Reperfusion Pulmonary Edema After Pulmonary Artery Thromboendarterectomy[1-3]

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SUMMARY Pulmonary artery thromboendarterectomy (PAT) is a potentially curative procedure in chronic, major vessel thromboembolic pulmonary hypertension. However, postoperative reperfusion pulmonary edema (RPE) has been a serious complication, often requiring prolonged mechanical ventilation. This entity has been described only anecdotally in the past. To characterize it more fully, we retrospectively analyzed the course and potential determinants of RPE after thromboendarterectomy in 22 patients who had PAT at our institution from 1969 through 1984. Particular attention was directed to clinical data, thrombus location, areas operated, postoperative roentgenograms, and preoperative and postoperative hemodynamic data. In all patients but 1, RPE developed within 72 h after surgery, corresponding to anatomic locations distal to vessels subjected to PAT. Regions of lung not reperfused at surgery were uniformly spared. Pulmonary capillary wedge and/or left atrial pressures preoperatively and postoperatively were not elevated. None of the preoperative data predicted which patients would develop more persistent RPE. These observations suggest that the phenomenon of RPE is a peculiar, focal form of pulmonary edema, the basis for which remains to be defined.

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Introduction

Chronic, large-vessel thromboembolic pulmonary vascular disease is one cause of pulmonary hypertension and chronic right ventricular failure (1). The demonstration that this form of pulmonary hypertension is potentially correctable by thromboendarterectomy (1-15) apparently has heightened interest in this entity. In the 14-yr period 1969 to 1983, we had 20 such patients referred; since 1983, more than 30 patients have been referred.

Although the long-term outcomes of thromboendarterectomy have been quite positive, the postoperative period has been difficult and characterized by sustained arterial hypoxemia and radiographic pulmonary infiltrates in regions distal to vessels subjected to endarterectomy. This syndrome of hypoxemia and focal pulmonary infiltration, which we have called reperfusion pulmonary edema (RPE), often requires prolonged mechanical ventilation and increased inspired oxygen concentrations for days to weeks after surgery.

This syndrome has been noted previously (6, 10, 11), but its incidence and characteristics have not been well defined. We have encountered RPE consistently after thromboendarterectomy, and we report here our experience with this syndrome in 22 patients. Our purposes were to define its incidence, location, and duration and to determine whether any preoperative data could be used to predict its duration.

Methods

The medical records of 22 patients who had undergone pulmonary artery thromboendarterectomy at the UCSD Medical Center between July 1970 and May 1984 were reviewed retrospectively. Patient characteristics (age, sex, duration of symptoms) and preoperative and postoperative pulmonary spirometry, gas exchange, and hemodynamic data were recorded. Preoperative ventilation/perfusion lung scans and pulmonary arteriograms, and serial postoperative chest roentgenograms and perfusion scans were reviewed by all of us, recording the preoperative and postoperative distributions of pulmonary blood flow and the nature, location, and duration of postoperative roentgenographic abnormalities. Chest radiographs were obtained daily during each patient's stay in the intensive care unit and at approximately 3-day intervals thereafter until discharge or clearing of pulmonary infiltrates. Arterial branches rendered patent by thromboendarterectomy were recorded, and the location of these vessels was compared with the location of the pulmonary infiltrates.

In all patients, thromboendarterectomy specimens were submitted for routine pathologic examination.

Statistical analysis was performed using standard regression analysis to obtain product-moment correlation coefficients; p values < 0.05 were accepted as significant.

Results

Patients were 21 to 69 yr of age (mean, 47 yr). Pertinent preoperative and postoperative data are listed in table 1. Radiographic abnormalities developed in all patients except 1 (Patient 22). All pulmonary infiltrates developed within 72 h of surgery. In some instances, the infiltrates were evident on the first postoperative radiograph, taken approximately 2 h after chest closure. The infiltrates persisted from 1 day to several weeks (mean, 9 days), and in all cases developed *only* in lung regions subtended by vessels subjected to endarterectomy. One representative set of angiograms and radiographs is shown in figure 1. Reperfusion edema developed only in regions of the lung supplied by arteries opened by endarterectomy. Postoperative pulmonary capillary wedge pressures or left atrial pressures, available in all patients, were normal (all < 14 mm Hg; mean, 10).

The pulmonary infiltrates were all similar. They were dense, alveolar-filling infiltrates. Air bronchograms often were visible. No Kerley B lives were present, nor was there significant volume loss. The infiltrates were consistently interpreted by us, radiologists, and attending physicians as "focal pulmonary edema."

Statistical analyses disclosed that the duration of postoperative reperfusion edema did not correlate with any preoperative clinical or laboratory feature, including age, sex, duration of symptoms,

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