

Carbon dioxide embolism during transanal total mesorectal excision (TaTME) : two case reports

O. LEMPEREUR (*), E. DECKER (**), J. JORIS (*)

Abstract : Transanal total mesorectal excision (TaTME), a new approach for rectal cancers, requires transanal carbon dioxide (CO₂) high-flow insufflation to create a workplace. Two patients scheduled for TaTME experienced CO₂ embolism during the anterior mesorectal dissection in contact with the prostate. CO₂ embolism resulted in a sudden drop of end-tidal CO₂, preceded by a short increase in one patient, and in oxygen desaturation. Hemodynamic alterations were minor. We report these two cases and discuss the pathophysiology of CO₂ embolism and risk factors that promote CO₂ embolism during TaTME to warn anesthetists of this serious complication, often unexpected and misdiagnosed.

Keywords : rectal surgery ; transanal total mesorectal excision ; carbon dioxide embolism ; end-tidal CO₂.

INTRODUCTION

Transanal total mesorectal excision (TaTME) is a new technique of mesorectal resection that is proposed as an alternative to the standard laparoscopic approach for mid- and low rectal cancers (1). As compared to the abdominal “top-down” pelvic dissection, the transanal approach offers clearer visualization of the dissection plane, reduces tumor manipulation, and allows reduction in positive circumferential resection margin (2). For the pelvic time, a transanal monotrocard system is used that combines a carbon dioxide (CO₂) high-flow insufflation to create surgical workplace, a smoke evacuation, and an access port for the surgical instruments. Among the 20 patients undergoing TaTME in our institution, two patients experienced CO₂ embolism during the anterior dissection of the mesorectum. Despite a few case reports (3-5), this complication of TaTME remains unexpected or even unrecognized by anesthetists and surgeons. We report these two cases to warn anesthetists of this serious complication, the incidence of which is probably not anecdotal during this surgery. Furthermore, pathophysiology of CO₂ embolism during TaTME may result in different clinical presentations, which explains misdiagnosis.

Both patients gave written consent to use their information for publication.

CASE REPORTS

Medical history

The first patient is a 69-year-old man with a mid-rectum cancer scheduled for a TaTME after preoperative radio-chemotherapy. Surgical history included several vascular surgeries and abdominal hernia repair. In his medical history, we highlight arterial hypertension, hypercholesterolemia, and asymptomatic prostatic hypertrophy.

The second patient is a 75-year-old man with a low-rectum cancer scheduled for a TaTME without preoperative radio-chemotherapy. Medical history included a carotid thromboendarterectomy, coronary balloon dilation with drug eluting stenting, and symptomatic prostatic hypertrophy.

Anesthesia and surgery

In both patients general anesthesia was maintained with sevoflurane in an oxygen air mixture. Lungs were ventilated using a lung protective ventilation strategy with a positive end-expiratory

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pressure of 7 mmHg. The first stage of surgery was a trans-abdominal laparoscopy to mobilize and prepare the sigmoid colon for the colorectal anastomosis. After release of the pneumoperitoneum the pelvic time was started. Patients had the legs elevated and were in a 30° to 45° Trendelenburg position. A transanal monotrocard system (AirSeal®, Conmed, Utica, NY, USA) was used for CO₂ high-flow insufflation and mesorectal excision. The anterior mesorectal dissection was complicated by the hypertrophic prostate in both cases. The surgeon had then to face bleeding. Signs of CO₂ embolism occurred 30 min and 20 min after the beginning of transanal insufflation in patient 1 and patient 2, respectively.

Description of carbon dioxide embolism

In patient 1, in six min end-tidal CO₂ (ETCO₂) fell from 37 to 29 mmHg while oxygen saturation (SpO₂) decreased from 97 to 95 % and mean arterial pressure from 93 to 88 mmHg with no changes in heart rate (Fig. 1 upper panel). Selective bronchial intubation and ventilation perfusion mismatch secondary to patient position were first considered. Selective intubation was disproved. Recruitment maneuver and switch from volume-controlled to pressure-controlled ventilation were tested but without benefit. Meanwhile ETCO₂ decreased further to 24 mmHg, SpO₂ to 91 %, and mean arterial pressure to 80 mmHg. CO₂ embolism was then diagnosed (Fig. 1 red arrow) and treated.

