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ECPR for cardiac arrest caused by postoperative massive pulmonary embolism in a super-morbidly obese patient: A case report

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ABSTRACT:

Introduction: Massive pulmonary embolism (PE) remains a leading cause of preventable perioperative mortality after bariatric surgery. Cardiopulmonary resuscitation (CPR) is challenging in patients with extreme obesity, and extra-corporeal cardiopulmonary resuscitation (ECPR) is increasingly used as a salvage therapy.

Case presentation: A 60-year-old woman with super-super obesity (body weight: 142 kg; body mass index [BMI]: 61 kg/m²) suffered pulseless electrical activity (PEA) cardiac arrest on postoperative day (POD) 3 following elective robotic Roux-en-Y gastric bypass.

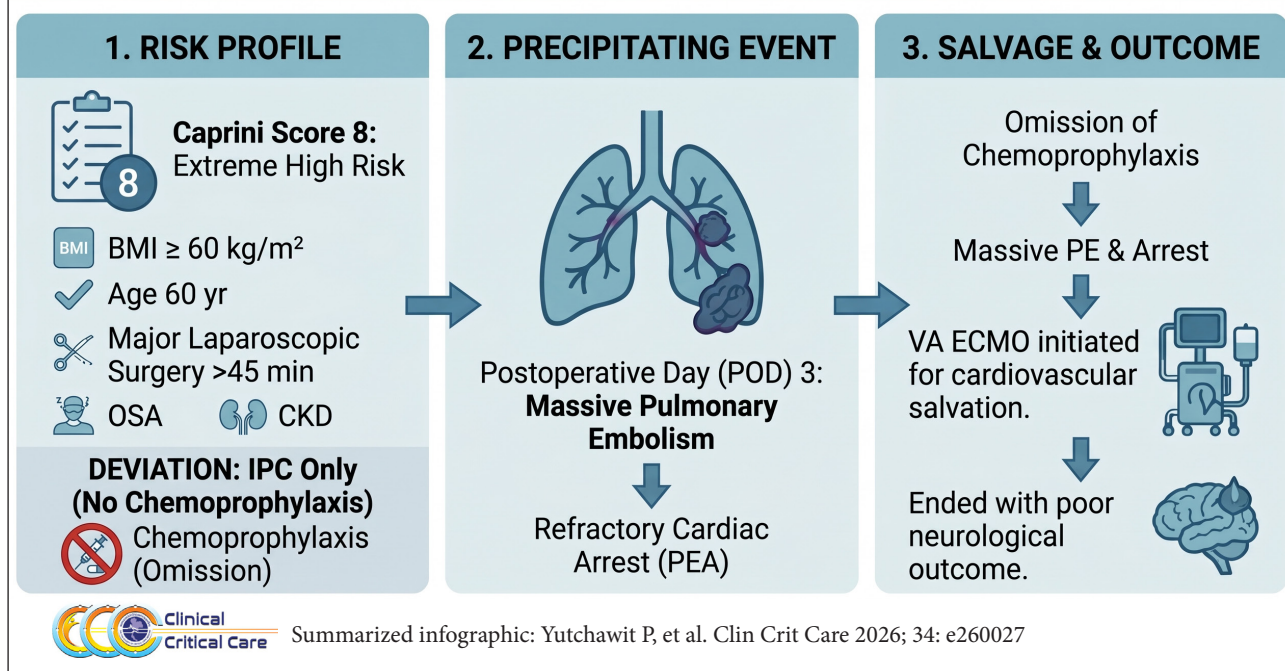
Management: ECPR was initiated using veno-arterial extracorporeal membrane oxygenation (VA-ECMO). Cannulation was difficult due to her extreme body habitus, resulting in a prolonged low-flow time of 85 minutes to reach target flow. Bedside echocardiography and pulmonary artery catheterization confirmed massive obstructive shock. Thrombolysis was deferred due to bleeding risks from the fresh bariatric incision, and she was successfully managed with conservative anticoagulation. Her ICU course was complicated by new-onset atrial fibrillation, septic shock, and Harlequin syndrome, all of which were successfully resolved.

