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Immediate Hemodynamic and Pulmonary Changes Following Pulmonary Thromboembolism

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The postembolic sequelae of pulmonary thromboembolism have been extensively reported.[1–4] However, the immediate hemodynamic response to pulmonary thromboembolism in man has not been described. Nor has rapid resolution within hours of the pulmonary vascular response been reported.

The following case illustrates the hemodynamic and pulmonary changes in a patient who was extensively monitored prior to and during an acute episode of pulmonary thromboembolism.

REPORT OF A CASE

A 66-year-old man was admitted to the hospital for elective repair of an abdominal aortic aneurysm. He had a two-year history of progressive claudication with no sign or symptom of neurologic, cardiac, or renal disease. The patient had a seventy-pack-year history of cigarette smoking with symptoms of chronic bronchitis. He was receiving no medication. Physical examination disclosed no abnormality except a 6-cm pulsatile abdominal mass. Routine laboratory tests, arterial blood-gas values, electrocardiogram, and roentgenogram of the chest were normal. Pulmonary function studies showed mild obstructive disease with a normal diffusing capacity.

Before induction of anesthesia, peripheral intravenous and radial-artery catheters were placed. A pulmonary-artery catheter was inserted via the right internal jugular vein. Blood pressure was 140/80 torr, pulse 90/min, central venous pressure (CVP), 6 torr, pulmonary arterial pressure (PAP) 20/8 torr, and pulmonary-artery wedge pressure (PAWP) 5 torr (table 1). Anesthesia was induced with morphine, 10 mg, iv, and thiopental, 100 mg, iv. Following pretreatment with 3 mg *d*-tubocurarine and topical lidocaine laryngotracheal spray, 80 mg, endotracheal intubation was accomplished without difficulty, facilitated by succinylcholine, 100 mg, iv. Anesthesia was maintained with 50 per cent oxygen, 50 per cent nitrous oxide, morphine sulfate, 10 mg, iv, and pancuronium, 4 mg, iv. Ventilation was controlled (table 1).

Anesthesia and operation proceeded uneventfully. An hour after abdominal incision, and before clamping of the aorta, both lumbar sympathetic chains were divided. No hemodynamic change was seen. Approximately 20 min later, without surgical or anesthetic incident, the blood pressure decreased from 120/70 to 50/40 torr over 10 sec (table 1). The pulse rate increased from 84 to 110/min; the electrocardiogram showed sinus tachycardia without other abnormalities. Concurrently, CVP increased from 7 to 16 torr, and the PAP from 18/8 to 44/38 torr. PAWP was essentially unchanged (increasing from 5 to 6 torr). Inspiratory airway pressure was unchanged (20 cm H_2O), and auscultation of the chest revealed no pulmonary or cardiac change.

Ephedrine (25 mg) was administered twice iv, but systemic blood pressure remained in the range of 50/40 to 60/45 torr. Despite an increase in inspired oxygen concentration to 100 per cent and an increase in minute ventilation (from 7 to 10 l/min) for approximately 10 min, arterial blood-gas analysis showed that Pa_{O_2} was 379 torr, Pa_{CO_2} 77 torr, *p*H 7.09, and base deficit 5 mEq/l (table 1). These findings were considered to indicate possible pulmonary embolus with acute right heart failure. Over the next 30 min, the patient was given dopamine (2–4 μg/kg/min), sodium bicarbonate (44 mEq), and calcium chloride (300 mg); anticoagulation was achieved with 10,000 units heparin, iv. Following this, arterial blood pressure, pulse rate, filling pressures, gas exchange, and inspiratory airway pressure improved. Over the subsequent three-hour intraoperative period, the hemodynamic and pulmonary status returned to the pre-incident values. The patient tolerated aortic clamping and unclamping, blood transfusion, and endotracheal extubation without incident. The operation was completed without additional complications.

Postoperatively, serial electrocardiograms and myocardial enzymes gave no evidence of myocardial injury. The only remarkable finding was the development of a left-lower-lobe wedge-like infiltrate on chest roentgenogram 48 hours postoperatively. (The pulmonary-artery catheter was located in the right pulmonary artery). This infiltrate resolved over the following six days.

