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Current Management of Pleural Disorders

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ABSTRACT

A wide range of conditions can present with plural involvement. For this reason patients with pleural disorders may be seen by a number of different specialists. The most common pleural disorders include pleural effusion and pneumothorax. Pleural effusion and pneumothorax are defined as accumulation within the pleural space of fluid and air respectively. The most common disorders responsible for over 90% of pleural effusions include congestive heart failure, malignancy, infection and pulmonary embolism. The pneumothorax can be divided into primary spontaneous, secondary, iatrogenic or traumatic. This review article will discuss our current understanding behind the pathophysiology of pleural effusion. Common causes and less common conditions resulting in pleural effusion will be described. Investigations and management of patients with pleural dsiorders will also be discussed. In addition, recent advances in our understanding of etiology and management of pneumothorax will be covered. Investigations and management of pleural disorders require an understanding of the underlying pathology as well as the expertise in currently available interventional procedures. The main challenge remains to manage patients in accordance to the current guidelines, which is best achieved through specialist services. However, the knowledge related to pleural disorders remains of importance to many specialists.

Keywords: Pleura, Pleural Effusion, Pneumothorax

1. INTRODUCTION

Pleural disorders remain a common clinical problem relevant to a number of specialists (Du Rand and Maskell, 2010; Light, 2011a). For example, around a quarter of referrals to respiratory clinics are related to pleural diseases (Light, 1997). The prevalence of pleural disorders has been estimated at around 3000 people per million population each year (Du Rand and Maskell, 2010). The annual incidence of pleural effusion in the United States of America (USA) may be as high as 1 million (Light, 1997; 2011b). The most common disorders responsible for over 90% of diagnosed pleural effusions include congestive heart failure, malignancy, infection and pulmonary embolism (Marel *et al*., 1993). In epidemiological studies congestive heart failure and pulmonary embolism were responsible for pleural effusions in 800,000 and between 300,000 to 500,000 cases annually in the USA respectively (Light, 2002; 2010; Marel *et al*., 1993; Porcel and Light, 2008). Malignant pleural effusion affects more than 175,000 people each year in the USA and more than 40,000 in the United Kingdom (Bennet and Maskell, 2005). Pulmonary infection such as pneumonia may result in pleural effusion in around 40% of patients with an estimated 60,000 cases of pleural infection per year in the USA and its associated mortality of approximately 15% (Chapman and Davies, 2004; Ferguson *et al*., 1996; Light, 2006a; Sahn, 1993). In the USA between 1993 and 2003 there were 7549 cases of pleural effusion due to tuberculosis representing 18% of all cases (Baumann *et al*., 2007). The incidence of pneumothorax has been described between 18-28/100,000 cases per annum for men and 1.2/100,000 for women (MacDuff *et al*., 2010). In the UK, hospital admissions due to pneumothorax have been estimated at 16.7/100,000 per year for men and 5.8/100,000 per year for women (Gupta *et al*., 2000). The same rates for a person consulting a physician were 24.0/100,000 each year for men and 9.8/100,000 each year for women. Slightly lower rates at 7.9/100,000 per year were reported in a study from the USA (Melton *et al*.,

1979). This review will discuss the current understanding, investigations and management of common pleural disorders such pleural effusion, pneumothorax, infection of pleura as well as some of the less common conditions that may affect the pleura.

1.1. Characteristics of Pleural Fluid

The pleura is a monolayer of mesothelial cells covering the lung and the inner surface of the chest cavity and creating the pleural space (Finley and Rusch, 2011). In healthy adults the volume of pleural fluid has been reported as 0.26±0.1 mL kg^{-1} with its main role being to lubricate the visceral and parietal pleural surfaces allowing for the breathing movement (Noppen, 2001). There are over 50 causes of pleural effusions. Pleural effusion occurs when there is more fluid entering the pleural space than that being removed (Porcel and Vives, 2006). This can take place when there is an increased formation of the fluid or reduced re-absorption (Hooper *et al*., 2010a). The precise mechanisms of pleural fluid formation have not yet been fully understood but vary depending on the aetiology. For example, increased pulmonary capillary pressure may be seen in cardiac failure, increased permeability in infection, decreased oncotic pressure in hypoalbuminemia, increased membrane permeability and obstructed lymphatic flow in malignancy (Porcel and Light, 2006; Hooper *et al*., 2010b). Light *et al*. (1972) described simple diagnostic criteria to differentiate plural effusions into trasudates and exudates. These 'Light's criteria' rely on sampling pleural fluid and analysing pleural effusion and serum ratio of protein and Lactate Dehydrogenase (LDH). Over the years these criteria have been proved to have diagnostic accuracy of 96% in separating transudates and exudates and compared favorably with other methods such as assessment of pleural fluid cholesterol or albumin (Heffner *et al*., 1997; Romero *et al*., 2000). The more common causes of transudative effusions are congestive heart failure and liver cirrhosis and those of exudative effusions malignancy, infection and pulmonary embolism.

