

Case report

Systemic thrombolysis for acute massive pulmonary embolism in the immediate postoperative period after bariatric surgery

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Patients who undergo bariatric operations are at high risk of venous thromboembolism (VTE), with the incidence of perioperative pulmonary embolism (PE) estimated at .85–.9% [1,2]. VTE remains the leading cause of death of the bariatric surgical patient during the perioperative period [3]. PE accounts for 50% of deaths after laparoscopic or open gastric bypass (Roux-en-Y gastric bypass [RYGB]) [4].

The clinical severity of PE can be highly variable, ranging from asymptomatic to severe hypoxemia and shock. Massive PE, however, characterized by circulatory collapse, is rare, and, as such, randomized controlled trials on its optimal management are lacking. We present a case of the successful use of thrombolytic therapy for acute massive PE in the immediate postoperative period after laparoscopic RYGB in a super-obese patient, highlighting the necessity for prompt diagnosis and treatment.

Case report

A 45-year-old man with arterial hypertension, bronchial asthma, sleep apnea, and a longstanding history of morbid obesity (body mass index 55.6 kg/m²) underwent laparoscopic RYGB. He had neither a significant past surgical history nor a history of VTE.

After an appropriate preoperative evaluation, antecolic laparoscopic RYGB, with a linear stapled gastrojejunal anastomosis, was performed. The procedure was uneventful and lasted approximately 54 minutes. Because of the massive central obesity, the greater omentum was split to facilitate the antecolic placement of the Roux limb. The perioperative standardized antithrombotic protocol of our department consists of subcutaneous administration of enoxaparin sodium (4000 anti-Xa IU/4 mL, Clexane 40

mg), administered subcutaneously once preoperatively (the evening before surgery) and once postoperatively on a daily basis until the day of discharge, the use of graded elastic compression stockings, and early ambulation, which is initiated the evening of the operation.

On the second postoperative day, the patient began to receive sips of clear fluids by mouth. On postoperative day 4, the patient exhibited clinical signs of an abdominal disorder, with acute and intense pain on the right lower quadrant of the abdomen. The physical examination revealed tenderness and guarding on palpation. He had no fever, but significant tachycardia (115 bpm) and mild leukocytosis (11,900 white blood cells/ μ L) were exhibited. We decided to proceed to the operating room for laparoscopic exploration for a presumed leak. During laparoscopy, ischemia with patches of necrosis was discovered in a part of the greater omentum, which had been previously divided. No evidence of leak was found, and a partial omentectomy was performed. This was the first complication of its kind noted in our department after >2000 cases of laparoscopic RYGB. The second operation lasted approximately 52 minutes, and after extubation, the patient was transferred to the intensive care unit. He returned to the surgical floor the morning of the following day in good condition and was fully mobile.

Approximately 15 hours after his return to the surgical floor, the patient became suddenly dyspneic. He was tachycardic (110 bpm) and tachypneic (35 breaths/min), and a significant decrease in his arterial pressure (95/55 mm Hg) was noted. After an initial assessment, the patient was transferred to the intensive care unit. Because the diagnosis of PE was strongly suspected (D-dimer assay level of 2700 ng/mL), therapeutic dose heparin anticoagulation was started. The diagnosis of PE was confirmed 4 hours later by helical computed tomography pulmonary angiography using multidetector computed tomography. Extensive pulmo-

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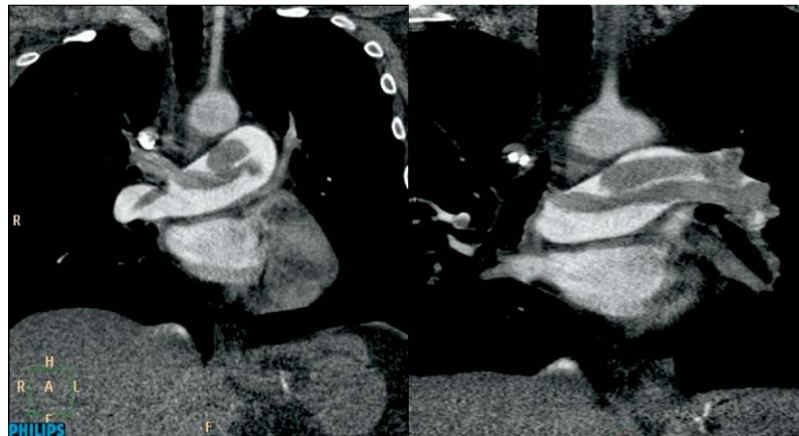