DISCUSSION

The initial event was the development of acute pulmonary hypertension in the face of a normal pulmonary-artery wedge pressure. The differential diagnosis for this occurrence during anesthesia includes: 1) hypoxemia or acidemia (*e.g.*, hypoventilation, endobronchial intubation); 2) pneumothorax; 3) non-cardiogenic pulmonary edema; 4) aspiration pneumonitis; 5) administration of vasoconstrictive medications; 6) air embolus; 7) pulmonary thromboembolism. There is no evidence for an etiology other than pulmonary thromboembolism in this man. The diagnosis of pulmonary thromboembolism was confirmed by the subsequent development of characteristic hemodynamic and respiratory sequelae.

Hemodynamic changes are explicable on the following basis. With pulmonary thromboembolism, the cross-sectional area of this patient's pulmonary arterial bed was abruptly reduced, and pulmonary hypertension ensued. This large increase in right ventricular afterload produced isolated right ven-

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TABLE 1. Hemodynamic and Arterial Blood-gas Values before and after Pulmonary Thromboembolism

	Hemodynamics					Arterial Blood-gas Values			
	Blood Pressure (torr)	Pulse (/min)	Central Venous Pressure (torr)	Pulmonary Arterial Pressure (torr)	Pulmonary-artery Wedge Pressure (torr)	Pa_{O_2} (torr) (Inspired Oxygen)	Pa_{CO_2} (torr) (Minute Ventilation)	pH	Base Deficit (mEq/l)
Pre-induction	140/80	90	6	20/8	5	82 (21 per cent)	34 (—)	7.38	2.5
Pre-incident	120/70	84	7	18/8	5	254 (50 per cent)	43 (7.1)	7.37	1.0
Post-incident									
Immediately	50/40	110	16	44/38	6	379 (100 per cent)	77 (10.1)	7.09	5.0
30 min	120/75	95	11	30/20	5	370 (100 per cent)	59 (14.1)	7.26	3.1
One hour	126/80	90	7	18/10	5	427 (100 per cent)	47 (10.1)	7.33	2.4
Three hours	145/95	95	8	20/11	6	523 (100 per cent)	39 (7.1)	7.41	0.1

tricular failure, manifested by an elevated filling pressure (CVP) in the face of an unchanged PAWP and systemic hypotension. However, with a decreased right ventricular output, the left ventricular filling pressure (reflected by PAWP) did not decrease significantly. One explanation, based on recent studies,[5] is that with an intact pericardium, isolated right ventricular distention can cause a shift of the interventricular septum, encroachment on the left ventricular cavity, and elevation of left ventricular filling pressure. Thus, even though PAWP did not change, left ventricular filling volume might have decreased with acute right ventricular failure.

To produce these hemodynamic changes, this embolus must have reduced the cross-sectional area of the pulmonary arterial bed by 40 per cent or more.[6] This large reduction could represent not only obstruction, but also vasoconstriction of the pulmonary vasculature. Resolution within hours of the hemodynamic changes has been found previously only in animal studies,[7,8] and has been ascribed to a transient vasoconstriction by a neurohumoral agent.[9,10] The rapid resolution of the hemodynamic findings in this case suggests that a similar neurohumoral mechanism may exist in man. It should be noted, however, that use of dopamine and calcium may have improved right ventricular ejection and promoted a more rapid resolution.

The respiratory sequelae were equally characteristic. We hypothesize that, as a result of embolic obstruction, a non-perfused but ventilated zone developed, leading to an increase in arterial carbon dioxide tension despite an increase in minute ventilation. Since there is no reason to suspect a concomitant acute increase in carbon dioxide production, these changes are consistent with a sudden, significant increase in intrapulmonary dead space. This has been reported to occur with massive emboli.[11] The return of carbon dioxide tension to control accompanied the return of normal hemodynamics over the subsequent three-hour period.

The change in Pa_{O_2} from 254 torr (50 per cent inspired oxygen) to 379 torr (100 per cent inspired oxygen) represents an increased alveolar-to-arterial oxygen tension difference, and is a frequent result of massive emboli.[6] Despite extensive investigation, the mechanism responsible for this remains uncertain. Suggested etiologies include: 1) reduction in cardiac output (secondary to right ventricular failure) producing increased tissue oxygen extraction, decreased mixed venous blood oxygen concentration, and exacerbation of the effect of pre-existing shunt, 2) overperfusion of the non-embolized lung zones that cannot be sufficiently ventilated to maintain adequate oxygenation of the blood they receive, and 3) with embolic resolution, reperfusion of poorly ventilated (pneumoconstricted) or nonventilated (congestive atelectatic) lung zones.

