

Sudden Profound Hypoxaemia in the Intensive Care Unit—A Case Report

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Abstract

Acute hypoxaemia is a life-threatening emergency. Diagnosis of the exact aetiology may be complicated by the presence of pre-existing lung conditions. A case report is presented of a non-intubated patient with a pre-existing lung tumour who developed sudden profound hypoxaemia 3 days after emergency abdominal surgery. Definitive aetiological diagnosis was delayed due to chest X-ray features suggestive of compression and erosion of tumour tissue into the airway. Emergency computerised tomography (CT) imaging however revealed mucous plugging leading to massive atelectasis as the main aetiology.

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Key words: Airway obstruction, Atelectasis, Fibreoptic bronchoscopy, Intensive care, Mucous plugging

Introduction

Significant arterial hypoxaemia is defined as a partial pressure of oxygen (PaO_2) that is less than 60 mmHg or a percentage of oxyhaemoglobin (% HbO_2) that is less than 90%.¹ The immediate response in acute situations is to rapidly exclude or treat common conditions such as airway obstruction, pneumothorax and pulmonary embolism. Occasionally, the diagnosis of the aetiology is compounded by the absence or masking of classical clinical signs or the occurrence of misleading radiological features by coexisting lung diseases. A case report is presented where a non-intubated, alert and conversant patient suddenly developed profound hypoxaemia. This did not improve to the usual resuscitative events and a chest X-ray done showed features of tracheal obstruction and free air adjacent to the main airways, prompting the diagnosis of airway involvement by the adjacent tumour tissue. Further investigations however revealed mucous plugging leading to massive atelectasis as the main aetiology.

Case Report

A 56-year-old male presented with symptoms suggestive of intestinal obstruction. He also gave a significant 6-month history of chronic cough with haemoptysis and recent right upper chest pains. A chest X-ray done on admission showed a mass in the upper lobe of the right lung with lytic lesions of the second and third ribs, suggesting a bronchogenic carcinoma. He failed to im-

prove with conservative measures and a laparotomy was scheduled on the third admission day (Fig. 1).

At laparotomy, a small bowel tumour with lymph node metastasis was found. This was later labelled histologically as a sarcomatoid carcinoma. Intraoperatively, the cardiovascular and respiratory systems were stable. Oxygen saturation was maintained between 97% to 100% on an inspired oxygen concentration of 31% to 35%. Peak airway pressures were between 18 to 22 cm H_2O . The patient was extubated and monitored in the intensive care unit.

Postoperatively, oxygen saturations were maintained above 94% on an inspired oxygen concentration of 40%. On the second postoperative day however, the oxygen saturation on the pulse oximeter was noted to be 90%. Clinical examination pointed to a right-sided pleural effusion. A chest X-ray confirmed this and 700 ml of straw coloured fluid was drained through a 28 FG chest tube inserted via the anterior axillary line approach into the right fifth intercostal space. This was left in situ and was oscillating well. A post insertion chest X-ray showed complete expansion of the right lung. Pleural fluid analysis was negative for malignant cells.

On the morning of the third postoperative day, while the patient was conversing with us during the ward round, he suddenly became tachypnoeic and the pulse oximeter registered a fall in the oxygen saturation from 95% to 87% within a minute and then over the next 5

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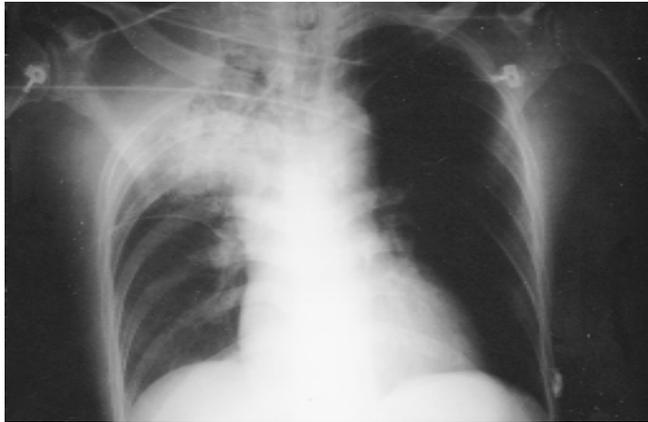


Fig. 1. Chest X-ray on admission showing right upper lobe tumour.