In healthy adults, pleural fluid contains less than 1800 cells μL^{-1}; mainly mesothelial cells, macrophages (75%) and lymphocytes (23%) as well as small amount of eosinophils and neutrophils (Noppen, 2001). Pleural fluid is normally alkaline at pH around 7.6. However in some conditions such as empyema, tuberculosis, cancer, rheumatoid arthritis or drug reactions pleural fluid pH may be below 7.2 (Good *et al*., 1980). In addition, to cellular component pleural fluid also contains bicarbonate, LDH, proteins and glucose (Noppen, 2001). There are a number of disorders that may result in abnormal cell composition of pleural fluid. Neutrophils are the most common cells in pleural effusion due to acute conditions and the presence of lymphocytes suggests more chronic process. Thus exudates with predominance (over 80% of the total cell count) of lymphocytes are suggestive of tuberculosis, lymphoma, leukaemia, rheumatoid arthritis or post cardiac surgery related pleural effusions (Sahn, 1995). Neoplastic pleural effusion can also be characterised by lymphocyte predominance usually 50-70% of total cell count. As most transudates are lymphocytic predominant, congestive cardiac failure remains the most common cause of this type of pleural effusion. Eosinophilic pleural effusion defined as more than 10% of total cell count is relatively rare and may be seen in up to 16% of exudative effusions with the most common causes being hemothorax, pneumothorax, malignancy, infection, drug reactions, pulmonary embolism or benign asbestos related pleural effusions (Martinez-Garcia *et al*., 2000; Kalomenidis and Light, 2003). Certain characteristics of cell count and other measurements such as low pH my point out towards specific conditions. For example, in the context of low pleural fluid pH, increased neutrophil count would suggest empyema, raised eosinophil count drug reaction and increased lymphocytes count tuberculosis or cancer. Rarely chyle a lymphatic fluid enriched in lymphocytes, immunoglobulins and lipids may be detected in the pleural space (Townshend *et al*., 2007). Chylothorax has been described mainly due to trauma, lymphoma and less frequently due to Lymphangioleiomyomatosis (LAM) or yellow nail syndrome. The treatment may include a low fat diet containing medium chain triglycerides, which reduces production of chyle or surgical ligation of the duct, which may be successful in up to 90% of patients.

1.2. Common Causes of Transudative Pleural Effusion

Cardiac failure and liver disease related pleural effusions are most commonly classified as transudates with the proposed underlying mechanisms being low albumin level and ascitic fluid movement across the diaphragmatic defect as a result of an increased intra-abdominal pressure (Kinasewitz and Keddissi, 2003). The management of pleural effusion due to liver disease may be difficult and relies on the treatment of its cause however, in refractory cases pleurodesis, indwelling

pleural catheter or repeated thoracocenthesis may be required (Milanez de Campos *et al*., 2000; Gur *et al*., 2004). Overall the most common cause of transudate remains congestive cardiac failure and in this context pleural effusions are usually bilateral although in a fifth of cases small unilateral effusions can occur (Porcel and Light, 2006). Interestingly a quarter of pleural effusions on the background of cardiac failure may be mislabelled as exudates (Johnson, 2000; Bielsa *et al*., 2012). In those patients therefore measuring the albumin rather than the protein gradient may be more accurate. Recent studies have shown that pleural fluid N terminal Pro-Brain Natriuretic Peptide (pro-BNP) levels may have high sensitivity and specificity for diagnosing cardiac failure related pleural effusion (Porcel *et al*., 2004). In the context of cardiac disease, pleural effusion related to Coronary Artery Bypass Grafting (CABG) surgery should also be mentioned. In most cases the amount of fluid is small and resolves spontaneously. Light *et al*. (1999) described two types of pleural effusion that occur post CABG surgery. The first type most likely traumatic in origin occurs within 28 days from CABG surgery and is usually blood stained and eosinophils predominant. The second type is most likely immunologic in origin and occurs at a later stage over 30 days following surgery and is lymphocytic in nature. In those patients, other causes of pleural effusion need to be excluded including infection, chylothorax, cardiac failure or pulmonary embolism. Pleural effusion due to isolated right heart failure has also been described and can occur with idiopathic pulmonary arterial hypertension or in association with connective tissue diseases (Brixey and Light, 2011; Luo *et al*., 2011).

