J Anesth (2008) 22:177–181
DOI 10.1007/s00540-007-0604-0

Journal of
Anesthesia

Unexpected complication of massive intraoperative pulmonary embolism following elective sigmoidectomy in the supine position

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Abstract
We describe a case of massive intraoperative pulmonary thromboembolism during elective sigmoidectomy in the supine position. During recovery from anesthesia, the patient developed hemodynamic compromise and severe hypoxemia. Intravenous inotropes and mechanical ventilation were instituted. The abrupt onset of symptoms and the pulmonary artery catheter, chest radiograph, and transesophageal echocardiography findings suggested massive pulmonary thromboembolism as a possible cause of the hemodynamic compromise and hypoxemia. Emergent angiography could not be carried out due to the patient's poor clinical status. Lack of experience in performing embolectomy, along with contraindication for thrombolysis, imposed the use of intravenous heparin and hemodynamic support as the only appropriate therapeutic modality. After 2 days' aggressive hemodynamic and ventilatory support, the patient had an uneventful course, and was discharged from the intensive care unit (ICU) 14 days later.

Key words Massive · Thromboembolism · Intraoperative · Supine

Introduction

Intraoperative acute pulmonary thromboembolism (PTE) is a rare entity during abdominal surgery. Prompt diagnosis and treatment, although often based on clinical suspicion, are mandatory for a successful outcome. In the absence of any current consensus on definite treatment, the management strategy, ranging from cardiovascular support and anticoagulation to more invasive interventions, is rather challenging [1]. Furthermore, the nature of the intraoperative period poses significant difficulties regarding diagnosis and treatment, especially after a major abdominal operation. We report a patient who sustained a massive PTE during elective sigmoidectomy in the supine position.

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Received: March 28, 2007 / Accepted: December 20, 2007

Case report

A 65-year-old woman (weight, 90 kg; height, 160 cm; body mass index [BMI] 35.2) presented for elective sigmoidectomy due to cancer. Her medical history, apart from the cancer, included only psychological disorders. Routine laboratory studies, electrocardiogram, and chest X-ray (Fig. 1), revealed no serious abnormalities. Low-molecular-weight heparin (enoxaparin, 4000 IU anti-activated factor X [Xa]) was administered, 12 hr prior to surgery.

General anesthesia was induced with fentanyl 400 μg, thiopental 325 mg, and rocuronium 70 mg. Following intubation, mechanical ventilation (tidal volume [V_{tidal}], 700 ml; respiratory rate [RR], $10 \cdot min^{-1}$; positive end-expiratory pressure [PEEP], 5 cmH_2O, fractional inspired oxygen ($F_{I_{O_2}}$), 1) was instituted. The patient was placed in the supine position and graduated compression stockings (GCS) were applied. Direct arterial pressure monitoring and right internal jugular vein catheterization (baseline central venous pressure [CVP], 7 mmHg), were instituted. Anesthesia was maintained with 0.8 minimum alveolar concentration (MAC) of sevoflurane and continuous infusion of remifentanyl. Following arterial blood gas (ABG) analysis (pH, 7.37, Pa_{O_2}, 439 mmHg; Pa_{CO_2}, 38 mmHg; base excess [BE], -3 $mEq \cdot l^{-1}$), $F_{I_{O_2}}$ was reduced to 0.6.

One hour after the induction of anesthesia, her blood pressure (BP) decreased precipitously to 75/38 mmHg, without any alteration in ABG. The incident was attributed to intraoperative blood loss (hematocrit value, 21%, vs 36% preoperatively). Infusion of phenylephrine was started at 6.7 $\mu g \cdot min^{-1}$, and two units of red

