

Control of Extensive Subcutaneous Emphysema by Balloon Trocar – A Case Report

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Abstract Subcutaneous emphysema (SE) is not an uncommon complication in laparoscopic surgery, it may cause severe hypercapnia and acidosis. The trocar related SE is usually caused by multiple attempts at the abdominal entry and leakage of gas into the subcutaneous tissue, this kind of SE usually causes refractory hypercapnia and may induce severe complications. However, Intraoperative treatment for this kind of SE is still limited. Here we present a case of refractory hypercapnia caused by trocar related SE, after failed attempts of hyperventilation and decreasing intra-abdominal pressure, the SE and hypercapnia were finally controlled by using a balloon trocar.

Keywords: *subcutaneous emphysema, balloon trocar, laparoscopy*

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1. Introduction

Subcutaneous emphysema (SE) is a complication in laparoscopic surgery, it may cause refractory hypercapnia that could hardly be controlled by hyperventilation. The cause of SE is various, such as high intra-abdominal pressure (IAP), traumatic trocar insertion, soft tissue dissection, fascial extension, etc [1]. The trocar related SE is usually caused by multiple attempts at the abdominal entry leading to the leakage of gas into the subcutaneous tissue. It may cause refractory hypercapnia, which could induce tachycardia, hypertension, and acidosis [1]. Intraoperative treatment for this kind of SE is still limited, the common strategy is interim deflation of the abdominal cavity combined with hyperventilation. In some circumstances, conversion to laparotomy may be necessary [2]. Here we presented a case of refractory hypercapnia caused by trocar related SE, after failed attempts of hyperventilation and reducing IAP, the extensive SE and hypercapnia were finally controlled by using a balloon trocar at the incision site.

2. Case Description

A 67-year-old female patient (152 cm/ 42 kg) diagnosed with hepatobiliary lithiasis was scheduled for laparoscopic hepatectomy. Her medical history and preoperative examinations were unremarkable. After anesthesia induction and tracheal intubation, the patient was mechanically ventilated with tidal volume (V_t) 300 ml

and respiratory rate (RR) 12 bpm. The peak airway pressure (P_{peak}) measured by the anesthesia machine was 14 cmH₂O and the initial end-tidal carbon dioxide tension (PetCO₂) measured by the monitor was 40 mmHg. A blood gas 20 min later after mechanical ventilation revealed pH 7.45, PaCO₂ 40 mmHg (PetCO₂ 35 mmHg), and HCO₃⁻ 27.7 mmol/L.

Then the patient's abdomen was insufflated by CO₂ and the IAP was set to 13 mmHg. Four standard trocars were placed. As a routine process to facilitate CO₂ expiration by hyperventilation, the RR was reset to 16 bpm and the P_{peak} altered to 18 cmH₂O. 50 min after the pneumoperitoneum, the PetCO₂ increased to 50 mmHg, a blood gas revealed pH 7.31, PaCO₂ 55 mmHg, and HCO₃⁻ 27.5 mmol/L. The CO₂ absorber was replaced after then.

Another 60 min later after pneumoperitoneum, the patient's PetCO₂ on the monitor further increased to 75 mmHg without significant changes of her vital signs. A palpation crepitation was felt on her abdominal wall, chest wall, neck, and lower face. An extensive SE was diagnosed. At this time, a surgeon said that he had previously retracted a trocar back to the subcutaneous tissue, slightly changed the direction, and reinserted it into the abdominal cavity. Hence, the cause of SE was suspected to be unintended CO₂ insufflation into the subcutaneous tissue from the port where the trocar was reinserted once. To escalate CO₂ expiration, the ventilation mode was reset to pressure-controlled (inspiratory pressure 21 cmH₂O) with RR 16 bpm, the expiratory V_t measured by the anesthesia machine was 350 to 400 ml. Meanwhile, the IAP was reset to 11 mmHg to reduce CO₂ absorption. However, despite decrease, the PetCO₂ was still higher than 66 mmHg. After discussion

with the surgeons, the patient's abdominal cavity was deflated so that the operation was stopped for nearly 10 min. At this interval, the SE was manually compressed for a while and the PetCO₂ gradually decreased to 53 mmHg by hyperventilation.

After the break, the abdomen was insufflated again with an IAP of 11 mmHg. Surprisingly, the SE aggravated simultaneously and the PetCO₂ increased to 57 mmHg shortly. At this time, a surgeon substituted the standard trocar with a balloon trocar at the suspected port. Then the PetCO₂ gradually decreased and was kept at 53 mmHg.

20 min after the use of the balloon trocar, the surgeons

requested an IAP of 13 mmHg for a better view of the surgical field and the PetCO₂ gradually increased to 63 mmHg with the SE aggravated again. The blood gas revealed pH 7.16, PaCO₂ 75 mmHg, and HCO₃⁻ 22.7 mmol/L. To avoid acidosis and SE progress, the IAP was set to 11 mmHg again. After then, the PetCO₂ gradually decreased and was kept between 44 and 57 mmHg till the end of the operation (Figure 1). A blood gas before the end of the operation revealed pH 7.21, PaCO₂ 63 mmHg (PetCO₂ 57 mmHg), and HCO₃⁻ 22.4 mmol/L. The patient's vital signs were relatively stable during the whole operation.