1.3. Malignant Pleural Effusion

Malignancy remains one of the most common causes of pleural effusion (Roberts *et al*., 2010). Pleural effusions occur in around half of patients with metastatic malignancy and usually presents with dyspnoea or chest pain (Shaw and Agarwal, 2004). Over three quarters of malignant pleural effusions are secondary to neoplasm of the lung, breast, ovaries, lymphomas, genitourinary and gastrointestinal tract (Heffner and Klein, 2008). Mesothelioma remains the most common primary pleural cancer associated with pleural effusion. Malignant pleural effusion characterized by the presence of cancer cells in the pleural space usually defines advanced stages of cancer with median survival around 3 to 6 months with palliation remaining the main goal of treatment (Lanken *et al*., 2008). It is worth remembering that patients with cancer can develop pleural effusion without the presence of cancer cells in the pleural fluid, so called paraneoplastic pleural effusion, as well as those associated with concomitant conditions such as pulmonary embolism or cardiac failure (Heffner, 2008). Malignant pleural effusions are exudates by nature due to the high protein and the LDH levels although they can present occasionally as transient transudates in cases of carcinomatous lymphangitis (Fernandez *et al*., 2000). Thoracocenthesis which is nowadays performed under ultrasound guidance allows for sampling of pleural fluid for cytological analysis (Kastelik *et al*., 2009; Phillip *et al*., 2003). The diagnostic yield from a single plural fluid cytological analysis has been quoted as up to 60% overall but with a much lower yield at only 20-30% in cases of mesothelioma. Occasionally, additional tests such as pleural fluid amylase may be of help (Fahim and Kastelik, 2010). A repeat aspiration only slightly improves the diagnostic yield. Thoracoscopy, surgical or medical, has been perceived as the ‘gold standard’ with a diagnostic yield of 93-97% (Rodriguez-Panadero, 2008). Medical thoracoscopy also called ‘pleuroscopy’ is performed under local anaesthesia and conscious sedation (Rodriguez-Panadero *et al*., 2006). Medical thoracoscopy involves insertion of a trocar through which then an optical telescope, a light source and biopsy forceps can be introduced into the pleural cavity. Surgical Video Assisted Thoracosopic Surgery (VATS) is performed by a thoracic surgeon in an operating theatre under general anaesthesia. Both techniques allow for drainage of the pleural fluid, pleural biopsies under direct vision and talc pleurodesis (Rodriguez-Panadero *et al*., 2006).

The management of patients with malignant pleural effusion requires expertise and a multidisciplinary approach in order to provide individualised high quality treatment that is in accordance with the current guidelines (Roberts *et al*., 2010). Patients with poor performance status who are expected to survive less than 3 months can be managed by performing repeated thoracocentheses if there is evidence of improvement in their symptoms (Heffner, 2010). In the majority of patients fluid will re-accumulate within 1 month, therefore thoracocenthesis is not an optimal therapy for patients with longer expected survival who may require more definitive treatment including medical or surgical pleurodesis or an insertion of indwelling pleural catheter (Heffner, 2008; Rodriguez-Panadero and Romero-Romero, 2011; Viallat *et al*., 1996). Pleurodesis is a procedure that allows for symphysis between the visceral and parietal pleura that prevents the accumulation of the pleural fluid (Rodriguez-Panadero

