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One-lung Anesthesia with Recent Pulmonary Embolism of the Ventilated Lung

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Acute pulmonary embolism is not uncommon.[1] The incidence at autopsy is 5–10 percent. The antemortem incidence is difficult to estimate due to the absence of classical signs and symptoms.[2] In contrast, pulmonary embolism developing during anesthesia and dual-lung ventilation is rare.[3,4] We report an unusual case in which one-lung anesthesia was administered in the presence of unrecognized pulmonary embolism of the ventilated lung.

REPORT OF A CASE

A 57-yr-old woman, hospitalized for 36 days, was found to have an asymptomatic, noncalcified 1-cm lesion in the left lung. She was scheduled for exploratory thoracotomy. She was a nonsmoker and her skin was noted to be well pigmented. Preoperative lung function studies showed an FEV_1 of 1.9 l (82% predicted) with a forced, vital capacity (FVC) of 2.5 l (76% predicted). Her hemoglobin was 10.3 $g \cdot dl^{-1}$. Her medical history revealed Crohn's disease of 10-yr duration for which she underwent proctocolectomy and ileostomy 27 days prior to her scheduled thoracotomy. In addition, 36 h prior to thoracotomy she had complained of a sudden onset of right-sided chest pain. The pain was exacerbated by deep breathing, application of local pressure, and lying on the right side. Examination of the heart and lungs as well as a chest roentgenogram and electrocardiogram had been noncontributory. The pain had resolved within 12 h while she received acetaminophen orally and was thought to have been musculoskeletal in origin.

On arrival in the operating room, she did not have overt cyanosis or tachypnea; however, a light-gray mottling of the skin and mucous membranes was apparent. Arterial blood pressure was 130/60 mmHg with a heart rate of 90 beats/min. Before induction of anesthesia, peripheral iv and radial artery catheters were inserted. Arterial blood was drawn for analysis. However, results of this sample did not become available until later. After breathing oxygen, anesthesia was induced with fentanyl, 0.2 mg iv, and thiopental, 250 mg iv. Succinylcholine, 80 mg iv, was used to facilitate right double-lumen endobronchial intubation. One-lung anesthesia (OLA) in the lateral position was maintained with isoflurane in 100% oxygen and pancuronium bromide, 5 mg iv. Results of the preinduction arterial blood gas analysis became available after the left chest had been closed. These revealed a Pa_{O_2} of 35 mmHg, Pa_{CO_2} 34 mmHg, and pH_a of 7.33 (inspired oxygen concentration (FI_{O_2}) of 0.21, alveolar–arterial oxygen tension difference ($PA\text{–}a_{O_2}$) 72 mmHg. The respiratory exchange ratio (R) was assumed to be 0.8 for all calculations of $PA\text{–}a_{O_2}$. An arterial blood sample taken during OLA had unfortunately clotted. However, blood shed at the operation site appeared well oxygenated. The lung lesion was benign (on frozen section) and surgery was limited to wedge excision. Arterial blood pressure remained at 120–130/70–80 mmHg and heart rate ranged from 90–105 beats/min throughout anesthesia. The chest was closed within 45 min of induction of anesthesia.