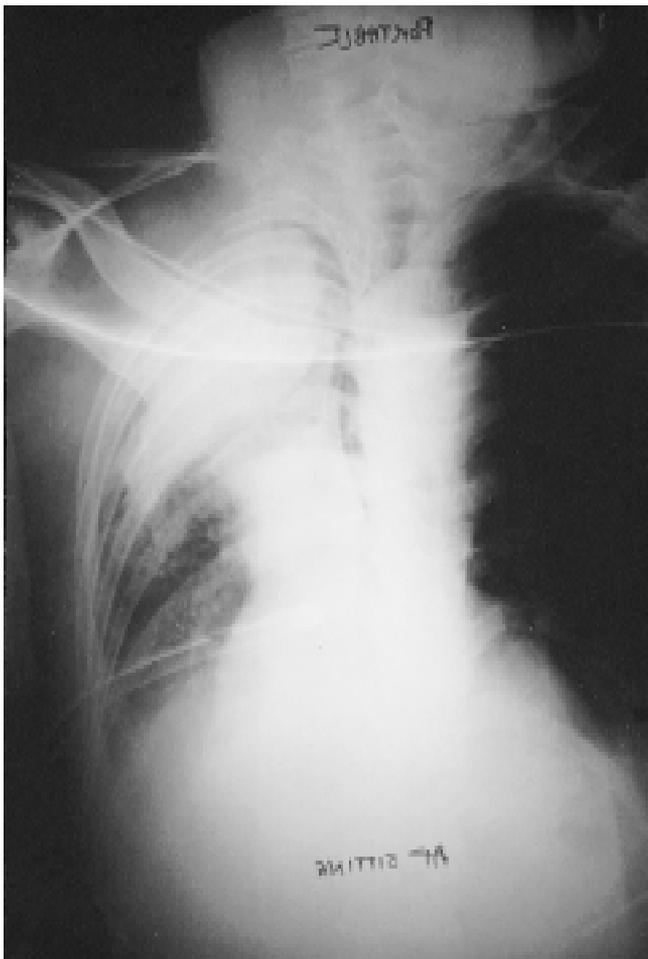


Fig. 2. Chest X-ray taken when events occurred. The right upper lobe is collapsed, there is difficulty in delineating the lower border of the trachea and there is suspicion of free air adjacent to the right heart border suggesting a pneumothorax. A chest tube is present.

minutes, to 80%. Physical examination revealed no tracheal shift, unilateral chest hyperresonance, bronchospasm or crepitations in the lung fields but there was decreased air entry on the right side. On the suspicion that the chest tube in situ was blocked as it was now not fluctuating well, a new 28 FG chest tube was immedi-



Fig. 3. This is a coronal reconstruction of transverse cuts of the CT scan images. The right main bronchus is filled with a soft tissue density suggestive of a mucous plug or aspirated material. The right lung is collapsed.

ately inserted through the previous chest tube track. There was however no air or fluid obtained. A chest X-ray done immediately thereafter showed no pneumothorax or pleural effusions on either side. There was however, both right upper and lower lobe collapses. Additionally, the lower margin of the trachea was not well visualised suggesting the possibility of extrinsic tracheal compression. There was also a need to exclude a loculated pneumothorax or pneumomediastinum adjacent to the right main bronchus (Fig. 2).

The patient was intubated but oxygen saturations remained between 80% to 85% on 100% oxygen. An arterial blood gas done read a pH of 7.409, PaO₂ 43.6 mmHg and a PaCO₂ 47.6 mmHg. Peak airway pressures were between 22 to 24 cm H₂O. The systolic blood pressure was 90 mmHg and the pulse rate 100/min. Fluid loading with colloids and the use of vasopressors increased the systolic blood pressure to 110 mmHg. At this point, the differential diagnosis was tumour invasion of the right main bronchus causing airway obstruction as well as a loculated pneumothorax or a massive

pulmonary embolism. An emergency computerised tomographic (CT) scan was done to aid in the diagnosis, to evaluate the possibility of emergency stenting of the airway and drain the possible loculated pneumothorax. This required minimal transport time and logistical arrangements in our institution as the scanner was located near the intensive care unit.