and Montes-Worboys, 2012). Talc is the most effective chemical pleurodesis agent and has been shown to be safe and effective treatment of malignant pleural effusion (Dresler *et al*., 2005; Janssen *et al*., 2007). Medical pleurodesis using chest drain and talc slurry is the least invasive method with acceptable success rates (Gondker *et al*., 2011; Dresler *et al*., 2005). There is evidence that short term chest tube drainage of malignant pleural effusion followed by talc slurry pleurodesis is as effective as longer protocols with the benefits of reduced hospital length of stay (Goodman and Davies, 2006; Spiegler *et al*., 2003). Thoracoscopy with talc poudrage pleurodesis has been shown to be possibly a more effective method for managing pleural effusion (Janssen *et al*., 2007). A number of studies suggest a success rate from thoracoscopy and talc poudrage at 1 month at 84% compared with those of 60% for pleurodesis with talc slurry through a chest drain (Rahman *et al*., 2010). However Dresler *et al*. (2005) demonstrated in a randomised controlled study that success rates at 1 month for thoracoscopic talc poudrage of 78% and that for talc slurry through intercostal chest drain of 71%. Although patients with lung or breast cancer had significantly higher success rates for thoracoscopic pleroudesis at 82% compared with that using intercostal chest drain and talc slurry. Comfort, safety and symptoms of fatigue seemed to favor thoracoscopy. Another option for the management of malignant pleural effusion involves insertion of small bore indwelling pleural catheters, which can be performed on outpatient basis and have been shown to improve quality of life and symptoms of breathlessness and have low complication rates (Putnam *et al*., 2000). The indwelling pleural catheters have acquired wide use especially in patients with malignant pleural effusion and trapped lung, those who had failed pleurodesis and have been shown to have 45% rates of spontaneous pleurodesis (Putnam *et al*., 2000; Van Meter *et al*., 2011). In a recent study indwelling pleural catheters were placed during medical thoracoscopy with talc pleurodesis with success rates quoted at 92% and associated low hospitalization stay of 1.79 days (Reddy *et al*., 2011).

1.4. Pleural Effusion Due to Pulmonary Embolism

Computed tomography pulmonary angiogram (CTPA) can detect pleural effusion in around 47% of patients with pulmonary embolism (Porcel *et al*., 2007; Bynum and Wilson, 1978). The mechanisms of pleural effusion in the context of pulmonary embolism are not fully understood but may be related to the presence of pulmonary infarction (Light, 2010). The fluid is usually a neutrophil predominant exudate (Porcel *et al*., 2007). Typically, pleural effusions are small and unilateral occupying less than 30% of the hemothorax, appear soon after the symptoms of thromboembolism have began and tend to reach their maximal size early in the course of the disease. Pulmonary infarction may be associated with larger pleural effusions that may clear more slowly (Bynum and Wilson, 1978). In patients who had massive pulmonary embolism pleural effusion may be seen on a CTPA in around 79% of cases (Findik *et al*., 2008). In cases when the diagnosis of pulmonary embolism was delayed pleural effusion may be loculated (Porcel *et al*., 2007). It is important to remember that potential complications of pleural effusion due to pulmonary embolism include hemothorax and infection. Therefore if pleural effusion on the background of pulmonary embolism is increasing in size diagnostic thoracoenthesis should be considered.

1.5. Pleural Infection

Whilst pleural infection remains relatively common, due to the advances in imaging techniques and antimicrobial therapies, the diagnosis and the outcomes of this condition have improved significantly (Davies *et al*., 2010; Heffner *et al*., 2010; Wrightson and Maskell, 2012). From the bacteriological aspects, for community acquired pleural infection the most common detected agents include Streptococcus milleri, Streptococcus pneumonia, Staphylococcus aureus and anaerobes (Maskell *et al*., 2005). For hospital acquired pleural infection the following bacteria have been commonly reported; staphyloccci including Methicillin-Resistant Staphylococcus Aureus (MRSA), gram negative bacteria such as Escherichia coli or Klebsiella species as well as anaerobes (Maskell *et al*., 2006). Light (1995) suggested a classification of parapneumoninc effusion and empyema into 7 classes depending on the size of the effusion, pH measurements, LDH levels and microbiological characteristics. The current guidelines describe three developmental stages of empyema associated with pneumonia; a simple exudate, fibrinopurulent stage and organising stage with scar tissue formation (Davies *et al*., 2010). Although parapneumonic effusion of less than 10 to 20 mm in depth, should resolve spontaneously majority of parapneumonic effusions would require sampling (Skouras *et al*., 2010). The sampling should be performed under image usually thoracic ultrasound guidance. Pleural fluid analysis including pH, glucose, LDH, microbiology including cultures for tuberculosis

and cytology provides important information regarding the management. The characteristics of complicated parapneumonic effusion include pH less than 7.2, glucose less 2.2 mmoL L^{-1} (40 mg dL^{-1}) and LDH greater than 1000 IU L^{-1} with empyema being defined by presence of frank pus. It should be noted that occasionally pH can be alkalotic in cases of empyema due to proteus species. There is evidence that in order to increase the diagnostic yield by around a fifth the aspirated plural fluid should not only be collected as standard culture but also inoculated into aerobic and anaerobic blood culture bottles (Menzies *et al*., 2011).

