Pulmonary Embolism after Cerebral Angiography
—Three Case Reports—

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Abstract

Three patients developed acute pulmonary embolism after cerebral angiography. The diagnoses were based on the clinical symptoms and echocardiography, chest roentgenography, blood gas analysis, and pulmonary perfusion scans after intravenous injection of 5 mCi of technetium-99m-labeled human albumin macroaggregates. Two of the three patients achieved clinical improvement, but one patient with severe embolization and circulatory deterioration died in spite of anticoagulation therapy. Recognition of the potential risk of pulmonary embolism after angiography and active prophylaxis are most important in preventing this complication.

Key words: pulmonary embolism, angiography, scintigraphy

Introduction

Thromboembolic complications are not unusual in patients with neurosurgical disorders, mainly occurring in the perioperative or post-traumatic period in patients with brain tumor, stroke, or spinal injury. Cerebral angiography performed by the transfemoral Seldinger technique also involves the risk of such complications, including deep vein thrombosis and pulmonary embolism. Clinical studies have revealed that up to 5% of neurosurgical patients develop detectable pulmonary embolism, with a substantial mortality ranging from 9% to 50%. The overall incidence of deep vein thrombosis is reported to be three- to five-fold greater than the incidence of pulmonary embolism. The incidence of undetected clinical pulmonary embolism is estimated at 14-45% in the general surgical population, possibly due to incomplete examinations resulting from rapid symptom progression as well as poor recognition of this complication.

We present three patients with pulmonary embolism which developed after transfemoral cerebral angiography, and discuss the cause, treatment, and prophylaxis of this life-threatening complication.

Case Reports

Angiography: Between January, 1990 and October, 1992, cerebral angiography was performed in 421 patients by the transfemoral Seldinger technique after routine prophylaxis against thromboembolism using 3-day Dextran infusion. After the procedure, the femoral artery was compressed with a 500 g weight and the patient remained prone in bed for about 16 hours. Pulmonary embolism developed in three of these 421 patients.

Case 1: A 78-year-old female with cerebral infarction underwent three-vessel cerebral angiography on October 7, 1992. The next morning, she experienced chest discomfort and mild dyspnea, and became hypotensive (systolic blood pressure 77 mmHg) about 30 minutes after removal of the femoral weight. No abnormal findings in the puncture region or the ipsilateral lower extremity were noted. Scintigraphy demonstrated a perfusion defect in the right lateral lung using intravenous injection of 5 mCi of technetium-99m-labeled human albumin macroaggregates (99mTc-MAA). Echocardiography showed
enlargement of the inferior vena cava and right atrium and ventricle, indicating pulmonary embolization (Fig. 1). The chest roentgenograph and blood gas analysis findings were normal. Her condition improved and she became normotensive about 1 hour later without anticoagulation therapy.

**Case 2**: A 62-year-old female with recurrent vertigo underwent bilateral vertebral angiography on June 10, 1992. The next morning, she noted chest discomfort and mild dyspnea, and became hypotensive (systolic blood pressure 48 mmHg) about 20 minutes after removal of the femoral weight. No abnormal findings in the puncture region or the ipsilateral lower extremity were noted. Scintigraphy revealed multiple perfusion defects in the lung after intravenous injection of 5 mCi of $^{99m}$Tc-MAA, indicating pulmonary embolization (Fig. 2). Chest roentgenograph and blood gas analysis findings were normal. Her clinical symptoms and the perfusion defects on scintigrams improved after daily intravenous administration of 120,000 U of urokinase for 1 week (Fig. 3).

**Case 3**: A 36-year-old female with subarachnoid hemorrhage underwent repeat four-vessel cerebral angiography on January 26, 1991. The first study, 1 week previously, had found no aneurysm and she...
had lain in bed during the intervening period. Eight hours after the second procedure, she complained of severe chest discomfort and dyspnea, and entered a state of shock (systolic blood pressure 38 mmHg). Blood gas analysis under endotracheal intubation and artificial ventilation (FiO₂ 1.0, respiratory rate 15/min, tidal volume 450 ml) revealed severe respiratory failure (PaO₂ 25.5 mmHg, PaCO₂ 109.0 mmHg, pH 6.680). The chest roentgenograph was not markedly abnormal (Fig. 4), but echocardiography disclosed marked enlargement of the vena cava and right atrium and ventricle (Fig. 5). No abnormal findings in the puncture region or the ipsilateral lower extremity were noted. Respiratory failure and circulatory deterioration progressed in spite of intravenous administration of 480,000 U of urokinase, and she died 6 hours after onset.

