Scholars Journal of Medical Case Reports

OPEN ACCESS

Abbreviated Key Title: Sch J Med Case Rep
ISSN 2347-9507 (Print) | ISSN 2347-6559 (Online)
Journal homepage: https://saspublishers.com

Severe Carbon Dioxide Retention during Laparoscopic Surgery: A Case Report

Ilyass Masad[1,2*], Atmani Walid[1,2], Anass Elbouti[1,2], Jaafari Abdelhamid[1,2], Mustapha Bensghir[1,2]

[1]Anesthesiology & intensive care department, Military Teaching Hospital Mohammed V of Rabat, Morocco
[2]Faculty of Medicine, Mohammed V University, Rabat, Morocco

DOI: 10.36347/sjmcr.2021.v09i05.020 | **Received:** 25.03.2021 | **Accepted:** 04.05.2021 | **Published:** 20.05.2021

***Corresponding author:** Ilyass Masad

Abstract **Case Report**

Excessive CO2 absorption, severe hypercapnia and acidosis may been countered in patients undergoing laparoscopic surgery. We have witnessed a patient, free of cardiac or pulmonary disease, undergoing laparoscopic surgery, who sustained severe hypercapnia and respiratory acidosis during operation.

Keywords: Carbon Dioxide Retention Carbon Dioxide Retention.

INTRODUCTION

The CO2 pneumoperitoneum induces hemodynamic, pulmonary, renal, splanchnic and endocrine pathophysiological changes. Most of them are not clinically significant if appropriate anesthetic care is provided. In some cases complications can develop depending on intra-abdominal pressure, the amount of CO2 absorbed, the circulatory volume of the patient [1]. In fact, Hypercapnia and acidosis are two physiological changes in response to carbondioxide (CO2) pneumoperitoneum during laparoscopic surgery. Inflation of the abdominal cavity with CO2 may be associated with pulmonary atelectasis, reduced functional residual capacity, increased airway pressure, increased CO2 absorption from the peritoneum and decreased diaphragmatic excursion caused either by positional change or increased intra-abdominal pressure. We report the case of severe hypercapnia with acidosis related to laparoscopic surgery for a patient with free cardiac and pulmonary diseases.

CASE REPORT

A 43-year-old female patient, admitted to the operating room for laparoscopic hysterectomy, pre-anesthesia evaluation found a patient without medical history, Body mass index at 26 kg/m2, cardiopulmonary evaluation with no abnormality, blood pressure at 120 mmhg systolic and 70 mmhg diastolic, heart rate 73 beats per minute, respiratory rate 14 cycles per minute, pulse saturation 99%, biological assessment without abnormality, chest X-ray and electrocardiogram normal. Patient classed ASA 1.

Admitted to the operating room, the patient was placed in the supine position and monitoring including HR, BP and SPO2 was initiated. Vascular access was provided by a 16G peripheral venous route and saline serum (500ml) vascular filling was done.

Premedication with 2 mg of midazolam, rachianalgesia with 100 µg of morphine and 25 µg of fentanyl, patient underwent general anesthesia with fentanyl 3µg/Kg propofol 2,5mg/Kg and rocuronium0, 6mg/Kg, Tracheal intubation remains easy not selective after auscultation, continuous anesthesia by isoflurane /O2. Laparoscopic hysterectomy was carried out under general anesthesia the aftermath of conditioning included monitoring end-tidal CO2 (EtCO2), and body temperature.

