Anesthetic Management of Pulmonary Thromboendarterectomy

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Acute pulmonary embolism is a common clinical entity occurring in over 600,000 symptomatic episodes each year in the United States.¹ In contrast, chronic pulmonary thromboembolism, resulting in pulmonary arterial hypotension and subsequent cor pulmonale, is a rare event.² Since 1958, approximately 50 cases involving attempts at surgical management of this condition have been reported, with a mortality rate approaching 25%.³⁴ None of the surgical reports have detailed the anesthetic management of this group of physiologically compromised patients.

We describe an approach to the anesthetic management of a patient with chronic embolic occlusion of the pulmonary arteries, who underwent successful pulmonary artery thromboendarterectomy.

REPORT OF A CASE

A 33-year-old, 85-kg man was scheduled for pulmonary artery thromboendarterectomy of branches of his right pulmonary artery utilizing extracorporeal circulation, followed by inferior vena caval interruption as part of the procedure. His medical history was unremarkable, with the exception of his pulmonary thromboembolic disease. This disorder began 2 years prior to admission, when he developed deep venous thrombosis. A ventilation/perfusion scan was consistent with acute pulmonary embolization, and the patient underwent anticoagulant therapy for 6 months following this acute illness. After incomplete recovery, he experienced another acute episode of pulmonary emboli, again treated with anticoagulants, 4 months prior to his pulmonary artery thromboendarterectomy. He also underwent a trial of streptokinase thrombolytic therapy preoperatively, which did not improve the angiographic paucity of his pulmonary arteries.

He denied cardiopulmonary symptoms on preoperative evaluation, though his chest radiograph evidenced cardiomegaly and a prominent central pulmonary vasculature. His electrocardiogram revealed right axis deviation, right atrial hypertrophy, and a right ventricular strain pattern, while echocardiography described massive right ventricular and atrial dilatation accompanied by right ventricular hypokinesis and paradoxical septal wall motion. The pulmonary function studies showed an FEV₁ₐₐ of 3.0 L (72% predicted) and a FVC of 3.62 L (67% predicted). With an FIO₂ of 0.21, PaO₂ was 51 mmHg, PaCO₂ 51 mmHg, and a pH of 7.50. Ventilation/perfusion scanning revealed multiple segmental lobar defects with the right lower lobe severely hyperperfused. Right heart catheterization values included a pulmonary arterial pressure (PAP) of 60/40 mmHg, a right ventricular pressure of 60/10 mmHg, and a central venous pressure of 10 to 12 mmHg. Pulmonary arterial angiography confirmed complete occlusion of the right lower and middle lobe arteries, with numerous other areas of acute and chronic changes compatible with embolic pulmonary disease.

Morphine sulfate 10 mg im diazepam 10 mg, and sodium citrate 30 ml orally were given 1 h before arrival in the operating room. Prior to the induction of anesthesia, monitoring was established with an ECG and percutaneous insertion of radial and pulmonary artery catheters. Special care was taken to achieve placement of the pulmonary arterial catheter tip in the most proximal portion of the main pulmonary artery, so as to interfere with the surgical dissection.

Anesthesia was induced with fentanyl 500 µg iv diazepam 10 mg iv and isoflurane in oxygen. Pancuronium bromide, 0.1 mg/kg, was used to facilitate tracheal intubation, which was accomplished using double-lumen endobronchial tube. Maintenance of anesthesia included inhalation of isoflurane in oxygen, pancuronium bromide, iv, and infusions of nitroglycerin and dobutamine to support cardiovascular function. The surgical procedure was performed via a right posterolateral thoracotomy with partial extracorporeal circulation using a right femoral arterial cannula and a right atrial venous cannula. The vena caval interruption was performed following completion of the thoracic procedure via a right flank incision.

The intraoperative anesthetic course proceeded uneventfully, with the exception of marginal urine output during cardiopulmonary bypass, which was augmented by the use of mannitol, 25 g iv, and an iv infusion of dopamine at 3 µg·kg⁻¹·min⁻¹. The operative course is outlined in figure 1. The patient underwent a 15-day intensive care unit stay, necessitated by the requirement for ventilatory support. This need for ventilatory support was presumed secondary to right phrenic nerve dysfunction, caused by operative dissection. A postoperative ventilation/perfusion scan confirmed reperfusion of the right lower lobe. The patient was discharged to home on postoperative day 24. Repeat right heart catheterization is planned when the patient returns for a 6-month follow-up examination.

