Case Report

Carbon dioxide embolism during posterior retroperitoneal adrenalectomy

M. Alexeev,[1] **E. Fedorov,**[2] **O. Kuleshov,**[3] **D. Rebrova**[4] and **S. Efremov**[5]

1 Resident, 3 Consultant and Head of Department, 5 Consultant and Professor, Department of Anaesthesiology and Intensive Care, 2 Resident, Department of Endocrine Surgery, 4 Resident, Department of Endocrinology, Saint-Petersburg State University Hospital, St. Petersburg, Russian Federation

Summary

We report a case of massive carbon dioxide embolism associated with injury to the inferior vena cava, during posterior retroperitoneoscopic adrenalectomy. The presenting clinical features were tachycardia, rapid oxygen desaturation and severe respiratory acidosis, without evidence of bleeding. The patient was resuscitated by increasing the fraction of inspired oxygen, administering intravenous fluid and converting to an open procedure to suture the vein. This case demonstrates that gas embolism due to vessel injury during posterior retroperitoneal adrenalectomy may arise without evidence of bleeding, severe hypotension or an abrupt increase in end-tidal carbon dioxide. Using a high carbon dioxide insufflation pressure in the retroperitoneal space enhances visualisation of the surgical field by decreasing small-calibre vessel bleeding. However, it can contribute to, and delay recognition of, carbon dioxide embolism. Knowledge of the clinical features of carbon dioxide embolism, careful monitoring and vigilance for intra-operative surgical challenges can assist with the detection of this rare but potentially fatal complication.

Correspondence to: M. Alexeev
Email: Miha_Decor@mail.ru
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Introduction

Adrenalectomy is the gold standard treatment of pheochromocytoma [1]. Although the surgical technique depends on tumour size, location and blood supply, in recent decades endoscopic approaches have allowed minimally invasive pheochromocytoma resection in appropriate cases [2]. Retroperitoneoscopic adrenalectomy, through either a posterior or lateral approach, has become a widely adopted technique with a good safety profile [3]. However, carbon dioxide (CO_2) insufflation of the retroperitoneal space can be associated with haemodynamic instability and complications related to absorption or embolism of CO_2 [4, 5]. Below, we describe a case of massive CO_2 embolism during posterior retroperitoneoscopic adrenalectomy.

Report

A 54-year-old man with pheochromocytoma of the right adrenal gland was admitted to hospital for an elective adrenalectomy. He had originally presented with headache and intermittent arterial hypertension, with maximal systolic and diastolic blood pressure values of 200 and 100 mmHg, respectively. Computed tomography imaging showed an adrenal neoplasm with 31x30x35 mm dimensions, without invasion into adjacent vessels or organs. Urinary metanephrines were elevated at 7.1 $mmol.l^{-1}$ (normal range: 0.0-3.5 $mmol.l^{-1}$). Doxazosin was commenced for pre-operative alpha adrenoceptor blockade, achieving a target blood pressure below 130/70 mmHg. Beta-blockade was not commenced as the heart rate remained below 80 $beats.min^{-1}$ [6].