After creation of a pneumoperitoneum by CO2 inflation, Intra-abdominal pressure was kept under 15 mmHg throughout the procedure and EtCO2 rose gradually from 30 to 38 mmHg. Adjustment of controlled hyperventilation by increasing the respiratory rate and tidal volume was made. During the maintenance of CO2 pneumoperitoneum, abnormally high EtCO2 was progressively generated and serial arterial blood gas analyses showed acute respiratory acidosis with insufficient metabolic compensation. The hypercapnia could not be eliminated effectively by adjusting the inspiration to expiration ratio, respiratory rate, tidal volume or positive end expiratory pressure of the mechanical ventilator while the application of CO2 pneumoperitoneum was still underway. The highest EtCO2 value registered was 115 mmHg. The patient

was ventilated with 100% O2 and the SpO2 could be maintained at 100%. Lactated Ringer's solution was given for hydration to achieve sufficient expansion of intravascular volume and adequate urine output. Arterial CO2 partial pressure (PaCO2) had escalated to 71.2 mmHg (Figure 1) during pneumoperitoneum while pH fell to 7.189 with bicarbonate (HCO3−) elevation to 27.3 mmol/L. The PaO2 was acceptable at 224.2 mmHg. The clinical examination does not find subcutaneous emphysema, thus, adjustment of the postion of the patient to reverse Trendelenburg position did not improve hypercapnia. The decision to stop the laparoscopy and the conversion to laparotomy was taken in the absence of improvement.

The patient remained intubated with mechanical ventilatory support after surgery. Excessive internal CO2 was washed out gradually and the patient was extubated successfully after four hours.

DISCUSSION

Laparoscopics surgery has largely replaced open one because of its minimal invasiveness, quicker recovery, markedly lower tissue trauma, and early discharge from hospital with the proviso that no complications occur during laparoscopic surgery [3].

The major factor which causes pathophysiologic changes, principally in hemodynamics and respiration, during laparoscopic surgery is pneumoperitoneum by CO2 inflation.

Both cardiovascular and respiratory systems are the most challenged systems of the human body during laparoscopy. Changes occurring during CO2 pneumoperitoneum result from two main factors: hypercarbia (and the subsequent acidosis) and increased intra-abdominal pressure [1].

In fact, CO2 is highly soluble and therefore is very rapidly absorbed from the peritoneal cavity into circulation. Because absorbed CO2 can only be excreted through the lungs, hypercarbia can only be avoided by a compensatory hyperventilation by increasing the tidal volume of ventilation in anesthetized patients. Hypercarbia can also develop as a result of a highly increased peritoneal absorption of CO2 and an insufficiently increased exhaustion of CO2. Absorption of CO2 is increased particularly during prolonged surgery using high intra-abdominal pressure. Exhaustion of CO2 is reduced in patients with compromised cardiopulmonary function and restricted CO2 clearance [4]. Hypercarbia and acidosis can cause hemodynamic changes by direct action on the cardiovascular system and by an indirect action through sympatho adrenal stimulation; Increased intra-abdominal pressure during pneumoperitoneum triggers several pathophysiological mechanisms in dependently of the type of used gas. The most important mechanism of the neurohumoral response of the vasopressin and renin-angiotensin-aldosterone systemis the sympathetic stress response including vagal reflexes [5].

During pneumoperitoneum the diaphragm is shifted up wards and the abdominal part of the chest wall is stiffened resulting a reduction of the total volume of the lungs, a significant decrease up to 35–40% in pulmonary compliance and a marked increase in the maximum resistance of the respiratory system [6, 7]. Although the decrease in functional residual capacity promotes a ventilation-perfusion mismatch and an intrapulmonary shunting which may lead to hypoxemia [8], this occurs rarely in patients with normal preoperative pulmonary function.

Without controlled hyperventilation an increase in the end-tidal carbondioxide pressure by 10 mm Hg develops. This is why the ventilatory pattern needs to be adjusted, and ventilation with large tidal volumes (12–15 ml/kg) tobe performed in order to prevent progressive alveolar atelectasis and hypoxemia and to allow CO2 elimination.

Although ventilation with positive end-expiratory pressure significantly improves pulmonary gas exchange [9, 10] and preserves arterial oxygenation during prolonged.

