Pulmonary Embolism during Spinal Anesthesia: Angiographic Diagnosis via a Flow-directed Pulmonary Artery Catheter

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Several authors have reported the occurrence of intraoperative pulmonary emboli during general anesthesia. Measurement of the pulmonary artery pressure and pulmonary capillary wedge pressure by means of a flow-directed, balloon-tipped pulmonary artery catheter has been used as an aid in the diagnosis of acute pulmonary emboli. The following case demonstrates the use of a Swan-Ganz catheter for obtaining a pulmonary angiogram to diagnose pulmonary emboli at the bedside in a patient who had severe hemodynamic compromise during spinal anesthesia.

REPORT OF A CASE

A 76-year-old woman was admitted for treatment of a fracture of the right hip. She had severe rheumatoid arthritis which had been treated for ten years with steroids. The patient also had chronic obstructive pulmonary disease with asthmatic bronchitis which required treatment with theophylline and bronchopulmonary toilet for 48 hours prior to surgical repair of the fracture.

Ninety minutes before surgery, the patient received 50 mg diphenhydramine and 10 mg prednisone orally. In the operating room, a radial artery catheter was inserted and while breathing room air, the PaO₂ was 74 mmHg, PaCO₂ 32 mmHg, and pH 7.46. Spinal anesthesia was planned for the proposed hip nailing. After infusing 500 ml of lactated Ringer’s solution, a subarachnoid injection of 10 mg tetracaine, 1 ml 10 per cent dextrose, and 0.1 mg epinephrine was administered with the patient in the left lateral decubitus position. The patient was then turned supine and the level of sensory anesthesia was determined to be at the sixth thoracic dermatome. The arterial blood pressure was 140/80 mmHg and the heart rate 95 beats/min. After the surgeon raised the affected leg to begin the surgical prep, arterial blood pressure decreased to 40/10 mmHg and heart rate increased to 120 beats/min. Cyanosis followed with marked distension of the neck veins and labored ventilation. The level of sensory anesthesia was found to be unchanged. Oxygen was delivered via face mask while the patient breathed spontaneously and phenylephrine (100 µg) was administered iv. Arterial blood pressure then increased to 100/50 mmHg, but the heart rate remained at 120 beats/min. Lead II of the electrocardiogram showed a widened QRS complex and T-wave inversion which represented a change from the preoperative tracing. Because of the venous engorgement and tachypnea, a nitroglycerin infusion was begun in an attempt to decrease venous pressures. To distinguish between myocardial infarction with left ventricular failure and pulmonary embolus, a Swan-Ganz catheter was inserted through the right internal jugular vein. Pulmonary artery pressure was 75/25 mmHg, the pulmonary capillary wedge pressure 16 mmHg, and central venous pressure 11 mmHg (table 1). Methylprednisolone, 250 mg, 5 mg furosemide, and 10,000 units heparin were administered iv and the patient was taken to the recovery room where arterial blood gases and hemodynamic variables remained stable. A twelve-lead electrocardiogram then showed sinus tachycardia and a right bundle branch block which had not been present preoperatively. Because of the clinical signs suggesting acute pulmonary embolus, a bedside pulmonary angiogram was performed. A supine portable chest roentgenogram was taken to verify the position of the pulmonary artery catheter. The catheter balloon was inflated until a pulmonary capillary wedge tracing was obtained. Sodium and meg-lumine diatrizoate, 10 ml, were injected through the distal catheter lumen and another chest roentgenogram was taken. During the injection the patient began to cough, but this stopped when the catheter balloon was deflated. The angiogram demonstrated several filling defects in a branch of the pulmonary artery to the right lower lobe (fig. 1). Only a segment of the right descending pulmonary artery was visualized using this technique. The patient was transferred to the Intensive Care Unit where heparinization was continued. The next day, a pulmonary angiogram done in the standard fashion demonstrated multiple pulmonary emboli in the right lung. With intravenous heparin and bronchodilator therapy, respiratory and hemodynamic status improved. The inspired oxygen concentration was decreased to 40 per cent and the PaO₂ was 75 mmHg, PaCO₂ 31 mmHg, and pH 7.52. With no pharmacologic support, the arterial blood pressure was 140/70 mmHg and the heart rate decreased to 106 beats/min. On the fourth day after the initial pulmonary embolus, the patient sustained a cardiopulmonary arrest presumably secondary to additional emboli. Resuscitation attempts were unsuccessful. An autopsy was not performed.

DISCUSSION

The diagnosis of pulmonary embolism is often difficult to confirm. In our case the acute onset of tachypnea, cyanosis, venous distension, hypotension, tachycardia, and right bundle branch block on ECG in an elderly patient who had been immobile for several days suggested pulmonary thromboemboli, but these could have resulted from acute left ventricular failure and pulmonary edema from an acute myocardial infarction. The hemodynamic changes after pulmonary emboli include pulmonary hypertension and increased right atrial pressure with a normal pulmonary capillary wedge pressure. In this patient, the elevated pulmonary arterial pressure may have resulted from chronic lung disease, and the increased pulmonary capillary wedge pressure was consistent with left ventricular dysfunction so that further diagnostic tests were necessary.

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Establishing the correct diagnosis in our patient was complicated by significant chronic pulmonary disease. After the acute intraoperative event, the arterial blood-gas values taken while the patient was spontaneously breathing 100 per cent oxygen indicated a large intrapulmonary shunt and an acute respiratory alkalosis. The P_{aCO_2} did not change significantly from the preoperative value, but the respiratory rate had increased, suggesting a greater pulmonary dead space. Although the patient had been heparinized in the operating room because of the clinical evidence suggestive of a pulmonary thromboembolism, the hemodynamic data obtained from the Swan-Ganz catheter and the arterial blood-gas values were not diagnostic. Further treatment required that a precise diagnosis be made. If a myocardial infarction had occurred, the risk of hip surgery would be significantly increased and conservative treatment with traction would be indicated. Prolonged anticoagulation would be required as treatment for pulmonary embolus, but corrective hip surgery could be performed so that the patient could be mobilized to prevent additional thrombus formation. Since a Swan-Ganz catheter already had been inserted, a definite diagnosis of pulmonary embolus could quickly be made by performing a wedged angiogram without a significant risk to the patient.

