CASE REPORTS

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Aneurysmal Compression of the Trachea and Right Mainstem Bronchus Complicating Thoracoabdominal Aneurysm Repair

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PATIENTS presenting for repair of thoracoabdominal aneurysms pose many challenges for the anesthesiologist. The procedure requires a complicated anesthetic technique that must be adapted to the needs of specific clinical situations. A common practice is the use of double-lumen endobronchial tubes (DLT) and one-lung ventilation (OLV) to facilitate surgical exposure of the aneurysm. This technique may be contraindicated in situations in which the aneurysm may cause compression of the trachea and left mainstem bronchus. We report a case in which compression of the right mainstem bronchus precluded the use of a DLT and OLV in the repair of a large thoracoabdominal aneurysm.

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Case Report

A 77-year-old man was admitted to the hospital for elective repair of an extensive dissecting thoracoabdominal aneurysm (DeBakey type IIIB). During the 3–4 weeks before surgery, the patient had been experiencing progressive dyspnea on exertion with increasingly limited activity. The patient had also complained of progressive dysphagia and experienced a 20-pound weight loss in the 2 months before operation.

Preoperative evaluation revealed a thin, ill-appearing man in no apparent distress. There were no gross abnormalities of the head and neck, and the trachea was midline. Examination of the lungs with the patient in the upright position was unremarkable. Laboratory studies were significant only for a hematocrit of 31%. The chest x-ray revealed a widened mediastinum with normal-appearing lung fields. There was no report of tracheal or bronchial compression. A CT scan of the chest showed a dissecting aneurysm of the descending thoracic and abdominal aorta that was 8 cm at its widest margin.

In the operating room, radial artery, central venous, and pulmonary artery catheters were placed. A catheter was placed in the lumbar subarachnoid space for drainage of cerebrospinal fluid. Anesthesia was induced uneventfully with thiamylal, fentanyl, and pancuronium. Because of the patient's symptoms of dyspnca, we elected to intubate the trachea with a 8.5-mm single-lumen endotracheal tube, and to perform bronchoscopy before placement of a left-sided DLT. After tracheal intubation, it was noted that chest movement was asymmetrical and that breath sounds were diminished on the right side. Bronchoscopy revealed that the lower one-third of the trachea was approximately 50% narrowed, and that the right mainstem bronchus was almost completely occluded by external compression (figs. 1 and 2).

Two concerns arose at this point. First, we were reluctant to pass a DLT beyond the tracheal and bronchial obstruction, because it was unclear to what extent the aneurysm may have eroded the airway structures. Secondly, had we placed a DLT in the left mainstem bronchus, ventilation of the right lung may not have been possible because



Fig. 1. The black arrow indicates narrowing of lower trachea caused by posterior compression from the aneurysm. The white arrow marks the takeoff of the right mainstem bronchus. (From a color photo taken through a Pentax VB2000 video-bronchoscope/EMP image manager, Orangeburg, NY.)

of the mainstem compression on that side. To evaluate the latter issue, we passed a Fogarty catheter into the left mainstem bronchus for use as a blocker. With the left lung occluded by the inflated balloon, we were unable to ventilate the right lung, despite airway pressures of > 50 cmH₂O.

In light of these findings, it was elected to proceed with the use of the single-lumen endotracheal tube and "two"-lung ventilation. If surgical exposure was deemed inadequate with the left lung ventilated, or if the patient's respiratory status deteriorated, we were prepared to institute full cardiopulmonary bypass. The patient was placed in the right lateral decubitus position and a left thoracoabdominal incision was carried out. We found that the patient could be easily oxygenated, that his lungs could be ventilated while he was in this position, and that the surgeons were able to adequately expose the entire thoracic aorta with simple traction of the left lung. Surgery proceeded uneventfully with the use of our standard procedures, which include the use of aortafemoral bypass and moderate hypothermia (30° C). The aneurysm extended behind the trachea and beneath the right mainstem bronchus. These structures were thin and the spine was eroded. After graft placement, bronchoscopy was repeated and marked relief of the tracheal and bronchial compression was noted.

Further questioning of the patient postoperatively revealed that he had been unable to sleep except when lying on his side. Retrospective examination of the preoperative chest x-ray and CT did reveal radiographic evidence of airway compression by the aneurysm (figs. 3A and B). The patient had an extended postoperative course, but was subsequently discharged from the hospital and was doing well at the 6-month postoperative visit.

Discussion

Large aneurysms of the thoracic aorta can impinge on virtually any structure in the chest, thereby resulting in a wide variety of symptoms.^{3,4} Dyspnea and dysphagia, as exhibited by our patient, are not uncommon findings in patients with this disorder because of tracheal, bronchial, and esophageal compression. Use of endobronchial tubes to facilitate surgical repair of thoracic aneurysms has been employed for the past two decades.5 However, compression of the airway by an aortic aneurysm is a relative contraindication to the use of endobronchial bronchial DLTs, because of concerns of bronchial and aneurysmal rupture and exsanguination associated with placement. It is, therefore, vital that the anesthesiologist thoroughly evaluate the trachea and mainstem bronchi before placement of an endobronchial tube. Preoperative evaluation of the patient should include a history and physical examination directed at these issues. Appropriate studies should be performed to determine the extent of aneurysmal embarrassment of surrounding structures. When uncertainty exists, the airway should be examined under direct vision.

Our case is unique in that the patient was experiencing compression of the right mainstem bronchus by a thoracic aneurysm that, in our judgment, pre-

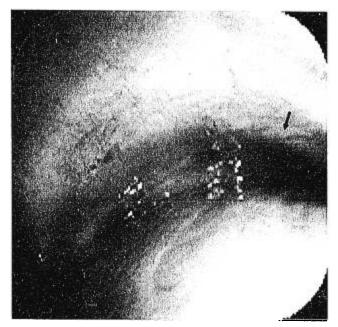


Fig. 2. Aneurysmal compression as seen from the entrance of the right mainstem bronchus. The arrow marks the entrance of the right upper lobe bronchus.

