Haemoptysis in a patient operated under regional anaesthesia: What could be the first diagnosis?

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Abstract

Introduction
Haemoptysis is always an alarming symptom but a rather rare event in the recovery ward following an uneventful regional anaesthesia. The aim of this case report is to highlight all possible causes of haemoptysis in an anaesthetist’s daily practice, and to emphasize that tuberculosis is one of the possible causes of haemoptysis and a re-emerging disease.

Case Report
A young male patient, heavy smoker, submitted for emergency surgery, presented massive haemoptysis after uneventful regional anaesthesia. He was admitted to the ICU and discharged 48 h later. Initial diagnosis was lidocaine-induced cardiomyopathy. He received anti-tuberculosis treatment based on a positive Mantoux test. He completely recovered a year later.

Conclusion
Since tuberculosis is a re-emerging disease, preoperative evaluation should be more careful.

Introduction
Haemoptysis is always an alarming symptom but a rather rare event in the recovery ward following an uneventful regional anaesthesia. In large pulmonary medicine structures, massive haemoptysis occurs in less than 0.5% patients due to chronic lung infections or neoplastic disease. The aim of this case report is to discuss haemoptysis in a patient operated under regional anaesthesia.

Case Report
Here we present an unusual case of haemoptysis in a patient admitted for emergency surgery carried out under epidural anaesthesia. Medical history of the case did not indicate any major disease.

A 27-year-old male, heavy smoker, working as manual labour, American Society Anaesthesiologists (ASA) category II, was admitted for emergency inguinal hernia repair. Physical examination, laboratory tests and chest radiograph revealed no abnormality at that time (Figure 1).

The patient was pre-mediated with diazepam 10 mg orally 1 h before epidural anaesthesia at L3–L4 interspace with 2% lidocaine 300 mg and fentanyl 0.05 mg.

The duration of the intervention was 75 min; Ringer’s lactate 750 ml and Gelofusine 250 ml were infused keeping haemodynamic and respiratory stability of blood pressure, 110/60 mmHg; heart rate, 75/min; SpO2, 100% and FiO2, 0.28. After completion of the surgery, the patient was transferred to the recovery room where he gained motility and sensation in the lower extremities approximately 30 min later.

The patient suddenly experienced vigorous dry cough, and 5 min later, he expectorated 30 ml of bright red blood, developing sinus tachycardia (125 pulses/min) and respiratory distress with dyspnoea, forceful inspiratory efforts and bilateral chest pain in the
superior mediastinal region and anterior thorax. He presented acute arterial desaturation: PaO$_2$, 55 mmHg; PCO$_2$, 36 mmHg and pH, 7.44 rapidly deteriorated to PaO$_2$, 48 mmHg; PCO$_2$, 33 mmHg and pH 7.2; the PaO$_2$/FiO$_2$ ratio was 80. The patient continued to expectorate fresh blood followed by pink exudates (70 ml), and he was admitted to the ICU.

Oropharyngeal examination was negative for injury and auscultation revealed bilateral crepitations in the upper pulmonary fields. Consciousness and haemodynamic stability were maintained, and arterial saturation gradually improved (SpO$_2$ 90 under Venturi mask 60%); thus, neither IPPV nor non-invasive mechanical ventilation were introduced and neither invasive haemodynamic monitoring nor Swan–Ganz catheter were used. Chest X-ray revealed bilateral infiltrates to the upper and median right pulmonary fields without cardiac enlargement (Figure 2). ECG was normal, but echocardiography revealed decreased left ventricular contractility (ejection fraction, 46%).

The patient showed rapid clinical improvement, the echocardiography was normalized. At 48 h after admission, he was discharged from the ICU with a diagnosis of lidocaine-induced cardiomyopathy.

Further investigation included Ga 67 pulmonary scanning, sputum direct stain, cytology and cultures for common bacteria, mycetes and mycobacteria, which all tested negative. The full urine tests, allergen tests and HIV serology were negative also well. Anti-tuberculosis (TBC) treatment was started based on a positive Mantoux test. The patient fully recovered a year later.

Discussion

Haemoptysis is a common non-specific alarming sign present in many pulmonary diseases, including chronic or acute infections (such as bronchiectasis, TBC, histoplasmosis, bronchitis and fungal infections)\(^3\), tumours\(^1\) and cardiovascular diseases (such as valvular diseases, pulmonary thromboembolism or malformations, pulmonary oedema and Goodpasture syndrome)\(^4\). It is also present in traumatic conditions such as in cases of lung contusions, air embolism, foreign body, Swan–Ganz catheter insertion\(^4\), after laryngeal mask application\(^2\) or in cases of post-intubation trachea rupture, predominantly in women more than 50 years of age\(^5\). Concerning the latest case in the last decade, the estimated incidence of trachea rupture presented with haemoptysis and emphysema, complicating endotracheal intubation, ranged from 0.05% to 0.37% of all orotracheal intubations\(^6\). Haemoptysis is reported in a case of diffuse alveolar haemorrhage due to pulmonary capillaritis\(^7\). It can also be of an unknown aetiology\(^8\).

