Journal of
Clinical & Medical Surgery

www.jclinmedsurgery.com

ISSN 2833-5465
Open Access
Volume 6

Case Report

Case report on effective cardiopulmonary resuscitation in an orthopedic case due to pulmonary embolism with peripheral arterial disease

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Article Information

Received: Jan 28, 2026
Accepted: Feb 19, 2026
Published: Feb 26, 2026
Archived: www.jclinmedsurgery.com

Abstract...

Pulmonary Embolism (PE) is a common condition, but this case discusses its occurrence during general anaesthesia. The patient was scheduled for spinal fixation under general anaesthesia when a sudden pulmonary embolism and cardiac arrest occurred shortly after induction. The patient was successfully resuscitated. Diagnosis was confirmed through Computed Tomography Angiography (CTA), blood investigations, ECG, and echocardiography. CTA revealed peripheral vascular disease with occlusion of major lower limb vessels. This case highlights that massive PE is life-threatening and emphasizes the importance of proper preoperative evaluation for Deep Vein Thrombosis (DVT), early diagnosis, and timely intervention.

Background

While numerous cases of pulmonary embolism are reported in the literature, perioperative PE remains rare. Indian data suggest an overall incidence of PE in adults at 15.9%, with PE contributing to 80% of mortalities among such patients [1]. The incidence of perioperative PE is higher compared to non-operative settings. Perioperative PE is particularly hazardous, as it presents with varied symptoms, making early diagnosis challenging. Failure to detect itpromptly often leads to fatal outcomes [2].

Case presentation

We report the case of a 74-year-old female patient with a history of hypertension, lumbar L5-S1 intervertebral disc prolapse with listhesis, and radiculopathy, who was scheduled for discectomy and spine fixation.

The patient had a history of intestinal liposarcoma surgeries in 2013 and 2021, followed by radiation therapy. She had been bedridden for 15 days due to severe back pain and had received a transforaminal epidural steroid injection. Her medical history included hypertension and type II diabetes mellitus. There was no history of DVT or family history of sudden cardiac death.

The patient, categorized as ASA III, exhibited normal pulse and blood pressure.

Preoperative findings

- Blood investigations: TC - 15,300 per cumm, ESR - 80 mm/hr, Hb - 12.8 gm/dl,
- Platelet count - 4.7 lakhs/cumm, PT/INR - 1.00, RBS - 126 mg/dL, Sr
- Creatinine - 1.2 mg/dl, LFT – within normal limit, Na - 130.2 meq/l, K^+ - 3.60 meq/l, Ca^{++} - 10.5 mg/dl
- ECG: Incomplete RBBB and sinus tachycardia.
- Echocardiography: Mild concentric LVH, trivial AR, LVEF - 60%, no RWMA.
- Radiological findings: Chest X-ray - Within normal limits.
- Lumbar spine MRI - L5-S1 disc compression and listhesis.

Citation: Gogoi D, Dutta A. Case report on effective cardiopulmonary resuscitation in an orthopedic case due to pulmonary embolism with peripheral arterial disease. J Clin Med Surgery. 2026; 6(1): 1225.

On the day of surgery

The patient's vital signs were stable. Preoperative management included IV cannulation, administration of Ringer's lactate, and premedications (glycopyrrolate, ondansetron, pantoprazole). Induction was achieved using propofol, midazolam, fentanyl, and rocuronium. Anaesthesia was maintained with sevoflurane, and a flexometallic tube (size 7) was inserted smoothly. Initial readings: SpO_2-100%, $EtCO_2$-35-40 mmHg, pulse - 92 bpm, BP-110/60 mmHg Intraoperative Event Approximately 10-15 minutes post-induction, the patient experienced sudden desaturation, hypocapnia, and a blood pressure drop to 90/45 mmHg, followed by Pulseless Electrical Activity (PEA) and a silent chest.

Management and resuscitation

- CPR was initiated as per ACLS protocol.
- One ampoule of adrenaline was administered intravenously.
- After 5-6 minutes of quality CPR, sinus rhythm was restored, carotid pulse became palpable, and SpO_2 and $EtCO_2$ normalized.
- Return of Spontaneous Circulation (ROSC) occurred, and 200 mg of sugammadex was administered intravenously.
- The patient was extubated and kept under observation.

gPost-Event investigations

- Point-of-care ultrasound: Thrombus detected in the right femoral artery and vein.
- ECG: RAD with ST elevation in inferior leads and V3-V6, RBBB observed.
- Bedside echocardiography: No RWMA, normal pulmonary artery pressures, and no right ventricular dilatation.
- Blood Gas Analysis (ABG): pH-7.401, pCO_2-44.6 mmHg, pO_2-338.3 mmHg, HCO_3-27.7 mmol/L.
- Post-arrest labs: TC-28,000 percumm, Hb-11.8 gm/dl, serum creatinine - 1.3 mg/dL, D-Dimer >10,000 ng/mL.
- Cardiac angiography: LAD - 50% occlusion, OM - 60-70%, RCA - mild diffuse calcification.
- Peripheral angiogram: Complete occlusion of the right internal and external iliac vessels, partial occlusion of the left internal iliac vessels.