Vascular catheters were placed into the right internal jugular vein and left radial artery before the induction of anaesthesia. General anaesthesia was induced with 2 $mcg.kg^{-1}$ fentanyl, 2 $mg.kg^{-1}$ propofol and 0.6 $mg.kg^{-1}$ rocuronium i.v. and maintained with sevoflurane at an end-tidal concentration of 2.1%, delivered in 50% oxygen at a fresh gas flow of 2 $l.min^{-1}$. Intra-operative monitoring included electrocardiography, pulse oximetry, capnography, oesophageal temperature measurement, arterial blood gas analysis and processed electroencephalogram (pEEG). During the retroperitoneal stage, the end-tidal CO_2 concentration was 4.7-6.0kPa. Pulse contour cardiac index (PCCI) monitoring through transpulmonary thermodilution was monitored using the PiCCOplus system (Pulsion Medical Systems, Munich, Germany). Haemodynamic support was provided with noradrenaline 0.05 $mcg.kg^{-1}.min^{-1}$ during induction and until surgery. During mobilisation of the adrenal gland, noradrenaline was discontinued and glyceryl trinitrate (titrated up to 1.49 $mcg.kg^{-1}.min^{-1}$) and esmolol (titrated up to 26 $mcg.kg^{-1}.min^{-1}$) were administered to maintain a mean arterial pressure below 100 mmHg. After 105 min of surgery, during surgical dissection of the pheochromocytoma away from the inferior vena cava, there was a rapid decrease in SpO_2 to 75% and in arterial blood pressure to 80/40 mmHg. The heart rate increased to 118 $beats.min^{-1}$ (sinus tachycardia), and end-tidal CO_2 increased from 5.7 to 8.7 kPa. Trajectories of heart rate, mean arterial pressure and end-tidal CO_2 since the injury are presented in Figure 1. There were no significant changes in pEEG or oesophageal temperature. The surgeon reported a 3 × 1 mm defect in the inferior vena cava, associated with clipping of the central vein of the right adrenal gland, which extended directly from the inferior vena cava. However, bleeding did not occur due to the tamponade effect of an insufflating pressure of 25 mmHg in the retroperitoneum. Carbon dioxide embolism was strongly suspected when arterial blood gas analysis showed acute respiratory acidosis and severe hypercapnia. The fraction of inspired oxygen was immediately increased to 1.0 and the noradrenaline infusion was increased to 0.1 $mcg.kg^{-1}.min^{-1}$. Haemodynamic and acid–base trajectories are shown in Table 1.

The surgeon performed haemostasis using an endoscopic clamp with a tampon while transferring the patient from the prone to lateral position, and thoracophrenolumbotomy was performed. The clamp was removed and the inferior vena cava was sutured before the adrenalectomy was completed. The minute ventilation was increased from 7 $l.min^{-1}$ to 12 $l.min^{-1}$. The peripheral oxygen saturations remained between 80 and 90% for 10 min before achieving a stable value of 94-97% that coincided with turning the patient lateral and commencing open surgery. The estimated blood loss was 600 ml. Postoperatively, the patient was transferred to the intensive care unit and his trachea was extubated 2 h later. He made a full recovery without any neurological deficit.

Discussion

Carbon dioxide embolism during videoendoscopic surgery is a well-described but rare complication [7]. It is important to differentiate between hypercarbia associated with gradual absorption of insufflated carbon dioxide and true gas embolism. The former is associated with a relatively slow increase in $PaCO_2$ without hypotension or oxygen desaturation. Low minute ventilation, excessive intraperitoneal or retroperitoneal pressure or leakage of carbon dioxide into fascial spaces may be contributary factors. In contrast, carbon dioxide embolism is associated with rapid disturbances in arterial pressure, cardiac

Table 1 Trends in haemodynamics and acid–base status.

Time of measurement	MAP (mmHg)	HR ($beats.min^{-1}$)	CI ($L.min^{-1}.m^{-2}$)	SVRI ($dyn \cdot s.cm^{-5}.m^{-2}$)	pH	$PaCO_2$ (kPa)	PaO_2 (kPa)	SaO_2, %	BE, ($mmol.l^{-1}$)
Pre-induction	104	87	4.8	1720	7.33	5.85	12.27	97.2	-2.1
Induction	64	102	1.7	2988	7.32	6.2	19.99	99.2	-2.1
Mobilisation	134	73	1.9	5482	7.54	4.93	17.87	97	-4.5
Injury of vein	101	114	3.0	2623	7.2	6.8	13.47	97.4	-3.5
3 -min post -injury	59	112	3.2	2213	6.79	26.93	7.05	57.5	-5.2
7 -min post -injury	84	126	4.1	1569	7.05	11.33	9.49	87.3	-6.3
Conversion to open procedure	67	76	3.4	1204	7.11	8.93	21.6	98.5	-7.7
Suture of vein	64	77	3.5	1096	7.31	7.03	63.7	99	0.9
End of anaesthesia	92	85	4.9	1268	7.45	4.61	18.53	98.2	0.4

SVRI: system vascular resistance index; MAP: mean arterial pressure; HR: heart rate; CI: cardiac index; BE: base excess