Postoperatively, dual-lung ventilation *via* an endotracheal tube was continued with an FI_{O_2} of 0.9 and PEEP of 7.5 cm. However, blood gas analyses were poor, with a Pa_{O_2} of 80 mmHg, Pa_{CO_2} 42 mmHg, pH_a 7.41, and $PA\text{–}a_{O_2}$ 508 mmHg. A thorough search failed to reveal a cause for the hypoxia. An oximetric pulmonary artery catheter was inserted *via* the right internal jugular vein. Pulmonary artery pressure (PAP) was 97/53 mmHg, central venous pressure (CVP) 27 mmHg, with a cardiac index of 2.6 $l \cdot m^{-2} \cdot min^{-1}$. Systemic vascular resistance (SVR) was 1260 $dyne \cdot s \cdot cm^{-5}$ and pulmonary vascular resistance (PVR) was 1000 $dyne \cdot s \cdot cm^{-5}$. Mixed venous oxygen saturation ($S\bar{v}_{O_2}$) was 47%. Pulmonary embolism was suspected. Perfusion scanning demonstrated a single, peripherally placed defect involving 40% of the right upper lobe. Ventilation scan was normal. Anticoagulation by a heparin infusion was initiated. Hypoxia and bilateral basal atelectasis continued to be a problem, and the patient required ventilatory support for 6 days. PAP and CVP remained elevated, and the pulmonary artery catheter was removed on the fourth postoperative day. At that time PAP was 45/17 mmHg and CVP 10 mmHg with an $S\bar{v}_{O_2}$ of 63%. PVR had decreased to 180 $dyne \cdot s \cdot cm^{-5}$. After tracheal extubation Pa_{O_2} was 67 mmHg, Pa_{CO_2} 46 mmHg, and pH_a 7.38 (FI_{O_2} of 0.6, $PA\text{–}a_{O_2}$ of 303 mmHg). Oral anticoagulation was continued with warfarin, and the patient required supplemental O_2 therapy for 3 weeks. She was discharged on the 36th postoperative day. Blood gases before discharge were Pa_{O_2} 67 mmHg, Pa_{CO_2} 38 mmHg, and pH_a 7.46 (FI_{O_2} of 0.21, $PA\text{–}a_{O_2}$ of 35 mmHg). The lung lesion was found to be a granuloma secondary to coccidioidomycosis.

DISCUSSION

This case demonstrates three unusual features. First, severe hypoxemia occurred with a small perfusion defect. Although there is no general agreement about the mechanism of hypoxemia from pulmonary embolism, right to left intrapulmonary shunting[5] and impairment of gas diffusion[6] are important factors. In the presence of an adequate cardiac output, pulmonary blood flow is redistributed to regions unaffected by embolism and leads to a disturbance of ventilation–perfusion (V/Q) matching.[7] Inhibition of hypoxic pulmonary vasoconstriction (HPV) by isoflurane, pulmonary hypertension, hypoxic compartments in the dependent lung, and surgical manipulation of the nondependent lung probably increased the V/Q mismatch.[8,9] Other mechanisms that may explain the severe hypoxemia include undetected minor pulmo-

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nary emboli, shunting of blood through a functionally closed but patent foramen ovale[10] and shunting through intrapulmonary arteriovenous anastomoses.[11] As the latter two causes were not suspected, appropriate diagnostic tests were not performed. Persistence of hypoxemia postoperatively was probably in part due to lung atelectasis.

Second, the severity of pulmonary hypertension is in marked contrast to the relatively small area of lung affected by embolism. Factors that might have exacerbated the pulmonary hypertension include hypoxemia, acidosis, undetected minor pulmonary emboli, and neurohumorally mediated vasoconstriction.[12-14] The relatively slow resolution of pulmonary hypertension in this patient is not incompatible with pulmonary embolism,[15] although postoperative atelectasis was also contributory.[14]

Third, pulmonary embolism in this patient probably occurred 36 h prior to thoracotomy. It was not detected preoperatively because cyanosis was probably obscured by anemia and skin pigmentation. In addition, chest wall tenderness suggested a local musculoskeletal disorder. Two cases have recently been reported where chest wall tenderness was the initial presenting feature of pulmonary embolism and led to a misdiagnosis.[16] The parietal pleura is innervated by intercostal nerves. Pulmonary infarction causes inflammation of the overlying pleura. Local pressure is thought to stretch the inflamed pleura and elicit pain.[16] Although unproven by venography, deep venous thrombosis probably was the source of pulmonary embolism. This patient had two major risk factors—immobility and a recent major abdominal operation.

In conclusion, this case provided a unique opportunity to observe the respiratory and hemodynamic changes associated with OLA and recent pulmonary embolism of the ventilated lung. Severe pulmonary hypertension and persistent hypoxemia were the cardinal features. In addition, chest wall tenderness proved to be a pitfall in the preoperative diagnosis of pulmonary embolism.

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