Pulmonary Embolism

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Pulmonary embolism is a common disease and continues as a serious complication of different medical and surgical disorders. The disease is often overlooked and causes a significant morbidity and mortality. Current diagnosis, management and prophylaxis of pulmonary embolism is reviewed in this study. [Journal of Turgut Özal Medical Center 1997;4(2):243-247]

***Key Words**: Pulmonary embolism, diagnosis, management, prophylaxis*

Pulmoner embolizm

Pulmoner embolism sık görülür ve değişik medikal ve cerrahi hastalıkların ciddi bir komplikasyonu olmaya devam etmektedir. Hastalık sıklıkla gözden kaçmakta ve önemli morbidite ve mortaliteye neden olmaktadır. Bu çalışmada pulmoner embolizmin güncel tanı, tedavi ve proflaksisi gözden geçirilmiştir. [Turgut Özal Tıp Merkezi Dergisi 1997;4(2):243-247]

***Anahtar Kelimeler**: Pulmoner embolizm, tanı, tedavi, proflaksi*

Pulmonary embolism (PE) is a common phenomenon and continues as a serious complication of a variety of primary medical and surgical disorders (1). Venous thromboembolism is the third most common acute cardiovascular disease after cardiac ischemic syndromes and stroke (2). Analysis of epidemiologic studies shows that the incidence of PE is underestimated clinically. The frequency of the diagnosis of PE at a given hospital greatly increases if a referral unit for PE is set up in the hospital (2). The disease is often overlooked because the symptoms and signs caused by PE are nonspecific and may be confused with a variety of other cardiopulmonary disorders that have similar presentations (3). The incidence of PE in an Italian area is about 100 cases per year per 100.000 persons with an overall short-term mortality (within 30 days from diagnosis) of 11.4% (9.2% in treated patients versus 25.2% in untreated patients) (4,5). The incidence of PE in postoperative patients remains significant and often presents as an emergency. Marpurgo et al reported that out of 92 postmortem cases of massive or submassive PE, only 28% were diagnosed before death, whereas the false-positives accounted only for 3% of cases (6). Microscopic examination greatly increases the number of observed recent and old thromboemboli, so that the percentage of PE in autopsy cases is between 52% and 90% (7,8,9).

ETIOPATHOGENESİS

A PE is defined as an occlusion of one or more pulmonary vessels by a material that has traveled there from outside of the lung and is usually caused by a dislodged thrombus that originated in the deep veins of the legs or pelvis (10). The majority of

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patients with PE have a lower extremity deep-vein thrombosis (DVT) as a source of origin. The main factors that influence thrombus formation are stasis, injury and hypercoagulability (Virchow's triad).

Older age, obesity, immobilization, heart disease, particularly congestive heart failure and atrial fibrillation, carcinomatosis, serious infections, and cerebrovascular accidents are often found to be predisposing causes of PE (10). Patients with various traumatic lesions, particularly fractures of the hip and extensive soft tissue damage to the lower extremities, have a higher incidence of PE. Patients with pelvic trauma are known to be at increased risk for the development of thromboembolic complications. The incidence of DVT in patients with pelvic fractures is 35% to 60%. Proximal DVT, which is most likely to result in PE, occurs in 25% to 35% of these patients, and almost 1/2 of all proximal thrombi will be in the pelvic veins. The incidence of symptomatic PE in the pelvic trauma population is 2% to 10% whereas a greater proportion of patients will have clinically silent PE. Fatal PE occurs in 0.5% to 2% of patients with pelvic trauma (11).

It has long been known that surgical procedures, particularly prostatectomy, hip fracture repairs, and operations on the lower extremities, are also associated with a higher risk of PE. Oral contraceptives are also known to increase the incidence of both thrombophlebitis and PE (10,11).

DIAGNOSIS

Pulmonary embolism is an often underdiagnosed disease resulting in significant excess morbidity and mortality rates. As symptoms and signs of PE are nonspecific, the diagnosis still remains a challenge to the attending physician. First we must suspect PE and consider its likelihood in the presence of a number of clinical signs and symptoms. Asymptomatic PE is also common and the most important causes of an incorrect diagnosis are failure to suspect PE, and the protean nature of the disease.

It is impossible to prove or disprove a diagnosis of acute PE on clinical grounds. Unexplained dispnea, chest pain, and hemoptysis are the most frequent presenting symptoms (12), and sudden onset of dyspnea and pleuritic chest pain are the most typical ones (13). Clinical findings include tachycardia, fever, rales, tachypnea, and evidence of DVT in the lower extremities.

The use of findings from chest radiograph, ECG, and blood gas analysis may raise clinical suspicion of PE and decide on therapeutic management (14). Although the plain chest radiograph may show evidence of diminished vascular markings in the area of PE (Westermark's sign), a completely normal chest radiograph may be seen in up to 40 percent of patients with PE (1,13,14).

A slight elevation in bilirubin, due to resolution of the emboli in the lungs, is sometimes present, and lactate dehydrogenase activity may be increased. In patients who are otherwise normal, reduced arterial PaO2 being helpful in suspecting PE, but a normal value does not exclude PE. As many as 30 % of persons with PE and no prior cardiopulmonary disease will have a PaO2 greater than 80 mm Hg, therefore arterial blood gas data are of insufficient discriminant value to permit exclusion of the diagnosis of PE (12,15).

