

Case Report

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Near Fatal Carbon Dioxide Embolism during Laparoscopy and its Successful Aspiration Using Ultrasound Guided Catheter

Naveen Aggarwal¹, Monica Gupta¹ and Surjya Prasad U^{2*}

¹Department of Anaesthesia, Al Salam International Hospital, Kuwait

²Department of Anaesthesia, NMC Hospital DIP, Dubai

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***Corresponding author:** Dr. Surjya Prasad Upadhyay; MD, Specialist Anaesthesiology, NMC Hospital DIP; Dubai investment park, Dubai, UAE, E-mail: run77in@yahoo.com

Abstract

Significant carbon dioxide embolism is an extremely rare but potentially fatal complication during laparoscopic surgeries. Until now, carbon-Di-oxide (CO₂) is the agent of choice to create the pneumoperitoneum; consequently, there is inherit risk of CO₂ embolism. Clinical symptoms of this embolism may be asymptomatic to cardiovascular collapse to neurological injury depending on the amount and rate of carbon dioxide absorption into body. Here we describe a case of near fetal gas (CO₂) embolism in a morbidly obese patient undergoing laparoscopic cholecystectomy.

Keywords: Carbondioxide embolism; Laparoscopy; Cholecystectomy; Carboperitoneum

Abbreviations: ASA: American Society of Anaesthesiology; BMI: Body Mass Index; INR: International Normalisation Ratio; IPPV: Intermittent Positive Pressure Ventilation; TOF: Train Of Four; CPR: Cardiopulmonary Resuscitation; ROSC: Return of Spontaneous Circulation; TEE: Transesophageal Echocardiography

Introduction

Laparoscopy has become a standard of care for most intra-abdominal diagnostic and therapeutic procedures as it has many advantages such as more rapid return to daily activities, less post-operative pain, shorter hospital stay, and significant cost savings. Until now, carbon-Di-oxide (CO₂) is the agent of choice to create the pneumoperitoneum; consequently, there is inherit risk of CO₂ embolism. Significant carbon dioxide embolism is an extremely rare but potentially fatal complication during laparoscopic surgeries [1-3]. The magnitude or severity of symptoms depend on the rapidity and volume of gas (CO₂) entry into the venous circulation. Large rapid entry of CO₂ can leads to predictable chain of pathophysiological events which may continue to cardiovascular collapse and death. Depending on the amount and rapidity of CO₂ absorption, symptoms vary from hypercapnia, hypoxemia, decreased in end tidal CO₂, arrhythmias, myocardial ischemia, cardiac arrest. Most cases of CO₂ embolism occurs due to its inadvertent injection of CO₂ directly into a vessel (large vein, artery) or very vascular solid organs during the initial insufflation

phase of creating pneumoperitoneum. We encountered a case of unanticipated massive gas embolism (CO₂) without many of the usual clinical features during the dissection phase in laparoscopic cholecystectomy.

Case Report

A 25 year super morbidly obese female weighting 110 kg, height of 158 cm (body mass index [BMI] of 44 kg/m²) and American Society of Anaesthesiology (ASA) physical status of 3 was posted for laparoscopic cholecystectomy. There was no other obesity associated medical problems except for occasional snoring but no other suggestive history of Obstructive Sleep Apnea. General and physical examination including airway assessment was unremarkable except for super morbid obesity. Routine preoperative laboratory investigation (complete blood count, serum creatinine, blood glucose) were within normal range. There were mild elevation of hepatic enzymes but prothrombine time and international normalisation ratio (INR) were within normal range.

General Anaesthesia (GA) with endotracheal intubation was planned. She was premedicated with ranitidine 50 mg along with 10 mg of metoclopramide intravenously (IV), 45 minutes prior to induction of GA. On arrival to preoperative holding area, 2 mg of intravenous midazolam was administered.

All gadget and equipment to manage super morbid obese were kept ready including difficult airway cart. Patient was induced on the operating table in ramped position. After applying the standard monitors (pulse oxymetry, non-invasive blood pressure, electrocardiogram [ECG]) and preoxygenation with 100 % O₂ for 3 minutes; GA was induced with fentanyl 150 microgram, followed by propofol 200mg; after confirming the ability to ventilate with bag-mask, 40mg of atracurium was administered to facilitate endotracheal intubation. The patient was intubated using portex cuffed 7.5mm orotracheal tube and confirmed with waveform capnography and presence of bilateral breath sound on auscultation. Maintenance of anesthesia was done by 60% oxygen in air with sevoflurane (end tidal 2%), and remifentanyl infusion @100-400 microgram/hour. Intermittent positive pressure ventilation (IPPV) done with tidal volume of 400 ml, respiratory rate of 18/minute and PEEP of 6cm water. Apart from capnography, temperature and neuromuscular in the form of Train of Four (TOF) monitoring was initiated soon after endotracheal intubation.

