Pneumothorax and pulmonary embolism complicating post-traumatic hip surgery

Donald C. Oxorn MD FRCPC, David Skurdal MD PH D, Howard Belzberg MD, Richard C. Rosenbaum MD

A case of intraoperative pneumothorax preceded by a pulmonary embolus is presented. A high index of suspicion led to further investigation, after resolution of the pneumothorax was not accompanied by clinical improvement.

The literature is reviewed, and the various methods of prophylaxis and treatment discussed.

Pneumothorax and pulmonary embolism, while relatively common occurrences in a general medical population, are infrequently seen during the course of general anaesthesia.[1] Clinical signs and symptoms are few, and their occurrence is often heralded by sudden cardiorespiratory instability.

We present a case of intraoperative pneumothorax superimposed on a recent, undiagnosed pulmonary embolus. The presence of the latter was suspected, when tube thoracostomy did not produce the expected improvement in gas exchange.

The sequence of events is discussed, the literature reviewed, and comments are made on appropriate forms of prophylaxis and treatment.

Case report

The patient was a 22-year-old female. She was admitted to the Maryland Institute for Emergency Medical Services Systems, a level one trauma centre, following a motor vehicle accident. Although she was alert and oriented at the scene, she had transient amnesia for events immediately surrounding the accident.

Key words

COMPLICATIONS: pneumothorax, pulmonary embolus.

From the Departments of Critical Care Medicine, Anesthesiology, and Nuclear Medicine, Maryland Institute for Emergency Medical Services Systems; Baltimore, Maryland.

Address correspondence to: Dr. Donald Oxorn, Department of Anaesthesia, Halifax Infirmary, 1335 Queen St., Halifax, Nova Scotia B3J 2H6.

Her initial evaluation revealed a severely displaced pelvic fracture, with disruption of the right acetabulum, and abdominal tenderness, with an elevated amylase, consistent with traumatic pancreatitis. There was no clinical or laboratory evidence of cardiopulmonary injury. Breath and heart sounds were normal, and the chest *x*-ray showed no contusion, effusion, fracture, or pneumothorax. Blood gases (FiO_2 0.21) were PaO_2 = 99 mmHg, pH = 7.41, $PaCO_2$ = 30 mmHg, base excess = −4.0. Haemoglobin was 127 $g \cdot L^{-1}$ and haematocrit 36.6 per cent. Serum electrolytes, BUN, creatinine and coagulation studies were within normal limits.

The past medical history was unremarkable. The patient had no allergies and her only medication was a birth control pill (ethinyl estradiol 35 μg and norethidrone 1 mg). She was a non-smoker and gave no personal or family history of phlebitis, asthma, bronchitis, pulmonary embolism or pneumothorax. Height and weight were 162 cm and 64 kg.

The patient was transferred to the subacute ward for observation. Her course over the next six days was unremarkable. There were no complaints of shortness of breath, chest pain, haemoptysis, calf pain, or leg oedema. An intermittent pneumatic leg compressor was placed on the left calf after a doppler study revealed no evidence of deep venous thrombosis.

By day seven, adequate bowel function had not returned. A right subclavian line was inserted with difficulty, and TPN started. A post-insertion chest *x*-ray revealed no pneumothorax or effusion.

On day eight, the patient was taken to the operating room for open reduction and internal fixation of the right hip.

Anaesthesia was induced with thiopentone, and tracheal intubation was facilitated by the administration of succinylcholine. Maintenance anaesthesia was provided with isoflurane, nitrous oxide and oxygen administered by mechanical ventilation through a circle absorber. Morphine and pancuronium were given intermittently for analgesia and muscular relaxation. Monitors included ECG, intra-arterial blood pressure, central venous pres-

sure, oesophageal temperature, an oxygen analyzer and a pulse oximeter.