Outcome: Although cardiopulmonary function recovered and follow-up computed tomographic pulmonary angiography showed complete thrombus resolution, she sustained severe hypoxic-ischemic encephalopathy (HIE) and died one year later.

Conclusion: Standardized adherence to combined mechanical and pharmacologic venous thromboembolism (VTE) prophylaxis is essential in patients with super-super obesity. While ECPR can restore systemic circulation, technical challenges can prolong low-flow time and compromise neurological outcomes.

Keywords: Bariatric surgery; Pulmonary embolism; ECPR; Super-super obesity; Hypoxic-ischemic encephalopathy

POST-BARIATRIC MASSIVE PE & SALVAGE WITH ECPR



INTRODUCTION

Venous thromboembolism (VTE) is a leading cause of preventable perioperative mortality after bariatric surgery [1], particularly in patients with super-super morbid obesity (defined as body mass index [BMI] $\geq 60 \text{ kg/m}^2$) who have an exceptionally high baseline thrombotic risk [2]. When acute PE precipitates cardiac arrest in this population, standard cardiopulmonary resuscitation (CPR) is mechanically challenging and associated with survival-to-discharge rates of less than 10%. [3]. Extracorporeal cardiopulmonary resuscitation (ECPR) using veno-arterial extracorporeal membrane oxygenation (VA-ECMO) has emerged as a crucial rescue treatment to restore systemic circulation and facilitate RV recovery, with reported survival-to-discharge rates of 30-40%. [4]

However, executing ECPR in patients with super-super obesity is technically challenging. The immense depth of subcutaneous tissue complicates vascular access, potentially prolonging low-flow time and compromising neurological recovery. [4, 5]

This case report describes the clinical course of a patient with super-super obesity who suffered a massive postoperative PE and was rescued with ECPR. We highlight the critical technical challenges of ECPR in extreme obesity, analyze the diagnostic sequence, and discuss the impact of omitting standard pharmacologic prophylaxis.

CASE PRESENTATION

A 60-year-old Thai woman with super-super obesity (142 kg; 152 cm; BMI 61 kg/m^2) presented for elective robotic Roux-en-Y gastric bypass (RYGB). Her comorbidities included chronic kidney disease stage 3b (baseline creati-

KEY MESSAGES:

- Omitting pharmacologic VTE prophylaxis in super-super obesity post-bariatric surgery risks catastrophic massive pulmonary embolism.
- ECMO cannulation challenges in extreme body habitus can prolong low-flow duration, compromising neurological outcome.
- Institutions must enforce mandatory chemoprophylaxis and maintain obesity-specific ECPR cannulation protocols to protect high-risk patients.

nine 1.05 mg/dL), hypertension, dyslipidemia, impaired fasting glucose (HbA1c 6.0%), and severe obstructive sleep apnea. Preoperative transthoracic echocardiography demonstrated a preserved left ventricular ejection fraction (LVEF) of 65%, mild left atrial enlargement, and mild pulmonary hypertension with mean pulmonary artery pressure (mPAP) of 24 mmHg. A lower-extremity duplex ultrasonography performed one year preoperatively was negative for DVT. However, this screening was too remote to be clinically relevant to her acute preoperative risk and could not exclude interval or acute DVT formation. Her preoperative Caprini risk score was calculated as 8, classifying her as an extreme-high-risk patient. However, no pharmacologic VTE prophylaxis was administered perioperatively. The medical records contained no documented clinical contraindications (e.g., active bleeding or thrombocytopenia) to explain this omission.

The robotic RYGB lasted 3 hours and 10 minutes uneventfully. Intermittent pneumatic compression was used intraoperatively and through postoperative day (POD 1). She was cleared for discharge on POD 3 with a cumulative fluid balance of 6,224 mL by POD3.

