Left Main-Stem Bronchial Stenosis Complicating Bronchial Artery Embolization*

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A 36-year-old patient was found to have severe left main-stem bronchial stenosis two years after bronchial artery embolization (BAE) for hemoptysis. Embolization-induced bronchial ischemia appeared to be the only potential cause for the observed lesions, and, to our knowledge, this constitutes the first report of late bronchial sequelae following BAE. Despite balloon-catheter dilatation of the stenosis, the severity of poststenotic lesions led to left pneumonectomy. The anatomic data further supported the hypothesis of a complication of BAE. Clinicians and radiologists should be aware of this potential complication of a widely used therapeutic procedure.

**CASE REPORT**

In April 1984, a 34-year-old male high-school teacher was admitted to another hospital for evaluation of a first episode of hemoptysis of moderate abundance. He had no remarkable medical history except for whooping cough at the age of 17 years without clinically detectable sequelae. Results of physical examination were normal, as were chest roentgenograms, arterial blood gas determinations, routine biologic measurements, and pharyngolaryngeal examination. Bronchofiberscopy failed to demonstrate active bleeding or cause of bleeding, but it showed small amounts of clotted blood in both right and left bronchial trees. The patient was discharged from the hospital with planned bronchoangiography, but he was readmitted in the same hospital a few days later because of recurrent hemoptysis. Emergency bronchofiberscopy again showed normal bronchi and failed to demonstrate the site of bleeding. Bronchial arteriography revealed a dysmorphic bronchial artery at the level of the left main-stem bronchus, with large proximal and peripheral branches (Fig 1). Although it was difficult to ascertain whether this artery was the source of bleeding, its angiographic aspect led to embolization that was performed with isobutyl-2-cyanoacrylate.† Immediately after embolization, the patient experienced violent retrosternal burning, but he was discharged from the hospital two days later. During the following weeks, however, the patient suffered permanent cough, mild fever, and intermittent left retrosternal burning, but he refused hospital admission or any additional investigation. After two months, apyrexia was obtained, but exertional dyspnea and wheezing were noted and remained stable over the following months.

The patient was first seen at our institution in November 1986 with complaints of wheezing and mild dyspnea on exertion and when he was teaching. Physical examination was unremarkable except for a left-sided wheezing. Chest roentgenogram and computed tomographic (CT) scan revealed signs of left upper lobe atelectasis and an aspect of localized stenosis of the left main-stem bronchus. Bronchofiberscopy showed a normal trachea, normal bronchi and carina, and a progressive funnel-shaped stenosis of the left main-stem bronchus reaching the lobar division (Fig 2). The exploration of the left bronchi distal to the stenosis was possible only with a pediatric bronchoscope (external diameter of 3.5 mm) passed by force through the stenosis and showed an upper and anterior attraction of the left upper lobe divisions, which could not be further explored; the left lower lobe divisions appeared essentially normal. Perfusion lung scintigraphy revealed the almost complete absence of perfusion to the left lung, but digitalized subtraction angiography confirmed the CT scan findings in demonstrating normal left pulmonary arteries. Because the patient was in relatively good health, without recent pulmonary infection or hemoptysis, and because he was reluctant to undergo any kind of additional investigation or intervention, simple clinical and radiologic surveillance was decided.

In July and September 1987, however, two episodes of ill-tolerated febrile bronchial infections slowly resolved with oral antibiotic therapy and the patient agreed to new investigations in October 1987. Chest roentgenogram and CT scan revealed complete left upper lobe and lingular atelectasis, and bronchofiberscopy disclosed an aggravated stenosis with distal retention of purulent secretions.

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**Figure 1.** Opacification of a dysmorphic left bronchial artery prior to embolization with isobutyl-2-cyanoacrylate. The main trunk of this artery is close to the roof of the left main-stem bronchus and presumably vascularizes it.
secretions. After preparation with oral antibiotics (amoxicillin) and physiotherapy, an attempt at bronchial dilatation was performed in January 1988 (Fig 3). A moderate and stable dilatation was obtained as confirmed by two repeated endoscopies within the 30 days following the procedure, with a diminished wheezing. However, the major bronchiectatic lesions revealed by per-dilatation bronchography in the lower lobe, persistent upper lobe and lingular atelectasis without any therapeutic possibility, and the recent ill-tolerated infections in the lung without any existing or potential functional value were considered an indication for left pneumonectomy. The operation was performed uneventfully in February 1988. Remarkable intraoperative findings included only a diffusely hypervascularized pleura and a close adherence of the main-stem bronchus to the superior pulmonary vein.

The histopathologic examination of the left lung confirmed diffuse bronchiectasis in both lobes and showed a localized hemicircumferential transpapillary destruction of the main-stem bronchus with intense endobronchial and peribronchial fibrosis evocative of post-necrotic healing. No potential cause of bronchial stenosis was found, but small amounts of endovascular amorphous material (presumably isobuty1-2-cyanoacrylate) were observed in small bronchial arteries close to the stenosed bronchial segment. Whether bronchiectasis was the cause of the initial hemoptysis or the consequence of the main-stem bronchial stenosis could not be established. The patient is currently doing well, and he works normally without new episodes of pulmonary infection or hemoptysis.

