CASE REPORT

Lazarus phenomenon revisited: a case of delayed return of spontaneous circulation after carbon dioxide embolism under laparoscopic cholecystectomy

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ABSTRACT

The Lazarus phenomenon is rare occurrence of delayed return of spontaneous circulation (ROSC) after cardiac arrest when the resuscitation efforts are discontinued. It can have serious medico legal consequences. There are various explanations proposed for this phenomenon. We report a case of intraoperative cardiac arrest due to carbon dioxide embolism under laparoscopic cholecystectomy where ROSC occurred when cardiopulmonary resuscitation was abandoned.

Key words: Lazarus phenomenon; ROSC; Carbon dioxide embolism; Anesthesia

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INTRODUCTION

The Lazarus phenomenon is rare occurrence of delayed return of spontaneous circulation (ROSC) after cardiac arrest when the resuscitation efforts are discontinued.[1] The scientific explanations for this phenomenon have been difficult to prove. There are only 39 cases reported so far in medical literature. [2,3] The outcome in these cases has been variable with about 45% of cases dying soon after. No significant correlation is observed between the outcome and duration of CPR, time interval for ROSC or the etiology of cardiac arrest.[2]

We report a case of delayed ROSC after a case of probable intraoperative carbon dioxide (CO_2) embolism during laparoscopic cholecystectomy.

CASE REPORT

A 25-year-old lady, weighing 60 kg was scheduled to undergo laparoscopic cholecystectomy. The preanesthetic clinical evaluation and routine laboratory investigations were unremarkable. In the operating room standard anesthetic monitoring including continuous electrocardiogram (ECG), pulse oximetry (SpO_2) and non-invasive blood pressure (NIBP) were started. For premedication inj midazolam 1 mg and fentanyl 100 μg were administered intravenously (IV). General anesthesia was induced using inj propofol 100 mg and endotracheal intubation was accomplished by inj atracurium 30 mg. For maintenance of anesthesia oxygen and nitrous oxide mixture (33:66) with sevoflurane 1.5% was started. The patient was then handed over to surgeons for the procedure. The volume control mode of ventilation with tidal volume of 500 ml and respiratory rate of 12/min was started. The surgeons started CO_2 insufflation at a pressure of 12 mmHg and rate of initially 1lit/min and then increased to 4 lit/min. During insufflations of CO_2, end tidal CO_2 concentration ($EtCO_2$) started to decrease gradually, we immediately checked for circuit connections. Within a minute heart rate (HR) and SpO_2 also began to fall. The

heart rate decreased to 50/min from 90/min and SpO_2 came down to 70% from baseline of 100%. The surgeons were asked to stop insufflations of CO_2 and evacuate the peritoneal cavity. The NIBP reading was 56/28 mm Hg. The patient was started on manual ventilation with 100% oxygen and other anesthetic gases were discontinued. However, the patient developed cardiac arrest with ECG showing asystole. Cardiopulmonary resuscitation (CPR) with chest compressions was started at a rate of 100 / min immediately. The patient was manually ventilated with 100% oxygen at a rate of 8-10 / min with low tidal volume just sufficient to produce a visible chest rise. The OT table was moved to head down and right up position. The feasibility of insertion of a central venous catheter in view of suspected CO_2 embolism was discussed but it was not favored as it would have required interruption in ongoing chest compression. During the resuscitative efforts continuous capnography monitoring was used to monitor quality of chest compressions. We followed the adult advanced cardiovascular life support algorithm for cardiac arrest for resuscitation. The rhythm check using paddles of defibrillator was done every 2 min after chest compressions. A pulseless ventricular fibrillation was detected in between for which a biphasic shock of 200J was given. The CPR was continued for about 40 min but spontaneous circulation could not be resumed. During this period of CPR, 4 doses of 1 mg adrenaline (1:10000) and a defibrillation shock were administered. After discussion with surgeons and the relatives of the patient the resuscitative efforts were discontinued. Incidentally the endotracheal tube of patient was not disconnected from the ventilator.

About 5-7 min after discontinuation of CPR the ECG on monitor started showing idioventricular rhythm. The carotid pulse was not palpable at this time. After 5 min the sinus rhythm at a rate of 96 / min appeared on monitor. The heart sounds by auscultation were confirmed and the carotid pulse albeit of low volume was also palpable. The NIBP reading was 60/32 mmHg. The patient was administered a bolus of 1 lit of crystalloid solution. The noradrenaline and dopamine infusions were started. Gradually the pulse oximeter plethysmography also appeared on the monitor. However, the BP continued in the range of 70-80 mmHg systolic. The patient was shifted to intensive care unit for further management. Mechanical ventilation with FiO_2 100% and infusions of inotropes were continued. Neurological examination revealed dilated non reacting pupils and a Glasgow coma score 3. The arterial blood gas analysis showed severe metabolic acidosis and hypoxia. Attempts for achieving therapeutic hypothermia with available resources were made. After 4 hours the patient again developed cardiac arrest and despite all resuscitative efforts could not be revived.