Pneumoperitoneum [11] it should be noted that positive end-expiratory pressure in the presence of elevated intra abdominal pressure, increases the intrathoracic pressure and produces marked reduction in cardiac output. Therefore,it should be applied cautiously [12-13].

CONCLUSION

In conclusion, excessive CO2 absorption, severe hypercapnia and acidosis may be encountered in patients undergoing laparoscopic surgery. We have witnessed a patient, free of cardiacor pulmonary disease, undergoing laparoscopic surgery, who sustained severe hypercapnia and respiratory acidosis during operation. Fortunately, with close intraoperative monitoring, supportive measures, and postoperative mechanical ventilatory assistance, Excessive internal CO2 was washed out gradually and the patient was extubated successfully the nextmorning.

REFERENCES

1. C.N. Gutta T. Oniub A. Mehrabia P. Schemmera A. Kashfib T. Krausa MW. BüchleraCirculatory and Respiratory Complications of Carbon Dioxide Insufflation Dig Surg. 2004;21:95–105
2. Leonard IE, Cunningham AJ. Anaestheticconsiderations for laparoscopiccholecystectomy. Best PractRes ClinAnaesthesiol. 2002;16:1−20.

3. Cunningham AJ, Brull SJ. Laparoscopiccholecystectomy:anesthetic implications. Anesth Analg. 1993;76:1120−33.
4. Hsieh CH. Laparoscopiccholecystectomy forpatients withchronic obstructive pulmonary disease. LaparoendoscAdvSurg Tech. 2003;13:5–9.
5. Neudecker J, Sauerland S, Neugebauer E, Bergamaschi R, Bonjer HJ, Cuschieri A, Fuchs KH, Jacobi CH, Jansen FW, Koivusalo AM, Lacy A. The European Association for Endoscopic Surgery clinical practice guideline on the pneumoperitoneum for laparoscopic surgery. Surgical endoscopy. 2002 Jul;16(7):1121-43.
6. Makinen MT, Yli-Hankala A. The effect oflaparoscopiccholecystectomy on respiratory compliance as determined by continuousspirometry. J Clin Anesth. 1996;8:119–122.
7. Garcia-Perez M, Belda F, Lla J, Aguilar G, Soro M, Marti F, Guillen A. Changes in chest wall and lungcomplianceduringlaparoscopiccholecystectomy. Rev Esp AnestesiolReanim. 2001;48:171–175.
8. Haydon GH, Dillon J, Simpson KJ, ThomasH, Hayes PC: Hypoxemiaduring diagnostic laparoscopy: A prospective study. Gastrointest Endosc. 1996;44:124–128.
9. Loeckinger A, Kleinsasser A, Hoermann C, Gassner M, Keller C, Lindner KH. Inert gas exchange during pneumoperitoneum at incremental values of positive end-expiratory pressure. Anesthesia & Analgesia. 2000 Feb 1;90(2):466-71.
10. Sandbu R, Birgisdottir B, Arvidsson D, Sjöstrand U, Rubertsson S. Optimal Positive end-expiratory pressure (PEEP) settings indifferential lung ventilation during simultaneous unilateral pneumothoraxand laparoscopy. Surgical endoscopy. 2001 Dec 1;15(12):1478.
11. Hazebroek EJ, Haitsma JJ, Lachmann B, Bonjer HJ. Mechanical ventilation with positive end-expiratory pressure preserves arterial oxygenation during prolonged pneumoperitoneum. Surgical endoscopy. 2002 Apr;16(4):685-9.
12. Moffa SM, Quinn JV, Slotman GJ. Hemodynamic effects of carbon dioxide pneumoperitoneum during mechanical ventilation and positive end-expiratory pressure. The Journal of trauma. 1993 Oct 1;35(4):613-7.
13. Kraut EJ, Anderson JT, Safwat A, Barbosa R, Wolfe BM. Impairment of cardiac performance by laparoscopy in patients receiving positive end-expiratory pressure. Archives of Surgery. 1999 Jan 1;134(1):76-80.