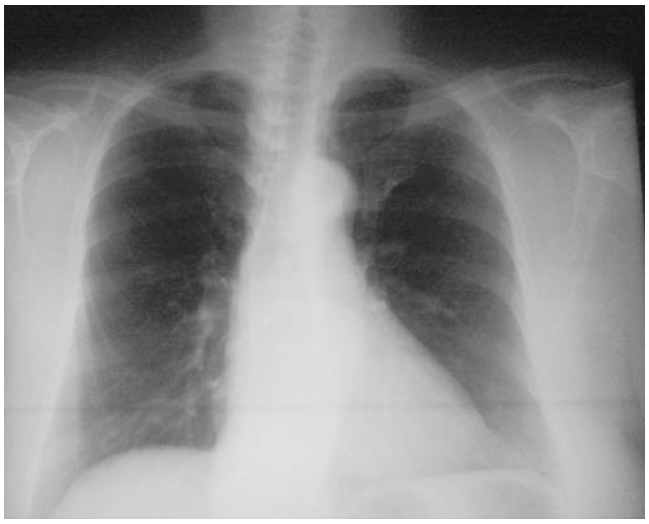

Fig. 1. Preoperative chest radiograph, with no significant pathologic findings

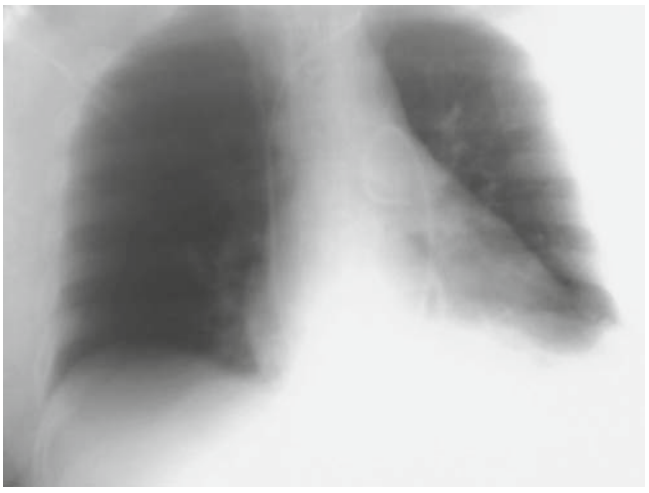

Fig. 2. Intraoperative chest radiograph, showing oligemia in the right hemithorax

blood cells were transfused. Hemodynamic parameters were restored and phenylephrine was withdrawn within 15 min.

Seventy-five minutes after this hypotensive episode, when the surgical procedure was reaching completion, routine ABG analysis revealed arterial oxygenation deterioration (Pa_{O_2} dropped from 262 to 95 mmHg), mild hypercapnia (Pa_{CO_2}, 42 mmHg); pH, 7.33; and BE, –4 $mEq \cdot l^{-1}$, with a concomitant elevation of CVP to 15 mmHg. At that time, the ABG alterations were not considered striking enough to lead to postponement of recovery from anesthesia.

During recovery from anesthesia and while spontaneous breathing was gradually being restored (V_{tidal}, 250–300 ml; RR, 22 $\cdot min^{-1}$; $F_{I_{O_2}}$, 1), an abrupt decrease in the pulse oximetry measurements (peripheral oxygen saturation [Sp_{O_2}], 99% to 85%) was noted. ABG analysis revealed serious deterioration of arterial oxygenation (Pa_{O_2}, 60 mmHg), severe hypercapnia (Pa_{CO_2}, 62 mmHg), acidosis (pH, 7.31; BE, –5 $mEq \cdot l^{-1}$), and a remarkable widening of the Pa_{CO_2}-end-tidal carbon dioxide (Et_{CO_2}) difference (20 mmHg versus 5 mmHg during the operation). Recovery from anesthesia was ceased, while sedation with sevoflurane 0.8 MAC, neuromuscular blockade with rocuronium 60 mg and mechanical ventilation were re-established (V_{tidal}, 750 ml; RR, 10·min^{-1}; PEEP, 8 cmH_2O; inspiratory-to-expiratory time ratio (I:E), 1:1.5; $F_{I_{O_2}}$, 1). A chest X-ray was performed, and although it was of low quality, it revealed apparent oligemia in the right hemithorax (Fig. 2).

Due to the patient's gradual hemodynamic instability (BP, 78/40 mmHg; heart rate [HR] ~ 110 beats $\cdot min^{-1}$), an intravenous infusion of adrenaline was applied, titrated to 20 $\mu g \cdot min^{-1}$. ABG measurement revealed: Pa_{O_2}, 108 mmHg with $F_{I_{O_2}}$, 1; Pa_{CO_2}, 53 mmHg; pH, 7.08; and BE, –15 $mEq \cdot l^{-1}$. In conjunction with alveolar plateau restoration on the capnogram, a further Pa_{CO_2}-Et_{CO_2} difference widening (~25 mmHg), was noticed, augmenting the diagnostic significance of the widening of the Pa_{CO_2}-Et_{CO_2} difference. A pulmonary artery catheter (PAC), placed in the right subclavian vein, revealed severe pulmonary hypertension up to 70/40 mmHg, so intravenous milrinone, 0.75 $\mu g \cdot kg^{-1} \cdot min^{-1}$, was added to the regimen. The 12-lead ECG showed marginal ST depression in all leads, with ST elevation in aVR (Fig. 3). The ECG findings were inconclusive, as they could have been attributable to any condition leading to myocardial overload. On the basis of the abruptness and magnitude of the clinical presentation and chest radiograph findings, acute PTE was suspected.

