Carbon dioxide embolism during laparoscopic sleeve gastrectomy

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Abstract

Bariatric restrictive and malabsorptive operations are being carried out in most countries laparoscopically. Carbon dioxide or gas embolism has never been reported in obese patients undergoing bariatric surgery. We report a case of carbon dioxide embolism during laparoscopic sleeve gastrectomy (LSG) in a young super obese female patient. Early diagnosis and successful management of this complication are discussed. An 18-year-old super obese female patient with enlarged fatty liver underwent LSG under general anesthesia. During initial intra-peritoneal insufflation with CO_2 at high flows through upper left quadrant of the abdomen, she had precipitous fall of end-tidal CO_2 and SaO_2% accompanied with tachycardia. Early suspicion led to stoppage of further insufflation. Clinical parameters were stabilized after almost 30 min, while the blood gas analysis was restored to normal levels after 1 h. The area of gas entrainment on the damaged liver was recognized by the surgeon and sealed and the surgery was successfully carried out uneventfully. Like any other laparoscopic surgery, carbon dioxide embolism can occur during bariatric laparoscopic surgery also. Caution should be exercised when Veress needle is inserted through upper left quadrant of the abdomen in patients with enlarged liver. A high degree of suspicion and prompt collaboration between the surgeon and anesthetist can lead to complete recovery from this potentially fatal complication.

Key words: Bariatric surgery, carbon dioxide embolism, sleeve gastrectomy

Introduction

Bariatric laparoscopic surgery is being increasingly performed all over the world, with the overweight and obese population out-numbering the malnourished people. Both restrictive and malabsorptive procedures are performed at our institute laparoscopically but laparoscopic sleeve gastrectomy (LSG) is the most common procedure performed at present.

Carbon dioxide (CO_2) or gas embolism during laparoscopic surgery has been reported for cholecystectomy,[1-4] radical prostatectomy,[5] hysterectomy[6] and even during pediatric laparoscopic procedures.[7-9] We did not come across a single case report of gas embolism during laparoscopic bariatric surgery through Medline search on 9 July 2010. We are reporting this unusual but potentially fatal complication of CO_2 embolism during LSG at our institute, where over 1500 LSG have been carried out in the past 2 years.

Case Report

An 18-year-old female patient with body weight 145 kg, height 164 cm, and body -mass index (BMI) 53.9 was posted for LSG. Preoperatively, her blood pressure was 118/68 mmHg, pulse 70/min, temperature 36.8°C, and she had a history of occasional attacks of bronchial asthma (seasonal). Her preoperative SaO_2% on room air was 99%, and there was no history suggestive of obstructive sleep apnea. Her pre-operative investigations did not reveal any abnormality except a large fatty liver. She was pre-medicated with omeprazole 40 mg IV and metoclopramide 10 mg IV 2h before surgery and midazolam 2 mg IV was administered in the receiving area of the operation theatre. She had received 3500 IU of tinaparin 2 h earlier. Graduated compression stockings were applied before the surgery. Ceftriaxone 1000 mg was administered intravenously an hour prior to the surgery.

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On arrival to the operation room, the monitors were attached and she was pre-oxygenated for 2 min. Anaesthesia was induced, with the patient in the 30° anti-trendelenberg position, with IV fentanyl 100 μg, IV lignocaine 60mg, IV propofol 200 mg, and cisatracurium 18 mg. Her trachea was intubated with a 7 mm ID oro-tracheal cuffed Portex tube. Anaesthesia was maintained with 80% oxygen, sevoflurane (end-tidal 2%), alfentanil infusion, and intermittent doses of cisatracurium, as required. The lungs were ventilated with tidal volume of 500 ml, at the rate of 14-18/min and PEEP of 7 cm of H_2O using a low flow technique. ECG, NIBP, SaO_2%, end-tidal CO_2, nasopharyngeal temperature, and TOF were monitored. She received 2000 ml of lactated Ringer's solution and 500 ml of 6% hydroxyl-ethyl starch (Haesteril 6%, Fresenius Kabi); during anaesthesia which lasted for 120 min.