DISCUSSION

Our patient's case illustrates a rare occurrence of return of spontaneous circulation (ROSC) after cessation of CPR attempts. This has been reported as Lazarus phenomenon in the literature and the first case was reported in 1982.[1,4] So far, only 39 cases of delayed ROSC have been reported with both in-hospital and out of hospital cardiac arrest. The majority of cases were diagnosed as myocardial infarction or obstructive airway disease.[2] Other etiologies include ruptured abdominal aortic aneurysm, hyperkalemia, gastrointestinal hemorrhage, fat embolism, trauma, sepsis and toxicity of digoxin, opiates and cocaine.

The exact mechanism of delayed ROSC is unclear. In patients with COPD rapid ventilation without adequate time for expiration during CPR can lead to dynamic hyperinflation (generation of auto- PEEP) of lungs which impairs venous return and cardiac output. Discontinuation of CPR and hyperventilation has been postulated to improve venous return and hence can result in delayed ROSC.[5] Other authors have suggested delayed action of drugs administered during CPR, hyperkalemia and myocardial stunning as mechanism of delayed ROSC.[6,7]

The carbon dioxide embolism during laparoscopic surgeries is rare complication with incidence of 0.001%. The clinically significant CO_2 emboli however, may be fatal with reported mortality rate of 28%.[8] The majority of cases reported in literature occurred at the beginning of surgery due to misplacement of Veress needle into vein or parenchymal organ. Because of its high solubility, CO_2 embolism has less marked effect compared to embolism of air. However, the large amount of CO_2 entering into circulation can be fatal. CO_2 embolism can manifest itself through a "gas lock" effect in the vena cava, right atrium or pulmonary artery, causing obstruction to venous return, RV ejection, right and left heart failure, paradoxical embolism, arrhythmia, pulmonary hypertension, systemic hypotension, and cardiovascular collapse.

In our case the embolism of CO_2 occurred probably

due to misplacement of tip of Veress needle into a blood vessel as on the retrospective examination it was noticed to be blood stained. During insufflations of CO_2 pressure was maintained at 12-13 mmHg and the flow rate was adjusted 1-5 L/mins. The diagnosis of CO_2 embolism was apparent when $EtCO_2$ started falling but simultaneously hemodynamic instability rapidly progressed to asystole. The CPR in our patient was immediately ensued after recognition of embolism. The decision to stop resuscitative efforts were made after 40 minutes of cardiac arrest as the $EtCO_2$ was persistently less than 10 mmHg, carotid pulse was not palpable and no cardiac rhythm appeared by this time. The surprise appearance of idioventricular rhythm and then sinus rhythm prompted us to resume steps for hemodynamic optimization of patient. As with majority of cases of Lazarus phenomenon the ROSC occurred within 10 minutes of cessation of CPR. We tried our best to ensure the optimal quality of chest compressions by maintaining adequate rate and depth, minimizing interruptions, keeping ventilation rate at 8-10 / min and by timely rotation of team members doing chest compressions by our team leader. The delayed ROSC could be related to high solubility of CO_2 leading to dissolution of gas lock effect with passage of time. The plausibility of this assumption is supported by the reports of reversal of CO_2 embolism with CPR.[9,10] The chest compressions during CPR would have also helped in fragmenting CO_2 embolism into small bubbles. It is also a possibility that more than one of above mechanisms could be involved in delayed ROSC.

After ROSC however, our patient developed persistent hypotension probably due to ischemia sustained due to gas lock by CO_2 emboli causing right ventricular strain or decreased cardiac output. The hypotension did not respond to fluid resuscitation and inotropic supports and ultimately led to second episode of cardiac arrest.

CONCLUSION

The delayed return of ROSC can have serious medicolegal and professional consequences. As majority of reported patients with Lazarus phenomenon have had ROSC within 10 minutes after cessation of CPR, one should always monitor a patient at least for ten minutes or longer following unsuccessful CPR.

Conflict of interest: None declared by the authors.

Author contribution: All authors took part in the management of the patient and preparation of the manuscript.

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