Case Report

Successful resuscitation after suspected carbon dioxide embolism during laparoscopic ureteric reconstructive surgery

Kalpana S. Vora, Veena R. Shah, Geeta P. Parikh, Pranjal R. Modi[1]

Abstract

Carbon dioxide (CO2) embolism is a rare but potentially fatal complication of laparoscopic surgery. We report a case of presumed CO2 embolism in a 35-year-old female during laparoscopic ureteric reconstructive surgery. After 2 h of operating time, a sudden decrease in end-tidal carbon dioxide and deterioration of hemodynamic status followed by cardiac arrest with pulseless electrical activity suggested gas embolism. Immediate cardiopulmonary resuscitation and inotropic support resulted in successful outcome. Thus, early recognition of the complication and prompt treatment can avoid catastrophy.

Keywords: Carbon dioxide embolism, end-tidal carbon dioxide monitoring, laparoscopy

Access this article online

Website: www.ijccm.org

DOI: 10.4103/0972-5229.117081

Quick Response Code:

Introduction

We report a case, carbon dioxide (CO_2) embolism a potentially devastating complication of laparoscopic surgery, during dissection phase and was successfully resuscitated.

Case Report

A 35-years-old female, weighing 42 kg was posted for transperitoneal laparoscopic Boari's ureteric reimplantation. She had undergone. cesarean section 6 months back. Her, physical examination and routine investigations were unremarkable.

With routine monitoring, balanced general anesthesia was given. Patient was positioned supine with 20° trendelenberg tilt. CO_2 Pneumoperitoneum was created uneventfully using open insufflation technique and sustained with pressure of 14 mm Hg. Patient was hemodynamically stable. After 2 h, when the surgeon was dissecting the ureters, there was a sudden drop in $ETCO_2$ (8 mm Hg), tachycardia (130/min) followed by multiple ventricular premature beats (VPBs) with blood pressure (BP) 110/72 mm of Hg and SaO_2-98%. Surgical bleeding and endotracheal tube blockage were ruled out, and gas embolism was suspected. No drug was being administered intravenously. Infusion tubing was patent. Pneumoperitoneum was terminated immediately and the patient was placed in 45° trendelenberg position. Injection lignocaine 80 mg intravenously was given for VPBs, and the regular rhythm was restored with the heart rate of 42/min. Injection atropine 0.6 mg intravenously was given. At this point, the SaO_2 decreased from 98% to 77% while the breath sounds were normal with no adventitious sounds. Also, the patient was cyanosed with non-recordable BP and heart sounds. ECG monitor suggested complete heart block with pulseless electrical activity. One liter of crystalloid was infused. We could not appreciate a "mill wheel" murmur on auscultation. Patient was manually ventilated with 100% O_2. Injection atropine, totally 1.8 mg intravenously was given. Cardiopulmonary resusicitation was commenced and injection adrenaline 1 mg intravenously was given. Since there was no response to these measures, injection dopamine 20 ug/kg/min and nor-adrenaline 0.36 ug/kg/min were infused. Multiorifice central

From:
Departments of Anesthesia and Critical Care, and [1]Urology, IKDRC and Dr. H. L. Trivedi Institute of Transplantation Sciences, Gujarat, India

Correspondence:
Dr. Kalpana S. Vora, 13, Choksi Park, Jivraj Park, Ahmedabad - 51, Gujarat, India. E-mail: drkalpana_vora@yahoo.co.in

venous catheter with its tip at the superior vena cava – right atrium (SVC-RA) junction was inserted via right internal jugular vein but air could not be aspirated. The central venous pressure (CVP) was 15 mm of Hg. After 20 min, resuscitative efforts were successful, patient had pulse rate of 120/min, BP 100/54 mm/Hg, SaO_2 100%, $ETCO_2$ 30 mm/Hg and ECG showed sinus rhythm with right ventricular strain pattern.At this time the CVP was found to be 10 mm of Hg. ABG analysis was normal. Surgery was abandoned. After ½ h, adrenaline was stopped. Two-dimension echocardiography (2-D Echo) showed no patent ductus arteriosus or any other congenital defects. She was shifted to intensive care unit with dopamine 5 ug/kg/min. which was tapered and stopped after ½ h. The patient did not receive hyperbaric oxygen as we did not have the facility. She regained consciousness after 2 h without neurological deficit and was extubated.

Discussion

CO_2 embolism can occur either during peritoneal insufflationor during the surgical dissection.[1] In this case, possible causes of sudden cardiovascular collapse like hypovolemia, hemorrhage, vaso-vagal reflex, pneumothorax and endotracheal tube blockage were ruled out before considering gas embolism The adhesions caused by previous surgery might have led to vascular injury during dissection and the operating site was above the level of the heart (trendelenburg position) creating a pressure gradient[2,3] Thus the open venous channels, at lower pressure were open to gaseous medium at a higher pressure leading to gas embolism.

Either, slow infusion of air (0.01-2.00 ml/kg/min) or bolus of gas (25-200 ml or 1-13 ml/kg), creates a "gas–lock" in the right atrium, obstructing the pulmonary outflow tract. Paradoxical air embolism was ruled out by a 2-D Echo that showed no intra-cardiac channels.

Any kind of pulmonary embolism leads to sudden deterioration of the hemodynamic status associated with decreased $ETCO_2$ levels. Since CO_2 is a highly soluble gas (54 ml/dl), prompt recovery of the vital signs is usual. Aspiration of gas through central venous catheter is the definitive means of treating gas emboli. In our case, failure to aspirate the gas may suggest that it might have been trapped in the pulmonary vasculature.

TEE is the gold standard for detection of pulmonary air embolism, however, it is not indicated routinely for intra-operative monitoring.[4] Precordial Doppler ultrasound can detect volume as low as 0.25 ml. Sudden decrease in $ETCO_2$ is an early sensitive but nonspecific sign of venous air embolismPrecordial stethoscope enhances specificity of $ETCO_2$ and mean pulmonary artery pressure values.[5]

Immediate treatment of gas embolism requires deflation of pneumoperitonium, placing the patient in Durant's position, hyperventilation and administration of 100% oxygen. Aggressive cardiopulmonary resuscitation should be continued till the acute effects of the embolism abate and one should maintain positive circulating volume to prevent further entrainment of the gas.

Thus, in laparoscopic surgeries early detection of gas embolism with prompt resuscitation can result in a favorable post-operative outcome.

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How to cite this article: Vora KS, Shah VR, Parikh GP, Modi PR. Successful resuscitation after suspected carbon dioxide embolism during laparoscopic ureteric reconstructive surgery. Indian J Crit Care Med 2013;17:190-1.

Source of Support: Nil, **Conflict of Interest:** None declared.