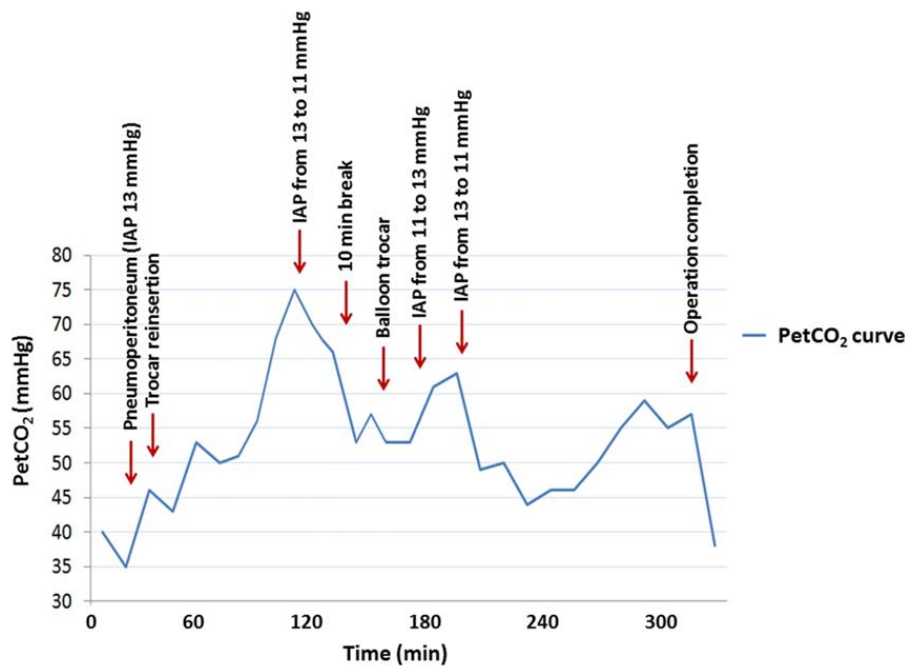


Figure 1. The change of PetCO₂ during the operation (IAP: intra-abdominal pressure. PetCO₂: end-tidal carbon dioxide tension)

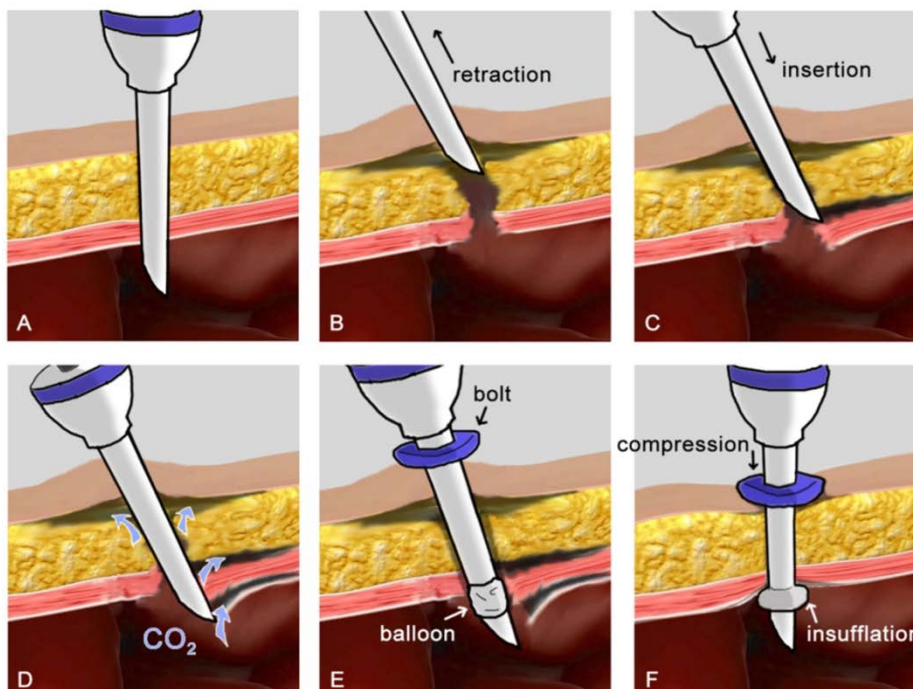


Figure 2. The illustration of the trocar related SE and the effect of the balloon trocar (A: the standard trocar is inserted snugly in the tissue. B: retraction of the trocar dissects the subcutaneous tissue. C: insertion of the trocar dissects the muscles and the peritoneum. D: CO₂ is insufflated into the dissected tissues. E: a balloon trocar is inserted. F: the balloon trocar plugs the leakage snugly.)

