SPONTANEOUS HEMOTHORAX DUE TO RUPTURED PULMONARY INFARCTION AFTER ANTICOAGULATION WITH ENOXAPARIN: A CASE REPORT

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SUMMARY

Pulmonary thromboembolism remains a leading cause of morbidity and mortality. Embolic obstruction of pulmonary vasculature may result in pulmonary hemorrhage but usually does not cause pulmonary infarction. Pulmonary infarction may occur only 10 % of pulmonary emboli but, the rupture of pulmonary infarction is a very rare condition in the chest diseases clinics. In the literature only a few case reports have been published. We report here spontaneous hemothorax that thought to be occurred due to ruptured pulmonary infarction in a case of pulmonary thromboembolism anticoagulated with enoxaparin (1mg/kg, bid, SC). The patient was successfully managed with discontinuation of anticoagulation, blood and plasma transfusions, chest tube drainage for evacuation of blood from the pleural space.

Key words: *anticoagulant, enoxaparin, pulmonary thromboembolism, ruptured infarction, spontaneous hemothorax*

ÖZET

Enoksaparin ile Antikoagülasyon Sonrası Rüptüre Pulmoner Enfarkta Bağlı Spontan Hemotoraks: Olgu Sunumu

Pulmonary thromboembolism remains a leading cause of morbidity and mortality. Embolic obstruction of pulmonary vasculature may result in pulmonary hemorrhage but usually does not cause pulmonary infarction. Pulmonary infarction may occur only 10% of emboli but the rupture of pulmonary infarction is a very rare condition in the chest disease clinics. In the literature only a few case reports have been published. We report here spontaneous hemothorax that thought to be occurred due to ruptured pulmonary infarction in a case of pulmonary thromboembolism anticoagulated with enoxaparin (1mg/kg, bid, sc). The patient was successfully managed with discontinuation of anticoagulation, blood and plasma transfusions, chest tube drainage for evacuation of blood from the pleural space.

Key words: *anticoagulant, enaxaparin, hemothorax, pulmonary thromboembolism, spontaneous*

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Alındığı tarih: 28. 03. 2005, kabul tarihi: 10. 10. 2005
28 Nisan-1 Mayıs 2004 tarihinde Belek/Antalya'da düzenlenen Toraks Derneği 7. yıllık kongresinde poster olarak sunulmuştur.

INTRODUCTION

Hemothorax is the presence of significant amounts of blood in the pleural space in which the pleural fluid hematocrit is 50% or more of the peripheral blood hematocrit. Spontaneous hemothorax is uncommon and occurs without an obvious trauma[1]. The most common cause of spontaneous hemothorax is the complication of the anticoagulation in the treatment of pulmonary thromboembolism (PTE)[1]. The cases of spontaneous hemothorax resulted from various anticoagulant therapy have been well documented associated with a primary diagnosis of pulmonary embolus[1-4]. PTE may be complicated by pulmonary infarction in only 10 % of cases, especially in the decreased left ventricular functions[5]. The rupture of pulmonary infarction is a rare, catastrophic clinical condition with usually high mortality.

CASE REPORT

A 70-year-old woman admitted to our hospital with a 3-day history of nonproductive cough, hemoptysis, and suddenly developed shortness of breath and pleuritic chest pain on the right hemithorax. No risk factor for venous thromboembolism was established except age. On admission to our hospital she was afebrile with respiratory rate of 24/min, a hearth rate 100/min, and blood pressure 150/90mmHg. The lung examination showed scattered expiratory ronchi and decreased breath sounds over right inferior hemithorax. On the laboratory examination: WBC: 8100/mm^3, hemoglobin 12.5g/dl, hematocrit: 35.7 %, ESR: 60 mm/h. There was no abnormality on electrocardiography and chest X-ray. Arterial blood gas values were pH: 7.40, PCO_2: 36.3 mmHg, PO_2: 61 mmHg, HCO_3: 22.2 mEq/L, and oxygen saturation of 91.9 % on room air. D-dimer test was 1054ng/ml. Computerized spiral tomography (CT) showed a pleural-based, wedge-shaped parenchymal consolidation, and multiple thrombi on the division of right pulmonary artery and on its inferior lobar segmental branchies (Figure 1). The result of doppler ultrasonography of both lower extremities was interpreted as normal for evidence of venous thrombosis. A diagnosis of PTE was made and enoxaparin therapy (1mg/kg, subcutaneously, every 12 hours) was initiated. At the fourth day of therapy, she was pale and dyspnoeic, respiratory rate 30/min.