CT scan of the chest showed complete obstruction of the right main bronchus by soft tissue densities suggesting aspirated material or mucous plugging. The apical lung tumour mass was noted to be compressing the right subclavian vessels and superior vena cava but there was no direct compression on the trachea or right main bronchus. There was no loculated pneumothorax or pneumomediastinum. The scan procedure took less than five minutes (Fig. 3).

Fibreoptic bronchoscopy performed immediately thereafter confirmed the presence of secretions in the mainstem bronchus. On removal of the secretions, the oxygen saturation immediately picked up to 95%. Bronchoscopy also revealed the right upper lobe bronchus to be obliterated by tumour. No tumour fragments were aspirated from the right main bronchus nor was there any tumour in its walls.

A blood gas sample done 15 minutes after bronchoscopic suction on 60% inspired oxygen showed a pH of 7.449, PaO₂ of 62.4 mmHg and a PaCO₂ of 40.6 mmHg. A chest X-ray done 1 hour post bronchoscopic suction showed complete expansion of the right lower lobe. The patient was kept intubated that day to facilitate bronchosuction should the event occurs again. He was extubated the next day and discharged to the general ward several days later. He subsequently underwent a course of palliative radiotherapy for the lung tumour.

Discussion

Acute hypoxaemia can be fatal if not alleviated rapidly. Possible differential diagnosis for acute hypoxaemia in non-intubated patients should include pneumothorax, aspiration pneumonitis, massive atelectasis, upper airway obstruction and pulmonary embolism. In this instance, with the presence of an upper lobe lung tumour, airway obstruction by tumour fragments²⁻⁵ leading to distal collapse, haemorrhage into the bronchial tree,⁶ and erosion into the right main bronchus deserve consideration.

This patient illustrates some of the difficulties in identifying the precipitating cause. Although the clinical examination was not suggestive of either a pneumothorax or re-accumulation of the pleural effusion, the existing chest tube was not fluctuating well and could have been blocked by fibrin deposits resulting in a loss of function.⁷

Aspiration pneumonitis was considered unlikely as

the patient had not been fed for three days, gastric motility had returned and he was alert with no impairment of the cough and gag reflexes. Atelectasis was thought unlikely as he had been extubated immediately postoperatively and in the intervening 3 days did not develop a chest infection. He was on chest physiotherapy and was never noted to be coughing out excessive sputum. Pain relief was achieved with a continuous morphine infusion of 0.5 mg/h. He was alert and could communicate throughout the period in the unit. There was no extrinsic compression from the pleural effusion as this had been fully drained previously. Peak airway pressures were not elevated and were between 22 and 25 cm H₂O. A review of the literature however shows that airway pressures may not be elevated in massive atelectasis.²

Pulmonary embolism was considered possible as the event occurred suddenly, was accompanied by hypotension, tachycardia and cyanosis. Although there was no leg swelling, clinical evidence of deep vein thrombosis was found in only 30% of patients with pulmonary embolism.⁸ Mitigating factors against it were the absence of a raised jugular venous pressure and a louder second heart sound.

Extrinsic airway obstruction by the right upper lobe tumour or hilar nodes⁹ was considered more likely as the right main bronchus could not be visualised clearly on the chest X-ray. Additionally, the radiological appearance of the loculated pneumothorax could be explained by erosion of tumour into the right main bronchus. By themselves, tumour fragments have been reported to cause airway obstruction²⁻⁵ but they cannot explain the presence of the loculated pneumothorax. Massive haemoptysis as a cause of airway obstruction was unlikely in view of its clinical absence.