DISCUSSION

Most investigators now feel that chronic embolic occlusion of the pulmonary arteries is a result of recurrent embolic events, rather than the incomplete lysis of a massive single embolic episode.⁵ The potential physiologic consequences of this condition include pulmonary hypertension, cor pulmonale, chronic hypoxemia, tricuspid insufficiency, right heart failure, and death.⁶ Medical management has consisted of the treatment of pulmonary hypertension⁷ and anticoagulant therapy.⁸ Efficacy of anticoagulant therapy has been assisted by the technique of inferior vena cava interruption.⁹ Recently, medical management of embolic disease has focused upon thrombolytic therapy, with most reports dealing with acute ep-
isodose of pulmonary emboli. The role of fibrinolytic therapy in this condition remains unclear.10

The surgical approach to chronic embolic occlusion of the pulmonary arteries first was suggested by Hollister and Cull in 1956.11 Since then, 52 patients, including our patient, have been reported with 39 of the 52 patients surviving the perioperative period. Relief from this operative technique has ranged from nearly complete to no change in symptoms.5,4 The surgical indication for this operative approach is angiographically documented occlusion of more than 50% of the segmental arteries in a patient who is clinically symptomatic with cardiopulmonary complaints.4 The pulmonary arteries are approached surgically, using either a unilateral thoracotomy or sternotomy incision, with varying degrees of circulatory assistance from extracorporeal circulation.6 Deep hypothermia, utilizing extracorporeal circulation, and induced circulatory arrest also have been used successfully to facilitate surgical management.4

A number of clinical correlates can be drawn from our patient and from the anesthetic management of patients with pulmonary hypertension.7 These patients with chronic cor pulmonale have, by definition, pulmonary hypertension with an accompanying increased pulmonary vascular resistance (PVR). Therefore, any pharmacologic or ventilatory management that increases PVR should be avoided. Those factors that may increase PVR include use of nitrous oxide,12 hypoxemia, hypercarbia, acidosis, and functional residual capacity alterations.13 Ventilation and perfusion matching also is disturbed in patients with chronic embolic occlusion of the pulmonary arteries. This is evidenced by chronic hypoxemia, which our patient clearly demonstrated.8 Since all potent inhaled anesthetics have been reported to inhibit hypoxic pulmonary vasoconstriction,14 especially isoflurane, their use could worsen V/Q matching.15 Other investigators, though, have shown that in dogs, and in humans, isoflurane does not appear to inhibit hypoxic pulmonary vasoconstrictive reflex.16,17 Despite these conflicting data, we chose to utilize isoflurane in oxygen as our primary anesthetic, since its cardiovascular effects were thought to be advantageous.18

Myocardial function also is compromised in patients with chronic cor pulmonale. Ventricular interdependence is likely important in patients with right heart failure secondary to pulmonary hypertension.19-21 This group of patients compromised myocardial function is secondary to the failure and dilation of the right ventricle, which may result in an interventricular septal shift that encroaches upon the left ventricular cavity and results in left ventricular dysfunction.21 We chose to place a heparin-coated pulmonary artery catheter in the proximal main pulmonary artery. This monitoring technique was chosen in order to obtain cardiac output and pulmonary artery pressure measurements in the perioperative period. Other authors, though, states that they avoid the placement of a pulmonary artery catheter to minimize the chance of postoperative recurrent thrombosis of the pulmonary arteries.4 No reports are available to support this latter approach to monitoring. In fact, pulmonary artery catheterization and monitoring has not been mentioned in any of the reports of patients undergoing pulmonary thromboendarterectomy. We feel the availability of cardiac output measurements was clinically important in the management of the vasodilator and inotropic agent infusions, both intraoperatively and postoperatively, in our patient.

The use of a vasodilator would appear indicated in an attempt to decrease PVR and impendence to right ventricular ejection. We used nitroglycerin (NTG) as a vasodilator. A 50% greater decrease in PVR with NTG compared with sodium nitroprusside (SNP) has been described.22,23 Another advantage of NTG is an improvement in cardiac output up to 60% greater than with SNP, which had little effect on this variable in patients with pulmonary hypertension.22,23

The use of dobutamine as an inotropic agent was supported by its ability to increase the cardiac output from 2.3 to 4.9 l·min⁻¹ using a dose of dobutamine of 3 to 6 μg·kg⁻¹·min⁻¹ and an NTG infusion of 2 to 4 μg·min⁻¹. Waller reported that dobutamine was partic-
ularly helpful in managing patients with right ventricular failure, both with and without accompanying pulmonary hypertension. 24

Our patient required postoperative ventilatory support for 15 days, while a temporary right phrenic nerve injury resolved. The duration of ventilatory support required in the right 51 patients reviewed, ranged from 12 h to 2 months. 4 Phrenic nerve injury also has been reported postoperatively by others. 6 Another problem that may be evidenced in the postoperative period is recurrent laryngeal nerve dysfunction, associated with compression of this nerve by a distended pulmonary artery. 26

A more common abnormality that may necessitate postoperative ventilatory support is reperfusion pulmonary edema, probably due to a capillary endothelial defect. 27 It is present in up to 100% of cases in which extracorporeal circulation is utilized and in as many as 36% of cases performed without extracorporeal circulation. 28 Persistent pulmonary hypertension may occur postoperatively, though the mean decrease in pulmonary artery pressures in the 22 patients available for review reveal a fall from 80/21 mmHg preoperatively to 47/17 mmHg postoperatively. These values represent a 41% decrease in pulmonary systolic pressure and a 19% fall in diastolic pulmonary artery pressure. During the 3 days the pulmonary artery catheter remained in our patient, no decrease in pulmonary artery pressures were recorded.

