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Transesophageal Echocardiography Confirms Atelectasis Due to Right Mainstem Bronchial Intubation

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UNINTENTIONAL mainstem bronchial intubation is a well recognized potential hazard of tracheal intubation. It may lead to atelectasis, ventilation-perfusion mismatch, and hypoxemia. This case report illustrates how transesophageal echocardiography (TEE) may confirm atelectasis of the left lung associated with right mainstem bronchial intubation.

Case Report

A 69-yr-old woman presented for coronary artery bypass grafting. She had a history of exertional angina and diabetes mellitus and a remote history of asthma that currently required no therapy. She had normal pulmonary function tests. After placement of a peripheral intravenous catheter, a radial artery catheter, and a pulmonary artery catheter, anesthesia was induced with fentanyl, midazolam, and pipercuronium. Her trachea was intubated easily with an 8.0-mm endotracheal tube, which was secured with tape at 2 cm at the lips, and mechanical ventilation was used. A biplane TEE probe was inserted as part of our routine monitoring. Fifteen minutes after induction of anesthesia and while fractional inspired oxygen tension (FIO₂) was 1, arterial blood gases were pH 7.40, arterial oxygen tension (Pao₂) 278 mmHg, and arterial carbon dioxide tension PaCO₂ 34 mmHg. TEE examination showed normal left ventricular function and trivial mitral regurgitation. Views of the left chest demonstrated a normal descending thoracic aorta and well inflated left lung (fig. 1).

Two hours after induction of anesthesia, repeat arterial blood gases were pH 7.43, Pao₂ 170 mmHg, and PaCO₂ 35 mmHg on an FIO₂ of 1.0. Peak inspiratory pressures (PIP) had increased from 22 cmH₂O to 34 cmH₂O. Coincidental TEE reexamination of the descending thoracic aorta and left chest showed atelectasis of the left lung (fig. 2). The surgical field and drapes limited access to the patient's thorax, and adequate auscultation of the chest was not possible. As part of the surgical draping and positioning, the patient's head was under an "ether screen," which limited access to the patient's face and made it difficult to determine whether the endotracheal tube had been advanced further into the trachea. However, it could be appreciated that the tape securing the endotracheal tube had lost some of its adherence to the patient's face. Manual ventilation with larger tidal volumes to PIP of 45 cmH₂O did not relieve the atelectasis. The "ether screen" was moved, and the endotracheal tube was found to be at 23 cm at the lips. The endotracheal tube was untaped, pulled back, and resecured at 18 cm at the lips. After a few more larger tidal volume "sighs" to PIP of 40 cmH₂O, the TEE views of the descending thoracic aorta and left lung immediately reverted to images similar to figure 1. The remainder of the case was uneventful.

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Fig. 1. Transesophageal echocardiography image (transverse) showing the descending thoracic aorta and adjacent normally inflated left lung, which is in the brighter area to the lower left of the aorta (7-8 o'clock relative to the aorta). No detail of the left lung's structure is seen because of nearly complete reflection of ultrasound energy from the surface of the lung's air filled spaces. The darker area to the lower right (about 5 o'clock relative to the aorta) is likely due to the presence of the spinal column in that vicinity, although no details of its structure are discernible.
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Fig. 2. Transesophageal echocardiography image (transverse) showing the descending thoracic aorta and adjacent atelectatic left lung (P). The four dark "holes" in the left lung are due to blood-filled pulmonary vessels within the essentially completely fluid-filled collapsed lung parenchyma. There is, as in figure 1, a darker region in the vicinity of the spinal column, but no details of this are seen.

Discussion

In the normal patient, the inflated left lung is adjacent to the descending thoracic aorta. Because the lung is composed mostly of small air-filled spaces, ultrasound energy does not penetrate the lung and is nearly totally reflected from the surface. As a result, no detailed structure is seen around the descending thoracic aorta (fig. 1). If the lung is atelectatic, the normally air-filled spaces are collapsed, and the remaining essentially fluid-filled organ does conduct ultrasound energy into its interior. Then the details of the pulmonary vasculature often can be detected within the parenchyma of the lung by ultrasound examination (fig. 2).

TEE examination should not be considered as a primary technique for the detection of pulmonary atelectasis or the diagnosis of endobronchial intubation. It may confirm atelectasis of the left lung when standard techniques are unavailable or equivocal. It is important to recognize and properly interpret incidental TEE finding of lung atelectasis. It should not be confused with true pathology (e.g., tumor), and with the possible exception of situations involving intentional differential ventilation of the lungs (e.g., double-lumen endotracheal tube with left lung collapsed), atelectasis is never desirable or expected and should be corrected by appropriate therapeutic intervention.

References


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