Fig. 1. Computed tomography pulmonary angiogram (coronal plane) demonstrating filling defects in right and left pulmonary arteries, diagnostic of extensive PE.

nary thromboembolism was revealed, with a near-total and a subtotal occlusion of the left and right pulmonary arteries, respectively (Fig. 1). The patient was hemodynamically unstable, and vigorous volume resuscitation with intravenous fluids and vasopressors was required (heart rate of 120 bpm and arterial pressure of 100/65 mm Hg). Arterial blood gas analysis (fraction of inspired oxygen 50%) revealed a base deficit of 6.5 mmol/L (pH 7.328, partial pressure of carbon dioxide in arterial gas 35.9 mm Hg, partial pressure of arterial oxygen 75.2 mm Hg). A continuous positive airway pressure mask was applied. However, despite all efforts, no major improvement in his respiratory function was observed. Echocardiography revealed right ventricular dilation and hypokinesis, as well as a severe elevation in pulmonary artery pressure (57 mm Hg).

The patient continued to remain in shock with an increased risk of early death due to the right ventricular enlargement. A decision was made to institute systemic thrombolytic therapy 5 hours after the onset of his symptoms. Embolectomy was not an option in our institution.

Heparin administration was discontinued, and an intravenous bolus infusion of alteplase 10 mg was administered, followed by 90 mg alteplase during a 2-hour infusion, after which, heparin was resumed. The patient tolerated the lytic therapy well, with no signs of hemorrhage or other complications. Follow-up echocardiography 24 hours later demonstrated a relief of the right ventricular overload with a pulmonary artery pressure of 30 mm Hg. His condition improved rapidly. He was transferred to the surgical floor 5 days later, after a follow-up computed tomography pulmonary angiogram demonstrated significant thrombus reduction in both pulmonary arteries (Fig. 2). On the 15th post-operative day, he was discharged in good general condition and with a vitamin K antagonist.

Discussion

Obesity constitutes a major risk factor for thromboembolic events [5]. Weight-related obesity changes such as the increase in blood volume, intra-abdominal pressure, and

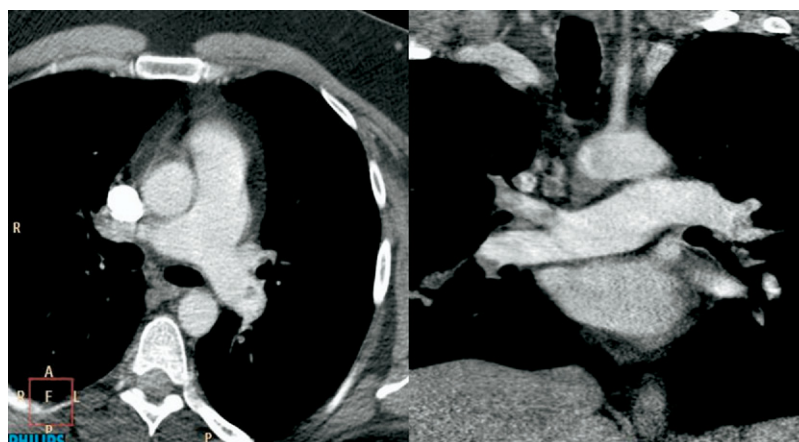


Fig. 2. Post-thrombolysis computed tomography scan (axial and coronal plane) demonstrating thrombus resolution.

