BRONCHIOLITIS OBLITERANS-ORGANIZING PNEUMONIA AFTER CORONARY ARTERY BYPASS GRAFT SURGERY

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Pulmonary edema, atelectasis, and pneumonia are frequent causes of respiratory dysfunction after cardiac surgery. Patients are usually dyspneic, hypoxic, and have pulmonary infiltrates. We describe the case of a patient in whom hypoxemia and pulmonary infiltrates developed as a result of bronchiolitis obliterans-organizing pneumonia (BOOP) after coronary artery bypass graft surgery (CABG).

Clinical summary. A 52-year-old woman had fever, shortness of breath, and bilateral alveolar infiltrates 1 day after CABG. The patient's medical history included coronary artery disease, peptic ulcer disease, gastroesophageal reflux disease, and a 10-year cigarette smoking history. She underwent quadruple CABG with an internal thoracic artery graft to the left anterior descending coronary artery and three saphenous vein grafts. The aortic crossclamp time was 60 minutes and the bypass time was 124 minutes. Intraoperative left ventricular ejection fraction estimated by transesophageal echocardiogram was 58%.

She was extubated on postoperative day 1 but had pleuritic chest pain. Physical examination revealed a well-developed woman in mild respiratory distress, alert and oriented, with a temperature of 101.3°F, a heart rate of 90 beats/min, a blood pressure of 109/50 mm Hg, a respiratory rate of 22 breaths/min, and an oxygen saturation of 89%. Lung auscultation revealed bilateral inspiratory crackles at the bases with decreased breath sounds throughout. The chest x-ray film showed decreased lung volumes, bilateral lower lobe infiltrates, and atelectasis. The arterial PO_2 while the woman was breathing room air was 63 mm Hg, with an alveolar-arterial oxygen gradient of 50 mm Hg and a white blood cell count of 16,200 cells/mL. She received nasal bilevel positive airway pressure ventilation (BiPAP; Respironic Inc, Murrysville Pa), furosemide, and ticarcillin/clavulonic acid empirically for possible nosocomial pneumonia. Eight days later she was still febrile. The white blood cell count had increased to 17,300 cells/mL, sputum cultures remained negative to date, blood cultures grew *Bacillus spp* in one aerobic bottle, and tobra-

Fig 1. Computed tomographic scan of the chest showing ground-glass opacities with areas of consolidation and bilateral pleural effusions.

mycin was added. A computed tomographic scan showed ground-glass opacities in the upper and middle lung fields, diffuse fibrotic changes, areas of consolidation, and small bilateral pleural effusions (Fig 1). No pulmonary emboli were seen on pulmonary angiography. No improvement was seen despite changing antibiotics to imipenem and cilastatin.

Transbronchial biopsy with a fiberoptic bronchoscope was performed on postoperative day 17. No endobronchial lesions were seen. Examination of the biopsy tissue revealed acute and subacute inflammation with fragments of fibromyxoid material in the bronchiolar lumen surrounded by granulation tissue, consistent with BOOP (Fig 2). Quantitative cultures of the bronchoalveolar lavage fluid were negative. The patient was started on a regimen of prednisone 30 mg by mouth twice a day, and her symptoms improved substantially. She was discharged 2 days later. A follow-up computed tomographic scan showed resolution of the ground-glass opacities and areas of consolidation (Fig 3). She then experienced a recurrence of symptoms once steroids were tapered, necessitating a prolonged second course of steroids.

Discussion. Many noninfectious processes may be mistakenly diagnosed as nosocomial pneumonia in patients in the intensive care unit who have dyspnea and worsening pulmonary infiltrates. These include atelectasis, pulmonary embolus with infarction, lung contusion (in trauma patients), and congestive heart failure. Cardiopulmonary bypass has

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Received for publication Aug 24, 1999; accepted for publication Oct 18, 1999.

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J Thorac Cardiovasc Surg 2000;119:382-3

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0022-5223/2000 \$12.00 + 0 12/54/103730

Fig 2. Histologic section of transbronchial biopsy tissue (hematoxylin-eosin stain, original magnification $\times 40$). A branching polypoid mass of loose myxoid fibroblastic tissue is attached focally to the air space wall in the upper right of the field.

been shown to alter pulmonary function. Acute lung injury and increased-permeability pulmonary edema occur more frequently with prolonged bypass periods.¹ This syndrome may be mediated by complement and neutrophil activation causing pulmonary epithelial and endothelial injury.

BOOP is a clinicopathologic syndrome that was described in 1985 by Epler and associates.² It is typified by dyspnea, fever, cough, hypoxemia, multiple patchy alveolar opacities on chest radiography, and histologic findings consisting of granulation tissue within the small airways and chronic inflammation surrounding the alveoli. Computed tomography may show areas of diffuse ground-glass opacities and/or areas of air-space consolidation.³ These infiltrates are generally peripheral. Although the classic clinical syndrome of BOOP is idiopathic, a variety of diseases have been associated with BOOP. These entities include connective tissue disorders, toxic gas inhalation, drug ingestion, organ transplantation, hematologic disorders, and malignant diseases. Infections have also been implicated as causes of BOOP.⁴

The diagnosis of BOOP has usually been made by open lung biopsy. The role of fiberoptic bronchoscopy has been evaluated by Polleti and associates.⁵ The presence of greater than 25% lymphocytes along with foamy macrophages and neutrophils or eosinophils in the bronchoalveolar lavage fluid is suggestive but not confirmatory of BOOP. Transbronchial biopsy specimens are often too small to allow diagnosis. However, if the specimens are large enough to contain all of the elements of BOOP, as in our case, transbronchial biopsy can be a diagnostic procedure.

The treatment of BOOP is prednisone, 1 mg/kg per day for 1 to 3 months followed by a slow taper for 6 to 12 months. The prognosis is good, with approximately 65% of patients

Fig 3. Computed tomographic scan of the chest showing resolution of the ground-glass opacities.

having complete clinical and physiologic recovery and another 30% having varying degrees of improvement.² In a small subgroup of patients the disease follows a rapidly progressive and fatal course.⁶ In some, the disease recurs when the dose of steroids is reduced; the dose must then be increased.

We have not found any reported cases of BOOP after cardiopulmonary bypass or any other forms of extracorporeal circulation. BOOP is an unusual postoperative complication of CABG and may be confused with nosocomial pneumonia, as in this case. The diagnosis should be considered if no other cause for hypoxemia, fever, and pulmonary infiltrates can be found. Effective treatment of this disease depends on an accurate diagnosis. Clinicians should use all available tools in pursuing a diagnosis, including bronchoscopy, thoracoscopic lung biopsy, and if necessary open lung biopsy.

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