CASE REPORT

Abolished ventilation and perfusion of lung caused by blood clot in the left main bronchus: auto-downregulation of pulmonary arterial blood supply

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SUMMARY

It is generally assumed that the lungs possess arterial autoregulation associated with bronchial obstruction. A patient with pneumonia and congestive heart failure unexpectedly developed frequent haemoptysis. High-resolution CT and diagnostic CT were performed as well as ventilation/perfusion (V/Q) scintigraphy with single-photon emission CT (SPECT)/CT. V/Q SPECT/CT demonstrated abolished ventilation due to obstruction of the left main bronchus and markedly reduced perfusion of the entire left lung, a condition that was completely reversed after removal of a blood clot. We present the first pictorially documented case of hypoxia-induced pulmonary vasoconstriction and flow shift in a main pulmonary artery due to a complete intrinsic obstruction of the ipsilateral main bronchus. The condition is reversible, contingent on being relieved within a few days.

BACKGROUND

Hypoxic pulmonary vasoconstriction is a fundamental physiological mechanism by which the lungs optimise ventilation/perfusion (V/Q) matching,¹ and redirect blood flow from poorly to better ventilated areas.² If airway obstruction is mild, it may have little effect on airflow and patients may be asymptomatic. Central airway obstruction may be extrinsic, intrinsic or mixed, fixed or dynamic. In this case, we present a benign, intrinsic obstruction of the main left bronchus, and show that the compensatory reduction in the pulmonary vascular circulation in response to sustained ventilation impairment is reversible.

CASE PRESENTATION

A patient treated with antibiotics for pneumonia was admitted to hospital with dyspnoea, high fever, dry cough and low arterial oxygen saturation.

On arrival, the patient had chills and a productive cough. Respiratory frequency was 32 breaths/min, temperature 40.2°C, heart rate 125 bpm, blood pressure 92/51 mm Hg, acidity (pH) 7.51, arterial oxygen partial pressure (P_aO_2) 6.98 kPa, arterial carbon dioxide partial pressure (P_aCO_2) 4.36 kPa, arterial oxyhaemoglobin saturation (S_aO_2) 90.7% and C reactive protein, 136. Chest X-ray showed cardiac augmentation, bilateral interstitial lines (Kerley B lines), pleural effusions and infiltrates, indicating pneumonia and congestive

heart failure. The patient was treated with nasal oxygen 3–4 L/min, and the antibiotic treatment was changed. In addition, a β blocker, an ACE inhibitor and diuretics due to signs of congestive heart failure were initiated. Owing to episodes of blood-stained mucus, a high-resolution CT (HRCT) scan was performed since pulmonary thromboembolism was suspected, and the patient was started on heparin treatment. The HRCT demonstrated bilateral pleural effusions and ground glass phenomenon. All arteries could be followed to the periphery with no signs of thromboembolism. Especially, no obstruction of the left main bronchus was registered (figure 1). The coagulation system was intact.

Owing to frequent haemoptysis, V/Q single-photon emission CT (SPECT)/CT was performed and demonstrated abolished ventilation due to obstruction of the left main bronchus and markedly reduced perfusion of the entire left lung (figure 2, 1a).

Figure 1 Diagnostic CT in transversal and coronal plane. Bilateral pleural effusions and a normal left main bronchus and contrast filling of arteries to both lungs.

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Figure 2 1a: Ventilation perfusion (V/Q) single-photon emission CT (SPECT)/CT. Ventilation scintigraphy (left picture) and perfusion scintigraphy (right picture) anteroposterior view demonstrating abolished ventilation of the left lung (L) and markedly reduced perfusion of the entire left lung (L). Images are made with more contrast in order to better illustrate the remaining but sparse perfusion of the left lung (L). 2a: V/Q SPECT/CT anteroposterior view. Ventilation scintigraphy (left picture) and perfusion scintigraphy (right picture). Homogenous and normal V/Q in both lungs (R and L). Six days after removal of an obstructive blood clot in the left main bronchus V/Q SPECT/CT demonstrating virtual normalisation of ventilation and perfusion. 1b: Diagnostic CT transversal and coronal plane demonstrating material in the left main bronchus (long arrow). Hounsfield units suggested blood. Notice the 'whirling' of contrast (short arrow) in left pulmonary artery and lack of contrast in the more peripheral arteries in left lung. 2b: Diagnostic CT transversal and coronal plane after removal of the blood clot demonstrating a normal left main bronchus and normalised blood flow in the left pulmonary artery.