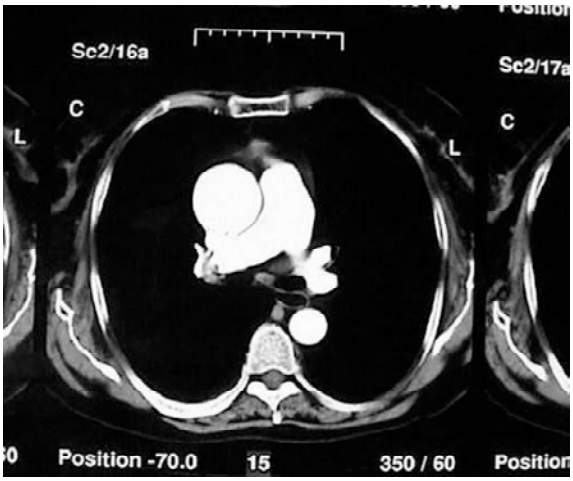

Figure 1: *Computed tomography of chest demonstrating thrombus on the inferior and superior lobar bifurcation of right pulmonary artery*

The blood pressure was recorded as 110/60mmHg with pulse rate 90 beats/min. Clinical examination was consistent with pleural effusion. Although coagulation studies were within normal limits, the hemoglobin value dropped to 7.9 g/dl. Chest X-ray showed a moderate right pleural effusion. CT showed a moderate pleural effusion with heterogeneous-dense appearance (Figure 2) and resolving on the thrombus area. Her anticoagulant therapy was immediately stopped in the view of the evidence of serious hemorrhage. Two units of whole blood and two units of fresh frozen plasma were transfused. A large bore chest tube was inserted into fifth right intercostal space and two liters of pleural effusion, which was shown to be grossly bloody (hematocrit level of pleural effusion was 14%) were evacuated in three days. After removal of the chest tube, small amount of pleural thickness remained at the base of right hemithorax. She was discharged on the 20th day of admission. She is still well with no respiratory complaint.

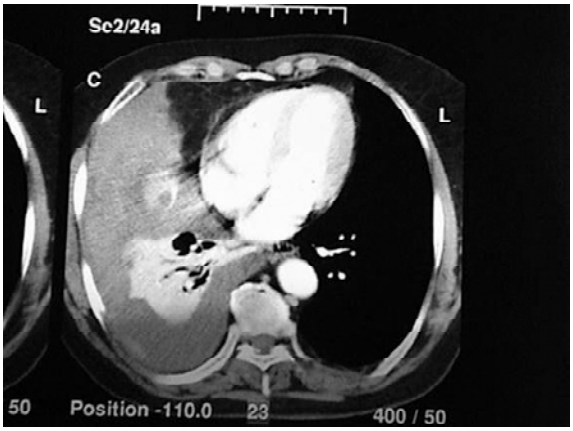

Figure 2: *CT scan of the chest showing atelectasis and pleural effusion with heterogeneous-dense appearance on the right hemithorax*

