113 年奇美醫院胸腔內科臨床病例討論會

日期：中華民國 113 年 02 月 06 日(星期二)

時間及地點：16:00-17:00

課程活動題目：胸腔影像綜合討論會：Aspiration syndrome

主講人　：謝俊民主任

主辦單位：奇美醫院胸腔內科

課程地點：10 樓討論室

教育積分：台灣胸腔暨重症加護醫學會

參加對象：主辦單位所屬院內醫師

聯絡人　：楊穎潔（06-2812811-57132）

摘要：

Introduction：

Aspiration of a sufficient amount of gastric acid produces a chemical pneumonitis characterized by acute dyspnea and wheezing with hypoxemia and infiltrates on chest x-ray in one or both lower lobes. Clinical findings depend on the extent of endobronchial obstruction and ranges from acute apnea to persistent cough. Although the aspiration of oral anaerobes can initially lead to infiltrates, it ultimately results in putrid sputum, tissue necrosis and pulmonary cavities. In 75% of cases, the clinical course of an anaerobic abscess is indolent and mimics pulmonary tuberculosis, with cough, shortness of breath, chills, fevers, night sweats, weight loss, pleuritic chest pain and blood-streaked sputum for several weeks. Patients with anaerobic abscesses are usually prone to aspiration of oropharyngeal contents and have peridontal disease.

Chest Radiography

Chest x-ray is more sensitive than physical exam for detection of pulmonary infiltrates. Chest x-ray can confirm the presence and location of the pulmonary infiltrate, assess the extent of the pulmonary infection, detect pleural involvement, pulmonary cavitation, hilar lymphadenopathy and gauge the response of antibiotic therapy. However, chest x-ray may be normal when the patient is unable to mount an inflammatory response or is in the early stage of an infiltrative process.

Chest computed tomography

High-resolution CT can improve the accuracy of the diagnosis of pneumonia, especially when the process involves lung obscured by the diaphragm, liver, ribs, clavicles or heart.　In the

patient with an uncomplicated course, chest x-ray need not be repeated before discharge, since the resolution of infiltrates may take up to 6 weeks after initial presentation. At times, CT may be especially helpful in distinguishing different processes: pleural effusion versus underlying pulmonary consolidation; hilar adenopathy versus pulmonary mass; and pulmonary abscess versus empyema with an air-fluid level.

The anatomic localization of the inflammatory process can have diagnostic implications. Most pulmonary pathogens produce focal lesions. A multicentric distribution suggests hematogenous infection. Hematogenous pneumonia appears as multiple areas of pulmonary infiltration that subsequently may cavitate. A diffuse distribution suggests P. jeroveci, CMV, hantavirus, measles virus or herpes zoster virus. Pleurisy and hilar nodal enlargement are unusual with PCP and CMV pneumonia. Oral anaerobes, S. aureus, S. pneumoniae serotype III, aerobic gram-negative bacilli, M. tuberculosis, and fungi can produce tissue necrosis and pulmonary cavities. In contrast, H. influenza, M pneumoniae, viruses and most other serotypes of S pneumoniae almost never cause cavities. Apical disease, with or without cavities, suggests reactivation tuberculosis. Anaerobic abscesses are located in dependent, poorly ventilated, and poorly draining bronchopulmonary segments and characteristically have air-fluid levels, unlike the well-ventilated, well drained upperlobe cavities caused by M. tuberculosis, an obligate aerobe. Mucor and Aspergillus invade blood vessels and cause pleural-based, wedge-shaped area of pulmonary infarction which may subsequently cavitate.