Immediately before discharge, she ambulated to the restroom and was found unresponsive approximately 10 minutes later (10:50 AM). Transferring her back to the hospital bed required the coordinated efforts of 10 staff members, introducing a crucial treatment delay (no-flow time: 10 minutes). Pulseless electrical activity was identified and CPR initiated (11:05 AM). After 20 minutes of CPR, a transient return of spontaneous circulation (ROSC) was achieved. However, she re-arrested into PEA minutes later, prompting ECPR activation at 11:35 AM. Bedside percutaneous femoral-femoral cannulation was performed under real-time ultrasound guidance using a 17 Fr arterial cannula and a 21 Fr venous cannula despite technically difficult cannulation. Low-flow VA-ECMO support was initiated at 12:15 PM (70 minutes low-flow duration), and full target flow was established at 12:30 PM (85 minutes total low-flow time from arrest). She was transferred to the ICU on ECMO with vasoactive support.

The timeline for cardiac arrest and extracorporeal cardiopulmonary resuscitation (ECPR) is summarized in Table 1.

ICU course and management

At the ICU, the patient was on full VA-ECMO support, achieving a flow of 2.5 L/min. Her body surface area (BSA) was calculated as 2.44 m² (using the Du Bois formula). The achieved flow of 2.5 L/min corresponded to an indexed flow of 1.02 L/min/m². She was deeply comatose with a Glasgow Coma Scale (GCS) of 3 (E1VTM1) and sluggishly reactive pupils. Her initial arterial lactate was 2.8 mmol/L. To maintain systemic perfusion, her vasoactive regimen was aggressively titrated based on actual body weight (142 kg): norepinephrine at 0.07 mcg/kg/min, epinephrine at 0.16 mcg/kg/min, and dobutamine at 3.5 mcg/kg/min. Transesophageal echocardiography demonstrated severe biventricular dilation with near-absent systolic function and spontaneous echo contrast, consistent with acute ob-

structive shock possibly from cor pulmonale. Pulmonary artery catheterization confirmed markedly elevated pulmonary artery pressures with normal wedge pressure. (CVP 12 mmHg, PAP 64/34 mmHg, PCWP 11 mmHg, BP 110/83 mmHg) Bilateral duplex ultrasonography revealed extensive DVT. Intravenous unfractionated heparin was initiated. Computed tomographic pulmonary angiography confirmed massive bilateral pulmonary embolism with right ventricular strain (Figure 1.)

Despite multiorgan dysfunction, gradual hemodynamic stabilization allowed progressive reduction of vasoactive support. A multidisciplinary team (MDT) comprising bariatric surgeons, cardiologists, and intensivists convened to determine the reperfusion strategy. Systemic thrombolysis was contraindicated due to the high risk of catastrophic intra-abdominal hemorrhage from her fresh bariatric staple line on POD 3. Catheter-directed therapy (CDT) was deferred because of distal clot extension, and surgical embolectomy was ruled out due to profound hemodynamic instability and extreme body habitus; anticoagulation was continued to achieve aPTT ratio of 1.5-2.5. Targeted temperature management (34–35°C) was maintained for 72 hours.

Her ICU course was complicated by atrial fibrillation, septic shock, and differential hypoxemia during ECMO weaning. After respiratory optimization, she was successfully decannulated from VA-ECMO on POD 8 with recovery of renal function.

Patient's pre-operative Caprini risk score

Total score = 8 points

Each risk factor represents 1 point

- ☐ / ☐ Age 41-60 years
- ☐ Minor surgery planned
- ☐ History of prior major surgery (<1 month)
- ☐ / ☐ Varicose veins
- ☐ History of inflammatory bowel disease
- ☐ Swollen legs (current)
- ☐ / ☐ Obesity (BMI >25)
- ☐ Acute myocardial infarction
- ☐ Congestive heart failure(<1 month)

Table 1. Cardiac arrest and ECPR timeline.

Time	Event	Intervention & Details
10:50 AM	Found unresponsive in restroom	Bed transfer delayed due to extreme body habitus (No-flow time: 10 mins).
11:05 AM	PEA arrest confirmed; CPR started	High-quality chest compressions; endotracheal intubation.
11:05–11:25 AM	Refractory PEA	Adrenaline 1 mg IV x 3, Calcium gluconate 2 g IV, NaHCO ₃ 50 mL IV administered.
11:25 AM	Transient ROSC achieved	Pulse present; preparation for ICU transfer.
11:30 AM	Re-arrest into PEA	CPR resumed. Endotracheal Adrenaline 2 mg x 5 given due to temporary loss of IV access.*
11:35 AM	ECPR protocol activated	Cardiovascular surgery team summoned to bedside.
11:45–12:15 PM	Bedside percutaneous cannulation	Percutaneous femoral-femoral cannulation under ultrasound guidance (17 Fr arterial, 21 Fr venous).