After the operation completed, the patient was transferred to post-anesthesia care unit (PACU). She was extubated after mechanical ventilation for another 45 min. The last blood gas before extubation revealed pH 7.32, PaCO₂ 51 mmHg, and HCO₃⁻ 24.8 mmol/L. After observation for 70 min, a mild palpation crepitation was on her body and she was transferred back to the ward with a good recovery. Follow-up in the next day, the patient recovered well with no obvious palpation crepitation, she was discharged 9 days after surgery.

3. Discussion

The incidence of grossly detectable intraoperative SE is relatively low in laparoscopic hepatobiliary surgery (0% ~ 0.8%) [3]. The original risk factors of hypercapnia and SE in this case are old age (> 65 years), high IAP (13 mmHg), and high PetCO₂ (> 50 mmHg) [1,3]. Ryoko et al. [4] reported a case of massive SE extending to the face during laparoscopic hysterectomy. They found no port was misplaced and their patient was intubated overnight after surgery in case of airway obstruction. In our case, 10 min break later, the SE from the abdomen to the lower face aggravated shortly after insufflation, the surgeon also declared there was a trocar reinserted before. Therefore, we consider that the tissue layer integrity was destroyed by trocar reinsertion and further dissected by CO₂ inflow from the port (Figure 2, A-D). Hence CO₂ was insufflated easily and SE occurred rapidly after the break. Coronil et al. [5] reported a case of life-threatening trocar related SE in a laparoscopic hemicolectomy, they solved the problem by hyperventilation, minimizing IAP, and needle drainage. However, the attempts of hyperventilation and decreasing the IAP did not work well in our case.

Permissive hypercapnia (PaCO₂ 56-65 mmHg) in laparoscopic abdominal surgeries was proved to be beneficial [6]. However, severe hypercapnia and SE may cause acidosis, cardiac arrhythmias, tachycardia, hypertension, and even pneumothorax or airway obstruction [1,5,7,8]. Therefore, hypercapnia with extensive SE should be treated properly. Interventions to attenuate hypercapnia and SE during laparoscopic surgery include hyperventilation, decreasing IAP and gas flow, needle drainage, and changing CO₂ absorber [1,6]. If ineffective, to stop the operation and deflate the abdominal cavity for a while was also feasible despite prolonged operation time. In some circumstances, to avoid major complications such as airway obstruction induced by extensive SE, changes in trocar positioning, or even converting to laparotomy may be other choices [2].

A balloon trocar consists of a standard trocar, a bolt, and a balloon, the bolt and the balloon help to stabilize the trocar on the abdominal wall [9]. Therefore, it is reasonable to presume that the bolt and the balloon could also plug the dissected tissue at the port and prevent CO₂ from going into the subcutaneous and preperitoneal tissue directly (Figure 2, E and F), yet relevant report is rarely seen. In this case, after applying a balloon trocar, the hypercapnia and SE were well controlled under an IAP of 11 mmHg. However, the effectiveness of the balloon

trocar was still limited because it failed to prevent the leakage of CO₂ if the IAP was reset higher (13 mmHg). The reasons were considered as follows: first, there was still a slight air leakage at the port that could not tolerate a higher IAP, second, the sealing pressure between the bolt and the balloon was not well controlled, third, the long operation time and the patient's individual risk of SE (age > 65 years) facilitated the accumulation of CO₂ [1]. Hence several factors could influence the outcome of this technique: first, trocar injury should be the leading cause of SE or hypercapnia, second, if the area of trocar injury is extensive, the inflated balloon may be unable to cover the leakage port, third, if the bolt is not well fixed to maintain an appropriate pressure with the balloon, it may be unable to prevent CO₂ leakage in a high IAP condition. Nevertheless, the balloon trocar minimized the progress of hypercapnia and SE with an IAP of 11 mmHg in this case, and it helped to avoid another incision port or a conversion to laparotomy.

Hypercapnia is known to be associated with increase in heart rate, blood pressure, and cardiac index [1,5], SE may further induce pneumothorax or airway obstruction [7,8]. On the other hand, SE, hypercapnia, and acidosis may not always be detrimental or life-threatening. In this case, the patient's hemodynamics was relatively stable during the operation with severe SE and hypercapnia. Merle et al. [10] reported a case of severe hypercapnia in a laparoscopic paraesophageal hernia repair, the patient also remained hemodynamically normal despite severe SE with pH below 6.81 and PaCO₂ higher than 115mmHg, and recovered after surgery uneventfully. The authors questioned that if the pH was an independent predictor of outcomes. However, we still recommend early preventing severe SE and hypercapnia that may adversely affect patients.

In conclusion, replacing a standard trocar with a balloon trocar may be an alternative to conventional methods for controlling trocar related SE and refractory hypercapnia. In addition, we recommend recognizing the leading cause of SE or refractory hypercapnia in routine clinical practice because of the limited effects and additional cost of a balloon trocar. A rapid increase of PetCO₂ or a rapid occurrence of SE from the abdominal wall after pneumoperitoneum establishment may be the indications of trocar injury, in this condition, a balloon trocar may help to avoid further complications.

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Yi Zou, Lai Wei, and Yanan Tang contributed equally to this report

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