Clinical Reports

Pneumomediastinum during general anaesthesia: a case report

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A case is reported of a pneumomediastinum which presented as an unexplained dysrhythmia during a routine general anaesthetic in a previously fit 19-year-old girl. The possible precipitating factors in this case are discussed along with a description of the signs and symptoms and an outline of its management.

Pneumomediastinum was first described in 1819 by Laennec[1] and it became well recognised as a clinical entity during the influenza epidemic of 1918. Macklin and Macklin presented their classical paper describing its pathophysiology in 1944.[2]

Case history

A 19-year-old girl weighing 62 kg, with an eight-year history of conductive deafness, was admitted for right radical mastoidectomy. Her general health was good and she had no significant past medical history. She had not been anaesthetised previously, was receiving no medication but smoked 15 cigarettes a day. Preoperative investigations were normal, although a chest *x*-ray was not felt to be required.

Following preoperative medication with papaveretum 15 mg and prochlorperazine 12.5 mg intramuscularly, anaesthesia was induced with fentanyl 100 μg, droperidol 2.5 mg and thiopentone 250 mg intravenously. D-tubocurarine 35 mg was administered for muscle relaxation and to assist in reducing the blood pressure during surgery. The trachea was intubated without difficulty using an 8.5 mm cuffed, red rubber endotracheal tube. On auscultation it was discovered that the tube had entered the right main bronchus. It was withdrawn until the breath sounds became equal and then secured. The patient was ventilated gently by hand via a Bain circuit in the anaesthetic room and therefore it was not possible to quantify the resulting airway pressures, although the expiratory relief valve was set at a maximum of 60 cm water pressure.

The patient's lungs were ventilated using a Brompton Manley ventilator provided with a fresh gas flow of 9 $L \cdot min^{-1}$, and the tidal volume was set to 700 ml. The resulting peak pressure during inflation was 20 cm water with a plateau pressure of 18 cm water. Anaesthesia was maintained with 66 per cent nitrous oxide in oxygen and supplements of fentanyl, droperidol and d-tubocurarine were given during the operation. Halothane 0.5–1 per cent was given to control mean arterial blood pressure at between 80 and 100 mmHg. The patient was monitored using an ECG (CM 5 position) and an automatic blood pressure recorder.

Seventy-five minutes after induction of anaesthesia the patient developed premature atrial contractions at the rate of approximately 10 to 15 per minute. This had no effect on the blood pressure. The adequacy of ventilation and the position of the endotracheal tube were checked by inspection and auscultation and found to be satisfactory. The concentration of halothane was reduced and then discontinued, but the premature atrial contractions persisted, although at a slightly reduced frequency for the rest of the operation, a further 30 minutes.

Neuromuscular blockade was reversed with neostigmine 2.5 mg and atropine 1.2 mg and it was noted that the T waves on the ECG became inverted with the resulting tachycardia. The patient's trachea was extubated uneventfully and she was transferred to the Recovery Room where her observations were found to be stable: a blood pressure of 130/60 mmHg and a regular pulse of 95 beats per minute. A chest *x*-ray, which was ordered to exclude alveolar collapse following the intubation of the right main bronchus, showed a pneumomediastinum.

After discussion with a cardiothoracic surgeon, the

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patient was treated conservatively and routine postoperative observations were carried out. Over the next 24 hours the patient did not have any chest pain or dyspnoea, had no evidence of subcutaneous emphysema and her cardiovascular variables remained stable. A further chest *x*-ray on the first postoperative day showed that the gas within the mediastinum had almost entirely disappeared. The patient made an uneventful recovery and was discharged home two days later.

Discussion

Pneumomediastinum may present either intraoperatively or immediately postoperatively from a number of possible causes. When a pressure gradient develops between marginal alveoli and the perivascular interstitial tissue, due to either an increase in alveolar pressure or a decrease in perivascular pressure, alveolar rupture occurs.[2] The gas takes the line of least resistance along the fascia surrounding the bronchovascular tree to reach the mediastinum.