Cardiac failure following this procedure, most often right heart failure, carries a mortality as high as 60%, if it occurs in the postoperative period. Other complications that may contribute to a prolonged postoperative course include pulmonary infarction, hemothorax, and possibly an increased incidence of pulmonary infection. The incidence of postoperative pulmonary infection appears to range from 12% to 25% in recent series, which is consistent with other reports of postoperative pulmonary complications following thoracic surgery. Review of the 15 cases in which preoperative and postoperative PaO2 are available reveal a mean increase of 25%, from 57 to 71 mmHg.

In conclusion, we have demonstrated that a patient, anesethetized with isoflurane in oxygen coupled with pulmonary and radial arterial catheter monitoring to guide vasoactive drug infusions, can be managed successfully while undergoing pulmonary thromboendarterectomy.

REFERENCES
Hypercoagulability in a Patient with a Brain Tumor

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The association of coagulopathy with neoplastic disorders or cranioencephalogenous trauma is well recognized.1–4 However, few cases of coagulopathy or hypercoagulopathy have been reported in patients with primary brain tumors.5,6 Hypercoagulability can occur with chronic activation of the coagulation system during episodes of chronic disseminated intravascular coagulation (DIC). We present a patient with a primary brain tumor, hypercoagulability, and chronic DIC.

REPORT OF A CASE

A 47-year-old woman with a frontal meningioma was scheduled for craniotomy and tumor resection. She was taking no medication and had no previous surgery. There was no history of bleeding disorders, phlebitis, or use of oral contraceptives. On physical examination, she was an alert and oriented obese woman. Vital signs were within normal limits. Cardiac and chest examination were normal. A neurologic assessment revealed optic atrophy in the right eye and a left eye, which visualized only finger counting. Although she had mild paresis of both lower extremities, no sensory deficits could be elicited. A chest roentgenogram and electrocardiogram both were normal. Computerized tomography of the head revealed a circumferential well-marginated mass over the right sphenoid ridge; angiogram confirmed abnormal vasculature in the same region. There was no clinical evidence of increased intracranial pressure.

Significant laboratory values were as follows: hemoglobin 11.1 g/dl, hematocrit 31.8%, platelet count 240,000/mm³, and white blood cell count 11,700/mm³ with a normal differential. The admission prothrombin time (PT) was 12.8/11.3 s. Measurements of the activated partial thromboplastin time (aPTT) were made with the MLA Electra 750® automated coagulation analyzer and were 79.5/24, 93.8/24.8, and 100/24 (patient/control) s. Plasma fibrinogen levels were 115, 120, and 92 mg% (normal 200–400 mg%). Further hematologic investigation revealed an aPTT of 15/24 and 16/26 (patient/control) s when measured by the Mechrolab Clot-Timer®, fibrin split products 1:64, plasma fibrinogen level 87 mg%, platelets 198,000/mm³, and Factor VIII level greater than 500% (normal 50–150%). Antithrombin III, a normal physiologic inhibitor of the coagulation cascade was measured as 116% (normal 80–120%).

She was premedicated with droperidol 2.5 mg and glycopyrrolate 0.2 mg iv. A difficult endotracheal intubation was anticipated because of obesity. An awake orotracheal intubation was accomplished using topical and transtracheal applications of lidocaine as well as dexamethasone 2.5 mg and fentanyl 0.2 mg iv. Subsequently, thiopental 500 mg in divided doses and pancuronium 10 mg were administered iv. Following intubation of the trachea, blood pressure remained stable at approximately 140/80 mmHg throughout the 12-h operation. Anesthesia was maintained by infusions of nitrous oxide and isoflurane in addition to fentanyl and pancuronium iv. Fibrinogen levels and platelet counts were obtained every 2 h throughout the case and ranged from 129 to 170 mg% and 155,000–186,000/mm³, respectively. Thirteen units of cryoprecipitate were given immediately prior to surgery, and an additional eight units were given intraoperatively because of the decrease in fibrinogen levels. Total fluids administered were crystalloids 5,550 ml, whole blood 500 ml, packed erythrocytes seven units, and fresh frozen plasma six units. Estimated blood loss was 1,000 ml. A complete resection of the tumor could not be accomplished because of marked perivascular extension and infiltration.

Continuous mild bleeding from the oropharynx was noted 5 h postoperatively, and laryngoscopy failed to reveal the site of bleeding. The platelet count was 60,000/mm³, and five units of platelets and two units of fresh frozen plasma were administered. Platelet count and fibrinogen level increased so that by the third postoperative day platelet count was 140,000/mm³ and fibrinogen 340 mg%. On the 6th postoperative day the platelet count again decreased to 92,000/mm³. Fibrinogen decreased to 120 mg% and fibrin degradation products increased. There were no abnormalities in the PT or aPTT. Administration of additional cryoprecipitate, whole fresh blood, fresh-