The electrocardiogram (ECG) is seldom specific for PE and not more than 20% of patients with proven PE demonstrate any ECG changes of significance. Electrocardiographic alterations include rhythm disturbances (atrial fibrillation, ectopic beats, heart block, tachycardia), T wave inversion in V1-V2, and PR displacement. The echocardiogram can be helpful in the diagnosis of massive PE. Echocardiographic detection of right ventricular strain in patients who present acute cardiopulmonary manifestations with no previous history of severe pulmonary disease may indicate the possibility of a PE (16,17).

Pulmonary scanning is helpful in diagnosis of PE, particularly if the plain chest radiograph is otherwise completely normal. To improve the diagnostic specifity of nuclear medicine techniques, ventilation imaging has been coupled to perfusion lung scanning to distinguish perfusion abnormalities secondary to ventilation disturbances (18-20). If the plain film is abnormal, one should not generally proceed with a radioactive pulmonary scan but move directly to pulmonary angiography. If significant PE is present, it will almost always be demonstrable by pulmonary angiography. The diagnosis of PE can be accurately made by perfusion lung scan (21-23) and pulmonary

angiography; however, when these diagnostic techniques are not promptly available, simple clinical procedures may be useful to identify patients with high probability of PE.

Recent technical advances in computed tomography (spiral CT, spiral computed tomographic angiography) and magnetic resonance (MR) imaging have spurred a renewed interest in these modalities for the diagnosis of PE (24-26).

MANAGEMENT

Deep venous thrombosis and PE are significant clinical problems. The objectives of treating patients with PE are to prevent death, to reduce morbidity from the acute event, and to prevent thromboembolic pulmonary hypertension. Awareness of risk factors can be helpful in prophylaxis and treatment plans. Oxygen and heparin therapy should be commenced as soon as the diagnosis of PE is suspected and additional basic support is mandatory (intubation and mechanical ventilation) if required. In the setting of hypotension, supportive therapy includes pressor agents and the preferred agents are norepinephrine (Levophed), isoproterenol hydrochloride (Isuprel), and epinephrine. Hypotension may be relieved by preload reduction or even by gentle diuresis. Intravenous fluids should be administered cautiously. In the absence of contraindications, thrombolytic therapy with urokinase or recombinant tissue plasminogen activator should be strongly considered in hypotensive patients (27). Major acute PE with haemodynamic instability responds well to thrombolysis (28). Thrombolysis can be applied with a 2 week "time window", no mandatory angiogaphy in many cases, a brief infusion through a peripheral vein, and no special laboratory tests (29). If thrombolysis fails or is contraindicated, catheter embolectomy or surgical embolectomy is indicated (17,30,31). Chronic thromboembolic pulmonary hypertension is a rare and aberrant outcome of PE and has become a potentially curable form of pulmonary hypertension, either with thromboendarterectomy or lung transplantation (28,32).

Vena cava filters are effective in preventing PE in patients with DVT or PE who either have contraindications to anticoagulation or have sustained a PE despite adequate anticoagulation. Although vena cava filters are not without complications, clinically significant morbidity and mortality are low (33). The placement of filters has progressively become a percutaneous procedure performed by interventional radiologists outside of the operating room (34).

In patients with PE, the amount of heparin administered depends upon the prolongation of the activated partial thromboplastin time to approximately twice the patient's pre-heparin value. The duration of heparin therapy is individualised depending upon the clinical course (35), and generally administered for 7 to 10 days (36). Oral coumarin administration is commenced several days prior to cessation of heparin therapy to allow the required period for adequate prolongation of the prothrombin time. The duration of coumarin therapy is controversial and most believe that a period of 3 to 6 months, if there are no further clinical manifestations at that time (36).

PROPHYLAXIS

Most deaths from PE can be prevented by providing adequate prophylaxis for hospital patients who are at high risk for venous thromboembolism. Physicians, however, must be able to identify high-risk patients and prescribe an appropriate prophylactic agent for each patient according to his or her level of risk. A number of prophylactic methods have been proven effective in the prevention of venous thromboembolism, including low-dose heparin, low-molecular-weight heparin (LMWH), oral anticoagulants, graduated compression stockings, and intermittent pneumatic compression of the legs and in some studies, inferior vena caval filters (11,36,37).

Heparin prophylaxis with subcutaneous low-dose heparin is adequate in some clinical settings. LMWH preparations are effective and safe for prophylaxis in certain medical patients as well as in general surgery and elective hip replacement. Subcutaneous administration of LMWH once or twice daily may prove more convenient from both the patient and nursing viewpoint, particularly in the treatment of established DVT. Monitoring is not necessary when using LMWH preparations as prophylaxis, and a fixed-dose without weight

adjustment has been used in most prophylaxis trials. When treating established DVT, less monitoring is likely to be required than currently is the case with low-dose heparin. Although the cost of LMWH preparations is greater than that of low-dose heparin, the decrease in the occurrence or recurrence of thromboses that has been demonstrated in some trials might prove an effective balance (38).

In view of the literature effective management of PE includes prophylaxis, screening, and appropriate treatment. Such a systematic approach to this potentially catastrophic problem may decrease the morbidity and mortality associated with thromboembolic complications in these patients.

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