprophytic fungi totalling over 200 species, 38 of which are known to cause disease. *Aspergillus fumigatus* causes most human disease, but *A. niger* and *A .flavum* are also encountered. They were first described in 1729 by Micheli, and named after the aspergillum or holy water shaker. They are widespread in nature, being found on foods, soil especially compost heaps, in water (tanks, swimming pools, heating systems) and grow on damp walls (mildew). The spore elongates and forms a hyphae (filament) which is capped by the conidiophores; these are fed by the hyphae until they are mature and released as new spores of ~2μm diameter. It is estimated that 100-200 spores are breathed in each day. Spore levels increase with building demolition, of practical importance in hospitals with immunosuppressed patients such as post transplant.

Incidence
It is difficult to give a figure for true incidence. One study suggests 11% of TB cavities become colonised by an aspergilloma. SCTS figures from 2002 suggest resection for aspergillus is rare, 12 cases that year.

Pathophysiology/Clinical
Three forms of disease are encountered:

(i) Allergic Bronchopulmonary Aspergillosis (ABPA)-an allergy to the spores, that usually occurs on a background of asthma or cystic fibrosis-up to 10% of CF patients may be affected. Raised IgE and *Aspergillus* precipitins may be present. Clinically the patients present with cough, with production of brown plugs of sputum with increased wheeze. Steroids, sometimes with voriconazole are used to control the disease.

(ii) Invasive aspergillosis is almost exclusively confined to immunosuppressed patients, especially transplant recipients. It is the commonest pulmonary fungal infection in this group of patients and is a significant problem numerically, with 8% of bone marrow transplant patients and 11% of lung transplant patients developing the disease. HIV patients are also susceptible but at lower risk (3%). Lung infiltrates occur, and can lead on to cavitation.

The fungal colonies invade blood vessels, causing thrombosis and local haemorrhage, (seen on CT as the "halo sign") and entry to these vessels allows dissemination to other organs eg brain, liver to occur. Bronchoalveolar lavage or even thoracoscopic lung biopsy may be required to establish the diagnosis. Voriconazole is recommended first line treatment. The mortality is high, around 50% and is particularly high (c.90%) in bone marrow transplant recipients and those with brain involvement.

(iii)Aspergilloma usually occurs as a colonisation of a pre-existing cavity (TB, sarcoid, lung abscess, cyst etc). It is sometimes classified into simple and complex following the classification of Belcher and Plummer. Simple aspergillomas develop in a cyst lined by ciliated epithelium, whereas complex ones arise in a cavity due to pre-existing disease, they are thicker with CT evidence of surrounding parenchymal disease, usually tuberculosis. The majority (80-90%) are the latter. Although the disease may be asymptomatic, most patients present with haemoptysis (80-90%) which may be life threatening. Almost all have positive *Aspergillus* precipitins. Chest X ray may show a (typically apical) mass surrounded by a crescent of air (Monod's sign). Most progress, even if initially asymptomatic although a few regress and heal.

Series comparing medical treatment and resection, show a benefit for surgery. Systemic antibiotics and intracavernous instillation of antifungals have variable results. Surgery is controversial in asymptomatic patients and in the very ill, it carries a high risk. Haemoptysis is the main indication. The operation can be technically challenging due to dense adhesions. Air leak and bronchopleural fistulas are a particular complication-the latter may require thoracoplasty to treat as the underlying residual lung may be poorly expansile due to disease.

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INTERSTITIAL LUNG DISEASE

Dr. J. Reynolds - Birmingham Heartlands Hospital.

The idiopathic interstitial pneumonias are a group of lung diseases which are characterised by varying degrees of inflammation and fibrosis involving the interstitial tissues and also the airways and air spaces. They have been studied for around 50 years but received their modern classification by joint working groups of the American Thoracic Society (ATS) and the European Respiratory Society (ERS) ratified in June 2001[1].

The ATS/ERS report stressed the importance of collaboration between clinicians, radiologists and pathologists in the management of these conditions, a stance which is reiterated in the recent British Thoracic Society guidelines for the management of interstitial lung disease [2]. The correct framework for the discussion of these patients is therefore multidisciplinary.

If the HRCT shows features characteristic of usual interstitial pneumonia (UIP) which would equate with a clinical diagnosis and if the clinical picture is also in keeping with idiopathic pulmonary fibrosis, then the patient can be treated as such without the need for a surgical lung biopsy.

The HRCT may show appearances suggestive of an interstitial pneumonia other than UIP or appearances may be equivocal between UIP and another interstitial pneumonia such as NSIP. In this case the patient will require further assessment, usually initially with transbronchial biopsy and bronchial lavage. If these should prove inconclusive then a surgical lung biopsy may be required.

Some patients may proceed directly from HRCT to a surgical biopsy, particularly if the radiological abnormality has a sub-pleural predominance. There is a window of opportunity for surgical biopsies and should be done earlier rather than later. The risk increase substantially when the DLCO is less than 35% predicted.

Imaging plays an important role in patients with a suspected idiopathic interstitial pneumonia [3]. The chest radiograph may provide the first indication of such a disease being present. HRCT has a pivotal role in helping distinguish patients with UIP from those with other interstitial pneumonias. For patients who go on to have a bronchoscopic or surgical biopsy the HRCT can guide the optimum type and location of the biopsy.

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SURGERY FOR INTERSTITIAL LUNG DISEASE

Mr. R. Steyn – Birmingham Heartlands Hosptial.