To elucidate the etiology of the cardiovascular collapse, emergent transthoracic echocardiography (TTE) was performed, which revealed right atrial and ventricular dilatation, moderate tricuspid regurgitation, right ventricular (RV) dysfunction, paradoxical interventricular septal flattening, and small left ventricular (LV) cavity size, without regional wall motion abnormalities, with normal function of the aortic and mitral valves (M-mode: ejection fraction [EF] 82%; left ventricular end-diastolic dimension (LVID), 4.55 cm; left ventricular end-systolic dimension (LVIS), 2.20 cm; interventricular septal wall thickness in diestole (IVSd), 0.85 cm; posterior wall thickness in systolic (PLWd), 0.9 cm). These findings could possibly be ascribed to massive PTE, so transesophageal echocardiography (TEE) was then performed in order to detect the presence of embolic material in the pulmonary artery. Although the

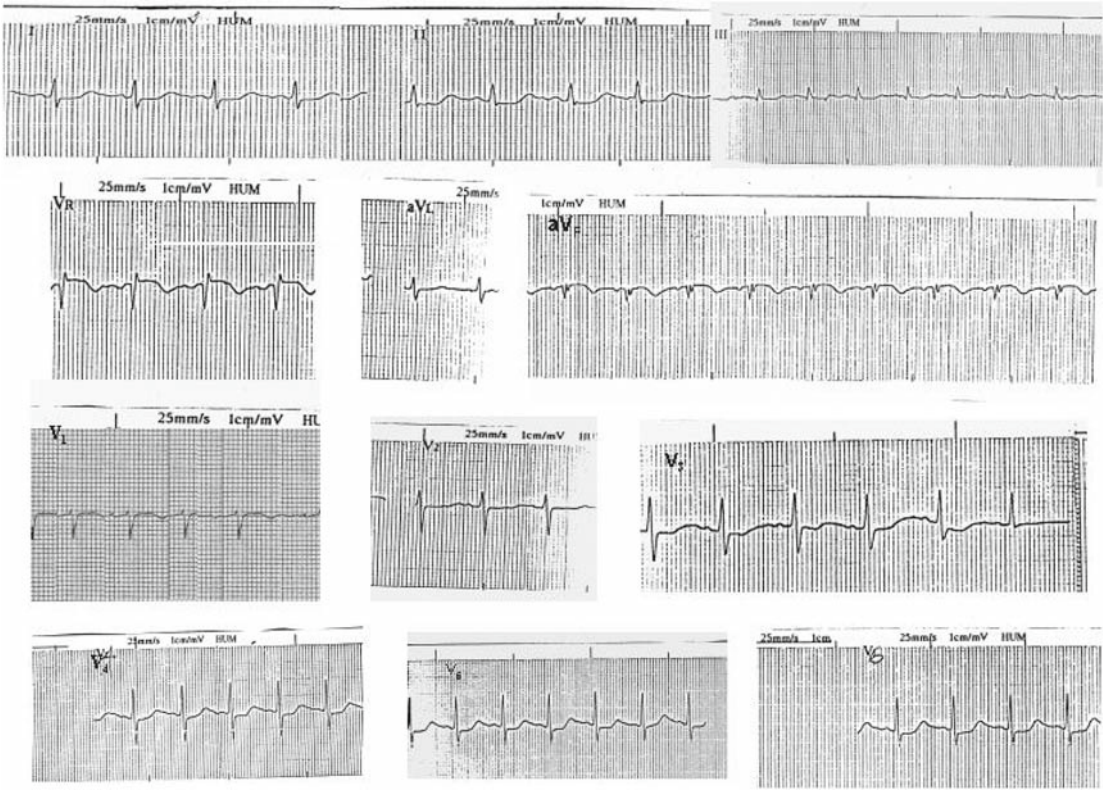

Fig. 3. ECG obtained during the circulatory collapse. aVR, ST elevation ST depression in all leads

