Fatal endotracheal haemorrhage in a patient undergoing repair of a large ascending aortic aneurysm

doi: 10.1017/S0265021506002171

EDITOR:

Endotracheal haemorrhage during cardiopulmonary bypass (CPB) is a rare complication. It may result from the effects of systemic heparinization and trivial airway injury that may occur during endotracheal intubation or erosion due to tracheal suction. Various lung parenchymal diseases known to cause haemoptysis may cause severe haemorrhage on CPB [1]. The most commonly described cause of endotracheal haemorrhage on CPB is pulmonary artery (PA) rupture complicating the use of PA catheter [2]. We report fatal endotracheal bleeding after manual inflation of a chronically collapsed lung in a patient who underwent ascending aortic aneurysm (AAA) repair.

A 56-yr-old woman weighing 50 kg underwent AAA repair. The salient clinical features were increasing breathlessness and grossly diminished air entry on right side of the chest. Laboratory test results and the electrocardiogram (ECG) were within normal limits. Computed tomography scan of the thorax showed a grossly dilated ascending aorta and compressed right lung. Transthoracic echocardiography showed the AAA; the aortic valve and the left ventricular function were normal. Pulmonary function tests showed moderately severe obstructive and restrictive lung disease.

Induction of anaesthesia, oral intubation with a 7.5-mm endotracheal tube and prebypass period were uneventful. Monitoring included ECG, heart rate, pulse oximetry, end-tidal CO_2, arterial pressure and central venous pressure. A PA catheter was not used. The airway pressure was 25–30 cm H_2O at a tidal volume of 400 mL and respiratory rate of 15 min^{-1}. Initially, the left femoral artery was exposed and prepared for cannulation to establish CPB. After systemic heparinization, CPB was initiated with left femoral artery and right atrial cannulation. On hypothermic (26°C) CPB and cold blood cardioplegic arrest, the diseased aorta was replaced with a 26-mm collagen-coated woven Dacron tube graft (Hemashield®; Boston Scientific, USA). After surgical repair, to de-air the heart, the lungs were manually inflated with the APL valve set at 30 cm H_2O. During ventilation, the right pleura was noticed to be not moving with ventilation. To ensure expansion of the right lung, the pleura was opened. Further inflations were given to re-expand the collapsed right lung. Suddenly, a large quantity of blood appeared in the endotracheal tube and the breathing circuit. The attempts to re-expand the lung were immediately stopped. Tracheal suctioning and lavage with cold saline were done repeatedly but the bleeding continued. Immediately, the endotracheal tube was replaced with a 37-Fr left-sided double-lumen endobronchial tube over a tube exchanger, and positioned in the left bronchus guided by a fibreoptic bronchoscope. Fibreoptic bronchoscopy showed continuous bleeding from the right bronchus; however, the precise bleeding site could not be located. The right pleural opening was further widened that showed extensive lung injury; the pleura was thickened and densely adherent to the lung and there was significant bleeding on pleural dissection. On manual ventilation, the left lung felt stiff, whereas, in the right lung, ventilation was almost impossible. In view of the extensive adhesion, lung parenchymal injury and bleeding while opening the mediastinal pleura, it was felt that the patient would not sustain pneumonectomy. Further, we hoped that protamine reversal of systemic heparinization would reduce or stop the bleeding from the right bronchus. Therefore, weaning of CPB was attempted with heavy inotropic support and the patient was weaned off CPB and anticoagulation was reversed. However, bleeding continued through the right lumen of the endobronchial tube. Arterial blood gas analysis on 100% O_2 showed a PaO_2 and $PaCO_2$ of 55 and 62 mmHg, respectively; the airway pressure at a tidal volume of 400 mL was 50 cm H_2O. Approximately 20 min after weaning from CPB, the patient developed ventricular fibrillation from which she could not be resuscitated. A review of the thoracic CT scan showed thickened mediastinal pleura and interlobar fissure. Evidently, it was a trapped lung and the implications of the thickened pleura and interlobar fissure were not realized.

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Accepted for publication 21 September 2006 EJA 4172
First published online 4 January 2007

Discussion

Lung compression is common in the presence of a mediastinal mass and large AAA. Generally, the compressed lung re-expands on manual inflation after removal of the cause of compression. The collapsed lung can cause inadequate gas exchange, raised pulmonary vascular resistance, raised afterload to the right ventricle and failure to wean from CPB; therefore, lung expansion is routinely ensured before termination of CPB. It should be noted that the blood gas disturbances due to chronic lung collapse are not significant since the normal lung usually compensates; however, chronic lung collapse should be differentiated from acute lung collapse, which results in hypoxaemia. In our patient, during de-airing of the heart, the chronically compressed right lung did not expand on manual inflation and sustained attempts using moderate inflation pressure resulted in lung trauma, bleeding and death. The most commonly described cause of endotracheal haemorrhage on CPB is PA rupture complicating use of a PA catheter [2]. Chauhan and colleagues [1] described iatrogenic creation of a fistula between the PA and left bronchus during placement of a PA vent for coronary artery bypass graft surgery. Long-standing lung collapse results in fibrotic changes in the lung parenchyma and in the overlying pleura that prevent lung inflation [3]. In patients presenting with a chronically compressed lung, during the preoperative visit the CT scan should be specifically examined for thickening of the interlobar fissure and mediastinal pleura and the possibility of the lung being trapped. In the presence of a trapped lung, manual inflations may not expand it. Forceful attempts to re-expand a chronically collapsed lung should be avoided. In our patient, presumably, the thickened pleura prevented lung reinflation and the pressure developed during manual inflation resulted in damage to the lung parenchyma.

The management issues include adequate oxygenation, prevention of soiling of the healthy lung and definitive treatment of bleeding. A management algorithm has been described [4,5]. Oxygenation can be ensured by continuing CPB. Temporary occlusion of the PA may reduce pulmonary bleeding while the patient is on CPB and may help replacement of the endotracheal tube with a double-lumen tube. Once oxygenation and lung isolation is achieved, attention should be paid towards definitive treatment of haemorrhage. The pleura on the side of the injury should be opened to assess lung parenchymal damage. Clamping and unclamping of the PA during fibreoptic bronchoscopy may identify the source of bleeding and help placement of a lobar bronchial blocker. Further, during weaning from CPB, the adequacy of the bronchial blocker should be assessed by bronchoscopy [1]. Weaning from CPB should be attempted only after achieving adequate bleeding control and oxygenation. Protamine should not be administered in the hope that once normal coagulation is restored the pulmonary bleeding will stop [1]. In case of extensive lung parenchymal injury or if the visceral pleura is ruptured, pulmonary resection should be considered; however, pulmonary resection in this setting carries a high mortality [1]. The bronchial blocker, if used, is deflated and removed after 24–48 h of postoperative ventilation under fibreoptic surveillance.

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https://doi.org/10.1017/S0265021506002171 Published online by Cambridge University Press