Mediastinal Emphysema following Extradural Analgesia in a Ventilated Patient

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SUMMARY: A case of mediastinal emphysema developing in a patient under general anaesthesia supplemented with epidural analgesia is described. A possible cause is discussed and its prevention considered.

Introduction

Surgical emphysema and pneumomediastinum as complications of general anaesthesia have been reported¹, as have cases of subcutaneous supraclavicular emphysema following epidural analgesia in obstetrics^{2,3,4}. A case of mediastinal emphysema in a surgical patient under general anaesthesia supplemented with epidural analgesia is described.

Clinical details

A 58 year old woman was scheduled for sigmoid colectomy for diverticular disease. She had no past history of any respiratory disease. Physical examination and routine investigations including lung function tests were normal and there was no other evidence of chronic obstructive airway disease. She was premedicated with lorazepam 2mg orally six hours pre-operatively and anaesthesia was induced with midazolam 2mg followed by alcuronium 20mg intravenously. Intubation was easy and atraumatic. The lungs were ventilated manually with nitrous oxide in oxygen 33% with the maximum peak inspiratory pressure of 30cm H₂O. Catheterisation of the epidural space was attempted at the 2-3 lumbar vertebral interspace in the left lateral position using the loss of resistance to air to identify the epidural space. The ligamentum flavum was easily felt and entered with the Tuohy needle and there was no suggestion of injection of air into subcutaneous tissue or the deep fascial planes. Despite the easy identification of the epidural space, the epidural catheter could not be passed. Incremental injections of air were made to facilitate the passage of the catheter, and these resulted in large volumes of air being injected into the epidural space, but they did not help in the passage of the catheter. The epidural space was identified at the level of the lumbar 1-2 interspace and catheterised for a distance of 35mm with the bevel of the Tuohy needle facing cephalad. More than 40ml of air were injected into the epidural space during the course of both attempts. Epidural analgesia was established in the supine position with 15ml of 0.5% plain bupivacaine and then ventilation of the lungs was continued with an East-Radcliffe ventilator with the maximum peak inspiratory pressure of 30cm H₂O.

Two hours later in the operating theatre, subcutaneous emphysema was noticed in the right

supraclavicular region. Examination of the patient showed that there was no clinical evidence of a pneumothorax. There had been no change in the maximum peak inspiratory pressure of 30cm H₂O or reduction in the tidal volume. Superior mediastinal obstruction was suspected because of the increased back pressure in the peripheral intravenous line which was sited on the dorsum of the left hand. There was also fullness and distension of the patient's neck veins. The blood pressure, pulse and ECG were unchanged and the signs of mediastinal compression disappeared within 10 minutes. Because the emphysema was not progressing the operation was continued. An X-ray of the chest was taken prior to the reversal of neuromuscular blockade with atropine 1.2mg and neostigmine 2.5mg. The X-ray showed widening of the superior mediastinum, tracking of air into the neck from the mediastinum and a small subcutaneous emphysema (Fig 1). Spontaneous breathing restarted and the trachea was extubated. The patient was returned to the ward for further observation breathing 40% oxygen by means of a mask.

Twelve hours later there was no evidence of subcutaneous emphysema and the previous X-ray changes had disappeared (Fig 2).

Discussion

Pneumomediastinum under general anaesthesia can occur following damage to the trachea, bronchi, alveoli or oesophagus. In this patient there was no evidence of damage to the respiratory tract. Tracheal intubation had been uncomplicated and atraumatic. There had been no history of respiratory disease. A nasogastric tube had not been passed and this made the possibility of oesophageal damage unlikely. If, however, the pneumomediastinum had been caused by damage to the trachea, bronchial mucosa or alveoli, then tension pneumomediastinum would have been likely with the continued positive pressure ventilation. Pneumothorax invariably accompanies alveolar damage associated with positive pressure ventilation. In this patient the pneumomediastinum was self limiting. Inadvertent transient hyperinflation in a lung with congenital bullae or air trapping behind secretions cannot be excluded. Nevertheless, in this patient a considerable volume of air had been injected into the epidural space.

Fig 1. Chest X-ray taken before reversal of neuromuscular blockade. Note widening of superior mediastinum, tracking of air into the neck and subcutaneous emphysema.

Evans *et al.*⁵ demonstrated radiologically that there was communication between the epidural space and the retroperitoneal area via the intervertebral foramina. Air⁶ and fluid⁷ from the retroperitoneal space can spread to the mediastinum. In this case, some of the large volume of air injected into the epidural space may have found its way into the retroperitoneal space via the intervertebral foramina and then into the mediastinum. Positive pressure ventilation could have assisted this spread by the transmission of a positive pressure to the epidural space⁸. This may be a rare complication of injecting large volumes of air into the epidural space. The danger of air emboli from the injection of air into the valveless venous plexus should also be borne in mind.

This case report shows a possible alternative cause for pneumomediastinum and it is suggested that if air is used for the identification of the epidural space, minimum volumes should be used.

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Fig 2. Chest X-ray taken 12 hours post-operatively. There is now no evidence of the changes seen in Fig 1.

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