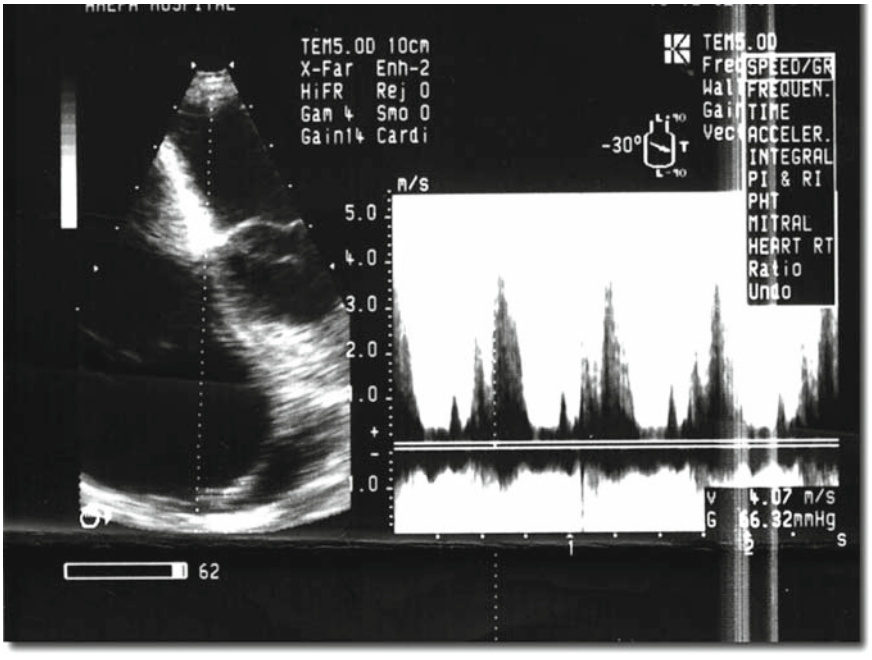

Fig. 4. Transesophageal echocardiography (TEE) findings: four-chamber view with small left ventricular cavity size, enormous enlargement of the right ventricle (RV) and right atrium (RA), and interventricular septum (IVS) flattening. Continuous-wave Doppler with tricuspid valve regurgitation, pressure gradient (PG), 66 mmHg

TEE findings were consistent with those obtained from TTE, we could not locate any large embolic material (Fig. 4). Due to the patient's poor hemodynamic and respiratory status, the idea of performing spiral computed tomography (CT) was abandoned, in order to avoid an extremely risky transfer to the CT suite. Consequently, the diagnosis of PTE was based mainly on clinical signs, PAC tracings, and indirect findings from the TTE and TEE, so a bolus of 5000 IU unfractionated heparin was administered, followed by continuous infu-

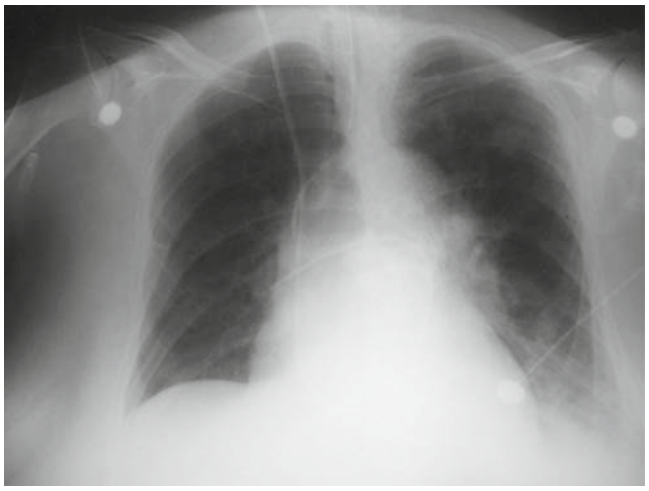

Fig. 5. Chest X-ray 3 days after the event, when the patient's condition had significantly improved

sion of 1000 IU · h^{-1}. Gradually, the hemodynamic performance of the systemic and pulmonary circulation was slightly improved, and the patient was transferred to the intensive care unit (ICU), 2 h after completion of the operation.

In the ICU, following a 2-day period of aggressive hemodynamic support, renal function deterioration, and poor systemic oxygenation, the patient had a quite uneventful clinical course, with gradual improvement of oxygenation and hemodynamic (pulmonary arterial pressure, 35/10 mmHg) performance, with concomitant normalization of chest X-ray (Fig. 5) and TEE findings. Unfractionated heparin was administered intravenously for 6 days and then low-molecular weight heparin was administered. After 14 days, the patient was discharged from the ICU in good clinical condition.

