Pathologic Bronchial Vasculature in a Case of Massive Hemoptysis due to Chronic Bronchitis*

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The cause of bleeding in a patient with recurrent massive hemoptysis was not apparent after bronchoscopy and gross examination of the lobectomy specimen. Histologic submission of all major bronchi uncovered dilated, tortuous bronchial arteries just below the bronchial mucosa with sites of both current and healing arterial rupture. This bronchial arterial abnormality is common to several chronic pulmonary diseases, but is rarely diagnosed as a cause of massive hemoptysis. Careful pathologic examination of major bronchi in the setting of hemoptysis of unknown causation is recommended.

Massive hemoptysis is usually due to primary lung cancer, tuberculosis, lung abscess, or bronchiectasis. More recently, such bleeding has also been ascribed to chronic bronchitis. The bronchial arteries are known to be increased in size and tortuous in chronic bronchitis. However, to our knowledge, the histologic demonstration of the bronchial vascular lesion producing massive hemoptysis in chronic bronchitis has not been reported. We describe and illustrate such a case.

CASE REPORT

A 68-year-old woman presented with hemoptysis and profound fatigue. Three years previously, she had her first episode of hemoptysis amounting to 500 ml. Stable left upper lobe streaky infiltrates were noted on her chest roentgenogram. Up until a year prior to hospital admission, she had smoked three cigarettes a day for 50 years. Her occupational history included six-months’ employment at a hot soldering plant. There was no history of tuberculosis.

Six months prior to her hospital admission, she developed a persistent cough without phlegm. She noted some slight wheezing and shortness of breath. Two months prior to the current admission, she again had another episode of hemoptysis. Bronchoscopic findings at that time were normal. Cytologic study and cultures were negative. After discharge and until this admission, she experienced intermittent episodes of 200 to 300 ml of hemoptysis.

On examination, inspiratory rhonchi were noted in the left upper and middle lung fields and there were scattered wheezes present throughout all areas. The chest roentgenogram was unchanged from three years ago.

The bronchoscopy was limited by thick plugs of mucus and blood. After acetylcysteine and bronchodilator therapy, bronchoscopy was again performed. No abnormal findings were observed. Smeads and cultures for acid-fast bacilli as well as routine cultures were negative. Cytologic study was negative. Her hemoglobin was 10.4 g/dl and

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FIGURE 1A (upper). Disrupted dilated bronchial artery communicates directly with bronchial lumen (arrow) and is largely filled with a recent fibrin clot (F) (hematoxylin-eosin, × 40). 1B (lower). Same artery exhibiting areas of mural fibrosis, loss of elastica on the bronchial luminal side, and perivascular neutrophils and lymphocytes. Much of the vascular lumen is filled with fibrin (F) (elastic stain, original magnification × 160).
Disc

Figure 2. Another bronchial artery, again just below the bronchial lumen, nearly completely occluded by organized fibrous tissue suggesting previous injury and thrombosis (elastic stain, original magnification × 160).

her white blood cell count was 5,600/cu mm. Results of her coagulation profile and other laboratory survey studies were normal. Hours after her repeated bronchoscopy, she coughed up 500 ml of bright red blood. A third bronchoscopy identified fresh blood clots in the left upper lobe bronchi; however, no active bleeding site was evident. A left upper lobectomy was then performed. She has remained free of hemoptysis at one year’s follow-up.

The surgical specimen consisted of a left upper lobe weighing 330 g. The parenchyma contained areas of recent hemorrhage without any other gross abnormality. Cartilaginous bronchi contained intraluminal blood, but there was no bronchiectasis, bronchial mural thickening, mucosal exudate, or other mucosal abnormality. The pulmonary arterial and venous systems were unremarkable.

Microscopically, the alveolar parenchyma showed only recent hemorrhage. Sections of all cartilaginous bronchi were examined, and elastic stains and step-sections were performed. Enlarged and tortuous bronchial arteries were seen in several bronchi just below their basement membrane. In one area, such a bronchial artery communicated directly with the bronchial lumen. This vessel wall showed focal disruption, areas of fibrosis, loss of the elastica in most areas, and patchy infiltrates of neutrophils and lymphocytes (Fig 1). Much of the vascular lumen contained a recent fibrin thrombus. In another bronchus, a bronchial artery located just beneath the basement membrane was nearly completely occluded by organizing fibrous tissue, suggesting previous injury and thrombosis (Fig 2). Bronchial glands in general were slightly enlarged, and lymphoplasmacytic inflammation was present in several foci, usually associated with bronchial mucosal squamous metaplasia. Malformed vessels were not a feature in other areas of the lung or bronchial sections.

Discussion

We are unable to find a similar report depicting the cause of massive recurrent hemoptysis in chronic bronchitis. Within the resected lobe, we found markedly dilated and tortuous arteries, including one with fresh rupture and another site with resolving disruption. Tortuous and dilated bronchial arteries have been associated with a wide variety of chronic pulmonary diseases, including chronic bronchitis. Two pathogenic mechanisms common to these conditions are (1) changes in the pulmonary artery pressure and (2) development of a precapillary bronchopulmonary anastomotic network. While most often widespread within the lung, one investigator has noted these bronchial arterial changes can be limited to one lobe. This may well be true in this patient.

The ascribing of hemoptysis to “bronchitis” raises some questions. In some studies, the diagnosis is made solely by the bronchoscopic observations. In our case, there were no mucosal abnormalities. The major pathologic finding that correlates with clinical chronic bronchitis is the enlargement of bronchial glands. However, the term is also used pathologically to denote chronic inflammation of the bronchi. Because of the mild bronchial glandular enlargement and increased inflammatory cells present in our patient, we regard this lobectomy specimen as coming from a patient with pathologic chronic bronchitis. Our use of a pathologic definition for the disease explains the insensitivity of bronchoscopy in this case.

Bronchial arteriography may have been diagnostic in our patient. Bronchial artery embolization, a potentially therapeutic option, has proved valuable in both immediate and long-term cessation of massive and recurrent hemoptysis.

The inability to find the cause of massive hemoptysis raises several issues. Since the underlying disease is not recognized, further management and diagnostic workup is problematic; the prognosis remains guarded. We emphasize the importance of embedding all large bronchi in a specimen to look for bronchial arterial lesions. This technique will also be valuable in cases of other types of small bronchial lesions, such as neoplasia or medial defects in bronchial arteries that may be otherwise missed even by careful gross examination.

References

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