In patient 2, ETCO₂ dropped abruptly from 40 mmHg to 19 mmHg while SpO₂ decreased from 99 to 88 %. Interestingly, a transient increase in ETCO₂ from 40 to 48 mmHg preceded the drop. Heart rate increased from 64 bpm to 75 bpm with no changes in arterial blood pressure (Fig. 1 lower panel). CO₂ embolism was rapidly diagnosed and treated.

In both cases, thoraco-pulmonary compliance and dynamic airway resistance did not change.

Treatment of CO₂ embolism

The oxygen inspired fraction (FIO₂) was increased to 1.0 and CO₂ insufflation was interrupted. Minute ventilation was increased. Patients already in head-down position were tilted to the left. ETCO₂ returned to the values from before the event in 12 and 8 min after initiation of treatment in patient 1 and patient 2, respectively. Within the same time, SpO₂ increased above 95%. In patient 1, delayed diagnosis might have contributed to a



Fig. 1. — Capture of the screen of the monitoring system at the time of carbon dioxide embolism in patient 1 and patient 2. Red arrow in upper panel = time of CDE diagnosis. FC = heart rate, PNI = non-invasive blood pressure.

slower correction of the parameters. We did not insert a central line to aspirate potential intracardiac CO₂ bubbles because of the absence of significant hemodynamic changes. After normalization of ETCO₂ and SpO₂, the operation was resumed in both patients. There were no postoperative sequelae.

DISCUSSION

Several operative conditions might facilitate CO₂ embolism during TaTME (3, 6, 7). With patients being in Trendelenburg position, gas embolism is facilitated through open pelvic veins with venous pressure lowered as the operative field is located above the level of the heart (8). High CO₂ insufflation flow, a small operative space, and dissection in the vicinity of high flow venous plexus of the prostate or paravaginal veins also promote CO₂ embolism. The contribution of these different factors in the surgical setting of TaTME may differ and result in variable rates of intravascular CO₂ accumulation and volumes of CO₂ entrainment. Consequently the clinical picture can vary making the diagnosis less conspicuous (8).

Our two cases with CO₂ embolism were associated with a sudden drop in ETCO₂ and SpO₂ resulting from the pulmonary pathophysiological changes after gas embolism (8, 9). Contrary to air emboli, CO₂ emboli do not result in an increase in airway resistance (9). As in most previously reported cases (6, 7), these two events occurred when the

surgeon was dissecting the anterior mesorectum in contact with the high flow venous plexus of the prostate and facing bleeding. Accordingly, the incidence of CO₂ embolism in case of laparoscopic prostatectomy is very high (10). In case of bleeding, suction and smoke evacuation are associated with CO₂ high flow insufflation to prevent a loss in surgical workplace. The resultant turbulent CO₂ flow in the small operative space promotes CO₂ intravascular entrainment. In other cases the CO₂ pneumoperitoneum can prevent back bleeding, impair recognition of injury of small veins, and allow unsuspected CO₂ entry (3). A small and slow CO₂ intravascular entry explains the increase in ET-CO₂ preceding the fall observed in patient 2 (11). Indeed, in these conditions exhalation of CO₂ may first increase because it is not yet impaired by a sudden enlargement of alveolar dead space. ET-CO₂ increase in this surgical setting should therefore warn the anesthetist. CO₂ embolism during TaTME results regularly from a slow intravascular entrainment of CO₂. Consequently hemodynamic changes are often minor as in our two patients. However severe hemodynamic disturbances, sometimes prolonged, and potentially leading to cardiac arrest were reported (3, 6). Paradoxical CO₂ embolism can occur through the opening of a patent foramen ovale secondary to the increase in right atrial pressure.

Dickson E. *et al.*, in a retrospective survey, reported an incidence of 0.4 % in the LOREC (tatme.medicaldata.eu) and OSTRiCh (www.ostrich-consortium.org) TaTME registries including 6375 cases (6). Harnsberger C. *et al.* reported an incidence of 4 % among 80 patients managed in a single institution (7). After reviewing the computerized anesthesia record of all 20 patients who underwent TaTME in our institution, we identified two more patients presenting with signs compatible with CO₂ embolism at a time corresponding to the pelvic time. The incidence of CO₂ embolism during TaTME is probably greater than previously reported.

