CASE REPORTS

Anesthesiology
1996; 84:1234–6
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Endobronchial Inflammatory Polyp after Thoracoabdominal Aneurysm Surgery: A Late Complication of Use of a Double-Lumen Endobronchial Tube

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ENDOBRONCHIAL inflammatory polyps, although rare, have been described after aspiration of a foreign body,[1,2] smoke inhalation,[3,4] and infection.[5–7] To our knowledge, there have been no reports of an endobronchial polyp after placement of a double-lumen tube. We recently cared for such a patient who underwent graft replacement for thoracoabdominal aneurysm. An endobronchial polyp was detected at the site of the cuff of the endobronchial portion of double-lumen tube 6 months after surgery.

Case Report

A 63-yr-old, 44-kg, 139-cm woman was admitted to the emergency department with a 2-day episode of hemoptysis. Six months before, she had undergone resection of a thoracoabdominal aneurysm involving most of the descending aorta and the upper abdominal aorta. Repair was performed *via* a left thoracotomy and one-lung ventilation, and femorofemoral cardiopulmonary bypass was used. The trachea had been intubated with a 35-Fr left-sided disposable double-lumen tube (polyvinyl chloride Mallinckrodt tube, St. Louis, MO). The tracheal and bronchial cuffs were inflated with 5 and 2 ml of air, respectively, confirming moderate tension in the pilot balloons on palpation. The position of the double-lumen tube was adjusted using fiberoptic bronchoscopy so that the proximal edge of the bronchial cuff was in the left mainstem bronchus just below the carina. The patient was placed in the right lateral decubitus position, and the position of the tube was reconfirmed by fiberoptic bronchoscopy and auscultation. The patient was cooled to a core temperature of 17°C with total bypass when the distal end of the graft was sutured and intercostal arteries were reattached. Bypass time, total cross-clamp time, and duration of deep hypothermic perfusion were 298, 227, and 15 min, respectively, and separation from cardiopulmonary bypass was uneventful. Because of persistent bleeding from the site of the reattached intercostal arteries and an apparent coagulopathy, massive blood transfusion was required, and her systemic blood pressure transiently decreased to 30 mmHg. Thus, the duration of anesthesia was extended to 18 h, with one-lung ventilation for 15 h. The endobronchial cuff was kept inflated throughout the one-lung ventilation. At the end of surgery, the endobronchial tube was replaced by a single-lumen endotracheal tube. The postoperative chest x-ray on admission to the intensive care unit revealed patchy infiltrate in the left lung field, consistent with an underlying pulmonary contusion. The patient continued to receive ventilatory support for 6 days. A chest x-ray taken 1 month postoperatively showed little improvement in aeration of the left lung field, which thereafter gradually cleared, and a complete recovery was seen on the discharge x-ray. Five months after surgery, the patient began to complain of a productive cough and whitish sputum, followed by occasional hemoptysis. Follow-up chest x-rays showed no abnormal findings. However, as episodes of hemoptysis worsened, a second admission was required.

The chest x-ray showed no apparent abnormality, but a computed tomogram of the chest revealed a mass in the left mainstem bronchus. Fiberoptic bronchoscopy confirmed a polypoid tumor at the upper portion of the left mainstem bronchus 1.5 cm below the tracheal bifurcation, readily bleeding and occupying almost the entire internal lumen of the bronchus (fig. 1). Because pathologic examination of a biopsy specimen revealed inflammatory granuloma with no evidence of malignancy, corticosteroids were topically applied with subsequent reduction of size of the tumor. However, at 4 months' follow-up, fiberoptic bronchoscopy revealed enlargement of the tumor, and the patient underwent bronchoscopic polypectomy with electrocoagulation under general anesthesia. After a rapid-sequence intubation with a 8.0-mm inner diameter endotracheal tube, anesthesia was maintained with sevoflurane, nitrous oxide, and oxygen. After intubation, bright red blood flowed from the lumen of the endotracheal tube. Fiberoptic bronchoscopy revealed endobronchial hemorrhage from the left mainstem bronchus, a granulomatous polyp with a diameter of 1.5 cm was successfully resected, and hemorrhage ceased. The patient was transferred to the intensive care unit, where mechanical ventilation and sedation were continued overnight. Her trachea was extubated the next morning, there was no further hemoptysis, and she was transferred to the ward on day 2. On the night after returning to the ward, she had a massive pulmonary hemorrhage of 500 ml followed by cardiorespiratory arrest. Cardiopulmonary resuscitation was not successful, and permission for autopsy was refused. Pathologic examination of the specimen revealed chronic inflammation and granulation tissue consistent with an inflammatory polyp.

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Received from the Department of Anesthesia, Kobe City General Hospital, Kobe, Japan. Submitted for publication October 2, 1995. Accepted for publication January 5, 1996.

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Key words: Complications: endobronchial polyp. Double-lumen endotracheal tube: cuff.

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Fig. 1. Bronchoscopic view of the endobronchial polyp located in the left mainstem bronchus.