Interstitial lung disease guideline: the British Thoracic Society in collaboration with the Thoracic Society of Australia and New Zealand and the Irish Thoracic Society
A U Wells, N Hirani and on behalf of the British Thoracic Society Interstitial Lung Disease Guideline Group, a subgroup of the British Thoracic Society Standards of Care Committee, in collaboration with the Thoracic Society of Australia and New Zealand and the Irish Thoracic Soc
Thorax 2008;63;v1-v58

Surgical lung biopsy in interstitial lung disease

- In prospective and retrospective studies, surgical lung biopsy has been shown to yield pathological diagnosis in 37–100% of cases.
- Two key considerations impact upon the decision to pursue a surgical lung biopsy in a patient with interstitial lung disease: (1) the risk associated with a surgical approach and (2) the recognition that histological assessment in interstitial lung disease has limitations and that the multidisciplinary integration of clinical and HRCT data, perhaps with the addition of transbronchial biopsy or bronchial lavage, is often sufficient to yield a confident diagnosis.
- Surgical lung biopsy, when required, should be performed before the initiation of treatment.
- A confident pathological diagnosis of interstitial pulmonary fibrosis or the other interstitial pneumonias can only be made if a surgical lung biopsy is obtained.
- A confident clinical diagnosis of interstitial pulmonary fibrosis can be reliably made in the presence of characteristic HRCT and clinical findings.
- If a surgical biopsy is performed in cases of suspected interstitial pneumonia, more than one biopsy specimen must be taken from more than one site, preferably from different lobes.
- Multiple multilobe lung biopsies are technically easier by video-assisted thoracoscopy (VATS) than by open lung biopsy.
- VATS is also associated with less early postoperative pain than open lung biopsy.
- It is recommended that the precise biopsy sites are based on HRCT appearances.
- In patients with suspected IIP, areas of intermediate abnormality or comparatively normal lung adjacent to areas of established honeycombing should be targeted with the specific aim of identifying UIP if present.

Referral for lung transplantation

- Patients should fulfil established selection criteria for transplant, thus generally excluding those over the age of 65 years and/or those with significant comorbidity.
- Referral to a transplant centre should be made if the disease is advanced (TLCO ,40% predicted) or progressive (>10% decline in FVC or >15% decline in FVC during 6 months of follow-up).

LUNG VOLUME REDUCTION SURGERY (LVRS) FOR END STAGE EMPHYSEMA

Mr. R. Page - Liverpool Heart and Chest Hospital.

Pathophysiology of Emphysema:-

Pathology	Physiological result	Clinical Features
Parenchymal loss	Alveolar loss□ □ surface area	□ pO2, □pCO2, □ DLCO,
	□ Elastic recoil > Bronchiolar compression	□ FEV1, □resistance, □work □ FVC, dyspnoea
Enlarged air spaces	Hyperinflation > uneven V/Q	□ pO2, □pCO2,□ RV, □ DLCO
	Chest hyperexpansion > Unfavourable mechanics	□ FEV1, □pCO2, dyspnoea

Surgical Technique

Resection of strip of most severely affected lung (70% of volume of upper lobe)
Shape residual lung to conform to thoracic cavity
Buttress staple / suture lines to reduce air leaks
Bilateral via sternotomy versus unilateral VATS

Mechanism of Action

1. Reduced airways resistance (increased outward traction)
2. Improved ventilation / perfusion matching
3. Improved chest wall mechanics (ribs less horizontal, diaphragm more domed)

Physiological benefits

1. Improved gas exchange
2. Improved gas flows
3. Reduced work of breathing
4. Subjective improvement in dyspnoea

Ideal candidate

"Pure" emphysema (dyspnoea, not sputum or bronchospasm = "pink puffer")
Hyperexpansion - clinical and radiographic, large residual volume, reasonable preservation of DLCO
Upper zone disease (CT + VQ scan assessment)
Absence of other disease (PA hypertension, ischaemic heart disease, diabetes, etc)
Significant limitation due to dyspnoea

NETT (National Emphysema Treatment trial. Ann Thorac Surg 2006;82:385-7)

1218 patients randomised to surgery or medical treatment
End-points = long-term survival and quality of life
Results:-
Upper lobe disease, poor exercise capacity:-
- LVRS better for survival and QOL
Upper lobe disease, good exercise capacity:-
- LVRS better for QOL, survival unaffected
Non-upper lobe disease:-
- Medical therapy better

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ENDOSCOPIC MANAGEMENT OF ESOPHAGEAL DISEASE

Dr. D. Low - Virginia Mason Medical Centre, Seattle, U.S.A.

Presentation Goals:

Comment and compare various outcome measures and approaches to endoscopic therapy for:

1. Zenker's diverticulum
2. Gastroesophageal reflux disease
3. Role of capsule endoscopy in esophageal disease
4. Definitive treatment of Barrett's esophagus, including discussion of:
    a. Argon beam coagulation
    b. Photodynamic therapy
    c. Endoscopic mucosal resection
    d. Radio frequency ablation
    e. Cryotherapy
5. Comparison of endoscopic and surgical management of Barrett's esophagus
6. Early esophageal adenocarcinoma
7. Esophageal perforation and anastomotic leaks
8. Benign esophageal tumors

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EMERGENCY TREATMENT OF DISEASE OF THE ESOPHAGUS

Dr. D. Low - Virginia Mason Medical Centre, Seattle, U.S.A.

Presentation Goals:

The assessment and treatment options for a variety of esophageal conditions will be reviewed including:

1. Esophageal perforation
2. Esophageal anastomotic leak
3. Esophageal foreign bodies
4. Esophageal trauma

References:

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