*Note: Endotracheal adrenaline administration was utilized as a non-standard rescue route during temporary loss of venous access, as supported by legacy resuscitation guidelines. Abbreviations: PEA: Pulseless electrical activity; CPR: Cardiopulmonary resuscitation; mg: Milligram; g: Gram; IV: Intravenous; ROSC: Return of spontaneous circulation; ETT: Endotracheal tube; ECPR: Extracorporeal cardiopulmonary resuscitation; VA-ECMO: Veno-arterial extracorporeal membrane oxygenation; L/min: Liter per minute; RPM: Revolutions per minute; ICU: Intensive care unit.

- ☐ Sepsis (<1 month)
- ☐ Serious lung disease incl. pneumonia (<1 month)
- ☐ Abnormal pulmonary function (COPD)
- ☐ Medical patient currently at bed rest

Only woman

- ☐ Oral contraceptives or hormone replacement therapy
- ☐ Pregnancy or postpartum (<1 month)
- ☐ History of unexplained stillborn infant, recurrent spontaneous abortion (≥ 3), premature birth with toxemia or growth-restricted infant

Each risk factor represents 2 points

- ☐ Age 60–74 years
- ☐ Arthroscopic surgery
- ☐ Malignancy (present or previous)
- ☐ Major surgery (>45 minutes)
- ☐ Laparoscopic surgery (>45 minutes)
- ☐ Patient confined to bed (>72 hours)
- ☐ Immobilizing plaster cast (<1 month)
- ☐ Central venous access

Each risk factor represents 3 points

- ☐ Age over 75 years
- ☐ History of DVT/PE
- ☐ Family history of thrombosis
- ☐ Positive factor V Leiden
- ☐ Positive prothrombin 20210A
- ☐ Elevated serum homocysteine
- ☐ Positive lupus anticoagulant
- ☐ Elevated anticardiolipin antibodies
- ☐ Heparin-induced thrombocytopenia (HIT)
- ☐ Other congenital or acquired thrombophilia

Each risk factor represents 5 points

- ☐ Elective major lower extremity arthroplasty
- ☐ Hip, pelvis or leg fracture (<1 month)
- ☐ Stroke (<1 month)
- ☐ Multiple trauma (<1 month)
- ☐ Acute spinal cord injury (paralysis) (<1 month)

OUTCOME

Although cardiopulmonary function recovered and follow-up imaging demonstrated near-complete thrombus resolution, the patient failed to regain consciousness. She developed severe hypoxic–ischemic encephalopathy (HIE) secondary to massive pulmonary embolism and prolonged cardiac arrest on POD5, with EEG showing a severely suppressed background pattern, compounded by delayed achievement of adequate ECMO flow. After a prolonged hospital course complicated by recurrent pneumonia requiring tracheostomy and in a persistent vegetative state, she died approximately one year after the arrest.

DISCUSSION

This patient had multiple risk factors for fatal perioperative pulmonary embolism, including BMI $>60 \text{ kg/m}^2$, prolonged laparoscopic bariatric surgery, obstructive sleep apnea with pulmonary hypertension, and limited mobility [5–9]. According to the American Society for Metabolic and Bariatric Surgery (ASMBS) position statement [5], patients with a Caprini score greater than 5 (as this patient = 8 points) should receive combined mechanical and pharmacologic prophylaxis unless contraindicated. In contrast, mechanical prophylaxis alone is recommended when the bleeding risk precludes chemoprophylaxis. In this case, only mechanical DVT prophylaxis in the absence of chemoprophylaxis may have contributed to the catastrophic event.

A DVT screen performed one year prior could not reliably exclude interval thrombosis.