**DISCUSSION**

Although a description of the early bronchial changes following BAE is not available in our patient, the direct causal relationship between BAE and the observed stenosis can be reasonably affirmed because of the following: initial chest roentgenogram and bronchofiberscopy were normal; the embolized artery was very close to the left main-stem bronchus and probably vascularized it (Fig 1); the patient experienced intense and prolonged chest pain immediately after embolization; the histopathologic aspect of the surgically removed stenosis is highly evocative of postinfarction healing; and no other obvious cause of bronchial stenosis can be evoked in this observation. The incidence of ischemic lesions after BAE is unknown. Transient chest pain following BAE appears rather common (up to 46 percent of patients is one series) and has been attributed to temporary ischemia of various intrathoracic structures; trachea, bronchi, pleura, esophagus, or chest wall. Regarding bronchial ischemia, the incidence of post-BAE necrotic lesions has never been evaluated by systematic endoscopy, and this complication might occur more often than can be diagnosed. Symptomatic severe lesions, however, appear extremely rare.

To our knowledge, our case illustrates the first report of severe late bronchial sequelae caused by BAE. To our knowledge, only four cases of ischemic lesions on a main-stem bronchus have been published, including one fatal, one severe, and two minor lesions. One patient died 25
days after BAE with alcohol from massive hemoptysis secondary to a fistula between the left pulmonary artery and the adjacent necrotic origin of the left upper lobe bronchus. In another case, BAE with an absorbable gelatin sponge (Gelfoam) resulted in a fistula between the esophagus and the left main-stem bronchus, with a very localized necrosis of the posterior wall of the bronchus and a larger ischemic area in the esophagus. Surgical repair with bronchial and esophageal sutures, however, appeared successful in this case. A zone of necrosis on a right main-stem bronchus was also reported after absorbable gelatin sponge embolization with a slow resolution within a few weeks. Finally, an asymptomatic 3 x 4-mm area of infarction of the mucosa of the left main-stem bronchus was described after embolization with a 10 percent solution of sodium chloride; two weeks later, however, the lesion was no longer detectable. Whatever the true incidence of severe ischemic lesions induced by BAE, the predisposing factors for such complications remain unclear. In our observation, the probable vascularization of the roof of the main-stem bronchus by the embolized artery trunk, together with the moderate hypertrophy of this vessel and the absence of evident anastomoses with other arteries perhaps constitute risk factors for BAE-induced ischemia. Furthermore, although ischemia has been described with virtually all embolization agents, the nonresorbable character of isobutyl-2-cyanoacrylate might be incriminated in the failure of posts ischemic healing mechanisms.

To our knowledge, only five cases of balloon-catheter dilatation of bronchial stenosis in adults have been reported in the literature.10,11 Partial or complete success was obtained in all cases, with times of follow-up ranging from a few weeks to 14 months. In two cases, however, laser photoresection was associated with balloon dilatation and the respective effects of each procedure were not clearly determined.11 In our patient, a small degree of dilatation was obtained with the balloon catheter alone, as proved by immediate postdilatation bronchography, diminished wheezing, and subsequent bronchofiberscopic controls. This technical success, however, was not beneficial to the patient because major distal bronchial lesions were evident already and left pneumonectomy could not be avoided.

We conclude that clinicians and radiologists must be aware of the possibility of severe late bronchial sequelae following BAE and that more information is needed regarding the pathophysiology and, consequently, the prevention of this rare but severe complication.

ACKNOWLEDGMENTS: The authors thank Drs. M. J. Botto and J. André for providing radiologic and anatomic data and Dr. D. Musset for his helpful comments.

REFERENCES


Pulmonary Edema Associated with Electrical Injury*

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The occurrence of cardiogenic pulmonary edema following alternating current electrical injury has not been reported. A patient developing severe pulmonary edema immediately following an electrical injury-induced episode of ventricular fibrillation is described. Evidence that the etiology of the pulmonary edema was cardiogenic is derived from both hemodynamic data and the calculation of the pulmonary edema fluid to serum colloid osmotic pressure ratio.

(Chest 1990; 97:1248-50)

Electrical injury in power line workers and the subsequent financial support from utility companies were instrumental in early modern resuscitation research and the development of defibrillators for clinical use.6 The application of direct current for the conversion of atrial fibrillation has been associated with pulmonary edema,7,8 but the mechanism of edema formation in these cases remains unknown.9 The cardiopulmonary sequelae of alternating current electrical injury have received little attention in the medical literature. The occurrence of pulmonary edema and the status of clinical cardiac function after injury have not been reported. A review of the treatment of lightning injury9 describes a patient who developed pulmonary edema, but provides no data supporting the etiology as cardiogenic or noncardiogenic. It is plausible that the

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