Pulmonary atelectasis is probably the most common respiratory complication occurring during the postoperative period in patients undergoing abdominal, thoracic or cranial surgery. Collapse is usually segmental and is generally not life-threatening, however some patients develop acute massive collapse involving an entire lobe or a whole lung, resulting in severe life-threatening hypoxaemia requiring emergency treatment.¹⁰ Similar acute situations have been reported in patients under anaesthesia.^{11,12} The exact incidence of massive atelectasis secondary to mucous plugging is unknown but in the paper by Marini et al,¹³ 4 out of the 31 patients selected for their trial over a 9-month period had lobar atelectasis involving an entire lung. It is unclear if mucous plugging was the aetiology in all 4 cases. In Mahajan's¹⁰ series of 10 cases, 2 had collapse of one entire lung with both having underlying pulmonary oedema. While the role of fibreoptic bronchoscopic suction for acute lobar collapse is controversial, its use in symptomatic whole lung collapse is reasonable.¹⁴

Early evaluation of the lung tumour in this asymptomatic patient presenting for emergency extrathoracic surgery, preoperatively either with a CT scan or fiberoptic bronchoscopy could possibly have allowed us to make a more rapid diagnosis especially if there was evidence of pre-existing airway compression or tumour tissue in the main airways.

In summary, a case report is presented in which mucous plugging led to massive atelectasis resulting in profound hypoxaemia. The diagnosis was compounded by its acute presentation in a non-intubated patient who was otherwise well and by the presence of a right upper lobe tumour which gave the initial radiological impression of extrinsic compression of the airways. It is suggested that even patients with asymptomatic lung tumours presenting for emergency extrathoracic surgery be evaluated with fiberoptic bronchoscopy, preoperatively or intraoperatively, to better define the presence of any airway obstruction.

REFERENCES

1. Shapiro B A, Peruzzi W T. Interpretation of blood gases. In: Ayres S M, Grenvik A, Holbrook P R, Shoemaker W C, editors. *Textbook of Critical Care*. 3rd ed. Philadelphia: W B Saunders Co, 1995:278-94.
2. Sakura S, Nomura T, Uchida H. Tumour fragments in the airway detected by intraoperative fiberoptic examination. *Anaesth Intensive Care* 1996; 24:265-6.
3. Tzabar Y. Intraoperative tracheal obstruction by tumour fragments. *Anaesthesia* 1995; 50:249-50.
4. Barat G, Ascorve A, Avello F. Unusual airway obstruction during pneumonectomy. *Anaesthesia* 1976; 31:1290-1.
5. Heydorn W H, Barry M J, Zajchuk R, Strevy T E. Tumour embolus as a cause of death during pneumonectomy. *J Thorac Cardiovasc Surg* 1974; 67:308-9.
6. Strollo P J. Haemoptysis. In: Civetta J M, Taylor R W, Kirby R R, editors. *Critical Care*. 1st ed. Philadelphia: J B Lippincott Co, 1988:1127-32.
7. Gregoire J, Deslauries J. Closed drainage and suction systems. In: Pearson F G, Deslauries J, Ginsberg R J, editors. *Thoracic Surgery*. 1st ed. New York: Churchill Livingstone, 1995:1121-35.
8. Freebairn R C, Oh T E. Pulmonary embolism. In: Oh T E, editor. *Intensive Care Manual*. 4th ed. Oxford: Butterworth-Heinemann, 1997:280-90.
9. Doyle L A, Aisner J. Clinical presentation of lung cancer. In: Roth J A, Ruckdeschel J C, Weisenberger T H, editors. *Thoracic Oncology*. 2nd ed. Philadelphia: W B Saunders Co, 1995:26-48.
10. Mahajan V K, Catron P W, Huber G L. The value of fiberoptic bronchoscopy in the management of pulmonary collapse. *Chest* 1978; 73:817-20.
11. Shaw I H. Collapse of the lung during hemithyroidectomy. *Anaesthesia* 1990; 45:61.
12. Samuels S I, Brodsky J B. Profound intraoperative atelectasis. *Br J Anaesth* 1989; 62:216-8.
13. Marini J J, Pierson D J, Hudson L D. Acute lobar atelectasis: A prospective comparison of fiberoptic bronchoscopy and respiratory therapy. *Am Rev Respir Dis* 1979; 119:971-8.
14. Dellinger R P. Fiberoptic bronchoscopy in critical care medicine. In: Ayres S M, Grenvik A, Holbrook P R, Shoemaker W C, editors. *Textbook of Critical Care*. 3rd ed. Philadelphia: W B Saunders Co, 1995:761-9.