Almost 20 min after induction of anaesthesia, when the patient was positioned for surgery, fentanyl 50 μg IV bolus was administered while the surgeon introduced the Veress needle through the upper left quadrant of the abdomen in the supine position. The intra-peritoneal insufflation using CO_2 was started with high flows to achieve intra-abdominal pressure of 20 mmHg. Within less than 1 min, the end-tidal CO_2 dropped from 35 to 9 mmHg, the patient was cyanosed and SaO_2% dropped to 67%. Her heart rate increased to 130/min from 78/min. However, her arterial blood pressure was maintained at 128/78 mmHg. The airway pressure was between 32 and 35 cm of H_2O.

Gas embolism was suspected and the surgeon was informed immediately. FiO_2 was increased to 1, fresh gas flow increased to 8 L/min, sevoflurane stopped, and manual bag ventilation was carried out. The surgeon stopped the insufflation. Tredelenburg position was given immediately and intravenous 6% hydroxyl-ethyl starch was infused fast to raise the venous pressure to prevent further entrainment of the gas. Within 5 min SaO_2% increased to 100%, and end-tidal CO_2 increased to 25 mmHg and later to 42 mmHg. The auscultation of chest revealed bilateral good and equal air entry, which excluded pneumothorax, and no mill-wheel murmur was detected. The pupils were bilaterally equal, small and reacting well to light. When heart rate dropped to 110/min, sevoflurane and alfentanil infusion 500 μg/h were resumed. Arterial blood gas analysis 15 min after the insult showed respiratory acidosis: pH 7.24, PCO_2 67mmHg, PO_2 220 mmHg, HCO_3 28.5 mmol/L. FiO_2 was decreased to 0.7 at this stage.

After almost 30 min, after the patient was stabilized, the surgeon directed the Veress needle more caudad and insufflated at lower flows to achieve the pressure of 10 mmHg under vision. A small hole on the anterior surface of the left lobe of the liver could be seen from where the gas was being entrained [Figures 1 and 2]. The surgeon covered this bleeding hole with a wet gauze [Figures 3 and 4] and applied pressure laparoscopically and later sealed it with surgicel. Since there was no further entrainment of the gas, he carried out the surgery uneventfully.

The arterial blood gas analysis after 1 h from the insult showed complete recovery: pH 7.36, PCO_2 42 mmHg, PO_2 147 mmHg, HCO_3 23.2 mmol/L; though the airway pressure maintained between 32 and 35 cm of H_2O and the heart rate was consistently around 110/min throughout the surgery.

At the end of the operation the neuromuscular blockade was reversed with atropine 1mg and neostigmine 2.5 mg. The trachea was extubated when the $T_1:T_4$ ratio reached 87% and she was completely awake.

The patient stayed in the PACU (post anaesthesia care unit) for 2 h under complete monitoring and observation. She was vitally stable. Blood pressure was 140/80 mmHg, SaO_2% was 100% on O_2 6 L/min through Hudson's mask,

Figure 1: A hole on the anterior surface of the left liver lobe

Figure 2: Gas being entrained from the hole on the anterior surface of the left lobe of liver

Figure 3: Wet gauze being applied on the damaged liver

Figure 4: Wet gauze being applied on the damaged liver

and 96% on room air. The pulse rate was 90-110/min and the respiratory rate was 16-18/min. Pain was relieved by IV infusion of paracetamol (Perfalgan, Bristol-Myers Squibb Pharmaceuticals Ltd) 1G and small boluses of IV pethidine.

The patient was shifted to the ward in stable condition under observation of vital signs and SaO$_2$% in the ward. O$_2$ 6 L/min through Hudson's facemask was given for 24 h. Incentive spirometry and chest physiotherapy were begun within 4 h after the surgery. The patient was discharged home after 2 days without any complications.