Complicated parapneumonic effusion and empyema will require intercostal chest drain insertion. Whilst historically a large bore chest drain was recommended recent evidence suggests that smaller size 10-14 F may be adequate in managing those patients (Light, 2011b). Antibiotic therapy should be initiated as soon as pleural infection is identified. The choice of antimicrobial agents will depend on the microbiological culture results. In community acquired pleural infection aminopenicillins with beta-lactamase inhibitor may be a good first choice of treatment although other antibiotics such as carbapenems, clindamycin or cephalosporins can also be considered. For hospital acquired pleural infection antimicrobial agents should also include MRSA cover until confirmation of pathogens from microbiological results becomes available (Maskell *et al*., 2006). If these measures are not sufficient and sepsis and the infected fluid are not controlled a proportion of patients may require surgery in the form of thoracoscopy with breakdown of adhesions or thoracotomy with decortications (Petrakis *et al*., 2010). Surgical options for an early usually fibrinopurulent stage of empyema may include VATS and in organised phase open thoracotomy and decortications may be the only option. The timing of surgery is a very important factor in influencing the mortality from empyema. There has been a long debate whether thrombolytic agents are of use in the treatment of pleural infection. In a recent study, intra-pleural tissue Plasminogen Activator (t-PA) and DNase were shown to reduce frequency of surgical referrals, duration of hospital stay and improved the drainage of infected fluid in patients with pleural infection (Rahman *et al*., 2011). Interestingly, treatment with DNase alone or t-PA alone was ineffective and the authors concluded that in patients in whom standard therapy failed a combination of intra-pleural t-PA and DNase may be considered. The outcomes of pleural infection with modern management are generally good with pleural thickening seen in 14% of cases, fortunately with very little functional impairment and the long term mortality of around 14% mainly due to co-morbidities (Davies *et al*., 1999; Farjah *et al*., 2007; Light, 2006a).

1.6. Pleural Effusion Due to Less Common Causes

There are a number of conditions causing pleural effusion, however amongst the less common causes connective tissue disorders, medications and pleural effusion in immunocompromised host are of particular interest. Around 4% of patients with rheumatoid arthritis may develop pleural effusion usually associated with the thickening of the visceral and parietal pleura (Avnon *et al*., 2007). Pleural effusion in rheumatoid arthritis is characterised by a low pH, low glucose with cell composition varying from lymphocyte to neutrophil predominant (Balbir-Gurman *et al*., 2006; Hunninghake and Fauci, 1979). In half of patients pleural effusion improves spontaneously within 4 weeks but in a fifth it may persist for over 12 months. Occasionally, patients with rheumatoid arthritis may develop chronic pleural effusion with a high content of lipids so called pseudochylus effusion. In addition, patients with rheumatoid arthritis have an increased risk of developing empyema. Pleural involvement may also be seen in Systemic Lupus Erythematosus (SLE) with around 10% of cases of pleuritis being reported as the first manifestation of this disease (Pines *et al*., 1985). Pleural effusion associated with SLE is characterised by reduced levels of complement and raised fluid to serum antinuclear antibody ratio of above 1. Pleural effusion has also been reported in association with conditions such as systemic sclerosis or dermatomyositis (Hunninghake and Fauci, 1979).

Clinicians should also be aware of pleural effusion due to medications and that occurring in immunocompromised host. Medications such as methotraxate, amiodarone, phenytoin or pergolide have been shown to cause pleural diseases including pleural thickening or pleural effusion (Kastelik *et al*., 2002). There are number of conditions that can result in pleural disorders in immunocompromised patients such as parapneumonic effusion, tuberculosis, Kaposi's sarcoma, thoracic non-Hodgkin lymphoma, primary effusion lymphoma and spontaneous pneumothorax (Beck, 1998; Afessa, 2000; 2001). There have been a small number of patients describe who have bilateral pleural effusions so called Contarini's syndrome when different cause for pleural fluid on each side can be confirmed (Porcel *et al*., 2012). In those cases common findings are parapneumonic effusion with contralateral transudate due to cardiac failure or malignant pleural effusion due to for example, ovarian cancer on one side with contralateral chylus pleural effusion as a result of

obstruction of thoracic duct from metastatic deposits (Lawton *et al*., 1985; Kutty and Varkey, 1978).