Surgery started with creation of pneumoperitoneum by CO₂ insufflation through varess needles targeting intraabdominal pressure of 15cm water; trocars were passed through the standard port under direct intraperitoneal vision. Patient remained stable during the gas insufflation and introduction of trocars; gall bladder was identified, operating table adjusted for proper surgical access. After clipping the cystic duct and artery; during the dissection of gall bladder from the liver bed, surgeon noticed profused oozing from the gall bladder bed; within a minute there was sudden drop in end tidal carbon dioxide from 37 to 7mmHg, the patient became cyanosed with saturation (SpO₂) below 50%, and heart rate dropped from 96/minute to 40/minute with very faint brachial pulse on palpation. Blood pressure recheck revealed a mere systolic reading of 60mmHg without recordable diastolic. Immediately called for extra help, inspired oxygen concentration increased to 100% and ventilator switch to manual mode; Surgeons were immediately notified and asked to hold the surgery and to release the carboperitoneum. A quick check for airway and circuit connection did not revealed any malfunction; a provision diagnosis of Carbon di-oxide embolism was made; rapid boluses of iv fluid flushed, decided to put the patient in left lateral position to prevent CO₂ lodging in the pulmonary artery. While attempting to give left lateral (Durant position), patient went into cardiac arrest (asystole in the ECG trace, with no palpable pulse). Immediately patient was repositioned supine, chest compressions started and asked for iv epinephrine 1mg. While Cardiopulmonary resuscitation (CPR) was ongoing, right internal jugular vein was cannulated quickly using ultrasound guidance and a triple lumen

central venous catheter inserted up to 20cm; immediately, all the three ports of central line were aspirated for gas. Approximately, a total of 25ml of gas was aspirated. Following aspiration of gas, CPR continued for another two cycles before getting the return of spontaneous circulation (ROSC). CPR lasted about 13min with four dosage of epinephrine (1mg each). Finally CPR terminated with ROSC with a heart rate of 130/minute, blood pressure of 139/86 mmHg and SpO₂ was 92%. Oxygen saturation slowly improved to 98% over 15min after increasing the PEEP to 10 cm of water. Arterial blood gas immediately after ROSC revealed mild respiratory acidosis with pH7.21, PaCO₂ 53 mmHg, PaO₂ 78 mmHg, HCO₃- 20 mmol/l. Blood glucose, electrolytes were all within normal limits.

Following successful resuscitation, it was decided for laparotomy and proceed with open cholecystectomy, surgery completed with surgicel (absorbable haemostat) pack in gall bladder bed and a surgical drain. After completion of surgery, patient was transfer to intensive care unit (ICU) for further management. Patient remained haemodynamically stable in ICU with normalization of ABG with adequate urine output. After 2 hours of ICU transfer, once patient started breathing on her own and followed commands, she was successfully extubated on facemask and was kept overnight for ICU monitoring. Further hospital course was unremarkable, the patient was discharged home after 6 days without any sequel.

Discussion

Incidence of clinically significant fatal Carbon dioxide embolism is a rare complication of laparoscopic surgeries (0.0014-0.6%) but with a high mortality of 28% [3]. There have been reports of carbon dioxide embolism occurring in various laparoscopic procedures [4-10].

Although, CO₂ embolism can occur at any time of surgery but majority of fatal embolism occur during initial insufflation of peritoneum due to inadvertent injection of CO₂ directly into a vessel (large vein, artery) or very vascular solid organs [1-3,9,11]. In our case, it was during the resection phase, there was a surgical raw area at the base of gall bladder and gas was entrained from there. Rapid entrapment of CO₂ in our case might be favored by high intraperitoneal insufflation (flow rate 6 l/min) to achieve a higher intra-abdominal pressure (15cm water).

