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Pulmonary embolism after laparoscopy-assisted colectomy

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Laparoscopic surgery has become the procedure of choice in the surgical treatment of early colon cancer as a minimally invasive technique [1,2]. However, the widespread adoption of laparoscopy for various surgical procedures has led to a higher incidence of life-threatening complications such as venous gas embolism and unintentional injury to vascular structures [3,4]. These adverse effects are generally considered to occur within several hours after surgery. We describe a case of pulmonary embolism that developed on the day after laparoscopic surgery.

Case report

A 57-year-old man weighing 67 kg underwent laparoscopy-assisted ileocecal resection for early colon cancer. His medical history was notable for hypertension treated with the Ca channel blocker nitrendipine. A preoperative electrocardiogram revealed complete right bundle branch block and sporadic ventricular premature beats. General anesthesia was induced with thiopental and vecuronium and the trachea was intubated with a cuffed endotracheal tube. Anesthesia was maintained with nitrous oxide (66%), sevoflurane (1%–2%), and continuous thoracic epidural block. Muscle relaxation was achieved with vecuronium, and the lungs were mechanically ventilated. During the laparoscopic procedures, intraabdominal pressure was elevated to 10 mmHg by peritoneal insufflation of CO_2 while keeping a slight head-up tilt position (approximately 10°). Pneumoperitoneum was initially maintained for 50 min to ease mobilization of the ileocecal structure and reinstituted for 12 min at the end of surgery to confirm hemostasis. There was no remarkable change in hemodynamics, end-expiratory carbon dioxide tension, or arterial oxygen saturation intraoperatively.

Soon after the operation, the patient regained consciousness and the endotracheal tube was extubated following reversal of the residual effects of the muscle relaxants. During the subsequent 80-min observation period in the postanesthesia care unit, there were no episodes of hypoxemia, abrupt changes in hemodynamics, or chest pain. The postoperative course was uneventful until the occurrence of a brief period of faintness at 8 P.M. of the day after surgery. A similar syncope attack reappeared the next morning at 6 A.M. and 10:30 A.M. when the patient stood up from his bed without any sign of respiratory distress or chest pain. Blood pressure and heart rate immediately after regaining consciousness were 90/60 mmHg and 84 beats · min^{-1}. Arterial blood gas indicated severe hypoxemia (Pao_2 41 mmHg) with normal $Paco_2$ (39 mmHg) despite oxygen therapy (3 l · min^{-1}; nasal cannula), suggesting acute pulmonary embolism, and the patient was transferred to the intensive care unit (ICU).

Anticoagulation therapy with heparin (15000 Units/day) was started immediately after arrival to the ICU, and a Swan-Ganz catheter was placed to monitor pulmonary arterial pressure. Since a pulmonary perfusion scan taken 3 h after ICU admission revealed multiple perfusion defects (Fig. 1), thrombolytic therapy with a tissue plasminogen activator (alteplase; 24 million U/day) was also initiated. As a result of the treatment, shunt fraction decreased from 48% to 10% and mean pulmonary arterial pressure decreased from 25 mmHg to 19 mmHg within the next 24 h. However, significant

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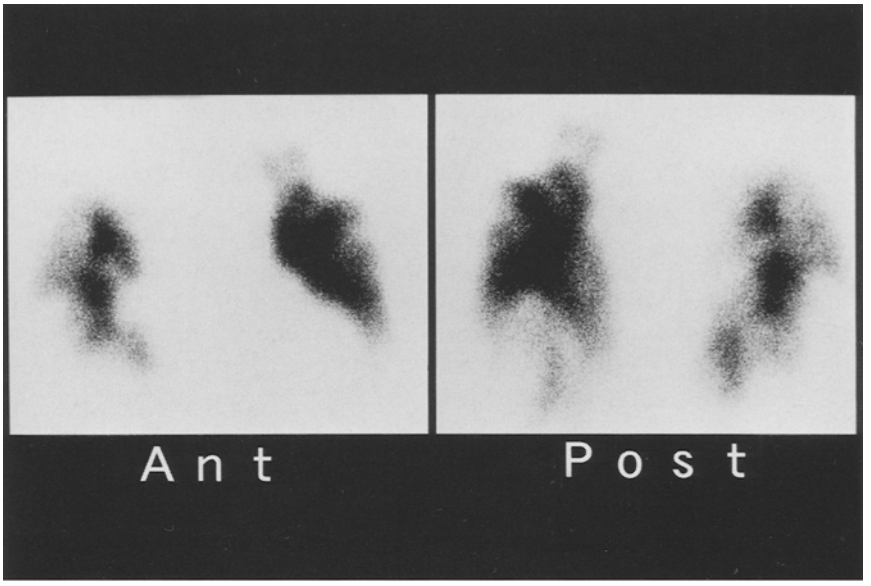

Fig. 1. Pulmonary perfusion scintigram performed on the day of admission to the intensive care unit (ICU). Multiple perfusion defects were noted in both lungs. *Ant*, anteroposterior view; *Post*, posteroanterior view