The risk of VTE after bariatric surgery remains highest in the early postoperative period, with a cumulative incidence up to 1.9% within 30 days, often after discharge [7]. This case underscores the need for institutional quality improvement initiatives in VTE prophylaxis protocols [10].

Cardiac arrest from massive pulmonary embolism carries a high risk of hypoxic–ischemic encephalopathy, particularly when systemic perfusion is not rapidly restored. Although ECMO was initiated within 30 minutes, maximal

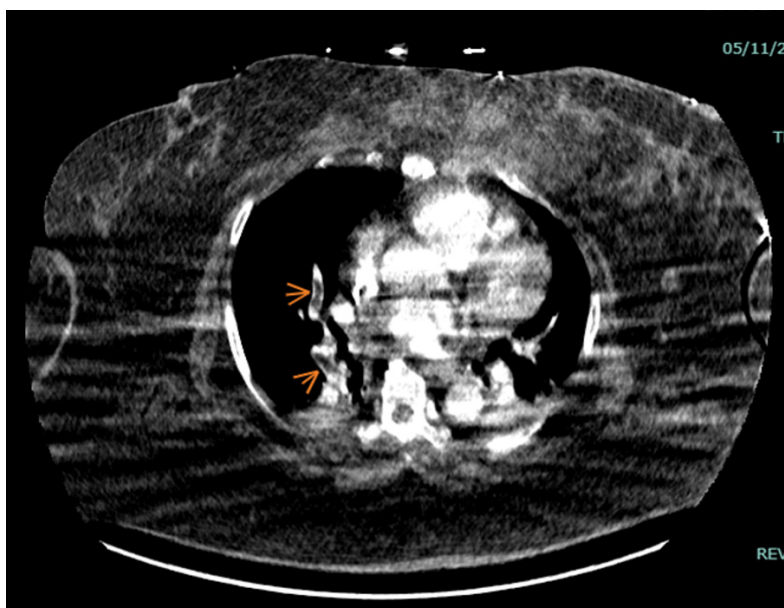


Figure 1. Computed tomographic pulmonary angiography (CTPA) demonstrated a massive acute pulmonary embolism involving the right interlobar artery with extension into the right middle and lower lobar branches, as well as thrombus within the left interlobar artery extending into the left lower lobar and lingular segmental branches.

Table 2. Literature review of ECPR for massive PE after bariatric surgery

Author & Year	Patient BMI (kg/m ²)	Arrest rhythm	Low-flow time (min)	Complications	Neurological outcome
Meneveau N et al., 2018 [11]	45	VF	45	Access site bleeding	Discharged home, intact
Pai et al., 2025 [4]	52	PEA	60	Acute kidney injury	Discharged to rehab, mild deficit
Current Case, 2026	61	PEA	85	Harlequin syndrome, AFib	Persistent vegetative state

flow was achieved after 85 minutes due to technical challenges related to extreme obesity. Prolonged low-flow time exceeded the optimal 60-minute window for neurological preservation in ECPR [11].

Super-morbid obesity further complicates ECMO through difficult cannulation, increased risk of mechanical complications, and complex pharmacokinetics affecting anticoagulation and vasoactive therapy. [12,13] These factors collectively increase procedural difficulty and neurologic risk in this population.

Lessons learned

- Institutional Protocol Adherence: Implementation of mandatory, EHR-triggered chemoprophylaxis orders for high-risk Caprini scores (≥ 5) is essential.
- Remote Screening Limitations: Preoperative DVT screening performed remotely (>1 month) must not be used to withhold perioperative chemoprophylaxis.
- ECMO Cannulation Preparedness: Bariatric centers must maintain obesity-specific cannulation kits, ultrasound protocols, and specialized teams to minimize low-flow times in extreme obesity.

CONCLUSION

This case highlights the catastrophic consequences of omitting pharmacologic VTE prophylaxis in super-super-obesity bariatric surgery. In cardiac arrest due to massive pulmonary embolism, early and effective ECMO support may restore circulation and RV function. Cannulation challenges in extreme body habitus can prolong low-flow duration, compromising neurological outcome.

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