Pulmonary oedema can be:

- cardiogenic, with or without fluid overload, from an increase in pulmonary capillary pressure originating from left ventricular dysfunction or from outflow obstruction with increased afterload;
- neurogenic, due to massive sympathetic neuronal discharge, resulting in systemic vasoconstriction and diversion of blood from the systemic to pulmonary circulation. In this setting, there are indices of increased capillary pressure and altered permeability\(^9\);
- associated with severe upper airway obstruction, as is the case in laryngospasm in adults, obstruction by a laryngeal mask or unilateral re-expansion of the lung. There are conflicting opinions concerning the mechanism of oedema, whether increased negative hydrostatic pressure resulting from inspiratory efforts against closed glottis co-exist with high capillary permeability or if aspiration in the bronchii and hypoxic vasoconstriction contributes to injury\(^8,9\);
- associated with beta-2 adrenergic receptor stimulants in premature labour\(^9\) or
- associated with naloxone reversal, in a manner resembling the neurogenic type\(^8\).

In many instances, it is difficult to estimate the quality and quantity...
Haemoptysis is defined as massive when the amount of daily blood volume expectorated exceeds 200 to 600 ml in 24 to 48 h, or when it results in acute airway obstruction, severe respiratory distress or major circulatory shock. In more than 90% of the reported cases, in immigrants in the West as well as in developing countries, massive haemoptysis is associated with underlying chronic pulmonary infection. Severe and untreated haemoptysis has a mortality rate of more than 50%.^6^7^.

Treatment of haemoptysis depends on the severity and persistence of the event, and the likelihood of recurrence.

In the literature, TBC haemoptysis is most often described as an event secondary to cavitary disease, aspergilloma, neoplasia or bronchiectasis. There is also a growing level of incidence of haemoptysis related to multi-drug resistant bacilli in patients with HIV infection even when TBC or other underlying lung disease has not been previously diagnosed. In young adults, haemoptysis due to non-caviar formatting TBC has been reported as a rare, devious and possibly lethal event. Angiographic findings in pulmonary TBC with massive haemoptysis include hypervascularisation, hypertrophy of systemic arteries, aneurysm, systemic to pulmonary anastomosis and rarely, contrast extravasation. Bronchial arteries are the source of haemorrhage in the majority of these cases.

Haemoptysis shows seasonal variation since exposure to cold, and the dry air may have a direct irritating action on respiratory mucosa. This could be worsened when there is underlying lung pathology, as is bronchial asthma. Respiratory tract infections, which also present seasonal variation, are known to affect the incidence of asthma exacerbations. Recurrent episodes of respiratory tract infections can result in haemoptysis even without TBC.

It has been reported that in patients with TBC, regional or general anaesthesia and positive pressure ventilation may promote pulmonary dysfunction as a consequence of drawing the patient’s own blood, and regional anaesthesia—even in combination with propofol—offers better operability. Regional anaesthesia, however, carries the risk of misinterpreting complications of the procedure with rare localisations of TBC, such as spinal or epidural.

Fibreoptic bronchoscopy accomplished with imaging studies (chest X-ray, CT), as it was in our case, is the diagnostic procedure of choice in an attempt to localize the bleeding site. However, difficulty to localize the bleeding site makes surgery a poor option. Bronchial arteriography and bronchial artery embolisation provide an effective means of non-surgical procedure for rapid diagnosis and treatment of severe haemoptysis.

In the reported patient, protein concentration in the expectorated was not measured, as the frothy sputum was misinterpreted as pulmonary oedema on admission to the ICU. Direct inspection and pH measurement in the recovery room confirmed haemoptysis. Reduced cardiac output and death have been reported in patients with hypertrophic cardiomyopathy receiving spinal or epidural anaesthesia, but in our case, no signs of low cardiac output, hypoperfusion, arrhythmia or abnormal cardiac sounds were observed; thus, such a diagnosis can be excluded. Myocarditis due to accompanying upper respiratory tract infection cannot be excluded, because recent reports present such patients with acute infection and transient left ventricular hypertrophy, impaired ejection fraction (EF, 40%–57%) and non-Q or Q wave in the ECG, with lower mortality in the non-Q group than in the Q group (8% vs. 27%).

In the present case, adequate intravascular volume expansion with colloids and crystalloids prevented vascular collapse, and spontaneous cessation of bleeding made the transfusion of whole blood unnecessary. We believe that haemoptysis most likely resulted from injury to the tracheobronchial vessels, enlarged from chronic inflammatory revascularisation due to a possible upper respiratory tract infection not reported by the patient. Also, haemoptysis could be presented due to persistent cough amplified by the cold environment and the presence of TBC. Haemoptysis during spontaneous breathing in a patient under epidural anaesthesia has not been previously reported.

**Conclusion**

TBC is a re-emerging disease and it is associated with possible serious complications from the respiratory system. It is imperative that the preoperative evaluation should be more careful, keeping in mind that complications exist—rarely or not—and they must be anticipated.

**Consent**

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal.

**References**