Bedside pulmonary angiography using a flow-directed, balloon-tipped, pulmonary artery catheter has been successful in diagnosing pulmonary emboli. Wilson and Bynum have utilized this angiographic technique in both laboratory animals and patients and claimed they were able to obtain better visualization of both the major and segmental pulmonary arteries while using a small volume of contrast material injected at a low pressure. This is possible because blood flow is stopped by the inflated catheter balloon during the injection. Repeated roentgenograms of the same vessel can be obtained since the catheter stays in place while the balloon is inflated. The procedure can be done at the bedside without moving the patient from an intensive care unit. This is important in critically ill patients who are likely to have cardiovascular instability.

Several authors have suggested criteria for diagnosing pulmonary emboli using this angiographic technique. A definite diagnosis can be made if there are intravascular filling defects or distinct vascular cutoffs with concave margins. In one report, nine patients in whom there was a suspicion of pulmonary emboli were studied. All the patients had a pulmonary artery catheter in place for other indications. A correct positive diagnosis confirmed by autopsy was made in three patients. In two patients in whom there was no evidence of pulmonary embolus by Swan-Ganz angiography, other tests confirmed these findings. There were three additional patients with negative bedside angiograms, and although this was not confirmed by other tests, their subsequent clinical course was not suggestive of pulmonary emboli. In the remaining patient, an angiogram interpreted as demonstrating a pulmonary embolus was shown at autopsy to be caused by an abscess which had distorted the

| Preoperative | 0.21 | 74 | 32 | 7.46 | 0 | 140/80 | 95 |
| Acute event | 1.0 | 78 | 30 | 7.46 | -2 | 40/10 | 120 |
| SG catheter insertion | 1.0 | 64 | 32 | 7.48 | -5 | 100/70 | 130 | 75/25 | 16 | 11 |
| Angiogram in recovery room | 1.0 | 71 | 28 | 7.43 | -5 | 110/70 | 135 | 64/32 | 18 | 15 |

BP = systemic blood pressure; PA = pulmonary artery pressure; PCW = pulmonary capillary wedge pressure; CVP = central venous pressure.

FIG. 1. An enlargement of the pulmonary artery visualized in the routine chest roentgenogram. Two intraluminal filling defects caused by pulmonary emboli are demonstrated (black arrows). The balloon of the Swan-Ganz catheter is noted by the white arrow. Excellent detail of the pulmonary artery is obtained with the technique because the contrast material is injected while blood flow is occluded.
pulmonary vasculature. This study demonstrates the accuracy of this technique.

Angiography by means of the Swan-Ganz catheter is safe when performed as described in the literature.\(^5\)–\(^9\) Five to 10 ml of intravenous contrast medium should be injected by hand through the distal catheter port after the balloon is inflated and the correct pressure waveform verified. A chest roentgenogram then is taken after the injection and the balloon is deflated. The patient will commonly cough during the injection. There have been no reports of complications related to this technique. Complications may result from the Swan-Ganz catheter insertion and long-term maintenance. Allergic reactions to the radiographic contrast agent are also possible.

In our case, the Swan-Ganz catheter was inserted after thrombi had embolized from the veins of the leg to the lung. Since the catheter is flow-directed, it will enter only a patent pulmonary artery. Thus, a false-negative diagnosis may be made if the catheter lodges in a segmental pulmonary vessel that does not contain an embolus. The tip of the Swan-Ganz catheter should be placed in as proximal a pulmonary artery as is possible to have the greatest chance of detecting an embolus. In this patient, standard pulmonary angiography demonstrated that the extent of the pulmonary emboli was much greater than visualized by bedside angiography. Pulmonary angiography using the Swan-Ganz catheter in this case substantiated the correct diagnosis so that appropriate treatment could be initiated and the patient stabilized prior to standard pulmonary angiography.

In summary, this case demonstrates the use of a Swan-Ganz catheter for diagnosing pulmonary thromboemboli which occurred during spinal anesthesia. The catheter was utilized for pressure measurements as well as pulmonary angiography. Bedside angiography using the Swan-Ganz catheter appears to be a safe, effective technique to diagnose pulmonary emboli.

REFERENCES


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Nitrous Oxide Plays a Direct Role in the Development of Tension Pneumocephalus Intraoperatively

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While previous reports have implicated nitrous oxide in the genesis of tension pneumocephalus postcraniotomy,\(^1\)–\(^4\) none provided direct evidence that nitrous oxide was involved. The following case provides direct evidence for a role of nitrous oxide in the genesis of tension pneumocephalus, and suggests that measuring CSF or intracranial pressure intraoperatively may contribute to early detection of this potentially hazardous problem.

REPORT OF A CASE

A 63-year-old woman was admitted for evaluation of hearing loss and dizzy spells. She had mild hypertension (140/85–170/95 mmHg) which was treated with aldactazide, one tablet orally each day. After a diagnosis of left acoustic neuroma was established, a craniotomy for tumor removal was scheduled to be performed in the seated position. Preoperative CT scan showed no intracranial air. Anesthesia was induced with thiopental and maintained with fentanyl and enflurane 0.25–0.5 per cent and nitrous oxide 66 per cent in oxygen. Intraoperative monitors included a right atrial catheter, and

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