Endobronchial streptokinase has been used previously to dissolve blood clots caused by massive spontaneous hemoptysis, in settings including sarcoidosis, cavitary histoplasmosis, and multiple myeloma. To our knowledge, however, the use of thrombolytic agents to dissolve clots following transbronchial biopsy has not been reported previously. We describe a patient in whom endobronchial urokinase was used for successful dissolution of clots secondary to massive bleeding after transbronchial biopsy.

CASE REPORT

A 56-year-old man was admitted to the hospital in October 1991 after 4 weeks of increasing dyspnea on exertion, 3 days of fever to 38°C, and minimal hemoptysis. Six months earlier he had received radiation therapy and chemotherapy for recurrent adenocarcinoma of the right lung. Fiberoptic bronchoscopy with TBB specimens, showing radiation pneumonitis, was complicated by heavy bleeding and respiratory failure. The patient was markedly hypoxic at the time of hospital admission. The hematocrit was 17.7%, and the platelet count was 97,000/mm³. The prothrombin time was 12.3 s (control, 10.9 s) and the partial thromboplastin time was 29 s. Chest radiograph showed a right parahilar mass and a new left lower lobe infiltrate.

Erythromycin, cefuroxime, and gentamicin (based on sputum Gram’s stain), pentamidine, vitamin K, and prednisone were administered. On the second hospital day, 6 U of platelets were administered, and FOB was performed 1 h later to diagnose the cause of the new left lung infiltrate. Oxygen, 100 percent, was given through an oral endotracheal tube (ETT) during the procedure. The left lung endobronchial mucosa was moderately inflamed and friable. Following bronchoalveolar lavage (BAL), a TBB was performed under fluoroscopic guidance in the lower lobe, basal segment. Massive hemorrhage immediately ensued, despite attempted tamponade with the bronchoscope tip. A large amount of blood was coughed up into the ETT tube and bag (Ambu-bag) and rapidly clotted. Ventilation quickly became impossible, even after removal of the bronchoscope. The ETT was therefore removed, and a 15-cm-long, very thick clot occluded the end of the tube. The patient could be ventilated only with great difficulty, through a bag-valve-mask. After sedation and paralysis, the trachea was then reintubated with a new ETT, also with great difficulty because of massive bleeding into the oropharynx. A larger-channel bronchoscope was then inserted through the new tube, and massive blood clots were found to occlude both main-stem bronchi. These could not be removed with suction, saline solution lavage, agitation with biopsy forceps, or a Fogarty balloon catheter. Oxygen-hemoglobin saturation dropped to 40 percent, and the peak inspiratory pressures rose to 120 cm H₂O. Urokinase (5,000 U/ml in sterile water, Abbokinase Open-Cath) was administered endobronchially, through the bronchoscope, directly onto the clots. Aliquots of 2,500 U were given, each diluted in 5 ml of sterile water, to a total of 15,000 U (30 ml) over 30 min. Further suctioning, saline solution lavage, and agitation with biopsy forceps resulted in almost complete lysis of the clots. ETT = endotracheal tube; TBB = transbronchial biopsy.
visible clots over approximately 75 min, without new bleeding. Remaining clots were then removed through a rigid bronchoscope. Subsequently, the oxygen saturation slowly rose to 80 percent, and later to 90 percent, giving a total duration of profound hypoxemia of approximately 3 h. The estimated blood loss was 750 ml.

By the following hospital day, the patient began to respond and went on to a full neurologic recovery and eventual hospital discharge.

**DISCUSSION**

Bronchial occlusion by blood clots following spontaneous hemoptysis, tracheal suctioning, and FOB has been reported, and there are reports of such occlusions treated by the endobronchial administration of streptokinase.

In the first reported case, a total of 30,000 U of streptokinase was given successfully over 1 h, with subsequent clearing of the airways and no short-term complications. In the second reported case, a total of 60,000 U of streptokinase (in 10,000-U aliquots) was used to dissolve endobronchial clot, which was then removed manually through an endotracheal tube. Finally, Maxwell and Stauffer reported two other cases of endobronchial administration of streptokinase (50,000 U in one case and 80,000 U in the other), followed by piecemeal removal of remaining clot. However, to date and to our knowledge, the use of endobronchial urokinase for dissolution of clots has not been reported, nor has the use of thrombolytic agents for clots following TBB.

Urokinase is frequently used to reopen occluded blood vessels and intravascular catheters. In the present patient, urokinase was administered when rigid bronchoscopy was temporarily unavailable and it appeared that the patient could not be ventilated without immediate intervention to reopen his airways. A total of 15,000 U in sterile water, given over 30 min directly through the bronchoscope onto the surface of the endobronchial clot, dissolved the clot without any apparent adverse effect.

Bleeding is a well-recognized complication of FOB, particularly in patients with qualitative or quantitative platelet abnormalities. The patient described herein had a prebiopsy platelet count of 97,000/mm³, and he bled massively in spite of platelet transfusion.

This case shows that endobronchial urokinase can safely and effectively be administered for large obstructing clots that can not be removed manually, despite the presence of a recent TBB site. The premixed urokinase preparation for catheter clearance may be used and diluted further with sterile water to make administration directly through the bronchoscope channel easier.

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**Use of Plasma for Arterial Blood Gas Analysis in Leukemia**

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A 63-year-old patient with chronic lymphocytic leukemia presented with severe hypoxemia. However, the patient’s hemoglobin saturation, measured by an ear oximeter, was normal. Although his WBC count was approximately 1,000,000/μl, hypoxemia could not be explained by the consumption of oxygen by leukocytes. Therefore, arterial blood gas values were analyzed in both plasma as well as in whole blood. The PaO₂ in the plasma was much higher than in whole blood and corresponded with the hemoglobin saturation measured by the ear oximeter. These findings suggest that very high leukocyte counts may interfere with the measurement of oxygen tension and that plasma may be used for blood gas analysis in this situation.

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The presence of hypoxemia in leukemic patients often warrants invasive investigation. However, determination of true PaO₂ in the blood gas sample can be difficult because of the high leukocyte counts in these patients. These leukocytes consume oxygen, and therefore, after the blood sample has been withdrawn, the PaO₂ progressively falls with time. The rate of fall in PaO₂ decreases if the blood sample is stored in ice.

This report describes severe pseudohypoxemia in the whole blood of a patient with chronic lymphocytic leukemia. In this patient, a low PaO₂ could not be explained by leukocyte oxygen consumption and was probably related to the high leukocyte count interfering with the oxygen electrode membrane.

**CASE REPORT**

A 63-year-old male smoker was admitted to another hospital with progressive dyspnea. Five years earlier, he had been diagnosed to have chronic lymphocytic leukemia. At that time, he was treated with prednisone and chlorambucil for 1 year but subsequently discontinued the treatment himself. He did not have a past history of cardiopulmonary disease. Room air arterial blood gas levels at that time were pH, 7.39; PaCO₂, 51 mm Hg; and PaO₂, 38 mm Hg. A chest roentgenogram showed mild cardiomegaly with increased interstitial markings at the bases. Results of other laboratory tests were within normal limits except a WBC count of 796,000/μl with...