intrathoracic pressure predispose to thrombogenesis. Moreover, thrombogenesis is a part of the inflammatory changes associated with obesity [6]. The risk for VTE in bariatric patients is multifactorial. Patients have a period of activated coagulation, transient depression of fibrinolysis, and temporary immobilization. Laparoscopic techniques might add to this risk, because, with the increased intra-abdominal pressure, the inferior vena cava is compressed, and thus, venous return from the lower extremities is diminished [7]. However, recent data have demonstrated equal or an even lower incidence of PE and VTE events in laparoscopic bariatric surgery compared with open surgery [1,4,5,8,9].

Unfractionated heparin, low-molecular-weight heparin, and mechanical prophylaxis have proved to be effective for the prevention of VTE in various clinical settings [10]. Currently, no consensus has been reached regarding the best method of prophylaxis against VTE in morbidly obese patients undergoing bariatric surgery [6]. It is also unclear whether low-molecular-weight heparins are the most efficacious using standard regimens or weight-based protocols [1]. Prophylactic placement of inferior vena cava filters might benefit bariatric patients with a history of VTE, evidence of venous stasis, or pulmonary hypertension, as well as super-obese patients with limited mobility [1,11].

PE is associated with a mortality rate exceeding 15%, primarily as a result of recurrent embolism [12]. However, an accurate diagnosis followed by effective therapy improves survival among patients with symptomatic PE [10]. Thus, it is imperative that effective therapy should be instituted as quickly as possible.

The role of thrombolytic therapy for acute PE has long been debated. Thrombolytic therapy can potentially achieve faster clot lysis, reverse the hemodynamic consequences of PE, and be lifesaving. Compared with heparin-treated patients, patients treated with thrombolysis showed a significant reduction in mortality, as demonstrated by a subgroup analysis of 5 trials that included patients with massive PE and shock. However, the benefit of thrombolysis was lost in the 6 trials that excluded hemodynamically unstable patients [13]. In patients with massive PE, who were nonrandomly enrolled in the International Cooperative Pulmonary Embolism Registry, thrombolysis did not appear to reduce mortality at 90 days compared with heparin alone [14]. Thrombolysis is generally recommended for patients with massive PE, arterial hypotension, and/or shock [15]. Hemodynamically stable patients with acute PE and right ventricular dysfunction might also benefit from thrombolysis [16]. However, the use of thrombolytic agents in this setting remains controversial [13,17].

The regimens that can be used include streptokinase, urokinase, and tissue plasminogen activators (e.g., alteplase). Thrombolysis is usually applied systemically. The optimal dosing regimen for alteplase in PE has not been determined. Instead, various protocols of administration have been used. Evidence of catheter-directed, thrombolytic therapy for acute

PE is very scarce. Although often relative, contraindications to thrombolysis should be observed. Contraindications such as recent major surgery (in the past 2 weeks), active or recent major bleeding, pregnancy, and clinically obvious risks of bleeding could preclude the use of thrombolysis [10]. However, under extreme circumstances, such as in our patient, the contraindications to thrombolysis should be weighed against the potential benefit. The most important complication of thrombolytic therapy is hemorrhage, with the most devastating being intracranial hemorrhage. In a recent meta-analysis, the incidence of intracranial hemorrhage was not increased in the thrombolysis group when compared with heparin alone [13]. Moreover, recent data from a Cochrane collaboration review confirmed the nonsignificant increase in the risk of major and nonmajor bleeding after thrombolytic therapy for PE [17].

Conclusion

Massive PE can result in a catastrophe if prompt diagnosis and therapy are not introduced. Thrombolysis is an established therapy for patients with massive PE. Systemic thrombolysis, despite its limitations, is a key element in our arsenal against acute massive PE in the postoperative period after bariatric surgery. Therefore, bariatric surgeons should be aware of this potentially lifesaving therapeutic option.

Disclosures

The authors claim no commercial associations that might be a conflict of interest in relation to this article.

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