Discussion

Many different gases have been used for intra-peritoneal insufflation for laparoscopic surgery, namely air, oxygen, CO$_2$, nitrous oxide, argon, and even helium though none has

proved ideal. CO$_2$ is cheap, does not support combustion, is non-irritant to the peritoneum, and has a high margin of safety due to its greater solubility (20:1 to oxygen and 25:1 to nitrogen), thus diffusing rapidly across the membrane to get excreted rapidly.

Intra-peritoneal insufflation of CO$_2$ can cause four principal respiratory complications: CO$_2$ subcutaneous emphysema, pneumothorax, endobronchial intubation, and gas embolism.[10] Although rare, gas embolism is the most feared and dangerous complication of laparoscopy. Incidence of gas embolism during laparoscopy is reported to be 15 per 100,000 cases per year.[11]

Gas embolism may occur due to intravascular injection of gas that follows direct needle or trocar placement into a vessel, or may occur as a consequence of gas insufflation into an abdominal parenchymal organ. This complication develops principally during the induction of pneumoperitoneum, particularly in patients with previous abdominal surgery. During cholecystectomy it usually occurs at a later stage when the base of the gall bladder is being dissected.[1-4] In pediatric surgery and in young adults, it can result from injection into a patent umbilical or paraumbilical vein.[7-9] Perhaps, due to very large amount of subcutaneous and intraperitoneal fat, no instance of Veress needle entering any blood vessel has been described.

In the above case report, CO$_2$ was entrained from the damaged liver parenchyma since the intra-abdominal pressure limit was set at a high level of 20 mmHg. The patient had a large fatty liver and it probably got damaged during the insertion of Veress needle. Slow entrainment of gas results in small emboli in the venous blood, which lodge more distally in the pulmonary circulation. Rapid entrainment of a large volume of gas can lead to large emboli formation, which may lodge in a large central vessel and even lead to cardiovascular collapse.[12] Our patient did not develop any cardiovascular collapse, as the entrainment of the gas was slow. Immediate cessation of insufflation on strong suspicion of gas embolism prevented further entrainment facilitating an uneventful recovery. During rapid embolization, gas under high pressure can cause a "gas lock" in the vena cava, right atrium, or pulmonary artery obstructing the venous return and result in a fall in cardiac output and circulatory collapse. Acute right ventricular hypertension may open the foramen ovale, allowing paradoxical gas embolization. Volume preload diminishes the risk of gas embolism and of paradoxical embolism.[13] Ventilation-perfusion (V/Q) mismatch develops from the increase in physiologic dead space and hypoxemia.

The low incidence of gas embolism during laparoscopy precludes

the routine use of invasive or expensive monitors to detect embolization of small quantities of gas. Capnometry and capnography are more valuable in providing early diagnosis of gas embolism and determining the extent of the embolism. As little as 0.25 to 0.5 ml/kg/min of gas entrainment in the venous circulation can be detected by a fall in the end tidal CO$_2$.[14] Pressure of EtCO$_2$ (PEtCO$_2$) decreases in the case of embolism owing to the fall in cardiac output and the enlargement of the physiologic dead space. Consequently, Δa-EtCO$_2$ increases. The severity of fall of EtCO$_2$ is related to the proportion of air present in the heart in relation to body weight.[15] The decrease in PEtCO$_2$ may be preceded by an initial increase secondary to pulmonary excretion of the CO$_2$, which has been absorbed into the blood.[16] Aspiration of gas or foamy blood from a central venous line establishes the diagnosis. Routine preoperative insertion of a central venous line, however, is not justified for these procedures.

Treatment of CO$_2$ embolism consists of immediate cessation of insufflation and release of the pneumoperitoneum. The patient is placed in steep head-down and left lateral decubitus position (Durant's maneuver). The amount of gas that advances through the right side of the heart to the pulmonary circulation is less if the patient is in this position because the buoyant foam is displaced laterally and caudally away from the right ventricular outflow tract. Discontinuing N$_2$O will allow ventilation with 100% O$_2$ to correct hypoxemia and reduce the size of the gas embolus and its consequences. Use of N$_2$O after 30 min after even air embolism is considered safe[17,18] but we did not use N$_2$O. We used 70% oxygen in air after the EtCO$_2$ and PaO$_2$ returned to normal values. In case the physiologic dead space increases, hyperventilation may help by increasing CO$_2$ excretion. If these simple measures are not effective, a central venous or pulmonary artery catheter may be introduced for aspiration of the gas.[16] Hyperbaric oxygen has been used in a few patients with some success.[12]

