Significance of Hemoptysis Following Thrombolytic Therapy for Acute Myocardial Infarction*

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Purpose: To describe the occurrence, cause, and significance of hemoptysis following thrombolytic therapy for acute myocardial infarction.

Patients and methods: We retrospectively reviewed 2,634 patients presenting with acute myocardial infarction who received thrombolytic therapy to determine the incidence of hemoptysis. Chart and radiographic review included the type, dose, and route of thrombolytic therapy. In addition, the onset, duration, and severity of hemoptysis were recorded and correlated with radiographic and bronchoscopic findings.

Results: Eleven patients (0.4%) developed hemoptysis following administration of thrombolytic therapy for an acute myocardial infarction. The duration and severity had a wide range, although no patient had significant hemodynamic compromise. The source of hemoptysis was identified in only one patient who had a tongue laceration following cardiopulmonary resuscitation, and blood was seen within the oropharynx and trachea. No definitive cause was identified in all other patients. There was no correlation between the different types or doses of thrombolytic therapy and the duration or severity of hemoptysis. Chest radiographs were nonspecific and demonstrated resolution within 11 days following hemoptysis. CT of the thorax in one patient and bronchoscopy in two patients confirmed chest radiographic findings and in no patient was an underlying pulmonary abnormality identified.

Conclusions: Pulmonary hemorrhage and hemoptysis are unusual complications of thrombolytic therapy in patients with acute myocardial infarction. Although hemoptysis may be the first indicator of an underlying pulmonary abnormality, we found no case in which a significant abnormality was unmasked. This study suggests that follow-up chest radiographs are recommended and further evaluation may be unnecessary if complete resolution is demonstrated.

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Key words: acute myocardial infarction; hemoptysis; thrombolytic therapy

Chocken administration of thrombolytic therapy for patients with acute myocardial infarction (AMI) results in restoration of coronary flow, improvement in left ventricular function, and a 23% reduction in 35-day mortality.1,2 Thrombolytic therapy, however, is not without significant risk, including a 0.65% incidence of intracranial hemorrhage, a 1.5% risk of major hemorrhage associated with hemodynamic compromise, and an 11% risk of moderate hemorrhage requiring transfusion.3,4

Intracranial bleeding is the most serious bleeding complication of thrombolytic therapy.4,5 Pulmonary hemorrhage and hemoptysis are much less common and frequently result in a search for an underlying pulmonary abnormality.7,9-13 The diagnostic investigation may be extensive, although the utility of evaluation including chest radiographs, thoracic CT, and bronchoscopy is uncertain. We retrospectively reviewed all patients who developed hemoptysis following thrombolytic therapy at our institution over a 5-year period to determine if significant abnormality existed and to determine the best method for evaluation.

Materials and Methods

From January 1989 to July 1994, 2,634 patients with AMI received thrombolytic therapy within 6 h of presentation. Eleven patients developed hemoptysis within 72 h of receiving thrombolytic therapy and constitute the current study group. Chart review included the type, dose, and route of thrombolytic therapy. The duration, cause, and severity of hemoptysis were also recorded as were the results of diagnostic and laboratory tests, including hemoglobin, hematocrit, prothrombin time, partial thromboplastin time, platelets, chest radiographs, thoracic CTs, and bronchoscopy. Using previously established parameters, hemoptysis was defined as “significant” if there was respiratory compromise believed due to the pulmonary hemorrhage or a reduction of hemoglobin level of 5 g/dL or more (or >15% in hematocrit) or “minor” if there was blood-tinted sputum and a decrease in hemoglobin level between 3 and 5 g/dL (or in hematocrit from 10 to 15%).14 All 11 patients had serial chest radiographs and 1 patient had a thoracic CT. Ventilation/perfusion study and pulmonary angiogram. All findings were read by two chest radiologists independently, and final interpretation was reached by consensus without knowledge of

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the clinical course. Radiographic findings were then correlated with the type of thrombolytic therapy, onset, duration, and severity of hemoptysis. Bronchoscopy was performed in two patients.

Results

Hemoptysis was a rare complication, occurring in 11 of 2,634 patients (0.4%). There were seven men and four women with an average age of 65 years (range, 49 to 79 years).

