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Video-assisted Thoracoscopy for Spontaneous Haemopneumothorax

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Spontaneous, as distinct from post-traumatic, haemopneumothorax is a rare but well-described clinical entity[1-4]. It presents classically with a picture of haemorrhagic shock superimposed on the chest pain and dyspnoea of a pneumothorax. Evidence of loss of circulating blood volume may be prominent early, but alternatively can be delayed many hours or days[1,3,5].

The treatment of haemopneumothorax has traditionally been the insertion of apical and basal chest drains followed by thoracotomy if bleeding continued or if drainage of the haemothorax was incomplete. Today, however, surgical intervention with the aid of video-assisted thoracoscopy is fast replacing the role played by open thoracotomy in the management of both pneumothorax[6-8] and haemopneumothorax[9].

We describe a patient with recurrent pneumothorax where delayed and unsuspected haemorrhage was first identified at video-assisted thoracoscopy. Details of anaesthesia for video-assisted thoracoscopic surgery in the presence of known pneumothorax are discussed, as is the importance of the anaesthetist being aware of the need to actively consider the possibility of accompanying and previously undiagnosed haemothorax.

CASE HISTORY

A fit, 23-year-old, 88 kg male developed left shoulder and anterior chest pain while exercising with upper body weights in a gymnasium. He gave a history of experiencing a radiologically confirmed left pneumothorax twelve months previously as well as another episode with identical symptoms about twelve months prior to that. He had been a smoker since the age of 16 years with a productive cough over recent months.

Medical help was not sought until next morning when he awoke with persisting left shoulder pain aggravated by movement and coughing. Chest X-ray showed a left pneumothorax with about 30% collapse, a small amount of pleural fluid assessed at 200 to 300 ml and evidence of apico-cystic disease.

He was admitted to hospital 22 hours after the onset of symptoms, and in view of the history of recurrent pneumothoraces, a video-assisted thoracoscopy with apical resection and apical talc pleurodesis was planned for next morning. Analgesia was provided with pethidine 100 mg IM four hourly prn with four doses being required over the intervening 18-hour period. Insertion of an intercostal drain was to be withheld unless justified by dyspnoea or a fall in oxygen saturation as monitored with pulse oximetry, together with radiological confirmation of an increase in pneumothorax size. Blood chemistry was normal, and Hb was 15.4 g/dl with a PCV of 0.452.

Next morning, though the pain in the left chest persisted there was no complaint of shortness of breath. Pulse rate was 80 per minute, blood pressure 160/80 mmHg and oxygen saturation 93% breathing air. The percussion note was recorded as being "resonant" with breath sounds "diminished plus or minus on the left side" and the trachea "deviated to the right".

Since a dose of pethidine had been required two hours before the scheduled time of surgery, no further premedication was administered. In the operating theatre the patient appeared anxious though not unusually so, was in obvious discomfort while moving from bed to the operating table, and was unwilling because of pain to cough on request. His pulse rate prior to induction of anaesthesia was found to be 120 per minute, with systolic blood pressure 110 mmHg and pulse oximetry 98% breathing oxygen.

Following pre-oxygenation he was given fentanyl 100 μg and also midazolam 3 mg because of his anxiety. Thiopentone 375 mg and suxamethonium 100 mg permitted rigid bronchoscopy at which copious

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amounts of muco-pus were suctioned from the left main bronchus. In the course of the bronchoscopy vecuronium 6 mg and a further 75 mg of thiopentone were given. A large left Robertshaw tube was inserted and, since there was no underwater-seal drain in place, ventilation with oxygen and enflurane was instituted to the right lung only.

Prior to positioning the patient on his side the pulse rate was unchanged on 120 per minute, systolic blood pressure was 100 mmHg, pulse oximetry 100%, and the end-tidal CO_2 36 mmHg. Papaveretum 10 mg was given IV.

Following the placement of the patient on his side the blood pressure had fallen to 80 mmHg systolic with the other measurements essentially unchanged. The rate of infusion of Hartmann's solution was increased, and though a fall in blood pressure of this order is not unusual following either positioning a patient on the side or the administration of IV papaveretum, the patient was given metaraminol 0.5 mg IV without delay since he had a known undrained pneumothorax and was on one-lung ventilation. The initial and a further dose of metaraminol 0.5 mg IV produced a less than expected response, improving the systolic blood pressure only to 90 mmHg with a pulse of 110 per minute. By this time the patient was prepared and draped and thoracoscopy commenced.