Change of angiography protocol: Since November, 1992, we have used a different postangiographic protocol to prevent venous thromboembolism. The femoral weight is now removed about 3 hours after angiography and the patient lies in bed for about 16 hours. None of 120 patients undergoing angiography under this protocol have developed clinical pulmonary embolism.

Discussion

Pulmonary embolism usually occurs during the perioperative period in patients with brain tumor, stroke, or spinal injury. Hamilton et al. reported an incidence of 3-3.8% in patients with brain tumor. Brisman and Mendell documented an 8.4% incidence at autopsy in 238 neurosurgical patients. Scmidt et al. reported that pulmonary embolism was the cause of death in 13.6% of 573 patients occurring within 3 weeks of stroke. Two of our three patients had also suffered from stroke (Cases 1 and 3), and one (Case 3) had lain in bed for 1 week after the onset of subarachnoid hemorrhage. The brain contains the highest concentration of tissue thromboplastin in the body, so intracranial surgery or trauma to the brain is thought to activate the coagulation mechanism through the release of tissue thromboplastin. The general immobility and advanced age frequent in neurosurgical patients may contribute to the occurrence of deep vein thrombosis and pulmonary embolism.

Transfemoral angiography also involves the risk of deep vein thrombosis or pulmonary embolism. Lang reported that venous system complications occurred in 14 of 1000 patients within 48 hours of percutaneous retrograde angiography. Ross reported
that 11 patients developed pulmonary embolism after cardiac catheterization, including four patients presenting with symptoms on the same day. Anno et al. compared lung perfusion scans before and after angiography in 60 consecutive patients (coronary arteriography in 24, and cerebral and visceral angiography in 18 each) to determine the incidence of pulmonary embolism after transfemoral angiography. New perfusion defects were demonstrated in 19 of 60 patients, although only one experienced clinical symptoms. These results suggest that pulmonary embolism is one of the more common complications of transfemoral angiography and that routine prophylaxis should be applied.

Thromboembolism, especially after transfemoral angiography, is believed to be caused by thrombophlebitis and/or leg vein thrombosis produced by femoral compression and extended periods of bed rest after the procedure. In our institution, reduction of the compression time after angiography from 16 hours to 3 hours has contributed to a decrease in the incidence of pulmonary embolism. Extended periods of compression of the femoral artery and bed rest should be avoided, especially in patients with risk factors for thrombosis. Lang recommended the use of heparin just prior to removal of the angiographic catheter, and reported that no clinical pulmonary embolism occurred after angiography in 2000 patients using this heparinization protocol.

Pulmonary embolism requires rapid and accurate diagnosis and treatment. The development of clinical symptoms, such as sudden onset of chest discomfort, dyspnea, cough, bloody sputum, or shock within 48 hours of angiography, suggests the occurrence of pulmonary embolism. Chest roentgenographs do not always provide diagnostic findings, because the classic "wedge-shaped" abnormal shadows are often not demonstrated on the lung field. Pulmonary perfusion scans after intravenous injection of 99mTc-MAA and pulmonary angiography are the most useful methods for the diagnosis of pulmonary embolism. Although pulmonary angiography is the optimum diagnostic examination, the procedure is invasive and therefore not easily performed in patients with angiographic complications. Pulmonary perfusion scanning is less invasive and is a reliable method for assessing the initial pulmonary vascular obstruction and quantifying any changes induced by or associated with the treatment. Perfusion scans should be performed in patients with acute pulmonary embolism to increase the accuracy of diagnosis.

Massive acute pulmonary embolism results in respiratory and circulatory catastrophe as in our Case 3. Re-establishment of circulation using dopamine or dobutamine and respiratory maintenance with artificial ventilation are necessary in such severe cases. Anticoagulant therapy is highly efficacious and prevents death from pulmonary embolism in more than 95% of patients. Anticoagulant agents, such as heparin, urokinase, and tissue plasminogen activator, should be administered immediately after confirmation of the diagnosis. However, prevention of pulmonary embolism is most important because this complication is life-threatening and resistant to treatment. Extended periods of femoral artery compression and bed rest should be avoided, and low doses of heparin may be administered after angiography in high risk patients. We observed no clinical pulmonary embolism in our 120 patients in whom the femoral weight was removed within 3 hours of angiography. Prevention, by recognition of the potential risk of pulmonary embolism after angiography and active prophylaxis, is more fundamental than rapid and accurate diagnosis and treatment.

References


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