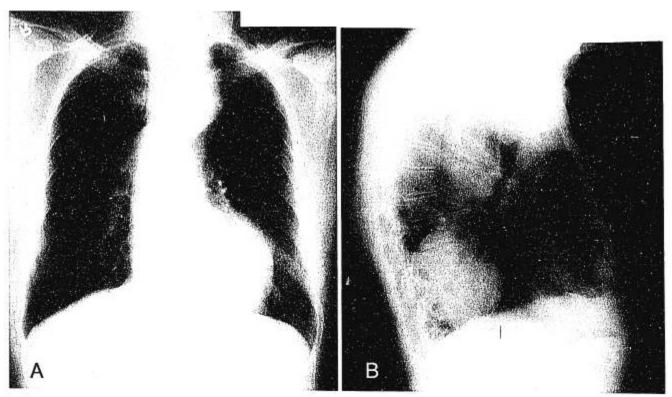


Fig. 3. (A) The PA chest film demonstrates the swing of the aneurysm from the midleft side to the lower right side of the trachea. (B) The lateral chest film demonstrates posterior tracheal encroachment by the aneurysm.

cluded the use of DLT and OLV. We found that, if we blocked the left mainstem bronchus with a Fogarty catheter balloon, the right lung could not be adequately ventilated. Therefore, a left-sided DLT would have been ineffective. We felt that placement of a right-sided DLT in this patient would have been both difficult and hazardous, because of the potential for bronchial or aneurysmal rupture. Deflation of the left lung facilitates surgical exposure to the descending thoracic aorta. For this reason, we were prepared to institute full cardiopulmonary bypass if exposure of the aneurysm had been inadequate with the left lung ventilated. Because the aorta had deviated into the right chest, surgery was possible with minimal traction and compression of the left lung.

This case, to our knowledge, represents the first report of right mainstem bronchial compression by a descending thoracic aortic aneurysm. Careful retrospective review of the patient's radiologic studies revealed the compression of the airway structures, which was not noted in the official readings. The clinician should, therefore, examine these studies preoperatively, es-

pecially in cases in which the patient's history and physical examination indicate compromise of the airway. Alternative methods of anesthetic and surgical management, including the use of cardiopulmonary bypass, must be anticipated in these settings to optimize patient safety and outcome.

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Determination of Intravascular Migration of an Epidural Catheter Using the Air Technique

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EPIDURAL hematoma associated with epidural anesthesia and anticoagulation can cause irreversible neurologic sequelae. This complication may occur in patients in whom anticoagulation with intraoperative heparin after atraumatic epidural catheterization had occurred;¹ intraoperative intraarterial thrombolytic therapy may pose an additional risk.²

We report a case in which we injected a small amount of air into the epidural catheter to confirm our suspicion that an epidural catheter had migrated into a blood vessel of a vascular surgery patient who was about to receive intraarterial urokinase. By making an unequivocal diagnosis of catheter migration, we avoided the use of the thrombolytic agent and the potentially disastrous consequences of epidural hematoma.

Case Report

Our patient was a healthy 22-yr-old man with a history of claudication in his right leg after blunt trauma, who presented for operative

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reconstruction of his right popliteal artery with a saphenous vein graft. He had several uncomplicated general anesthetics in the past, and his only medical problem was an asymptomatic variant of β -thalassemia. Medications before surgery included naproxen 500 mg bid, and intravenous heparin, which had been discontinued the day before surgery. Preoperative laboratory results included a hematocrit of 40%, PT (prothrombin time) of 13 s (normal 10–14 s), and PTT (partial thromboplastin time) of 29 s (normal 24–36 s).

We planned to use a combination of epidural and general anesthesia intraoperatively, and to infuse dilute local anesthetic *via* the epidural catheter to provide sympathetic blockade postoperatively. After administering 800 ml of lactated Ringer's solution intravenously, we used a loss-of-resistance technique to insert a 20-G nylon multiorifice catheter (Portex, Keene, NH) at the L3–L4 interspace. Neither blood nor cerebrospinal fluid could be aspirated from the catheter, no paresthesias occurred, and two 3-ml test doses of lidocaine 1.5% with 5 µg/ml epinephrine did not cause tachycardia⁴ or symptoms of local anesthetic toxicity. After administration of 6 ml 0.5% bupivacaine with 5 µg/ml epinephrine, the patient developed a sensory level of T-6 on the left and T-10 on the right.

We induced general anesthesia and muscle relaxation with 0.25 mg fentanyl, 350 mg sodium thiopental, and 10 mg vecuronium, and maintained anesthesia with 0.5–1.3% isoflurane in a 50% N_2O/O_2 mixture during the harvesting of the saphenous vein graft. An additional 3-ml dose of 0.5% bupivacaine with 5 $\mu g/ml$ epinephrine administered during the dissection did not cause an increase in heart rate. We then turned the patient to the prone position for the vascular reconstructive procedure.

Shortly thereafter, we administered an additional 3-ml dose of 0.5% bupivacaine with 5 μ g/ml epinephrine. This time, however, the injection was followed, within 20 s, by an increase in heart rate from 71 to 82 beats/min. Although suggestive of intravascular epidural catheter migration, this 11-beats/min increase in heart rate after epinephrine seemed inconclusive compared with the 32-beats/min mean maximum increase in heart rate described by Moore and Batra. Arepeat test with epinephrine-containing solution was similarly inconclusive. No change in blood pressure occurred after either injection. We were unable to aspirate blood from the catheter, even after