Discussion

Venous thromboembolism (VTE) is a major complication after abdominal surgery for cancer. The reported incidence of PTE for patients undergoing gynecologic abdominal surgery for malignancy is estimated to be approximately 4.1%, compared to 0.4% for nonabdominal or minor abdominal surgery [2]. Nevertheless, the incidence of intraoperative PTE during general abdominal surgery for cancer is unknown, as such cases have been rarely reported in the literature. Prolonged immobility, obesity, and age more than 60 years are considered as additional risk factors for VTE [3]. According to these data, our patient was stratified as being at high risk for VTE, so a single dose of enoxaparin 4000 IU anti-Xa was administered, preoperatively. This high dose is considered to reduce clinical VTE incidence by 71% and to be more effective than a low dose of unfractionated heparin in abdominal surgery (relative risk, 0.18) [4].

Acute intraoperative hypotension has multifactorial origins, such as anesthetic overdose, hypovolemia, pneumothorax, vena cava compression, myocardial dysfunction, anaphylaxis, and PTE. Regarding our patient, the initial hypotensive episode was attributed to blood loss, so it was treated with blood transfusion and inotropes. The ABG deterioration, seen 75 min later, could be attributed to hypoventilation and functional residual capacity (FRC) reduction. Furthermore, this situation could lead to an alteration of pulmonary vasculature status and a subsequent CVP elevation. However, the hemodynamic and ventilatory compromise during recovery from anesthesia alerted us towards a more serious complication, such as massive atelectasis, myocardial dysfunction, or PTE.

The absence of noteworthy radiographic signs of atelectatic regions, compared to findings on the preoperative chest X-ray, made the diagnosis of massive atelectasis unlikely. Furthermore, chest X-ray, PAC, and TTE findings were not supportive of left myocardial dysfunction. Consequently, the clinical suspicion of PTE became stronger.

Despite aggressive hemodynamic support, our patient's clinical status deteriorated. Transport to the angiography or CT suite, in order to achieve a definite diagnosis, could have been disastrous. Under these circumstances, TEE, because of its portability and image quality, was considered to be a rapid bedside diagnostic tool and an alternative to spiral CT and angiography for the evaluation of suspected PTE in the operating room. Previous studies have reported 80% sensitivity and 100% specificity for TEE, compared to angiography, for the diagnosis of PTE, and sensitivity and specificity comparable to those of spiral CT [5,6]. TEE has high sensitivity in revealing RV dysfunction, tricuspid regurgitation, and leftward interatrial septal bowing (96%, 50%, and 98%, respectively), which are indicative signs of PTE. Nevertheless, the sensitivity of TEE to diagnose acute PTE via direct visualization of emboli at any specific location is only 26%, while left PA thromboemboli are located in just 17% of PTE cases. Therefore, intraoperative TEE is of limited use to diagnose acute PTE, based on direct visualization of embolic material, while indirect TEE evidence of PA obstruction may be supportive of a diagnosis of PTE. This was the case in our patient, where, although intraoperative TEE did not reveal any thrombotic material in the right heart cavities or pulmonary vasculature, indirect findings, such as RV dilation, interventricular septal deviation, and decreased LV preload, in conjunction with the hemodynamic profile, hypoxemia, and widening of the Pa_{CO_2}-Et_{CO_2} dif-

ference were highly supportive of a diagnosis of acute PTE.

It is reported that the use of 3000 IU anti-Xa, 2 h prior to abdominal surgery, eliminates the risk of PTE [7,8]. Even though we adhered to all recommendations for VTE prevention (4000 IU anti-Xa enoxaparin administration and GCS use), and although the patient was initially stratified as being at high risk for PTE, these measures were inadequate to prevent such a serious complication from occurring.

Treatment of massive intraoperative PTE remains controversial. It is acknowledged that thrombolytic therapy is the treatment of choice in hemodynamically unstable patients [9, 10]. Nevertheless, a surgical procedure performed within 7 to 10 days is considered as a relative contraindication for thrombolytic therapy, and risks of early thrombolysis must be weighed against possible benefits [11,12].

In the literature there are several reported cases of acute symptomatic PTE that were successfully treated with intravenous unfractionated heparin [13,14]. Unlike the results with thrombolysis, uncontrolled hemorrhage is unlikely after heparinization, because disruption of the intrinsic coagulation cascade does not perturb the clot previously formed at the site of surgery [15,16]. In our patient, considering the absence of large emboli in the PA trunk, we directed our therapeutic approach towards heparin administration, combined with hemodynamic support [15].

In conclusion, massive intraoperative PTE is a rare but potentially lethal entity during abdominal operations, with an increased incidence in obese, elderly patients suffering from cancer. Moreover, the intraoperative period itself poses significant challenges, mainly due to limitations in definite diagnosis and therapeutic options. Based on these facts, clinical suspicion is often mandatory, so that diagnosis can be made and treatment can be established promptly.

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