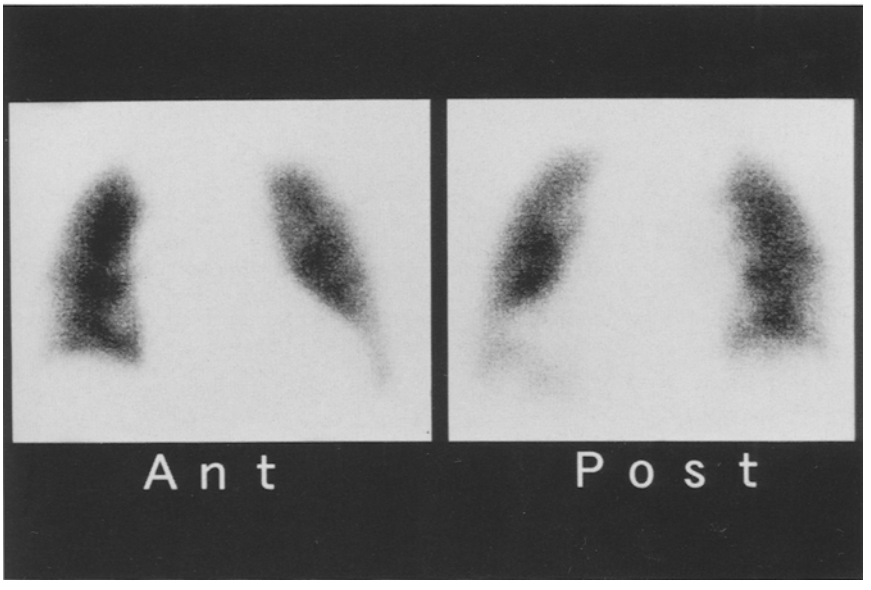

Fig. 2. Pulmonary perfusion scintigram performed the day before discharge from ICU. Note marked improvement of perfusion. *Ant*, anteroposterior view; *Post*, posteroanterior view

postoperative bleeding necessitating a blood transfusion became apparent with a marked increase in fibrinogen degradation products (FDP) (53.8 mcg/ml) and a decrease in antithrombin III (57%), and we discontinued the administration of heparin and tissue plasminogen activator within 40 h. Aside from these hemorrhagic complications, the patient's clinical conditions including hemodynamics and oxygenation were stable thereafter, and he was transferred to the ward on the 7th postoperative day. Venography of the legs performed on the 6th postoperative day showed no deep vein thrombosis. Pulmonary perfusion scan on the 7th postoperative day indicated a marked improvement in blood flow (Fig. 2).

Discussion

Pulmonary embolism associated with laparoscopic procedures is generally considered to be the consequence of carbon dioxide embolism [3,4]. It develops mainly during the induction of pneumoperitoneum with a sudden drop in cardiac output. Gas embolism may also occur later during surgery or may even be delayed after laparoscopy by trapping in the portal circulation [5]. The use of nitrous oxide during anesthesia may further delay the onset of gas embolism. However, in our case, it is unlikely that gas embolism resulted in pulmonary embolism since there was a time lag of at least 30 h after laparoscopy before the clinical signs of embolism be-

came overt. The satisfactory response to thrombolytic therapy further supports the hypothesis of pulmonary embolism secondary to venous thrombosis.

With regard to the cause-effect relation between laparoscopic procedures and the development of thromboembolism, Nagata et al. have examined pulmonary perfusion scintigraphy in 12 consecutive patients undergoing urological laparoscopic surgery and found radiologically evident pulmonary embolism in 7 patients postoperatively [6]. Although they attributed the results to carbon dioxide embolism, the clinical course was rather unspecific to gas embolism, having been more like thromboembolism. Indeed, Joris mentioned the possible risk of venous thrombosis and pulmonary embolism after laparoscopic surgery especially in the head-up position with the lower legs in flexion [7]. However, as far as we know, there is no published report comparing the incidence of postoperative pulmonary embolism between open and laparoscopic procedures.

Experimentally, definitive evidence has been obtained of venous stasis with pneumoperitoneum. Beebe et al. [8] have reported that peritoneal insufflation of CO_2 to 14 mmHg pressure during laparoscopic cholecystectomy resulted in a significant increase in femoral venous pressure (10 mmHg to 18 mmHg) and a significant decrease in peak velocity of flow (25 $cm \cdot s^{-1}$ to 19 $cm \cdot s^{-1}$). Furthermore, they noted reduced pulsatility in 75% of patients. These results suggest that pneumoperitoneum does cause venous stasis, which may increase the risk of deep vein thrombosis and subsequent development of pulmonary embolism. Such a mechanism might have played an important role in the development of pulmonary embolism in our patient. The fact that we could not detect any deep vein thrombosis by venography may have been the consequence of thrombolytic therapy. It is worth noting that our patient developed severe hypoxemia without any typical signs or symptoms of pulmonary embolism. We believe that continuous epidural morphine administration may have obscured most of the clinical features such as chest pain, dyspnea, or tachypnea. It is also noteworthy that the characteristic ECG abnormality for pulmonary embolism was also absent even under pulmonary hypertension.

Although thrombolysis with tissue plasminogen activator is localized at the thrombus site and thus hemorrhagic complications are considered rare [9], we encountered a significant postoperative hemorrhage requiring blood transfusion. Concomitant administration of heparin may have potentiated the bleeding tendency. Periodic assessment of the activated coagulation time shoud have been performed to avoid such complications in the postoperative state.

In clinical practice, to decrease the potential risk of pulmonary embolism after laparoscopic surgery, factors causing venous stasis in the legs should be avoided during the procedure. These include positioning of the patient (i.e., avoid extreme head-up tilt position, avoid tight strapping of the legs, prevent pressure on the popliteal space), as well as regulation of intraabdominal pressure and duration of pneumoperitoneum. It may be worth adopting measures known to reduce venous stasis such as pneumatic compressive stockings perioperatively.

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