1.7. Investigations of Pleural Effusion

Investigation of pleural effusion should involve a systematic approach (Porcel and and Vives, 2006; Hooper *et al*., 2010a). Clinical history remains important in determining the underlying cause of pleural effusion and should include assessment of medications and occupational aspects such as asbestos exposure. Chest radiograph can detect pleural effusion greater than 200 ml in volume. In recent years bedside thoracic ultrasonography has become more widely performed by pulmonary specialists (Kastelik and Arnold, 2012). Thoracic ultrasound allows for diagnosis and characterisation of pleural effusion (Feller-Kopman, 2009; Koenig *et al*., 2011). More importantly there is good evidence to suggest that ultrasound guided pleural procedures is associated with lower rates of complications (Grogan *et al*., 1990; Jones *et al*., 2003). When performed by an experienced operator certain findings on thoracic ultrasound may suggest the origin of pleural effusion such as malignancy, which may be supported by the presence of more than 1 cm pleural thickening, pleural nodularity and diaphragmatic thickening of more than 7 mm (Qureshi *et al*., 2009). Similarly, specific findings on CT may suggest the origin of pleural effusion such as pulmonary embolism, infection or malignancy. Thus presence on CT images of pleural nodules, nodular thickening infiltration of the diaphragm or circumferential or mediastinal pleural thickening may support the diagnosis of malignancy (Arenas-Jimenez *et al*., 2000).

Thoracocenthesis allows for sampling of pleural effusion and biochemical, cytological and microbiological analysis that assists in establishing the underlying cause. In around 15% of cases with exudates pleural effusion diagnosis cannot be reached (Light, 2006b). Medical thoracoscopy under local anaesthesia has been shown not only to improve diagnostic rates but also to influence the management outcomes in the majority of patients (Blanc *et al*., 2002; Harris *et al*., 1995). The diagnostic yields from pleural biopsies taken during medical thoracoscopy are at around 97% and for this reason this procedure is becoming more widely used in the investigations of patients with pleural effusion (Lee *et al*., 2010; Rahman *et al*., 2010; Sakuraba *et al*., 2006). Ideally, patients with pleural disorders should be investigated through specialist pleural services that include designated pleural clinics to provide rapid diagnosis using ambulatory manner and limited number of invasive procedures (Hooper *et al*., 2010b). The specialist pleural services should be run by pulmonologists with support from radiologists, pathologists, oncologist and thoracic surgeons. This multidisciplinary approach to manage patients with pleural disorders would aim to provide rapid diagnosis and the most appropriate therapy.

1.8. Pneumothorax

Pneumothorax defined as an air in the pleural cavity can be divided into primary secondary or traumatic (MacDuff *et al*., 2010; Tschopp *et al*., 2006). Patients usually present with pleurictic chest pain and dyspnoea and diagnosis is confirmed on a chest radiograph. Pneumothorax has to be distinguished from conditions such as pneumomediastinum (Faruqi *et al*., 2006). Primary spontaneous pneumothorax is not associated with underlying lung diseases whereas secondary pneumothorax occurs on the background of existing lung disorders most commonly chronic obstructive pulmonary disease, cystic fibrosis or interstitial lung diseases. Recent evidence suggests that primary spontaneous pneumothorax may be associated with changes within the pleura as well as being caused by the rupture of the blebs or bullae with the most commonly described abnormalities including emphysema like changes, plural inflammation and porosity which are areas of inflammation on the visceral pleura most likely due to mesothelial cells being replaced by inflammatory cells (Grundy *et al*., 2012; Haynes and Baumann, 2011). However, there is evidence that in patients with primary spontaneous pneumothorax there is an increased risk of recurrence on the ipsilateral as well as contralateral site if the blebs are confirmed on the CT imaging (Sihoe *et al*., 2000; Huang *et al*., 2007).