To summarize, CO$_2$ embolism can occur during bariatric laparoscopic surgery, like in any other laparoscopic surgery. Caution should be exercised when Veress needle is inserted through upper left quadrant of the abdomen in patients with enlarged liver. A high degree of suspicion and prompt collaboration between the surgeon and anaesthetist can limit morbidity in this potentially fatal complication.

References

1. Scoletta P, Morsiani E, Ferrocci G, Maniscalpo P, Pellegrini D, Colognesi A. Carbon dioxide embolization: Is it a complication of laparoscopic cholecystectomy? Mierva Chir 2003;58:313-20.
2. Wenham TN, Graham D. Venous gas embolism: An unusual complication of laparoscopic cholecystectomy. J Minim Access Surg 2009;5:35-6.
3. Khan AU, Pandya K, Clifton MA. Near Fatal gas embolism during laparoscopic cholecystectomy. Ann R CollSurg Eng 1995;77:67-8.
4. Abut YC, Eryilmaz R, Okan I, Erkalp K. Venous air embolism during laparoscopic cholecystectomy. Minim Invasive Ther Allied Technol 2009;18:366-8.
5. Hong, Fischler M. Detection of subclinical Carbon dioxide embolism by transesophageal echocardiography during laparoscopic radical prostatectomy. Urology 2010;75:1240-1.
6. Dubar G, Fischler M. Venous air embolism during total laparoscopic hysterectomy. Anaesthesiology 2010;112:497-8.
7. Lalwani K, Aliason I. Cardiac arrest in the neonate during laparoscopic surgery. Anesth Analg 2009;109:760-2.
8. Kudsi OY, Jones SA, Brenn BR. Carbon dioxide embolism in a 3 week old neonate during laparoscopic pylorotomy: A case report. J Pediatr Surg 2009;44:842-5.
9. MatteiP, Tyler DC. Carbon dioxide embolism during laparoscopicholecystectomy due to ptentpara umbilical vein. J Pediatr Surg 2007;42:570-2.
10. Sprung J, Whalley DG, Falcone T, Warner DO, Hubmayr RD, Hammel J. The impact of morbid obesity, pneumoperitoneum, and posture on respiratory system mechanics and oxygenation during laparoscopy. Anesth Analg 2002;94:1345-50.
11. Orebaugh SL. Venous air embolism: Clinical and experimental observation. Crit care Med 1992;20:1169-77.
12. McGrath BJ, Zimmerman LE, Williams JF. Carbon dioxide embolism treated with hyperbaric oxygen. Can J Anesth 1989;36:586-91.
13. Tuppurainen T, Makinen J, Salonen M: Reducing the risk of systemic embolization during gynecologic laparoscopy-effect of volume preload. Acta Anaesthesiol Scand 2002;46:37-42.
14. Kirby RR. Respiratory System. In: Gravenstein N, editors, Manual of Complications during Anesthesia. Philadelphia: JB Lippincott Company; 1991. p. 302-52.
15. Capnography KZ. A sensitive method of early detection of air embolism. Acta Anaesthesiol Belg 1975;23:78-85.
16. Morgan GE, Mikhail MS, Murray MJ. Carbon Dioxide is cheap, does not support combustion-Clinical Anesthesiology. 4th Ed, New York: McGraw-Hill; 2006. p. 582-4.
17. Lasasso TJ, Muzzi DA, Dietz NM, Cucharia RF. Fifty percent nitrous oxide does not increase the risk of venous air embolism in neurosurgical patients operated upon in sitting position. Anesthesiology 1992;77:21-30.
18. DeSousa K, Apte J. Complications in Patients undergoing Posterior Fossa Craniectomy in a Modified Recumbent position. Indian J Anaesth 2001;45:205.