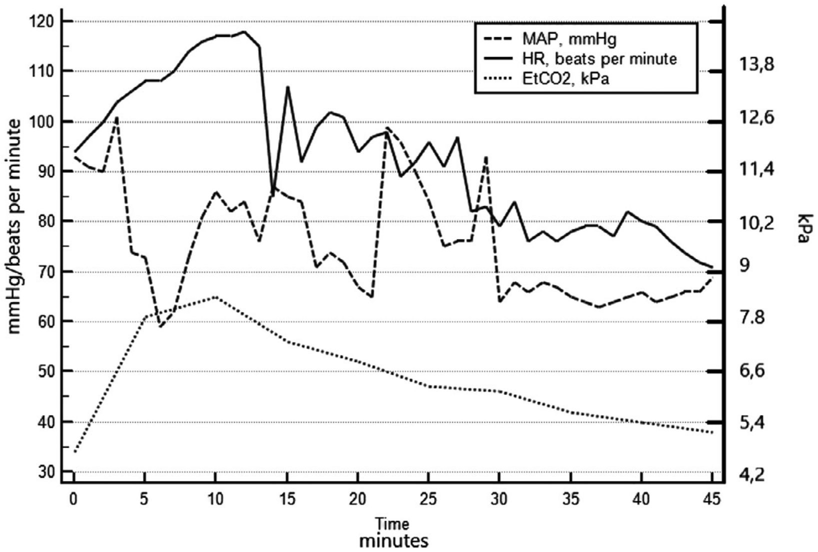

Figure 1 Trajectories of heart rate, mean arterial pressure and end-tidal CO_2. MAP - mean arterial pressure; HR - heart rate; EtCO2 - end-tidal CO_2

rhythm, oxygenation and acid–base status. Rapid CO_2 ingress into a vessel may occur following vascular puncture during needle insertion or through entry into low-pressure vessels during the intervention [4,8] Figure 1.

This clinical case is notable because CO_2 embolism occurred after a defect was created in the inferior vena cava, which is a relatively high-pressure vein. However, this did not lead to the development of clinically significant bleeding during the videoendoscopic stage of the procedure because the pressure in the vena cava (typically 10-15 mmHg) is exceeded by the pressure generated in the retroperitoneal space during this type of surgery (20–25 mmHg) [9]. The first symptom of gas embolism was a decrease in the oxygen saturation, followed by tachycardia and an increase in end-tidal carbon dioxide measurement. The use of a high insufflation pressure in the retroperitoneal space enhances visualisation of the surgical field by decreasing small-calibre vessel bleeding due to compression. However, it can contribute to gas embolism and creates a diagnostic challenge due to the apparent absence of bleeding [9].

Lethal cases of gas embolism are thought to arise due to the accumulation of gas bubbles in the right ventricle, leading to a reduction in cardiac output and arrhythmia [7,10]. Management includes left lateral positioning of the patient, attempted aspiration of gas through the central venous catheter and ventilation with 100% oxygen [10]. However, this manipulation is more relevant for air embolism, as air has much lower blood solubility than carbon dioxide [11]. Aspiration of CO_2 through a venous catheter and immediate lateralisation of the patient were not attempted in this case. The clinical signs of embolism regressed with supportive therapies within 5 min of onset. Arterial blood gas analysis showed a severe respiratory acidosis combined with a relatively mild metabolic component. Significant alterations in cardiac output, preload and vascular resistance were not observed with haemodynamic monitoring.

In addition to the haemodynamic effects of CO_2, a possible reason for the reduction in blood pressure during this event may be the coincidence of CO_2 embolism and clipping of the blood supply to the adrenal gland, with concomitant reduction in the secretion of endogenous catecholamines.

In conclusion, CO_2 embolism is a rare but potentially fatal complication of videoendoscopic procedures. In the context of posterior retroperitoneoscopic pheochromocytoma resection, it is important to understand the reason behind the embolism and potential clinical consequences. High retroperitoneal pressure may conceal bleeding due to vascular compression. Fluctuations in arterial pressure, cardiac output and heart rhythm may be anticipated by the anaesthetist during the operation for other reasons including the release of catecholamines, use of antihypertensive drugs, vascular injury and adrenalectomy leading to relative adrenal insufficiency. Teamwork between surgeons and anaesthetists in adrenal surgery is essential. Knowledge of the course of posterior retroperitoneoscopic adrenalectomy and the presenting features of CO_2 embolism can assist with the detection of this rare but potentially fatal complication. Timely recognition and treatment may be life-saving.

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