Surgery was performed with the patient in the left lateral position. Ventilatory parameters were: tidal volume 800 ml, respiratory rate 10 breaths·min^{-1}, FiO_2 0.35, PEEP 5 cm H_2O, peak airway pressure 18 cm H_2O. Breath sounds were equal bilaterally. After initial blood gases were obtained (Table, set 1), the FiO_2 was increased to 0.5, the PEEP to 7 cm H_2O, the tidal volume to 900 ml and the respiratory rate to 12. The next arterial gases (set 2) revealed further deterioration in oxygenation, and after a brief improvement on an FiO_2 of 1.0 (set 3), the PaO_2 fell even further (set 4). Breath sounds remained equal and clear bilaterally, and the airway pressure increased from 18 to 22 cm H_2O. No equipment malfunction was detected. Asucultation revealed bilateral end-expiratory wheezes, although peak airway pressure remained at 22 cm H_2O. The inspired isoflurane concentration was increased from 0.5 to 1.5 per cent, orciprenaline 2.25 mg was administered via the endotracheal tube, 44 m·Eq of sodium bicarbonate was given, and an infusion of isoproterenol was started at a rate of 1.0 μg·min^{-1}. The endotracheal tube was suctioned for a moderate amount of clear sputum, and the tidal volume was increased to 1000 ml. Despite these measures, oxygenation remained poor, and CO_2 elimination continued to worsen (set 5). The nail beds were cyanosed.

Haemodynamics at this time did not reflect the patient's precarious position. Blood and fluid replacement were commensurate with measured losses. Blood pressure and pulse had remained at approximately 120/70 mmHg and 100 beats/min since the beginning of the case. Central venous pressure, which had shown little fluctuation from the initial reading of 8 mmHg, rose to 15 mmHg.

Surgery was rapidly completed, and the patient was turned supine. A pulmonary artery catheter was inserted via the left internal jugular vein. Initial pulmonary artery pressure was 53/25 mmHg. Although passage of the catheter into the pulmonary artery was accomplished with ease, the pulmonary artery pressures and waveform persisted, and no tracing consistent with a wedge position could be obtained, despite the repeated passage of 20–25 cm of additional catheter, with a balloon volume of 1.5 ml. Eventually, a satisfactory tracing, with a wedge pressure of 17 mmHg was recorded.

A chest *x*-ray taken in the OR revealed a right pneumothorax, and the PA catheter was in the distal right pulmonary artery. A right-sided chest tube was inserted with resolution of the pneumothorax, although the right lung remained oligemic. Gas exchange improved slightly (set 6), although this benefit was short lived (set 7).

Because of the high index of suspicion, arrangements were made for a perfusion lung scan in the recovery room. This revealed no blood flow to the right lung. A digital subtraction angiogram was performed with injection of contrast through the distal port of the PA catheter, which had been pulled back into the right main pulmonary artery. This demonstrated a large embolus in the right pulmonary artery, occluding 75 per cent of the lumen (Figure).

Following these studies, blood gases steadily improved (sets 8 and 9). Because of the extensive surgery just completed, heparin and streptokinase were contraindicated. A Greenfield filter was positioned in the inferior vena cava via the right femoral vein. The next day, anticoagulation was achieved with heparin. Followup chest films were unremarkable, except for lucency of the right lung. The patient was weaned from mechanical

TABLE Intraoperative arterial blood gases

	*Set number**								
	1	*2*	*3*	*4*	*5*	*6*	*7*	*8*	*9*
FiO_2	0.33	0.50	1.00	1.00	1.00	1.00	1.00	1.00	1.00
PEEP (cm H_2O)	5	7	7	7	10	10	10	8	8
PaO_2 (mmHg)	72	69	349	58	74	129	95	236	468
pH	7.30	7.42	7.41	7.24	7.13	7.29	7.28	7.28	7.49
$PaCO_2$ (mmHg)	53	33	32	55	85	58	61	57	32
O_2 Sat (Measured)	92.00	94.00	99.00	84.00	87.00	98.00	96.00	99.00	99.00
O_2 Sat (Oximeter)	93.00	88.00	100.00	88.00	90.00	100.00	100.00	100.00	—
B.E.	−2.0	−2.0	−3.0	−5.0	−5.0	−0.5	−0.5	−1.5	2.0
HCO_3 (mEq·L^{-1})	25.0	20.5	20.0	23.0	27.0	27.0	28.0	26.0	23.0
CVP (mmHg)	11	8	9	11	15	19	19	18	14
Arterial pressure (mmHg)	122/78	140/80	140/80	122/70	125/65	100/55	100/50	120/60	120/70
Airway pressure (cm H_2O)	18	18	18	22	22	25	25	25	25
V_E (L·min^{-1})	8	10.8	10.8	12	12	15	15	15	15

*See text for timing of samples.