Discussion

Endobronchial inflammatory polyp is a rare occurrence usually attributable to any local irritation on the bronchial mucosa. Causes include foreign bodies, such as a chicken bone, a green pea, and a broncholith; inhalation of heated, irritant, or toxic gases[1–4]; and asthma.[5,6] In addition to irritation as a predisposing factor, the subsequent or underlying infection plays an important role in progress of the lesion. Local bronchial inflammation usually commences with edema or erosion and progresses to ulceration and, eventually, to a hyperregenerative process. Pathologic studies show that the inflammatory polyp consists of chronically inflamed vascular and edematous granulation tissue covered by normal ciliated respiratory-type epithelium.[8]

In the current case, the clinical course and the pathologic findings strongly suggested that injury by the endobronchial cuff of double-lumen tube was a causative factor of the inflammatory polyp. Although cuff-related granuloma associated with either tracheostomy or endotracheal tubes is well known, we found no reports of endobronchial inflammatory polyp related to the use of a double-lumen tube. The pathogenesis of a cuff-related lesion after an endobronchial tube may be similar to that related to an endotracheal tube. The pressure exerted by the cuff is transmitted to the bronchial mucosa, where ischemia-related injury may ensue. In this patient, a left-sided double-lumen tube had been placed during surgery; the endobronchial polyp was located in the left mainstem bronchus 1.5 cm below the carina, the approximate level of the tip or the cuff of the endobronchial tube. Considering the location of the polyp and distance from proximal left cuff to left lumen tip, damage appears to be related to the cuff rather than to the tube tip. In the current case, the volume of the endobronchial cuff was determined empirically; therefore, overinflation of the bronchial cuff cannot be excluded. The double-lumen tube remained in place for 18 h, whereas the endobronchial cuff was kept inflated for 15 h. Concomitant hypothermia during cardiopulmonary bypass and transient hemorrhagic hypotension might have reduced blood flow of the bronchial mucosa and contributed to the development of mucosal erosion at the site of endobronchial cuff. As the postoperative pulmonary infiltrate in the left lung resolved over a period of months, chronic inflammatory conditions of the bronchus may have promoted local inflammation, particularly at the level of the cuff, with resultant formation of granulation tissue. Inflammatory polyps may regress spontaneously without specific treatment once the causative factor is removed. Although corticosteroids are effective in regression of a polyp,[1] the polyp with a hypervascular nature in our patient did not resolve with steroid therapy.

In summary, we presented a case of endobronchial inflammatory polyp in the area consistent with the position of the endobronchial cuff of double-lumen tube, resulting in a fatal hemoptysis in a patient who underwent resection of a thoracoabdominal aneurysm with one-lung ventilation and femorofemoral cardiopulmonary bypass. The most probable cause for this delayed complication of endobronchial intubation was ischemia-related injury induced by the endobronchial cuff associated with a long period of cardiopulmonary bypass and hemorrhagic hypotension.

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Anesthesiology
1996; 84:1236–9
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Lippincott–Raven Publishers

Heparinase and Thromboelastography in Liver Transplantation for a Patient with von Willebrand's Disease

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THE role of heparin in impaired hemostasis during orthotopic liver transplantation (OLT), either exogenously derived from prior administration to the donor or endogenously derived from the donor liver, is controversial.[1,2] A report of improved clinical and laboratory indexes of bleeding after postreperfusion administration of protamine was not accompanied by objective evidence of the presence of heparin,[3] although Kang has promoted comparison of the untreated and protamine-treated thromboelastograph.[4]

Because the use of bacterial derived heparinase has been reported in cardiac surgery with the activated clotting time (ACT)[5] and thromboelastography,[6] heparinase was used to assess potential heparin effect with the thromboelastograph during OLT in a patient with type IIA von Willebrand's disease (vWD), when potential difficulty was expected in the diagnosis of heparin effect after reperfusion.

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Received from the Department of Anesthesiology, University of Texas Health Science Center, Houston, Texas. Submitted for publicatio August 8, 1995. Accepted for publication January 9, 1996.

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Key words: Blood: coagulation; heparin. Monitoring: vascular. Surgery: transplantation.

Case Report

A 46-yr-old woman (weight 53.5 kg, height 1.5 m) with primary biliary cirrhosis presented for OLT. She had portal hypertension that required previous sclerotherapy for bleeding esophageal varices and a transjugular intrahepatic portosystemic shunt (TIPS) procedure. She had vWD type IIA, the diagnosis having been made on the basis of normal von Willebrand factor (vWF) antigen level, low ristocetin cofactor activity indicative of low vWF function, and no platelet aggregation in response to low concentrations of ristocetin.[7]

At preoperative assessment, apart from the stigmata of end-stage liver disease, the patient had poor dentition, with slight bleeding from the base of the left upper premolar tooth without other sites of hemorrhage, and no evidence of cardiorespiratory disease. The patient had received one-deamino-8-D-argininevasopressin (DDAVP) for oral bleeding some years previously but had not reported bleeding complication with the TIPS procedure. Laboratory results included prothrombin time (PT) 11.7 s (normal range 11.1–13.1 s), partial thromboplastin time (PTT) 40.3 s (25–34 s), platelet count 110×10^3 mm^{-3}, hematocrit 31.6%, and normal serum electrolytes.

Prophylactic DDAVP (20 μg, intravenous) was administered before anesthetic induction because of the anticipated bleeding and the previous reported use. Standard anesthetic agents, adjuvants (sodium thiopental, fentanyl, pancuronium, and isoflurane), and monitoring were used.

Thromboelastographs were recorded concurrently with and without heparinase (4 IU tube^{-1}). A 1-ml blood sample was introduced into the heparinase tube, mixed by inversion, incubated for 3 min, and 0.36 ml withdrawn for insertion into the thromboelastography cuvette. No heparin was used in the flush solution for invasive monitors or during venoveno bypass, and the donor liver was flushed with 500 ml albumin (5%) before portal vein anastomosis. Perioperative hematologic variables are presented in table 1.

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