Clinical manifestation of CO₂ embolism depends on the amount rapidity of gas entering into the venous system. Slow entrainment of gas results in small emboli of CO₂ and the clinical effects depends on the balance between the volume of gas entering and remove from the venous system. CO₂ entrapment exceeding 1ml/kg/min may be fatal in majority of cases, which may occlude pulmonary circulation, whereas rapid embolization of a large volume of gas (Gas lock) causes right ventricular outflow tract obstruction and leads to cardiovascular collapse [11,12]. Clinically, CO₂ embolism can present as haemodynamic changes in the form of bradycardia, tachycardia, hypotension, arrhythmias and respiratory changes

that include hypoxemia (desaturation) with increase or decrease in end tidal CO₂ tension. A “mill-wheel” murmur can also be auscultated [2,3]. High intra-abdominal pressure and patient position further exaggerate the haemodynamic alteration due to massive CO₂ embolism. Peritoneal insufflation below 12cm water may decrease the incidence of significant CO₂ embolism [13].

Sub-clinical and minor gas embolism during laparoscopy can be detected early by using invasive monitors like Transesophageal echocardiography (TEE), esophageal doppler, esophageal and precordial stethoscope which are more sensitive and allow for early detection of even minor embolism [2,14-17]. However, routine use of such invasive monitoring in laparoscopic procedures are not indicated as the incidence of significant CO₂ embolism is very rare.

Capnography provides or a valuable tool for early diagnosis of gas embolism [14]. End tidal CO₂ (ETCO₂) monitoring has been suggested as a sensitive and non-invasive means of detecting gas embolism [14,15]. CO₂ embolism can cause either increase or decrease of ETCO₂ depending on amount and rapidity of gas embolism [3]. Small transient rise in ETCO₂ may be seen due to increase in the dissolved CO₂ in blood. Significant CO₂ embolism is detected by sudden decrease in ETCO₂ along with haemodynamic changes and desaturation [2,3] although, other mechanical causes like endotracheal tube kinking, malposition, pneumothorax should also be ruled out as we did in our case.

The prognosis of these fatal complication depends on early detection and immediate intervention in order to prevent further gas entry, reduction in the volume of gas entrained and haemodynamic support. Series of interventions are very crucial for successful resuscitation. First of all, immediate cessation of insufflation with decompress carboperitoneum should be done. Patient should be ventilated with 100% oxygen to improve hypoxemia and wash out of CO₂. Aggressive IV fluid boluses to increase central venous pressure to prevent further embolism of gas [18]. The patient may be placed in steep head down left lateral position (Durant maneuver) and this position relieved right ventricular outflow obstruction by emboli and also resumption of pulmonary blood flow [15]. Multi-orifice central venous or pulmonary artery catheter may be inserted for confirming diagnosis and may allow aspiration of gas from the right heart, thereby relieving the gas lock status [15,19,20]. Data from experimental laboratory has shown that during venous air embolism, air remained more on the right ventricle for a long period (average 12 min) [21]. We inserted the central line fully (up to 20 cm mark) and was able to aspirate gas from all the three port. Direct aspiration of gas from both right atrium and ventricle may be best treatment for immediate improvement in the haemodynamic parameters and this can be done rapidly using ultrasound guided placement of multilumen catheter through right internal jugular vein. This intervention was the most critical in our case and without this we might had lost the patient.

Ultrasound guidance (USG) during central line insertion is a real help as it reduces the procedural time, number of attempt, decrease the failure rate and complications [22-25]. Use of USG in emergency central venous access has been included as the core or primary emergency ultrasound application by American College of Emergency Physician (ACEP) [26]. Although, there is no current guideline or recommendation for emergent central venous catheter placement for aspiration of gas during an acute setting of gas embolism with haemodynamic instability, it may be very useful treatment when clinical condition deteriorate rapidly despite other resuscitative measures.

In our case, prompt clinical diagnosis based on rapid cardiovascular collapse, with decrease in end tidal carbon dioxide (ETCO₂) with desaturation and timely interventions using ultrasound guided emergency placement of central line and aspiration of CO₂ helped in successful resuscitation.

Conclusion

Carbon dioxide embolism is an inherent risk of laparoscopic surgeries, it can happen at any point of time during the laparoscopic procedures. Continuous vigilance and prompt interventions is the key to successful resuscitation. Ultrasound may be a great tool in immediate placement of central venous catheter and direct aspiration of gas from the heart in massive embolism may be the most effective intervention in haemodynamically compromised patient.

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