International guidelines provide recommendations for the management of patients with pneumothorax that include either observation, thoracocenthesis with aspiration of air or insertion of an intercostal chest drain depending on symptoms and the size of the pneumothorax (Baumann *et al*., 2001; Macduff *et al*., 2010). Patients with small pneumothorax without significant breathlessness as well as a selected group of patients with a large pneumothorax who are asymptomatic can be observed and may not require any intervention. For patients with large primary spontaneous pneumothorax an aspiration using 16-18 G cannula can be considered and if not successful an insertion of chest drain should be performed. British Thoracic Society (BTS) guidelines recommend for a large over 2 cm in

size primary spontaneous pneumothorax and for the secondary pneumothorax chest drain insertion although for a smaller size pneumothorax a simple aspiration may be considered (Macduff *et al*., 2010). The American College of Chest Physicians (ACCP) guidelines differ from the BTS guidelines with regards to their description of a large pneumothorax, which they describe as equal as or more than 3 cm apex-to-cupola distance. More importantly the ACCP panel overall for treatment of pneumothorax favored insertion of intercostal chest drain with a simple aspiration being thought to be rarely appropriate. The reason for this difference reflects the paucity of data from randomised control studies. The recurrence of primary spontaneous pneumothorax after treatment with insertion of chest drain remains between 30 and 55% and it increases to over 62% for the second primary spontaneous pneumothorax raising the debate whether surgical treatment of primary spontaneous pneumothorax should be offered more widely (Chambers and Scarci, 2009).

Surgical management including VATS is relatively safe and results in a definitive treatment of pneumothorax (risk of recurrence of less than 3%) through the resection of a bullous lesion, emphysematous pleural blebs and emphysema like changes together with pleurodesis (Chambers and Scarci, 2009). There is no evidence that the size of the blebs affects the rates of recurrence of pneumothorax but recurrence may be higher if bullectomy is performed without pleurodesis 20% versus 4%, as bullectomy is thought to be unable to remove all the areas of emphysema like changes. It is generally agreed that surgical treatment should be considered in the recurrent ipsilateral pneumothorax, contralateral pneumothorax or bilateral simultaneous pneumothoraces (Baumann *et al*., 2001; Macduff *et al*., 2010). Currently surgery is suggested for the management of patients with first primary spontaneous pneumothorax in whom there is persistent air leak lasting for more than 5 to 7 days or in cases with professions at risk such as pilots or those occurring during pregnancy (MacDuff *et al*., 2010). The literature suggests that VATS is economically justified as an initial treatment option of primary spontaneous pneumothorax (Chou *et al*., 2003; Rena *et al*., 2008; Schramel *et al*., 1996). In fact, there is evidence that VATS may be more effective than intercostal chest drain drainage for treatment of first episode of primary spontaneous pneumothorax resulting in 40% reduction in costs and reduction in the length of hospital stay (Schramel *et al*., 1996; Sawada *et al*., 2005). Pleurodesis with large-particle talc performed during thoracoscopy has been shown to be a safe and cost-effective as well as preventing recurrence of primary spontaneous pneumothorax (Cardillo *et al*., 2006). There is evidence that talc pleurodesis during medical thoracoscopy may be more cost effective than drainage with intercostal chest drain (Tschopp *et al*., 2002). Chen *at al*. (2008) observed that following unsuccessful aspiration of primary spontaneous pneumothorax, VATS compared with chest drain treatment was associated with a shorter hospital stay and lower rates of overall failure and recurrence. Others recommended VATS for the first episode of primary spontaneous pneumothorax if the CT of the thorax identified blebs or bullae (Sawada *et al*., 2005). Amongst the ACCP panel 15% of members would consider offering patients a surgical intervention to prevent a recurrence after the first episode of spontaneous pneumothorax. The BTS guidelines suggest that increasingly patient choice may affect the decision whether to proceed with surgical treatment of a first episode of primary spontaneous pneumothorax. Newer techniques for managing primary spontaneous pneumothorax are becoming available such as needlescopic VATS procedure or insertion of endobronchial valves for patients with persistent air leak and these will without doubt influence our treatment strategies for this common problem (Chou *et al*., 2009).

2. CONCLUSION

In conclusion, pleural diseases are common. With new evidence emerging the management of patients with pleural disorders is changing. Increasingly the evidence is provided through randomised controlled studies. Many physicians and surgeons are involved in managing patients with pleural disorders. The main challenge remains in making sure that patients are managed according to the current guidelines. This is probably best achieved if the patients with pleural diseases are treated by specialists with access to a multidisciplinary approach of pleural services. Therefore, clinicians should be aware of the current management options for patients with pleural disorders.

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