CASE REPORT

Seesawing end-tidal carbon dioxide: portent of critical carbon dioxide embolism in retroperitoneoscopy

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SUMMARY

An abrupt increase in end-tidal CO₂ (EtCO₂; from 35 to 58 mm Hg) followed by a sudden fall (to 18 mm Hg) was noted during retroperitoneoscopic adrenalectomy under general anaesthesia in a 23-year-old patient with adrenal hyperplasia. This was accompanied by hypotension (systolic blood pressure of 60 mm Hg), desaturation (88% SpO₂) and ST depression (3.5 mm). The patient was resuscitated with fluids and vasopressor drugs and about 4 mL of air was aspirated through the central venous catheter, confirming the diagnosis of an intraoperative gas embolism. Later, a rent in the adrenal vein extending into the inferior vena cava was discovered and sutured. The blood pressure, EtCO₂, ST segment and pulse oximetry returned to normal after 15 min. This case demonstrates that gas embolism may transpire during retroperitoneoscopic adrenalectomy and an acute rise followed by a sharp fall in EtCO₂ should alert the anaesthesiologist to this rare but potentially fatal complication.

BACKGROUND

Carbon dioxide embolism is an uncommon occurrence in retroperitoneoscopic surgery, unlike in conventional transperitoneal laparoscopy. The most sensitive tool to diagnose intraoperative gas embolism is a transoesophageal echocardiography (TEE), which is relatively expensive, technically complex and necessitates operator expertise. A standard and routine monitor used during general anaesthesia, the capnogram, shows a characteristic pattern during intraoperative

carbon dioxide gas embolism, which may help in early detection of this potentially lethal complication. A high index of suspicion followed by prompt treatment may avert serious complications and mortality due to clinically significant CO₂ embolism.

CASE PRESENTATION

A 23-year-old female patient (weight 82 kg and height 152 cm) presented with progressive weight gain, excessive facial hair growth, hyperpigmentation of the skin over face and limbs for 3 years. She had been evaluated and diagnosed elsewhere with an adrenocorticotrophic hormone (ACTH)-dependent pituitary adenoma and had undergone transnasal trans-sphenoidal excision of the same 7 months prior. Her symptoms had transiently resolved, and then recurred in the past 3 months at which time an MRI of her brain revealed a residual mass in the inferior aspect of the pituitary gland and her biochemical profile was consistent with ACTH-dependent Cushing's syndrome. She was initiated on ketoconazole 200 mg orally two times per day and prednisolone 5 mg orally once daily and referred to neurosurgery for further management. At our centre, she underwent endoscopic trans-sphenoidal radical excision of the residual tumour under general anaesthesia. Intraoperatively, the tumour was removed completely, including a rim of normal adenohypophysis along the periphery of the tumour. On routine follow-up, 3 months later, she was noted to have elevated 24 hours urine cortisol (1125 µg, normal value 10–100 µg/24 hours): she had failed to achieve remission despite pituitary tumour excision and medical management. She was planned for bilateral adrenalectomy via the retroperitoneoscopic approach under general anaesthesia.

INVESTIGATIONS

Her preoperative haemoglobin was 10 g/dL with a mean corpuscular volume (MCV) of 65 fL (normal 80–100 fL), serum sodium 142 mEq/L, serum potassium 3.0 mEq/L, 8 am serum cortisol 19.7 µg%, ACTH 124.0 pg/mL (normal value 0–46), total T4 6.8 µg% (4–12), free T4 concentration was 0.83 mg% (normal 0.8–2), serum creatinine 0.6 mg/dL and normal liver function test results. Her ECG showed normal sinus rhythm with a rate of 80/min, chest X-ray was unremarkable and transthoracic echocardiogram showed normal right and left ventricular function.

Figure 1 Positioning in retroperitoneoscopic surgery: patient was placed in the prone position with hips flexed, anterior abdomen free from pressure and upper limbs flexed at the shoulders and elbows to optimise surgeon's access to the adrenal gland below the 12th rib.

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Table 1 The trends of the main intraoperative parameters

Parameter	Baseline	60 min	90 min	95 min	100 min	105 min	110 min	End of surgery
Heart rate (beats per minute)	96	82	98	99	99	96	99	95
Blood pressure (mm Hg)	140/82	130/68	60/30	64/36	70/38	100/60	110/70	120/76
EtCO ₂ (mm Hg)		30	35	58	18	20	24	34
Oxygen saturation	99	100	NR	88	94	99	100	100
ST segment (lead II)	−0.1	−0.3	−3.5	−3.5	−3.4	−3.1	−1.6	−0.1

NR, not recordable.