This obstruction was confirmed by diagnostic CT scan, which also demonstrated reduced blood flow in the left pulmonic artery and a whirling contrast phenomenon with almost non-existent contrast filling of the more peripheral arteries (figure 2, 1b). There was no sign of associated atelectasis, emphysema or consolidation of the lung parenchyma itself.

During bronchoscopy, a completely obstructing major blood clot (100 mL) and amounts of viscous mucous were removed from the left main bronchus. There was no sign of any malignancies and the patient fully recovered.

A control CT scan revealed normalisation of blood flow (figure 2, 2b) in accordance with control V/Q-SPECT/CT (figure 2, 2a), which also demonstrated normalised V/Q.

INVESTIGATIONS

HRCT and diagnostic CT scan were performed. V/Q scintigraphy with SPECT/CT was performed with inhaled 35 MBq ^{99m}Tc -Technegas and 185 MBq macroaggregated albumin ^{99m}Tc MAASOL injected intravenously.

TREATMENT

The patient was initially treated with amoxicillin/clavulanic acid (500 mg+125 mg 3 times per day).

In our hospital, the patient received nasal oxygen 3–4 L/min, and antibiotics were changed to piperacillin/tazobactam (4 g +0.5 g 4 times per day) and gentamicin (300 mg daily). In addition, carvedilol (3.125 mg 2 times per day), ramipril (2.5 mg 2 times a day) and furosemide (40 mg daily) were administered.

Bronchoscopy was performed with removal of aspirated material.

OUTCOME AND FOLLOW-UP

The patient slowly recovered from the pneumonia and congestive heart failure, but recovery was more rapid after removal of blood clot and viscous material from the left main bronchus.

DISCUSSION

Hypoxia has a marked effect on distribution of pulmonary blood flow.¹ In the systemic circulation, arterial blood pressure allows alterations in local vascular resistance to redirect blood flow to areas of increased demand such as, for instance, to muscles during exercise or mesenteric arteries after a meal. In the pulmonary circulation, arteries are all similar and there is no need to redirect flow, except when it comes to hypoxia. Hypoxic pulmonary vasoconstriction is an important mechanism for reducing the perfusion of inadequately ventilated lung tissue mediated through modulation and release of several partly unknown bioactive substances in combination with endothelial signal transduction to pulmonary arterioles.^{1–4} There are studies with some conflicting results due to failure to account for the reduction in cardiac output seen during anaesthesia.¹ Inhaled anaesthetics may depress hypoxic pulmonary vasoconstriction. Decrease in cardiac output may reduce the mixed venous oxygen tension with consequential generalised pulmonary vasoconstriction.

This report describes a patient with marked diminution of pulmonary capillary blood flow in the left lung as determined by macroaggregated ^{99m}Tc -albumin scintigraphy. This may be ascribed to hypoxia caused by almost total obstruction of the main bronchus secondary to a large intraluminal blood clot. The bleeding into the bronchial tree may have been worsened by the anticoagulant. Since the main pulmonary arteries were unremarkable on CT and the arteries could be followed to the periphery a few days before, there probably was insufficient blood flow to the lung capillaries as illustrated by pulmonary V/Q scintigraphies.

Experimental observations have suggested that the effect of hypoxia on pulmonary circulation is mediated by vasomotor signals. We suggest that the blood clot caused alveolar hypoxia and that the probable arteriolar vasospasm was completely reversed after removal of the blood clot.

We present the first pictorially documented case of hypoxia-induced pulmonary vasoconstriction and flow shift in a main pulmonary artery due to a complete intrinsic obstruction of the ipsilateral main bronchus. The condition is reversible, contingent on being relieved within a few days.

Learning points

- ▶ Hypoxia-induced pulmonary vasoconstriction and flow shift in a main pulmonary artery may be due to a complete intrinsic obstruction of the ipsilateral main bronchus.
- ▶ The condition is reversible, contingent on being relieved within a few days.

Contributors AB was involved in data collection, data analysis and data interpretation. JHH was responsible for data interpretation and writing.

PA participated in study design, data collection, data analysis, data interpretation, writing, principal investigator and corresponding author guaranteeing the overall content of the manuscript.

Competing interests None declared.

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