DISCUSSION

The differential diagnosis of causes of spontaneous hemothorax includes metastatic malignant pleural disease, anticoagulant treatment, complication of a bleeding disorder, bleeding from systemic vessels (aortic dissection, ruptured patent ductus arteriosus, leaking internal mammary artery aneurysm), bleeding from pulmonary vessels, active tuberculosis, costal exostoses and sub-diaphragmatic causes (endometriosis, splenic artery aneurysm)[1,6]. Complication of anticoagulant therapy for PTE has been described as one of the most common causes of spontaneous hemothorax[1]. Eleven cases of hemothorax as a complication of anticoagulant therapy had been reported by Rostand et al[2]. Of the patients reported, 5 were receiving heparin only, 4 were receiving heparin and warfarin, and 2 were receiving warfarin only. To our knowledge, however, there is only one case report of spontaneous hemothorax in a patient receiving enoxaparin in the literature[7]. In this report, patient had spontaneous bilateral hemothorax and a large retroperitoneal hematoma after 4 days of anticoagulation therapy (enoxaparin, 1 mg/kg, SC, bid) for suspected PTE. In our case, hemothorax was not bilateral and abdominal CT did also not detect retroperitoneal hemorrhage.

According to Rostand et al[2], hemothorax usually becomes apparent 4 to 7 days after anticoagulant therapy is initiated. The coagulation studies in patients with this complication are usually within therapeutic range and hemothorax is almost always on the side of the original pulmonary embolus, suggesting intrapleural rupture of a hemorrhagic pulmonary infarct[2]. In the presented case the values of coagulation tests, the side and the time of occurrence of spontaneous hemothorax were consistent these findings.

The rupture of pulmonary infarct should be suspected when a patient with a proven or suspected PTE experiences abrupt circulatory and respiratory insufficiency in association with rapidly accumulation unilateral pleural effusion. The diagnosis is further suggested by observation of frank blood in thoracentesis [2]. In our case, there was a PTE with pulmonary infarction proven by CT. A clinical deterioration, by means of a fall in hemoglobin level with a suddenly occurred pleural effusion, and a proven hemothorax by thoracentesis, suggests us that this was a rupture of pulmonary infarction. Wick et al[8] reported that ruptured pulmonary infarction was a rare and fatal complication of thromboembolic disease and distinguishing secondary hemothorax in pulmonary infarction cases from overt rupture of the infarcted area was extremely difficult. Ruptured pulmonary infarction may occur spontaneously, like our case, or iatrogenically as an overaggressive anticoagulation. Reperfusion of necrotic tissue in the infarcted zone of the lung increases vascular hydrostatic pressure and the possibility of pleural rupture.

Most important complication of anticoagulant therapy is bleeding which is classified as major; if it is intracranial or retroperitoneal, if it directly lets to death, or if it results in hospitalization or transfusion[9]. Risk factors for Low Molecular Weight Heparin (LMWH) induced hemorrhage are thought to be similar to those already linked to hemorrhage induced by unfractioned heparin: recent surgery or trauma, concomitant use of aspirin, thrombolysis, concurrent glycoprotein IIb/IIIa inhibitor use, and age >70 years[10]. In our patient there were no risk factors for LMWH induced hemorrhage, except age. LMWH is not associated with increased major bleeding compared with standard heparin in acute venous thromboembolism[10]. In most cases, major bleeding syndromes are procedure related but bleeding may also occur spontaneously. Almost half of all enoxaparin related major bleeding complications occur within 3 days of therapy[11,12]. Our patient had hemothorax at the fourth day (after seven doses) of enoxaparin (1mg/kg, subcutaneously, every 12 hours) and needed for transfusion of two units of whole blood and plasma.

Although LMWH has been shown to be as effective and safe as unfractionated heparin for the treatment of acute venous thromboembolism[13,14], it is important to take into consideration its hemorrhagic side effects such as spontaneous hemothorax in patients with pulmonary infarction.

The presence of hemothorax necessitates immediate withdrawal of thrombolitic or anticoagulant medications and evacuation of pleural space[2]. A massive transfusion may be required in some cases and it seems reasonable to obtain a thoracic surgical consultation for urgent thoracotomy and possible lobectomy of the infarcted site[8].

Ruptured pulmonary infarction should be kept in mind when a suddenly developed hemotorax determined in

a patient with PTE especially if clinical deterioration has been occurred.

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