At thoracoscopy 3,800 ml of heavily blood stained fluid and clot was suctioned from the chest cavity. A bleeding site was identified by the presence of blood oozing past a discrete clot overlying the parietal pleura at the apex of the thoracic cavity. There was evidence of apico-cystic disease and fibrous thickening of the visceral pleura at the lung apex, but no evidence of an additional bleeding site.

One litre of Haemaccel and a litre of Hartmann's solution restored the blood pressure to 110 mmHg systolic and the pulse rate fell progressively to 80 per minute. Prior to the transfusion of four units of reconstituted red cells, a blood sample was taken for full blood estimation.

By the time the apico-cystic disease had been excised using three applications of the Endo GIA 30 stapler (Auto Suture, United States Surgical Corporation) the ooze from the bleeding site had stopped. In view of the structures underlying the parietal pleura at the apex of the chest cavity the site was not diathermied. After observation for a further ten minutes, plain talc was insufflated into the upper one half of the chest cavity and the lung re-expanded under thoracoscopic vision following the insertion of carefully positioned apical and basal drainage tubes. Pulse oximetry had remained 99 or 100% throughout the whole procedure.

The postoperative course was uneventful. Pain was well controlled with a papaveretum infusion and the chest drains were removed on the first postoperative day. The histopathology of the excised apex of the left upper lobe confirmed the macroscopic impression and no bleeding site was identified within the lung tissue examined.

Hb and PVC readings were 15.4 g/dl and 0.452 respectively on the day before surgery, 10.1 g/dl and 0.293 following 1 litre Haemaccel and 1 litre of Hartmann's solution, 13.7 g/dl and 0.404 one hour following the further transfusion of four units of reconstituted red cells, and 14.0 g/dl and 0.410 on the day following surgery.

The patient was discharged from hospital on the fifth postoperative day and at follow-up four weeks later was very well with no chest pain. Chest X-ray revealed an air/fluid level at the left apex "consistent with a small loculated hydro-pneumothorax". The remaining lung fields and pleural recesses appeared clear apart from evidence of apico-cystic disease at the apex of the right lung.

DISCUSSION

Spontaneous pneumothorax is frequently accompanied by a small amount of blood in the pleural cavity, and it has been wisely suggested that a diagnosis of spontaneous haemopneumothorax should be restricted to the few patients where a large volume of blood is present[5]. In the absence of a history of trauma or intrinsic lung disease such as tuberculosis or carcinoma, an accompanying haemothorax is found in only 2-5% of spontaneous pneumothoraces admitted to hospital[1,2,4].

With a 3,800 ml volume of sero-sanguineous fluid and clot in the pleural cavity, our patient certainly had a haemopneumothorax. Cardiovascular and serial Hb and PCV measurements suggest that the blood loss did not occur early and acutely, but must have progressed throughout the hospital admission, with perhaps an increase in bleeding rate over the two hours or so immediately preceding surgery.

The site of bleeding in spontaneous haemopneumothorax is usually found to be a small, non-contractile vessel on the parietal pleural surface where vascularized adhesions between the visceral and parietal pleura have been torn as the lung collapsed[3]. As a consequence, haemopneumothorax might be expected to occur more frequently with recurrent pneumothorax.

When blood loss is severe initially, the patient with haemopneumothorax will present with a clinical picture of haemorrhagic shock, with hypotension, tachycardia and peripheral vasoconstriction. This picture can be confused with that of tension pneumothorax, and indeed the two could co-exist. On the other hand,

evidence of appreciable bleeding may not be seen early and can be delayed many hours or days[1,3,5].