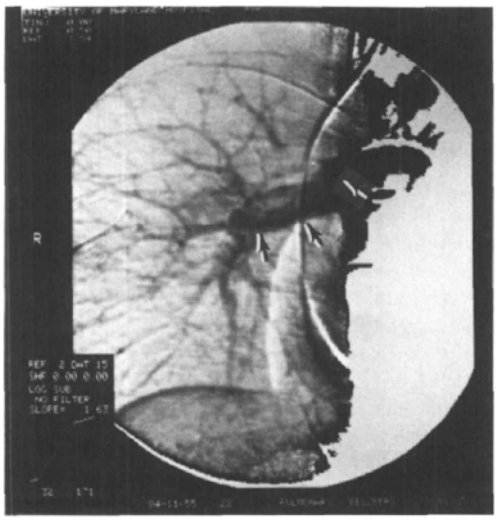

FIGURE Digital subtraction angiogram (DSA) performed in recovery room.

ventilation and extubated seven days postoperatively. Coumadin was begun and she was discharged 26 days after admission.

Discussion

The two causes of profound intraoperative hypoxaemia which were present in this case may not be detected easily under anaesthesia. Pneumothorax may present with diminished breath sounds and impeded venous return, but is often clinically silent. Pulmonary embolism is frequently a diagnosis of exclusion, and may remain unconfirmed until post mortem.

There have been approximately 26 reported cases of coexistent pulmonary embolism and pneumothorax.[2] The most commonly reported sequence of events is as follows: pulmonary embolism followed by pulmonary infarction with or without pulmonary sepsis; pneumothorax followed days to weeks later, presumably from breakdown of necrotic lung tissue.[3–5]

Our patient had risk factors for both conditions. A subclavian line had been inserted with difficulty the previous day. Although no pneumothorax was evident on the post-insertion chest film, it is likely that the lung parenchyma had been violated. With the use of positive pressure ventilation and nitrous oxide, a significant pneumothorax developed. Patients at high risk for pneumothorax should be managed without use of nitrous oxide, and with techniques that minimize peak airway pressure.

Multiple predispositions existed for venous thrombosis and pulmonary embolism: the recent use of the birth control pill,[6] immobilization and venous stasis,[7] and hip surgery.[8] In this clinical setting, effective prophylaxis has been reported with low dose intravenous heparin,[9] dextran,[7] anti-embolic stockings,[10] acetylsalycilic acid,[7] and intermittent pneumatic limb compression.[11] The effect of this latter method is to augment limb blood flow, and increase systemic fibrinolysis. The effects of compression on one limb are not known. The method of anaesthesia may also affect the incidence of deep venous thrombosis after hip surgery; it appears to be lower with major regional as opposed to general anaesthesia.[12,13]

The diagnosis of venous thrombosis may be made invasively by venography, or non-invasively by impedance plethysmography, which has proven reliable in certain groups of patients.[14] Once thrombosis has occurred, prevention of pulmonary embolism rests with systemic anticoagulation; if this is contraindicated, inferior vena caval interruption may be employed.[15] The diagnosis of pulmonary embolism is made with ventilation perfusion scanning, and if this is equivocal, pulmonary angiography. Treatment is again with systemic anticoagulation, or streptokinase infusion through a pulmonary artery catheter, if the embolus is large and haemodynamically significant. If gross cardiorespiratory instability is present, thoracotomy and pulmonary embolectomy may be used as a last resort.[16]

By the time our patient presented for surgery, the embolism had probably already occurred. The $PaCO_2$ of 53 mmHg despite a minute ventilation of 8 $L \cdot min^{-1}$ supports this possibility. Increased dead space, with CO_2 retention is a common presentation of large pulmonary emboli.[17] After the intraoperative pneumothorax, perfusion to the affected lung would have further decreased. This stasis may have facilitated propagation of the clot.

In retrospect, considering the severity of the insult, the paucity of clinical clues is surprising. Pulmonary hypertension and failure to obtain a wedge pattern, as was seen in our case, is reported as suggestive of embolic occlusion of a major pulmonary artery distal to the catheter.[18]

The natural history of pulmonary embolism is spontaneous lysis[16] to which the rapid improvement in our patient attests. The lucent appearance of the right lung on chest *x*-ray suggests that the blood flow to that lung was diminished for several days postoperatively.[19] The fortunate outcome was likely related to the rapidity with which the correct diagnoses were made.

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Résumé

Un cas de pneumothorax per opératoire précédé d'une embolie pulmonaire est présenté. La littérature est revue et les différents moyens de prévention et de traitement sont discutés.