Four thrombolytic agents, streptokinase, urokinase, tissue plasminogen activator (t-PA), and anisoylated plasminogen streptokinase activated complex were used in these patients. Six patients (54%) received a 15-mg IV bolus of t-PA followed by an 85-mg IV infusion over 90 min (total, 100 mg). Two patients (18%) received 1.5 million units IV streptokinase delivered in 1 h. Two patients (18%) received IV urokinase and then 100 mg IV t-PA as above. All of these patients also received routine heparin, with a 5,000-IU IV bolus followed by a continuous IV infusion at an approximate range between 1,000 and 1,500 IU/h titrated to maintain an activated prothrombin time of 1.5 to 2.5 times control. One patient initially received anisoylated plasminogen streptokinase activated complex alone, but was started on a regimen of heparin after she suffered an embolic stroke.

The duration and severity ranged from a single episode of small amounts (five patients) to protracted intermittent hemoptysis (six patients). The cause was thought to be identified in only one patient who had a tongue laceration following cardiopulmonary resuscitation (CPR) and nasal intubation. Blood was identified in both the oropharynx and trachea. One other patient with hemoptysis had CPR, and was subsequently intubated. While these may have been contributing factors, no definitive source was identified. There was no correlation between the different types or doses of thrombolytic agents used in this study and the duration or severity of hemoptysis.

The initial chest radiograph following hemoptysis demonstrated diffuse, bilateral, symmetric pulmonary opacities in four patients, scattered homogeneous opacities in four patients, atelectasis in two patients, and one patient had normal results from the study. Resolution of chest radiographic abnormalities appeared to depend on the patient’s underlying cardiac function, although all cases resolved within 11 days. Follow-up chest radiographs failed to reveal any definitive underlying cause for hemoptysis (eg, neoplasm, arteriovenous malformation [AVM], bronchiectasis) in any patient. Similarly, clinical follow-up failed to demonstrate persistent hemoptysis, continuous hematocrit drop, or specific cause accounting for the hemoptysis.

CT was performed in one case and demonstrated pulmonary edema, bilateral multifocal subsegmental consolidation, emphysema, and coronary artery calcification. There were no masses or endobronchial lesions, bronchiectasis, AVM, or other significant abnormalities. The patient also had an indeterminate ventilation/perfusion scan and subsequent pulmonary angiogram that was normal.

Bronchoscopy was performed in two patients. One patient had a small amount of blood within the right mainstem bronchus, and one patient had blood in the left mainstem bronchus. No endobronchial lesions, bronchiectasis, or other abnormalities were detected.

Discussion

Hemoptysis can be caused by a variety of different pulmonary abnormalities, including neoplasms, bronchitis, AVM, tuberculosis, pulmonary infarction, and bleeding disorders brought on by autoimmune diseases or drugs. Radiographic and clinical evaluation for hemoptysis typically entails chest radiographs, thoracic CT, and/or a bronchoscopy, although in many cases, the exact cause is not discovered. Our study confirms that hemoptysis following thrombolytic therapy for AMI is a rare complication and suggests that it does not require immediate diagnostic investigation looking for an underlying pulmonary etiology. In only one of our patients was a cause, most likely CPR and nasal intubation, determined. While these features may play a role in some cases, most patients had no identifiable abnormality.

The radiographic manifestations of pulmonary hemorrhage are varied and have been well described. The findings are frequently nonspecific, consisting of poorly defined opacities or consolidation, although they often depend on the underlying cause, including bronchiectasis, infection, neoplasm, or AVM. Chest CT has been strongly advocated as an important modality in evaluating patients with hemoptysis and may provide information not appreciated on chest radiographs. In our series, however, while the radiographic findings were typically nonspecific (except in those patients with pulmonary edema most likely secondary to their cardiac disease), an underlying abnormality was not identified. Furthermore, thoracic CT in this patient population did not add additional information.

Follow-up clinical investigation also failed to reveal any underlying cause for pulmonary hemorrhage and hemoptysis in our patients or, to our knowledge, in any of the patients described in the literature. In all patients, hemoptysis resolved without sequelae or significant additional complications. We thus conclude that patients with pulmonary hemorrhage and hemoptysis following thrombolytic therapy may be treated conservatively with observation and sequential chest radiographs until complete resolution of any pulmonary abnormality.
REFERENCES


