ACUTE GASTRIC DILATATION IN AN INCARCERATED HIATUS HERNIA

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The pathophysiology of acute intra-abdominal gastric dilatation following general anaesthesia has been reviewed previously [1]. This condition may be complicated by hypovolaemic shock, electrolyte imbalance and aspiration pneumonia [2]. Complications arising from acute dilatation in an incarcerated hiatus hernia have not been reported previously in the anaesthetic literature.

SUMMARY

A case is reported of an unusual complication in which acute gastric dilatation occurred in an incarcerated hiatus hernia, resulting in left ventricular failure in the postoperative period. Failure to recognize this gave rise to concern when the patient re-presented for further surgery.

CASE REPORT

A 76-yr-old female was admitted to hospital with a fractured neck of femur. She was unable to give a history, but had been suffering from senile dementia for many years. She was scheduled for dynamic hip screw fixation under general anaesthesia.

Physical examination revealed a frail physique with dorsal scoliosis and pectus recurvatum. Cardiovascular examination revealed a regular heart rate (70 beat min⁻¹), normal jugular venous pressure, arterial pressure 140/80 mm Hg, no sacral or peripheral oedema, cardiac apex not palpable and normal heart sounds with no added sounds. Haematological and biochemical indices were within normal limits for the patient's age. The electrocardiogram showed sinus rhythm with no evidence of ischaemia, previous myocardial infarction or conduction abnormality. The chest radiograph was not reported formally before operation (fig. 1).

No premedication was prescribed. The patient's lungs were pre-oxygenated with 100% oxygen before induction of anaesthesia with thiopentone 125 mg i.v. Intubation of the trachea was facilitated by use of suxamethonium 75 mg i.v. Anaesthesia was maintained with oxygen, nitrous oxide and enflurane. Neuromuscular blockade was maintained with atracurium 30 mg and analgesia supplemented with morphine 4 mg.

ECG, heart rate and arterial pressure were monitored throughout and remained within 10% of preinduction values. Surgery lasted 70 min. A total of 400 ml of whole blood was infused to replace measured blood loss. At the end of the procedure residual neuromuscular blockade was antagonized with neostigmine 2.5 mg and glycopyrrolate 0.5 mg. Recovery was uneventful and the patient was transferred to the ward 1 h after operation. Thirty-six hour later she became increasingly dyspnoeic, heart rate was 130 beat min⁻¹, arterial pressure 170/90 mm Hg with an increased jugular venous pressure. Auscultation revealed poor air entry bilaterally, with expiratory wheeze and fine rales, and an audible third heart sound. ECG showed sinus tachycardia with no evidence of recent ischaemic change. Arterial blood-gas analysis showed pH 7.41, Pao₂, 14 kPa (FIO₂ 0.6), PaCO₂, 4.2 kPa, standard bicarbonate 21 mmol litre⁻¹ and base deficit −4 mmol litre⁻¹.

Portable chest x-rays were said to be technically very poor, but no pneumothorax was seen (figs 2, 3).

Treatment comprised frusemide 80 mg i.v.,
aminophylline 250 i.v. over 15 min, hydrocortisone 200 mg i.v. and nebulized salbutamol, in addition to diamorphine 5 mg i.v. The patient improved gradually and required no further treatment overnight. The following day it was noticed that the hip had become displaced and an anaesthetic assessment was requested regarding further surgery in the light of presumed recent left ventricular failure.

On examination there were no new physical findings. Heart rate was 80 beat min⁻¹, arterial pressure 130/70 mm Hg, with no jugular venous distension. Auscultation revealed normal heart sounds, but active bowel sounds were heard over the left chest. The interpretation of events based on a review of all x-rays was that, on admission, gastric and bowel gas was seen clearly around the cardiac outline (fig. 1)—evidence of an incarcerated hiatus hernia. The x-rays during the acute deterioration showed marked gaseous distension occupying most of the mediastinum with associated pulmonary congestion (figs 2, 3). On the following day there was less gaseous distension and the lung fields were clear.

At this stage the patient resisted all attempts to pass a nasogastric tube, but it was felt that there was no contraindication to a further operative procedure under general anaesthesia. A rapid sequence i.v. induction with cricoid pressure was performed after pre-oxygenation, using etomidate 10 mg i.v. and suxamethonium 50 mg i.v. A nasogastric tube was passed subsequently without difficulty. Anaesthesia was maintained with nitrous oxide in oxygen supplemented by enflurane. Vecuronium 5 mg was given to facilitate IPPV and morphine 3 mg given during operation. Residual neuromuscular blockade was antagonized by neostigmine 2.5 mg with glycopyrrolate 0.5 mg after 60 min. Postoperative x-ray taken in the recovery area showed there was still some bowel gas present in the mediastinum, but that the stomach remained decompressed (fig. 4). The nasogastric tube remained in place and there were no further problems in the early postoperative period. In spite of intensive chest physiotherapy and antibiotics, the patient died 6 weeks later with bronchopneumonia.
DISCUSSION

The pathophysiology of acute gastric dilatation is well known and has been reviewed [1]. We have been unable to find any reference to this problem occurring in an incarcerated hiatus hernia in the postoperative period. In its usual position, rapid distension of the stomach leads to mucosal congestion and bleeding, with considerable third space fluid sequestration. In a Type II or paraoesophageal hiatus hernia, there is a defect in the phreno-oesophageal membrane, permitting a peritoneal sac to enter the thoracic cavity. If acute dilatation occurs in this case, expansion of the stomach would be limited by the borders of the mediastinum. It could affect cardiac output by reducing ventricular filling by direct compression or by interfering with systemic or pulmonary venous return. A visceral–cardiac reflex has been suggested as the cause of haemodynamic alterations in intra-abdominal dilatation [3].

In our patient, the response to diuretic, morphine and aminophylline therapy suggests that there was an increase in left ventricular filling pressure. A more rapid response may have been achieved if the x-ray appearances had been interpreted correctly and a nasogastric tube passed.

The role of nitrous oxide in this case is
debatable. During the first operation there may have been diffusion of nitrous oxide into the incarcerated stomach which already contained a gas bubble as seen on the preoperative x-ray. This could have initiated a sequence of distension, mucosal ischaemia and increased secretion which continued into the postoperative period. The majority of cases of intra-abdominal gastric dilatation are reported to occur in the first few days following surgery [4].

The lessons to be learned from this patient are that a gas bubble in the mediastinum indicates a potential for intrathoracic visceral dilatation in the postoperative period, requiring that a nasogastric tube should be passed and aspirated continuously; and that apparent acute left ventricular failure in patients with an incarcerated hiatus hernia also indicates that a nasogastric tube should be passed as part of the management of the patient.

REFERENCES