CASE REPORTS

DELAYED SURGICAL EMPHYSEMA, PNEUMOMEDIASTINUM AND BILATERAL PNEUMOTHORACES AFTER POSTOPERATIVE VOMITING

W. G. M. BREMNER AND C. M. KUMAR

SUMMARY

We describe a case of surgical emphysema, pneumomediastinum and bilateral pneumothoraces which occurred some hours after general anaesthesia for a repeat laparoscopy and followed persistent nausea and vomiting. We report the case because of the unexpected and delayed appearance, which led to delay in diagnosis and management. We suggest that this intrathoracic air leak was a consequence of postoperative vomiting rather than a complication of laparoscopy. (Br. J. Anaesth. 1993; 71: 296-297)

KEY WORDS

Complications: nausea, vomiting, surgical emphysema, pneumomediastinum.

Although surgical emphysema, pneumomediastinum and pneumothorax are well recognized complications of laparoscopic procedures, they usually become manifest during or immediately after operation [1-4]. The delayed development of these complications has not been described after general anaesthesia associated with postoperative vomiting.

CASE REPORT

A 37-yr-old woman under investigation for pelvic pain presented for a third laparoscopy in 4 years. She had undergone two previous laparoscopies which were uneventful except for postoperative nausea and vomiting. She weighed 55 kg, did not smoke and was otherwise healthy.

She was premedicated with temazepam 20 mg and metoclopramide 10 mg orally 1 h before operation. An induction sequence of fentanyl 75 μg, propofol 150 mg and atracurium 20 mg was followed by atraumatic tracheal intubation with a cuffed, 8-mm oral tube. Breath sounds were auscultated and ventilation (Manley Pulmovent MPP) to normocapnia achieved with a fresh gas flow of 7 litre min⁻¹. Tidal volume was 600 ml and peak airway pressure 18 cm H₂O. Anaesthesia was maintained with 1% enflurane and 66% nitrous oxide in oxygen. Monitoring included electrocardiography, automated arterial pressure (Dinamap), pulse oximetry, capnography (Nornocap, Datex), airway pressure, expired tidal volume (Wright Respirometer) and a nerve stimulator (Bard).

A pneumoperitoneum was formed with ease by the insufflation of carbon dioxide 3 litre and the insufflator pressure alarm was set at 15 mm Hg. After 20 min of laparoscopy (no intervention), the pneumoperitoneum was released and residual neuromuscular block antagonized with neostigmine 2.5 mg and glycopyrronium 0.5 mg. There were no problems associated with artificial ventilation during the procedure. The patient's trachea was extubated uneventfully after return of spontaneous ventilation. She was transferred to the recovery room, administered oxygen 4 litre min⁻¹ (Hudson mask) and routine clinical observations were satisfactory (heart rate, arterial pressure, oxygen saturation and ventilatory frequency). Morphine 15 mg and metoclopramide 10 mg were administered i.m. for abdominal pain in the recovery area. The patient complained of nausea and vomited several times.

Three hours after transfer to the ward, the patient complained of central chest pain, tightness across her chest and mild dyspnoea. Surgical emphysema restricted to her face and neck developed. An urgent chest radiograph revealed subdiaphragmatic gas and subcutaneous emphysema, but no pneumothorax. Conservative management involved reassurance, analgesia and oxygen therapy. There was no further vomiting. The chest discomfort and tightness continued overnight. A chest radiograph on the first day after operation revealed mediastinal emphysema and bilateral pneumothoraces, in addition to the pneumoperitoneum and subcutaneous emphysema. The larger pneumothorax was less than 25% of the lung field. Chest drains were not inserted. After 4 days of observation during which the subcutaneous emphysema and pneumothoraces resolved slowly, the patient was discharged home. At review 1 month later she was well with a normal chest radiograph and normal lung functions tests.

DISCUSSION

Several mechanisms associated with laparoscopy may result in the abnormal presence of gas in the subcutaneous tissue, mediastinum and pleural cavity. Local subcutaneous emphysema may arise if
carbon dioxide is insufflated too superficially because of incorrect positioning of the Verres needle [3]. One case of subcutaneous emphysema of the anterior chest wall has been attributed to inaccuracy of the intra-abdominal pressure gauge on the carbon dioxide insufflator machine resulting in an unintentionally high intra-abdominal pressure [2]. Pneumomediastinum may be produced by cephalad dissection and tracking of gas insufflated either preperitoneally or retroperitoneally through an unintentional peritoneal tear [3]. Mediastinal gas may then rupture through the mediastinal pleura to cause a pneumothorax. Pneumothoraces may result also from the transdiaphragmatic passage of insufflating gas through an acquired or congenital pleuroperitoneal defect [5—7]. However, it is anticipated that the problems caused by the passage of insufflated gas (carbon dioxide) during operation into the extraperitoneal area are transient because of the relatively great solubility of carbon dioxide [3, 8].

Although mediastinal emphysema may be spontaneous in the absence of underlying disease, it occurs most frequently in one of several situations which have in common an increase in alveolar pressure such as sneezing, straining, the Valsalva manoeuvre and protracted vomiting [9]. Other possible causes of this patient’s pathology not related to gas insufflation include trauma, penetrating injury or damage during instrumentation to the pharynx, larynx, trachea and oesophagus [9, 10] and pulmonary barotrauma [11]. However, intubation was atraumatic, airway pressure not excessive and the patient did not develop the serious clinical sequelae of an oesophageal tear.

The most common cause of mediastinal emphysema is alveolar rupture [9], but for this to occur, the pressure within the alveolus has to exceed that in the surrounding tissue and air from the ruptured alveoli then tracks along the bronchovascular sheaths to the lung hilum and mediastinum, whence it may spread along fascial planes to cause subcutaneous emphysema in the face, neck, chest or abdomen [9]. However, if the rate of escape of air from mediastinum is insufficient, the mediastinal pleura may rupture to cause pneumothorax [12, 13]. In our patient, we believe that there was an excessive increase in alveolar pressure after vomiting, leading to alveolar rupture, and that the gas took the line of least resistance along the fascia surrounding the bronchovascular tree to reach the mediastinum [14, 15]. It seems likely that the offending gas was oxygen-enriched air, so that subcutaneous emphysema, mediastinal emphysema and pneumothoraces were slow to develop and resolve because of equilibration of gases with blood [16]. However, we did not obtain any samples which might have revealed a reduced carbon dioxide tension.

We conclude that subcutaneous emphysema, mediastinal emphysema and pneumothorax may develop after postoperative vomiting. There may be no obvious associated factor and the complete picture may not develop in the immediate postoperative period. A case of delayed subcutaneous emphysema after operation should be investigated intensively and the surgical procedure, such as laparoscopy, should not always be assumed to be the cause.

REFERENCES


