Intracavitary Aminocaproic Acid for Massive Pulmonary Hemorrhage

To the Editor:

Medical therapy for massive endobronchial hemorrhage from an intracavitary mycetoma has been very disappointing at best. Surgical therapy is limited to the minority of patients who have significant cardiopulmonary reserve and are therefore candidates for partial lung resection. This case demonstrates the successful and quick control of life-threatening pulmonary hemorrhage by intracavitary injection of aminocaproic acid.

CASE REPORT

A 48-year-old man was admitted for treatment of pulmonary aspergilloma with hemoptysis. He had left superior sulcus carcinoma treated with resection and radiation therapy 13 years prior to admission, with a residual cavity at the left apex. Nine months prior to admission, cavity aspergilloma was diagnosed by needle puncture. Because of symptoms (fever and hemoptysis) thoracotomy was performed, but lung resection could not be accomplished due to marked radiation fibrosis. The cavity was explored, confirming the presence of *Aspergillus fumigatus*, and a large 30 ml balloon Foley catheter was placed in the cavity. The patient was treated for several weeks with both intracavitary and systemic amphotericin B, and he improved temporarily. On the day of admission, he noted increasing hemoptysis plus bloody drainage from the catheter. He promptly developed acute massive intracavitary hemorrhage and hemoptysis that required intubation with a double lumen endotracheal tube to isolate his left lung. He received 13 units of packed red blood cells, 6 units of fresh frozen plasma and 14 liters of saline solution. Bronchoscopic examination ruled out bleeding from the right lung, and attempts to tamponade the bleeding with the Foley catheter’s balloon were unsuccessful. Despite transfusions and intravenous vasopressors, he remained markedly hypotensive and continued to bleed briskly from the cavity into the intracavitary catheter. Surgery was not considered possible in this patient. Five grams (20 ml) of aminocaproic acid was injected into the cavity via the intracavitary catheter and the tube was clamped for several minutes. The bleeding slowed down immediately and within a few hours it had completely stopped. The patient was stabilized, and weaned off the ventilator a few days later. Unfortunately, he developed nosocomial pseudomonas pneumonia in his right lung and died from this three weeks after admission. Cultures from the left cavity continued to grow Aspergillus.

DISCUSSION

This is the first report of the use of aminocaproic acid for control of intracavitary pulmonary hemorrhage. Commonly, patients with fungus balls have significant pulmonary dysfunction and surgical therapy for significant hemoptysis involves unacceptable risks. Mild-to-moderate hemoptysis can usually be managed with conservative measures. Massive hemoptysis, of course, is a highly lethal condition, but frequently the underlying condition makes surgical risk extremely high. In cases of massive hemoptysis from cavitary fungal infections, the high-risk patient may benefit from a trial intracavitary aminocaproic acid.

Aminocaproic acid inhibits fibrinolysis principally via inhibition of plasminogen activating substances and through its antifibrinolytic activity. It readily penetrates red blood and tissue cells, and is rapidly excreted in the urine mostly unmetabolized. Five or more grams by intravenous infusion are usually required to control bleeding from systemic fibrinolysis. It has been postulated that extravascular clots formed in vivo with incorporated aminocaproic acid may not undergo spontaneous lysis as do normal clots.

We cannot prove that the drug led to the cessation of bleeding, but our observation in this particular case strongly suggests aminocaproic acid played an important role in controlling the bleeding. Obviously, more information and investigation of this therapeutic modality is needed before it can be recommended, except in extreme cases such as the one reported here.

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would have been interesting to note the differences between these two groups. Second, visual monitoring as opposed to continuous electrocardiographic (ECG) monitoring was used. We have previously demonstrated that one can easily underestimate the actual incidence of arrhythmias, including serious ones, if continuous monitoring is not used. Several studies have demonstrated a higher incidence of ventricular arrhythmias using continuous electrocardiographic monitoring. Elliott showed that cardiac complications were frequent (not infrequent as stated by the authors) with a deviation from baseline rhythm in 76 percent, premature ventricular contractions in 46 percent and ventricular tachycardia in 25 percent of 116 critically ill patients. We have demonstrated a 53 percent incidence of advanced ventricular arrhythmias using continuous ECG monitoring in 119 critically ill patients with shock, complicated myocardial infarction or ischemia and respiratory failure. Therefore, physicians treating seriously ill patients should not become complacent with this reported low incidence of ventricular ectopy, as continuous monitoring was not used and many of the patients were not critically ill. Ventricular arrhythmias remain a serious and frequent complication of catheterization. Interestingly, we have recently demonstrated that prophylactic lidocaine can decrease the incidence of advanced ventricular arrhythmias in critically ill patients undergoing catheterization requiring less than 20 minutes.

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To the Editor:

We agree with Dr. Sprung and his colleagues that the incidence of ventricular arrhythmias observed during passage of a pulmonary artery catheter is dependent upon the type of monitoring employed, as well as upon the severity of the patient’s underlying disease. A continuously recorded electrocardiogram documents more catheter-induced ventricular arrhythmias than does simple visual inspection via an oscilloscope.

The hemodynamic consequences of ventricular ectopy seem to depend primarily upon the presence or absence of acute cardiac or pulmonary disease. Catheter-induced ventricular arrhythmias in patients undergoing catheterization prior to cardiac surgery (a “good risk” group without acute disease) cause minimal, if any, hemodynamic compromise and are unlikely to progress to sustained ventricular tachycardia or fibrillation. In our institution, in over 5,000 such catheterizations, we have never observed ventricular fibrillation. The absence of acute disease or ischemia may explain the inability of lidocaine, administered immediately prior to catheterization, to control or abolish ventricular arrhythmias in this group of patients. Lidocaine may be more efficacious in the patient with acute ischemia or hypoxemia by suppressing arrhythmias due to pre-existing disease which may be exaggerated during catheter placement.

The reports of Dr. Sprung and his colleagues describe the difficulties that may be encountered when catheterizing acutely ill patients. The occasional need for pharmacologic, electrical or mechanical therapy for ventricular irritability emphasizes that such treatment aids must always be immediately available during placement of a pulmonary artery catheter. In addition, the operator must pay continuous attention to the electrocardiogram and to the clinical condition of the patient and not become totally involved with catheter insertion to the exclusion of all else.

The risk of death secondary to catheter placement is still minute. The deaths reported by Dr. Sprung occurred in patients who died from ventricular fibrillation but who were already in cardiogenic shock with little chance of survival.

Stephen J. Thomas, M.D.; and Arthur D. Boyd, M.D., New York University Medical Center, New York

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1 Salmenspera M, Peltola K, Rosenberg P. Does prophylactic lidocaine control cardiac arrhythmias associated with pulmonary artery catheterization? Anesthesiology 1982; 56:210-12

Cavitation in Acute Histoplasmosis

To the Editor:

Bennish et al in their case report, "Cavitation in Acute Histoplasmosis" (Chest 1983; 84:496-97) indicate that they . . . could not find well documented report of . . . "cavity formation in acute histoplasmosis."

Two large outbreaks of histoplasmosis occurred in Indianapolis in 1978-79 and 1980-81 affecting about 150,000 people. Cavitary histoplasmosis occurred in 8 percent of these cases. Upper lobe infiltrates with thin or thick walled cavities or cystic changes were common. Air fluid levels also occurred. One third of the patients improved without treatment. Amphotericin B and ketoconazole were useful in cavitary histoplasmosis.1,2

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