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 6 月 11 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：Pulmonary Embolism

主講人　：蔣士仁主任

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

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摘要：

Introduction:

When venous thrombi dislodge from their site of formation, they embolize to the pulmonary arterial circulation or, paradoxically, to the arterial circulation through a patent foramen ovale or atrial septal defect. About half of patients with pelvic vein thrombosis or proximal leg deep venous thrombosis (DVT) have pulmonary thromboembolism (PE), which is usually asymptomatic. Isolated calf vein thrombi pose a lower risk of PE, but they are the most common source of paradoxical embolism. With increased use of chronic indwelling central venous catheters for hyperalimentation and chemotherapy, as well as more frequent insertion of permanent pacemakers and internal cardiac defibrillators, upper extremity venous thrombosis is becoming a more common problem. These thrombi may also embolize and cause PE Dyspnea is the most frequent symptom of PE, and tachypnea is its most frequent sign. Whereas dyspnea, syncope, hypotension, or cyanosis indicates a massive PE, pleuritic pain, cough, or hemoptysis often suggests a small embolism located distally near the pleura. On physical examination, young and previously healthy individuals may simply appear anxious but otherwise seem deceptively well, even with an anatomically large PE. They may only have dyspnea with moderate exertion. They often lack “classic” signs

such as tachycardia, low-grade fever, neck vein distention, or an accentuated pulmonic component of the second heart sound. Sometimes, a paradoxical bradycardia occurs.

In older patients who complain of vague chest discomfort, the diagnosis of PE may not be apparent unless signs of right heart failure are present. Unfortunately, because acute coronary ischemic syndromes are so common, one may overlook the possibility of life-threatening PE and may inadvertently discharge these patients from the hospital after the exclusion of myocardial infarction with serial blood tests to detect cardiac injury and serial electrocardiograms.

Chest radiographic findings:

A normal or near-normal chest x-ray in a dyspneic patient suggests PE. Well-established abnormalities include focal oligemia (Westermark's sign), a peripheral wedged-shaped density above the diaphragm (Hampton's hump), or an enlarged right descending pulmonary artery (Palla's sign).

Computed tomographic findings:

Computed tomography (CT) of the chest with intravenous contrast (ordinarily, 100 mL administered at 3 to 4 mL/s via an antecubital vein) is superseding lung scanning (see below) as the principal imaging test for the diagnosis of PE. Chest CT effectively diagnoses large, central PE. New generation multislice scanners image the entire thorax with 1-mm thin sections during a single 12- to 15-s breath-hold and can detect peripherally located thrombi in fifth order branches. In patients without PE, the lung parenchymal images may establish alternative diagnoses not apparent on chest x-ray that explain the presenting symptoms and signs, such as pneumonia, emphysema, pulmonary fibrosis, pulmonary mass, or aortic pathology.

Primary Therapy versus Secondary Prevention

Primary therapy consists of clot dissolution with thrombolysis or removal of PE by

embolectomy. Anticoagulation with heparin and warfarin or placement of an inferior vena caval filter constitutes secondary prevention of recurrent PE rather than primary therapy.

Risk Stratification

Risk stratification is crucial in determining treatment strategy. The presence of hemodynamic instability, right ventricular dysfunction, or elevation of the troponin level due to right ventricular microinfarction can identify high-risk patients.

Primary therapy should be reserved for patients at high risk of an adverse clinical outcome. When right ventricular function remains normal in a hemodynamically stable patient, a good clinical outcome is highly likely with anticoagulation alone

Adjunctive Therapy

Important adjunctive measures include pain relief (especially with nonsteroidal anti-inflammatory agents), supplemental oxygenation, and psychological support. Dobutamine — a β-adrenergic agonist with positive inotropic and pulmonary vasodilating actions—may be effective in the treatment of right heart failure and cardiogenic shock. Volume loading should be undertaken cautiously because increased right ventricular dilatation can lead to even further reductions in left ventricular forward output. Heparin binds to and accelerates the activity of antithrombin III, an enzyme that inhibits the coagulation factors thrombin (factor IIa), Xa, IXa, XIa, and XIIa. Heparin thus prevents additional thrombus formation and permits endogenous fibrinolytic mechanisms to lyse clot that has already formed. After 5 to 7 days of heparin, residual thrombus begins to stabilize in the endothelium of the vein or pulmonary artery. However, heparin does not directly dissolve thrombus that already exists.