Noncardiogenic Pulmonary Edema Following Laser Therapy of a Tracheal Neoplasm*

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Nd-YAG laser phototherapy has been demonstrated to be effective for the treatment of airways obstruction caused by tracheal tumors. Pulmonary edema has been reported as a complication following acute relief of an upper airway obstruction. We describe a case where laser photoablation of an endotracheal malignancy resulted in acute pulmonary edema. It is important to recognize this potential cause of respiratory deterioration following successful phototherapy to obstructing lesions of the upper airways.

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Pulmonary edema following relief of an upper airway obstruction (UAO) has been observed in both the pediatric and adult populations.1-3 Although the mechanism by which this occurs has been debated, there is general agreement that the pulmonary edema develops immediately or shortly after relieving the UAO.1-3 We recently observed this complication in a patient who underwent successful resection of a critical endotracheal occlusion with the Nd-YAG (neodymium-yttrium aluminum garnet) laser. Anticipation of this potential complication will help identify and possibly avert its occurrence.

CASE REPORT

A 71-year-old woman with adenocarcinoma of the right lung was referred to our institution for Nd-YAG laser therapy of a recurrent mass at the lower end of the trachea. Admission physical examination was remarkable for inspiratory stridor and signs of right lung atelectasis. A chest roentgenogram confirmed the atelectasis and was otherwise normal. There was no prior history of heart disease, and the ECG did not reveal evidence of acute ischemia or previous myocardial infarction. Serum albumin level was 3.3 g/dl.

The patient was premedicated with 5 mg of IV morphine sulfate and corticosteroids. Vital signs were continuously recorded throughout the procedure with a single-lead cardiac monitor and an automated sphygmomanometer. A 5 percent dextrose in water solution at a rate 10 ml/hour was used to maintain peripheral venous access. Fiberoptic bronchoscopy via an endotracheal tube revealed almost complete occlusion of the lower trachea by tumor that extended into and completely occluded the right main-stem bronchus. Nd-YAG laser therapy successfully reestablished 50 percent of the tracheal lumen. No attempt was made to open the right main-stem bronchus. There was no intraoperative bleeding or excessive airway lidocaine instillation. During most of the procedure, the patient was spontaneously breathing through the endotracheal tube with supplemental oxygen. Occasional respiratory assistance was provided with coordinated Ambu bagging. The total duration of phototherapy to the tumor was approximately 1.5 h.

Immediately following extubation, she was noted to be in acute respiratory distress, with arterial blood gas values of pH, 7.29; Pco2, 43 mmHg; Paco2, 42 mmHg (F1o2, 1.0 by face mask). She was immediately reintubated and transferred to the ICU.

Postoperatively, the patient was alert and had a normal blood pressure. Diffuse rales and rhonchi were heard in the left hemithorax, and there was no evidence of a cardiac gallop. The ECG was unchanged and the chest roentgenogram revealed diffuse alveolar opacification of the aerated left lung consistent with pulmonary edema. The ECG did not reveal evidence of acute ischemia or previous myocardial infarction.

FIGURE 1. Roentgenogram reveals diffuse alveolar opacification of the aerated left lung consistent with pulmonary edema.

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Over the following 24 h, she was able to tolerate progressive reduction of the F1o2, and was successfully extubated. Follow-up chest roentgenogram revealed complete resolution of the pulmonary edema (Fig 2), and she was discharged the following day.

DISCUSSION

Pulmonary edema following relief of UAO has been associated with various conditions such as epiglottitis, laryngotracheobronchitis, strangulation, interrupted hanging, and tumors.1-3 Galvis et al postulated that highly negative transpulmonary pressures generated during inspiration against an obstructed airway may disrupt the microvascular integrity and increase venous return. These events are hemodynamically compensated during expiration, when positive transpulmonary pressures serve to decrease venous return. Relief of the UAO may lead to a sudden increase in venous return, which, in the presence of “damaged” capillaries, results in pulmonary edema. Alternatively, Sofer and coauthors1 proposed that pulmonary edema actually develops during the obstruction, when as a consequence of highly negative pleural pressures, there is an increased preload and impaired left ventricular ejection. The pulmonary edema becomes roentgenographically apparent after removal of the obstruction due to a decrease in lung volume consequent to removal of the patient’s “intrinsic PEEP.”

In our patient, a paradoxical respiratory deterioration occurred immediately following successful laser photoabec-
Cardiac Amyloidosis Causing Ventricular Tachycardia

Diagnosis Made by Endomyocardial Biopsy

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In patients with malignant ventricular arrhythmias, endomyocardial biopsy may be helpful when all other findings from the workup are negative. A case of nonsustained polymorphic ventricular tachycardia is presented. The findings from an echocardiogram, coronary angiogram, and cardiac catheterization were negative. An electrophysiologic study showed inducible nonsustained ventricular tachycardia. A right ventricular endomyocardial biopsy was diagnostic of cardiac amyloidosis. The findings from a workup for systemic amyloidosis were negative. Primary cardiac amyloidosis should be considered in patients with malignant arrhythmias and no documented heart disease, and endomyocardial biopsy is helpful in making this diagnosis.

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In patients with serious ventricular arrhythmias and no obvious heart disease, it has been shown that an endomyocardial biopsy may be helpful in identifying underlying cardiac pathologic abnormalities. It has also been well established that cardiac amyloidosis may be a cause of ventricular arrhythmias and sudden cardiac death; however, the diagnosis of cardiac amyloidosis in patients with ventricular arrhythmias has been one suspected from biopsy of other organs and confirmed by postmortem examination.

We report herein a case of cardiac amyloidosis presenting with malignant cardiac arrhythmias in which the pathologic diagnosis was made ante mortem by endomyocardial biopsy of the right ventricle.

CASE REPORT

A 66-year-old man was admitted to the hospital for atypical chest pains. He had been well previously with no prior cardiovascular disease or complaints. The patient denied dyspnea, fatigue, palpitations, dizziness, or syncope. He had noted an upper respiratory infection during the past two weeks. The findings from physical examination were unremarkable. The 12-lead ECG showed left axis deviation and a QS complex in leads V1 and V6; the QRS voltage was normal.

While on telemetry monitoring, runs of nonsustained polymorphic ventricular tachycardia were noted (Fig 1). Cardiac enzyme levels were negative for infarction. The ESR was elevated at 60 mm/hr. A two-dimensional echocardiogram showed mild concentric left ventricular hypertrophy but no other abnormalities.

A treadmill test was equivocal for ischemia. Because of continued chest pain and ventricular arrhythmias which were poorly controlled by administration of procainamide or flecainide, cardiac catheterization was performed. Hemodynamic findings at catheterization revealed a right atrial pressure of 4 mm Hg, right ventricular...

REFERENCES


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