Alveolar rupture may have been precipitated by two possible causative factors in this patient: firstly, the initial positioning of the endotracheal tube in the right main bronchus would have caused relative overinflation of the right lung. Secondly, intermittent positive pressure ventilation increases the intra-alveolar pressures and also reduces the perivascular pressure by impeding venous return and reducing cardiac output.

Throughout anaesthesia the inflation pressures were not excessive and we felt that this played little part in the aetiology in this case. One other case of pneumomediastinum following atraumatic intubation has been reported in a patient who had respiratory depression following a drug overdose and although the authors did not highlight this as a possible cause, the right main bronchus also was intubated accidently.[3]

Air may also reach the mediastinum by tracking down the fascial planes of the neck or through any of the diaphragmatic hiatuses but as surgery was remote from these areas it is unlikely in this case.

Trauma to the oesophagus or trachea following difficult intubation can cause a pneumomediastinum.[4] However, laryngoscopy and intubation were achieved easily and atraumatically. In addition no increased resistance to expiratory gas flow occurred from bronchospasm, coughing or faulty equipment.

Any air reaching the mediastinum will increase in size during the first hour by up to 200 per cent[5] when nitrous oxide is used. This may account for the late appearance of signs during anaesthesia and for the relatively rapid resolution of the pneumomediastinum postoperatively.

Pneumomediastinum may be difficult to detect during anaesthesia. Subcutaneous emphysema is a classical sign, but it is more likely to suggest a pneumothorax, which itself may be the result of a pneumomediastinum which has decompressed into the pleural cavity. Electrocardiographic changes occur in up to 25 per cent of patients and although no characteristic pattern has been described, depressed ST segments, inverted T waves, low voltage recordings and shifts of electrical axis are common.[6] A wandering pacemaker has also been reported in a previous series of patients.[7] The mechanism of these ECG changes is not known but air around the heart may act as an irritable focus. It is not possible to diagnose pneumomediastinum from the ECG but an unexplained dysrhythmia may suggest that further postoperative investigation is required.

Postoperatively the patient may complain of stabbing substernal or precordial pain. On auscultation of the heart Hamman's sign may be heard, which is a crackling or bubbling sound best heard at the left sternal edge synchronous with systole.[8] This sign is not pathognomonic and is only found in 50 per cent of cases.[9]

The diagnosis is confirmed by chest radiography. Free gas is seen as a parallel line of lucency outlining the cardiac border and is best visualised on an expiratory film. A lateral projection is also required to demonstrate air around the aorta or retrosternally.

Appropriate treatment of pneumomediastinum is determined by the underlying cause. Any precipitating or contributing factors such as bronchospasm, inhaled foreign bodies, vomiting or high airway pressures during intermittent positive pressure ventilation should be treated promptly. Life-threatening complications such as pneumothorax, which may occur in up to a third of cases, should be relieved immediately by the insertion of an underwater sealed chest drain. A tension pneumomediastinum, which occurs very rarely, is treated by cervical mediastinotomy.[10] However, the majority of cases of spontaneous pneumomediastinum resolve with conservative management: bed rest, oxygen, adequate analgesia and reassurance.

This case demonstrates one of the more unusual complications of endotracheal intubation and intermittent positive pressure ventilation, where a pneumomediastinum developed following inadvertent intubation of the right main bronchus. This condition is rare but perhaps occurs more often than is reported and may remain undetected postoperatively. In the majority of cases it is a benign condition resolving spontaneously with conservative management.

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Résumé

Un cas de pneumomédiastin est rapporté s'étant présenté comme une dysrythmie inexpliquée lors d'une anesthésie générale de routine chez une jeune fille de 19 ans en bonne santé. Les facteurs déclenchant possibles dans ce cas sont discutés avec une description des signes et symptômes et de la conduite anesthésique générale.