Over recent years the management of both spontaneous pneumothorax and haemopneumothorax has changed with the introduction and widespread availability of video-assisted thoracoscopy[6-9]. Prior to its introduction, management involved the percutaneous placement of an apical intercostal catheter to drain a pneumothorax together with a large-bore basal catheter to drain a haemothorax. Thoracotomy was indicated if either the pneumothorax was recurrent or the air leak persisted, or if either bleeding continued or the haemothorax failed to drain completely since incomplete drainage could lead to restrictive pleural thickening and fibrosis, or even empyema[9-13]. Today, the availability of video-assisted thoracoscopy now enables not only a visual assessment of underlying lung pathology such as apico-cystic disease, but also the early and precise identification of a bleeding site. Cautery of a bleeding site where appropriate, excision of apico-cystic disease, pleurodesis or pleurectomy, and precise placement of apical and basal chest drains are all readily achievable. By avoiding rib separation, postoperative pain is reduced and the duration of hospital stay shortened[6-8]. Occasionally following video-assisted thoracoscopic talc pleurodesis there is an inordinate degree of pleural pain which may necessitate thoracic epidural blockade for one or two days.

The use of video-assisted thoracoscopy in the diagnosis and management of a wide range of conditions including spontaneous pneumothorax, is associated with excellent outcomes and low complication rates[7,8,14]. Though complications can be surgical, they can also relate to problems with the double-lumen tube and one-lung anaesthesia, or to the practice of insufflating CO_2 into the thoracic cavity on the side of the thoracoscopy in order to facilitate lung collapse. This latter practice has been associated on occasion with convincing accounts of cardiovascular collapse[15-17], in particular profound bradycardia and hypotension[15,17]. In a pig model, insufflation of CO_2 resulted in marked reductions in cardiac output and systemic blood pressure[18]. Not surprisingly, several centres have recommended low CO_2 flow rates and limitation of insufflation volumes and pressures[15-17,19,20]. With our patient, no CO_2 was insufflated and, as is usually the case with a properly positioned double-lumen tube, the lung collapsed satisfactorily. The post-induction fall in blood pressure was not associated with the thoracoscopy and responded promptly to fluids and blood, with small increments of metaraminol being used prior to the diagnosis of hypovolaemia.

For thoracoscopy in the presence of an undrained pneumothorax, it is advisable to pre-oxygenate the patient, use suxamethonium if not otherwise contra-indicated, and avoid positive pressure ventilation until the double-lumen tube is satisfactorily positioned. This enables positive pressure ventilation to be restricted to the side without the pneumothorax. Since double-lumen tubes, even the anatomically shaped Robertshaw, can pass inadvertently into the wrong bronchus[21-23], a strong case can be made for using a fibreoptic bronchoscope to guide the double-lumen tube into the correct main bronchus[24,25]. If, however, a suitable fibreoptic bronchoscope is not available, we consider that the Robertshaw tube is preferable to a less firmly shaped plastic double-lumen tube, and also that a right-sided Robertshaw should be at hand in case the left-sided tube persists in entering the unintended bronchus.

Rigid bronchoscopy prior to the insertion of the double-lumen tube is invaluable in allowing the efficient removal of phlegm or muco-pus, as occurred in this case, and in identifying any atypical bronchial anatomy.

Once the patient is anaesthetized and the thoracoscope introduced into the chest cavity, it is our usual practice to add nitrous oxide to the inspired gas mixture and reduce the inspired oxygen concentration to 50%. The view of Conacher et al[25] is supported, that with a double-lumen tube well positioned there is rarely need or advantage during one-lung anaesthesia in maintaining the FiO_2 greater than 50%.

It could be argued that in the presence of a known pneumothorax, anaesthesia should not have been induced without a functioning underwater-seal drain[26]. However, with the thoracic surgeon standing by, it is our usual practice to induce anaesthesia in the manner described and provide ventilation to the non-pneumothorax lung only. If the patient does have an underwater-seal drain in situ, this is removed by the surgeon as soon as one-lung ventilation is established.

With the wisdom of hindsight it is obvious that hypovolaemia should have been suspected from the pre-induction tachycardia. Bleeding into the thoracic cavity should be suspected when a patient with known pneumothorax, especially a recurrent pneumothorax, develops an otherwise unexplained tachycardia or hypotension. Chest percussion with the patient in the sitting position prior to induction would certainly have warned of the presence of a large haemothorax. Advance knowledge of the presence of a large haemothorax may not have influenced the decision to perform a video-assisted thoracoscopy, though it would certainly have resulted in active preoperative blood volume replacement and central venous pressure monitoring.

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