Post-resuscitation care

The patient was transferred to the ICU and treated with:

- Oxygen supplement by face mask
- Ecosprin and clopidogrel loading doses.
- Low molecular weight hearin
- Fluid resuscitation and inotropic support.
- DVT mechanical support

Discussion

We could manage the case without ventilatory support / ECMO as in other studies. Here our patient presented with desaturation, hypocapnia, and a silent chest and unrecordable BP and pulse. PE may present with silent chest as acute bronchial asthma crisis [3]. Pharmacologically-active substances may be released at thrombo-embolic sites, leading to the excitation of specific chemoreflexes [4]. Such agents include acetylcholine, histamine, serotonin, and the plasma kinins, all of which have bronchoconstrictor properties, suggesting that bronchoconstriction occurring with pulmonary emboli may be related to the presence of a substance whose activity or release is blocked by heparin, as evidence of bronchoconstriction in selected patients appears to represent an objective and sensitive, although nonspecific, index of PE. A massive PE may mimic ACS, with chest pain, hypotension, tachycardia, hypoxia. Such features are S1Q3T3, complete RBBB, TWI, ST-segment depression in V4 through V6, STE-V1, STE-III, Qr-V1, RAD, AF, and RV transmural ischemic pattern [5].

In massive APE the ST elevations in lead aVR is frequently seen, and is associated with an overall worse prognosis. Sudden death due to PE can occur in the presence of ACS, developing ventricular arrhythmia, or during non-shockable rhythm. Obstruction of the pulmonary trunk or other large pulmonary vessels, coupled with the release of vasoconstrictive mediators from thrombotic tissue, leads to a rapid increase in right ventricular afterload. The resultant decrease in venous return to the left heart, and subsequent reduction in left ventricular end diastolic volume, is further exacerbated by the pressure related leftward shift of the interventricular septum. As a result, circulatory shock and eventually cardiac arrest ensues [6].

The cause of cardiac arrest is not easily recognized, especially when the initial rhythm is different from ventricular fibrillation; in the majority of cases, the mechanism of cardiac arrest in massive PE is Pulseless Electrical Activity (PEA) [7]. The various Point of Care Ultrasound (POCUS) protocols that have been described to assess the hemodynamically unstable patient in the, also include asearch for the presence of deep vein thrombosis and pulmonary embolism [8]. The presence of DVT, right heart thrombi and other suggestive features of PE such as increased pulmonary artery or right ventricular pressures, right ventricular dilatation, tricuspid regurgitation and interventricular septal deviation with the aid of POCUS [9,10].

Peripheral Arterial Diseases (PAD) overall confers a modest increase in VTE risk compared to patients without PAD. In Peripheral Arterial Disease, endothelial dysfunction secondary to widespread vascular dysregulation, decreased nitric oxide bioavailability, and inhibition of endothelial nitric oxide synthase have been implicated [11].

Endothelial dysfunction, one of the three crucial pillars for thrombus formation, is also a key pathophysiologic process of PAD. The subsequent arterial calcification process is likewise thought to result from widespread derangement of inflammatory cytokines, cholesterol transport, and very low-density lipoprotein metabolism. PAD can manifest with either low ABI (<1.0), due to atherosclerosis, or elevated ABI (>1.4), due to arteries and arterial calcification Immobility is a key risk factor for VTE and is incorporated in the widely validated Wells' Criteria

for DVT risk prediction [11]. It is important to consider that patients with severe PAD may be highly debilitated and immobile due to claudication, further increasing their VTE risk.

CTPA is considered the gold standard technique for diagnosing patient with suspected acute PE. But negative CTPA generally means that the test did not detect any blood clots in the pulmonary arteries at that time of imaging which may be due to many factors both technical and clinical [12]. Current PE management guidelines do not recommend specific treatments for patients with high-risk perioperative PE [13].

Differential Diagnosis of this case are pulmonary embolism, acute myocardial infarction, aortic dissection, pericardial tamponade.

Summary

Pulmonary embolisms are the third most important cause of cardiovascular death, particularly in high-risk patients with right ventricular dysfunction and elevated cardiac biomarkers. Peripheral artery disease accounts for an important part of morbidity and hospitalizations in cardiovascular patients, and the mortality associated with it—in contrast to coronary disease—is not falling. Pulmonary embolisms are the third most important cause of cardiovascular death, particularly in high-risk patients with right ventricular dysfunction and elevated cardiac biomarkers. Peripheral artery disease accounts for an important part of morbidity and hospitalizations in cardiovascular patients, and the mortality associated with it—in contrast to coronary disease—is not falling.

PAD itself doesnot lead to pulmonary embolism but the shared risk factors between the two condition is present.

Pulmonary Embolism (PE) can be a severe disease and is difficult to diagnose, given its nonspecific signs and symptoms. Because of this, testing patients with suspected acute PE has increased dramatically [14].

Patient with high risk for thrombosis like malignancy, bed ridden should be evaluated for deep vein thrombosis and impending PE by POCUS. In our case, all the findings following cardiac arrest favour PE, except negative CTPA which showed no pulmonary embolus. It showed all occlusion of major vessels of lower limbs and cardiac vessels. Our ECG, echocardiography and D-Dimer suggest embolism.

Conclusion

So, in orthopaedic cases with past history of malignancy and prolonged bedrest it should be mandatory to do the screening of DVT, angiography to rule out any embolic phenomenon in perioperative period.

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