Development of the wedge-like infiltrate 48 hours post-incident is characteristic of embolism even without infarction, and is attributed to a loss of surface-active material with secondary atelectasis.[12] Resolution of this infiltrate over a six-day period is consistent with prior studies in man.[13]

In conclusion, this case provided a unique opportunity to follow the development and resolution of the hemodynamic and respiratory changes associated with acute pulmonary embolism in man. Both the immediate hemodynamic changes (pulmonary hypertension, right ventricular failure, systemic hypotension) and the immediate respiratory changes (increased alveolar dead space, hypoxemia, atelectasis) are consistent with those previously reported in animal and human studies during the late postembolic period. However, the rapid resolution of these changes, previously reported to occur only in animals, suggests that a similar course is possible in man.

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Intraoperative Diagnosis of a Gastrobronchial Fistula

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Gastrobronchial fistula is a rare condition. Frequently it is a complication of subphrenic abscess.[1,2] Other causes include direct trauma to the chest and abdomen, infiltrating neoplasm, and perforation of the stomach while incarcerated in a diaphragmatic hernia (DH).[3] Despite its rarity, gastrobronchial fistula is suggested by a history of recurrent pulmonary infections resistant to therapy or the coughing up of gastric contents. The diagnosis can usually be established by roentgenographic contrast studies or endoscopy. The following report describes the intraoperative diagnosis and management of a patient with an unsuspected gastrobronchial fistula.

REPORT OF A CASE

A 53-kg white man was first seen at the Mayo Clinic at the age of 33 years because of persistent gastroesophageal reflux. The patient was a self-described alcoholic who had smoked two packs of cigarettes per day for the preceding 15 years. At 4 years of age, because of bleeding esophageal ulcer, the patient had undergone a distal esophageal and proximal gastric resection with esophagogastrostomy reconstruction. At the age of 26 years he had sustained a gunshot wound to the abdomen and undergone small-bowel resection. By the age of 31 years he had noticed regurgitation of gastric contents when he assumed the supine position. He had become progressively dyspneic and orthopneic. Studies of the upper gastrointestinal tract and barium-enema examination had revealed a large amount of stomach and small and large intestine herniated into the left hemithorax. The patient had undergone repair of the diaphragmatic hernia as well as gastric fundal plication via left thoracotomy.

The patient was admitted to the Mayo Clinic complaining of recurrent gastroesophageal reflux followed by the progressive onset of dyspnea, productive cough, and fever. Physical examination revealed slight respiratory distress. Roentgenogram of the chest demonstrated a diaphragmatic hernia and an interstitial infiltrate in the left lung base with an elevated left hemidiaphragm (fig. 1). *Escherichia coli* were cultured from the sputum. Arterial-blood gas values during breathing of room air were Pa_{O_2} 53 torr, Pa_{CO_2} 37 torr, and *p*H 7.41. Pulmonary function studies revealed total lung capacity, forced vital capacity (FVC), first-second forced expired volume (FEV_1) and residual volume to be 72, 50, 70, and 45 per cent of predicted values, respectively. The FEV_1/FVC ratio was 0.74. Flow rates in the mid-lung volume range were 20 per cent of predicted, with no improvement following administration of an aerosolized bronchodilator. These data suggested a combined restrictive and obstructive lesion. Routine laboratory values were all within normal limits. An upper gastrointestinal tract study demonstrated gastroesophageal reflux and a large diaphragmatic hernia with most of the stomach in the left hemithorax. Esophagogastroscopy was performed, with no evidence of esophagitis.

The diagnosis of gastroesophageal reflux with chronic aspiration pneumonia was made. Intravenous hyperalimentation was initiated and oral feedings were discontinued. Two weeks after admission, the patient was brought to the operating room for diaphragmatic hernia repair and an anti-reflux procedure. Following placement of a precordial stethoscope, blood pressure cuff, six-lead electrocardiogram, and 14- and 18-gauge intravenous catheters, the right radial artery was cannulated with a 20-gauge Teflon® catheter. Following 5 min of preoxygenation, a 50-mg test dose of thiopental and pancuronium, 0.5 mg, were administered, followed in 3 min by 250 mg thiopental and 100 mg succinylcholine. Cricoid pressure was continuously applied. The trachea was easily intubated with a 9-mm endotracheal tube, without evidence of regurgitation or aspiration. The endotracheal tube cuff was balotted at the suprasternal notch. The patient was ventilated with a mechanical ventilator‡ with a minute volume ($\dot{V}_E$) of 7 l (4 l nitrous oxide and

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