The learning message of our case reports is that CO₂ embolism is an underrecognized complication of TaTME. Several reasons contribute to misdiagnosis. TaTME being a new procedure, providers (surgeons and anesthesiologist) need to be aware of its possible complications. Furthermore, CO₂ embolism has not been described in case of other techniques of total mesorectal excision (6). CO₂ embolism is a well-known, fortunately rare, complication associated with laparoscopy, but during TaTME it occurs after release of the pneumoperitoneum (6). While in case of laparoscopy CO₂ embolism most often coincides with the creation of CO₂-pneumoperitoneum, in case of TaTME this complication is delayed compared to the beginning of transanal insufflation of CO₂.

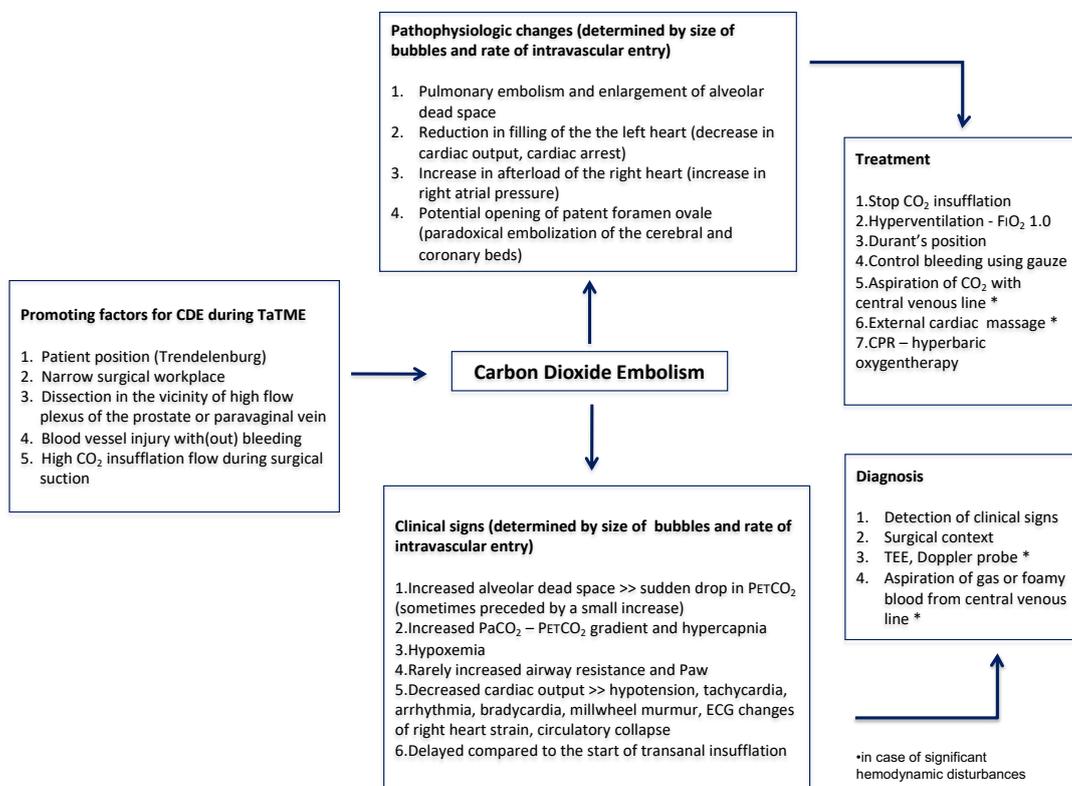


Fig. 2. — Synthesis figure with diagnostic and treatment algorithm for the carbon dioxide embolism (CDE) during TaTME.

Finally, pathophysiology of CO₂ embolism during TaTME may result in different clinical presentations, which contributes to unrecognition of this complication.

Treatment of CO₂ embolism during TaTME is not specific and combines interruption of CO₂ insufflation, inhalation of oxygen at 1.0 FIO₂, hyperventilation to compensate for the enlargement of alveolar dead space, patient positioning in Durant's position, circulatory support with fluids and inotropes if necessary. If CO₂ embolism is associated with bleeding, suction must be interrupted, the operative field flooded with saline, and bleeding site controlled using small gauze. Aspiration of intracardiac CO₂ bubbles with a central venous line, cardiopulmonary resuscitation, and hyperbaric oxygenation will be initiated in case of major hemodynamic and neurologic consequences (8, 9). Diagnosis and treatment algorithm for CDE during TaTME are synthesized in Fig. 2.

In conclusion, TaTME combines several risk factors for CO₂ embolism. Its incidence is probably not anecdotal. Anesthetists must be aware of this complication occurring during transanal insufflation particularly during dissection of the anterior mesorectum and in case of bleeding.

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