EtCO₂, end-tidal CO₂.

TREATMENT

After premedication with oral lorazepam 2 mg and omeprazole 20 mg prior to arrival in the operating theatre, the patient was induced with propofol (2 mg/kg), fentanyl (2 µg/kg) and vecuronium (0.1 mg/kg), along with 100 mg of hydrocortisone for perioperative steroid cover. After placement of the endotracheal tube, anaesthesia was maintained with 0.8–0.9 minimum alveolar concentration (MAC) of isoflurane in a 50% oxygen/air mixture and morphine (up to 0.15 mg/kg) was titrated to optimise haemodynamic response to surgical incision. Intravenous fluids including normal saline and ringer lactate (6–8 mL/kg/hour) were started and adjusted according to clinical requirements. A target of 35–45 mm Hg of end-tidal CO₂ (EtCO₂) was maintained throughout the surgery. Other monitors consisted of five lead ECG, invasive arterial blood pressure (via radial artery cannula), pulse oximetry, MAC, nasopharyngeal temperature, urine output and arterial blood gas analysis. After internal jugular central venous catheter insertion, the patient was carefully positioned in the prone position with the hip flexed 30° for the procedure (figure 1). The arterial blood gas after induction of anaesthesia was unremarkable (pH 7.37, PCO₂ 38 mm Hg, PO₂ 202 mm Hg, bicarbonate 26.4 mmol/L and base deficit −1.8).

The hyperplastic left adrenal gland was removed uneventfully via the retroperitoneal approach before starting the right side adrenalectomy by the same technique. A 10 mm/30° endoscope was used via port site entry at the tip of the 12th rib, and gas insufflation was carried out at 200 mL/min at a pressure of 20–24 mm Hg. The retroperitoneal pressure was maintained at 15 mm Hg using a CO₂ insufflator. The patient remained haemodynamically stable during the left adrenal dissection. About 90 min into the surgery, when the surgeon clamped the right adrenal vein, a sudden rise in the EtCO₂ from 35–58 mm of mercury was noted, followed by an abrupt drop to 18 mm of mercury. This was almost instantly accompanied by an ST segment depression from 0.0 to −3.5 mm and a drop in blood pressure from 120/70 mm Hg to 60/30 mm Hg, without any change in the heart rate as depicted in the table below (table 1).

Figure 2 Capnogram depicting the sudden rise in end-tidal CO₂ followed by an abrupt fall well below normal, characteristic of clinically significant carbon dioxide embolism.

As a gas embolism was suspected, the surgeon was informed promptly. The FiO₂ was increased to 1.0 and the patient was turned to the left lateral and Trendelenburg position. While the hypotension was aggressively treated with a bolus of 500 mL of crystalloid and intermittent boluses of ephedrine (a total of 25 mg was given in incremental doses), simultaneous aspiration via the central venous line was commenced. About 4 mL of air was removed through the central line. Ten minutes later, her blood pressure, ST segment and EtCO₂ normalised. Surgery was resumed and successful retrieval of the adrenals was performed. The patient was extubated in the operating room and monitored in the postoperative care unit for 2 hours, where no neurological deficits were noted.

OUTCOME AND FOLLOW-UP

Her postoperative stay in the surgical ward was uneventful, though some bruising was noted in the right operated site, suggestive of mild bleeding. This was managed conservatively successfully with stable haemoglobin not requiring transfusions. She was discharged from the hospital on the fourth postoperative day and was confirmed to be well at 1 month.

DISCUSSION

Although laparoscopic surgeries are associated with a substantially low morbidity and mortality compared with open procedures, the potential for fatal complications must not be overlooked.¹ Carbon dioxide embolism with clinically significant haemodynamic compromise is a rare, although potentially lethal, phenomenon. It is caused by entrapment of the gas into a solid organ, injured vein or artery, and results in obstruction of outflow from the right ventricle or pulmonary artery.² Carbon dioxide embolism may occur at various steps of laparoscopic surgery: during insufflation to create the pneumoperitoneum

Figure 3 Endoscopic image of the right adrenal vein, just prior to dissection of the tumour off the adjacent segment of the inferior vena cava.

