Severe Hypoxia Following Spinal Anesthesia: Possible Association with Pulmonary Embolism

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(Key words: spinal anesthesia, hypoxia, pulmonary embolism)

Although pulmonary embolism during or after spinal anesthesia is very rare, it has been reported in two orthopedic cases following hip fracture[1,2]. Acute onset of dyspnea, tachypnea, chest pain, cyanosis, tachycardia, and hypotension suggest pulmonary embolism, however, pulmonary radioisotope scan and pulmonary angiography are required for the definitive diagnosis. A case of severe hypoxia following spinal anesthesia, in a patient whose disease was diagnosed as pulmonary embolism a few weeks after surgery by lung scan, is presented here.

Case Report

A 48-yr-old, 58-kg, 150 cm man, suffering from left leg ulcer of unknown cause, was scheduled for skin graft of the left leg under spinal anesthesia. He had been taking clocapramine (neuroleptic) and mianserin (antidepressant) for more than 10 years for schizophrenia. Debridement had been performed under spinal anesthesia without any complications 3 months ago. Preoperative physical examination and laboratory data indicated no particular problems, although arterial blood gas tensions were not measured and the venogram of the left leg revealed some obstructive areas around the ulcer. Bloood pressure (BP) was 102/60 mmHg and the heart rate was 80 beats·min^{-1}.

Following intramuscular administration of 100 mg of secobarbital, as premedication, spinal anesthesia was performed with subarachnoid injection of 10 mg of tetracaine in 2.5 ml of 5 percent dextrose. 15 min later, the sensory skin anesthetic level, evaluated by pin prick, was Th10. Operation lasted 20 min and ephedrine 10 mg and atropine 0.25 mg were administered intravenously for the treatment of mild hypotension (BP 90/50 mmHg) and bradycardia (heart rate 55 beats·min^{-1}) during surgery. On admission to the recovery room, BP was 100/60 mmHg and heart rate 82 beats·min^{-1} and the level of sensory anesthesia was Th10. Approximately one hour after the admission to the recovery room, the patient suddenly became restless and complained of dyspnea, with dry cough. The heart rate transiently increased to 125 beats·min^{-1} while BP and the level of sensory anesthesia showed no marked change. Oxygen saturation, measured with a pulse oximeter, was 78% and arterial blood gas tensions, while breathing room air, were pH7.36, Pa_{O_2} 43 mmHg, and Pa_{CO_2} 45 mmHg. Oxygen was administered via a face mask. Twelve-lead electrocardiogram and chest x-ray showed no abnormal finding. Oxygen saturation increased gradually and central venous pressure was 1.5 cmH_2O. Thirty minutes later, the patient no longer complained of dyspnea when O_2 saturation increased to 96% while breathing approximately 40% oxygen via a face mask.

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J Anesth 5:303–305, 1991

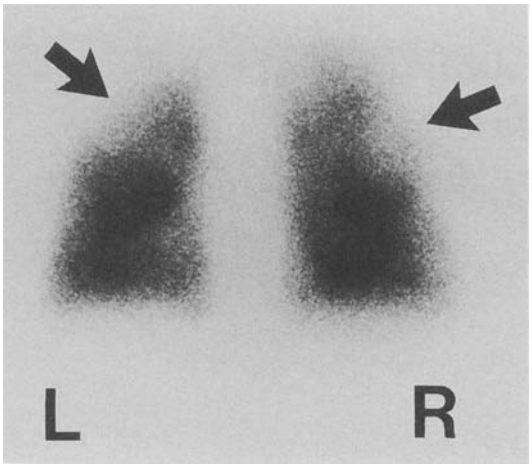

Fig. 1. A perfusion scan shows multiple defects (arrows) in both lungs. A ventilation scan was normal (not shown). They indicate high probability for the existence of pulmonary emboli.

The patient was transferred to the ward. Further dyspnea was not reported. The following day an arterial blood sample showed mild hypoxia (pH7.37, Pa_{O_2} 60 mmHg, and Pa_{CO_2} 45 mmHg) while breathing room air.

A few weeks later, radioactive ventilation-perfusion pulmonary scan was performed. The scan revealed multiple pulmonary emboli in both lungs (fig. 1). Anticoagulation therapy, with continuous intravenous heparin, was begun by the cardiologist. The cardiologist suggested that the pulmonary emboli came from venous thrombosis in the left leg and the patient had been suffering from asymptomatic small emboli before surgery.

Discussion

There are a number of factors, such as height of spinal block, patient position, hypotension or sedation, that may contribute to the development of hypoxia during or after spinal anesthesia. Davies et al.[3] reported that 42% of patients who underwent surgery under spinal anesthesia, in the lithotomy position, showed either transient episodes of arterial O_2 desaturation or trends of gradually progressive desaturation while breathing air, although only a few patients had clinically significant O_2 desaturation.

In the present case, severe hypoxia and tachycardia, without hypotension, occurred following spinal anesthesia, and pulmonary embolism was suspected by means of radioactive ventilation-perfusion pulmonary scan. Since there was no evidence for an etiology other than pulmonary embolism in the patient, it is probable that the severe hypoxia was a result of transient emboli. McIntyre et al.[4] reported that systemic arterial hypoxemia was the earliest, most sensitive manifestation of pulmonary embolism and hypotension was less frequent and transient in patients with no prior cardiopulmonary disease. Michael et al.[5] reported a case of intra-operative pulmonary embolism who developed a sudden, marked reduction in arterial oxygen saturation, detected by a pulse oximeter, without hemodynamic change. However, in the two cases of pulmonary embolism reported previously[1,2], both circulatory collapse and hypoxemia developed during spinal anesthesia. The hypoxemia improved rapidly without any medical treatment except O_2 administration in the present case. Mangano[6] reported rapid resolution of hemodynamic and respiratory changes, associated with acute pulmonary embolism, in a human subject. Although the exact cause is unknown, venodilatation, as a result of spinal anesthesia, might have been contributed to the transient central migration of a thromboembolus in this case.

Pulmonary embolus should be suspected in a patient who suddenly develops dyspnea, tachypnea, tachycardia, and cyanosis during spinal anesthesia or in the recovery room, even when cardiovascular collapse does not appear. Continuous monitoring of arterial O_2 saturation, by pulse oximetry, may be useful for early detection of pulmonary embolism in a patient with venous thrombosis.

(Received Aug. 1, 1990, accepted for publication Nov. 22, 1990)

References

1. Newens AF: Pulmonary embolism following spinal anesthesia: Report of a case. Anesthesiology 35:646–647, 1971
2. Berry AJ: Pulmonary embolism during

spinal anesthesia: Angiographic diagnosis via a flow-directed pulmonary artery catheter. Anesthesiology 57:57–59, 1982
3. Davies MJ, Scott DA, Cook PT: Continuous monitoring of arterial oxygen saturation with pulse oximetry during spinal anesthesia. Regional Anesth 12:63–69, 1987
4. McIntyre KM, Sasahara AA: The hemodynamic response to pulmonary embolism in patients without prior cardiopulmonary disease. Am J Cardiol 28:288–294, 1971
5. Michael S, Fraser RB, Reilly CS: Intraoperative pulmonary embolism: Detection by pulse oximetry. Anaesthesia 45:225–226, 1990
6. Mangano DT: Immediate hemodynamic and pulmonary changes following pulmonary thromboembolism. Anesthesiology 52:173–175, 1980