carbon dioxide may directly enter into a blood vessel or solid organ due to misplacement of the Veress needle; lesser volume of gas may be entrained into circulation through an opening in injured blood vessels either in the abdominal wall or at the site of surgery³; direct absorption of carbon dioxide into a venous plexus may result when the rate of gas flow is high resulting in insufflation of gas at a higher pressure than the vascular pressure⁴; this forces the entry of carbon dioxide as bubbles into the circulation and results in a gas lock in the right ventricle, right ventricular outflow tract and pulmonary arteries resulting in ventilation perfusion mismatch.⁵

The reported incidence of carbon dioxide embolism during laparoscopic surgery ranges from 0.001% to 0.59% before the era of TEE.^{6,7} Presently, TEE is the most sensitive technique to detect intravenous gas embolism, as small as 0.1 mL/kg, thus, projecting the incidence of CO₂ embolism from 17.1% to 100% in specific types of laparoscopic procedures.^{8,9} The drawbacks of TEE include high cost, technical complexity, need for continual operator attention and unavailability in every operating room.¹⁰ Nonetheless, clinically apparent CO₂ embolism is associated with a 28% mortality rate.¹¹ Near fatal cases of gas embolism have been reported in retroperitoneoscopic and laparoscopic procedures in the lateral or supine positions.^{12,13} In this case, the suspected CO₂ embolism occurred during posterior retroperitoneoscopic adrenalectomy, due to a rent in the right adrenal vein. Up till now, to our knowledge, this is only the second case reported during this surgical technique.

The clinical scenario in this case was exemplified by a momentary rise in EtCO₂ (from 35 to 58 mm Hg) followed by a fall in EtCO₂ (to 18 mm Hg), with concurrent and marked hypotension and ST depression over a few minutes. Having clinically ruled out a pneumothorax and subcutaneous emphysema, which might also have impaired ventilation in a similar, although slower manner, we considered the possibility of a gas embolism. An abrupt fall in EtCO₂, of this nature, may be caused by a fall in cardiac output, or by an interruption to pulmonary blood flow. The pattern of a quick rise in EtCO₂ followed immediately by a fall of this magnitude prompted a diagnosis of acute massive absorption of carbon dioxide and gas embolism (figure 2). Other case reports also cite a similar pattern of acute rise and then fall of EtCO₂ in CO₂ embolism.^{14,15} The embolism had probably transpired while dissecting the right adrenal vein (figure 3) which is very short and directly enters the inferior vena cava (IVC) from the adrenal gland, as it was discovered later that there was a small rent in the adrenal vein that extended up to the IVC. While retracting the adrenal gland and during dissection the tumour off the IVC near the adrenal vein, there was traction injury to the adrenal vein which was noted to be partly avulsed at the superior junction with the IVC. Normally the high insufflation pressure during the retroperitoneoscopic approach keeps the IVC compressed and does not permit significant bleeding or gas embolism; however, in this case, there was a short period of accidental traction on the site of the rent and that kept it open permitting the embolism. Since it was a junctional partial rent, it was possible to use metal clips to close it and divide the vein distal to the clip obviating the need for intracorporeal suturing. In this case, immediate release of the insufflation pressure, provision of 100% oxygen, implementing the Durant's manoeuvre (Trendelenberg and left lateral position), prompt boluses of fluids and vasopressors and aspiration of air via internal jugular central venous catheter all aided in swift restoration of haemodynamic stability. Although TEE has been established as the most sensitive tool in detection of gas embolism, it may not be available in every operating room. Therefore, the anaesthesiologist must

Learning points

- ▶ Carbon dioxide embolism is a rare but potentially fatal complication of posterior retroperitoneoscopic adrenalectomy.
- ▶ While the expertise and availability of transoesophageal echocardiography to detect this complication may not be ubiquitous, the standard and basic ASA (American Society of Anaesthesiologists) monitor, end-tidal CO₂ (EtCO₂), may help in early detection of this possibly lethal complication, based on a transient and quick rise in EtCO₂ followed by an abrupt fall.
- ▶ Communication between the surgeon and anaesthesiologist at crucial steps of the surgery, vigilant monitoring, a high index of suspicion based on EtCO₂ and haemodynamic fluctuations and prompt institution of appropriate treatment may play a pivotal role in lowering patient morbidity and mortality due to this infrequent complication.

have a high index of suspicion of gas embolism in the specific clinical setting, and must be prepared to recognise and manage this acute event.

Contributors MAA was the anaesthesiologist for the reported case. The operating surgeon was MJP. RJ was instrumental in writing up the case report and preparation of the table and graph.

Competing interests None declared.

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