Journal of the
Bahrain Medical Society

Butt M et al., J Bahrain Med Soc. 2022;34(2):57-61

CASE REPORT

Management of Acute Massive Pulmonary Embolism During Pharmaco-mechanical Thrombectomy for Acute DVT: A Case Report

Mehtash Butt[1,*], Rashad Al Qasim[2], Dhafer M Kamal[3]

[1]Senior Resident, department of anesthesia & ICU, Royal medical services Bahrain defense forces hospital. PO Box 50710; Tel: 0097336681277; E-mail: dr.mehtashbutt@yahoo.com

[2]Senior consultant, Department of anesthesia & ICU, Royal medical services Bahrain defense forces hospital. Add: Villa 248, Road 4303, Block 943; Tel: 0097333336970; E-mail: bahrainrashadalqasim@hotmail.com

[3]Senior Consultant Vascular and Endovascular Surgeon, Bahrain Defense Force Royal Medical Services Associate Professor of Surgery, The Royal College of Surgeons of Ireland - Medical University of Bahrain. Add: Villa 2473G, Road 4575, Block 745, Sanad, Kingdom of Bahrain. Tel: 0097336466300; E-mail: dhaferkamal@hotmail.com

***Corresponding author:**
Dr. Mehtash Butt, Senior Resident, department of anesthesia & ICU, Royal medical services Bahrain defense forces hospital. PO Box 50710, Tel: 0097336681277; E-mail:dr.mehtashbutt@yahoo.com

Received date: April 19, 2022; **Accepted date:** August 30, 2022; **Published date:** December 31, 2022

Abstract

This case report describes an incident of MPE during lower limb endovascular pharmaco-mechanical thrombectomy under sedation, which was immediately identified and promptly managed with cardiopulmonary resuscitation. After return of spontaneous circulation, ECMO was initiated, as her haemodynamics were unstable, and MPE was diagnosed based on transoesophageal echocardiography findings. During intensive care unit stay, she was successfully weaned off from ECMO and ventilatory support. However, the patient developed right-sided body weakness. Echocardiography showed a patent foramen ovale, and stroke due to paradoxical embolism was diagnosed. She was transferred to the ward in a stable condition and later discharged home.

Abbreviations: MPE: Massive pulmonary embolism, ECMO: extracorporeal membrane oxygenation.

Keywords: MPE: Massive pulmonary embolism, ECMO: extracorporeal membrane oxygenation, PFO: patent foramen ovale, PMT: pharmaco-mechanical thrombectomy, Sedation

Introduction

Acute pulmonary embolism (APE) is a medical emergency with significant morbidity and mortality, accounting for more than 500,000 deaths per year in Europe.[1] In the United Kingdom, the annual incidence of APE is 60–70/100,000, and approximately 25,000 patients die every year from preventable acquired venous thromboembolism (VTE).[2] There is a 100-fold increased risk of VTE in surgical patients.[3] The mortality rate of patients with massive pulmonary embolism (MPE) with cardiac arrest on arrival is 95%; those with out-of-hospital cardiac arrest, 85%; those receiving mechanical ventilation, 80%; and those who received cardiopulmonary resuscitation within the first 24 hours, 77 %.[4] The present case report describes

an incident of massive APE during right lower limb percutaneous pharmaco-mechanical thrombectomy (PMT) for symptomatic acute ilio-femoral-popliteal venous thrombosis.

Consent for publication

Written informed consent for publication of this case report was obtained from the patient.

Case presentation

A 47-year-old woman with American Society of Anesthesiologists physical status II was scheduled for right lower limb percutaneous PMT for the treatment of provoked acute deep venous thrombosis (DVT). Seven days prior, the patient had fractured her fifth metatarsal bone on the right foot. The DVT was thought to be caused by either the fracture itself or too tight cast application after open reduction and internal fixation (ORIF). The patient had a medical history of mild anaemia and gastro-oesophageal reflux disease. She had no drug or food allergy and was a non-smoker and non-alcoholic drinker. Her laboratory and chest radiography findings were normal, and electrocardiography showed a normal sinus rhythm.

Doppler ultrasound showed DVT over the common femoral vein, popliteal vein, and sapheno-femoral junction, with thrombus extensions to the great saphenous vein. The patient also had severe lower limb pain and significant oedema. Our experience with such cases is that anticoagulation alone is not usually as effective as venous thrombectomy combined with anticoagulation. Therefore, venous thrombectomy was performed to alleviate her symptoms. Generally, percutaneous PMT is effective and less invasive than open surgical thrombectomy. The risks and benefits of this procedure were fully explained to the patient, and informed consent was obtained. Heparin bolus injection followed by infusion was initiated intravenously according to the protocol along with analgesia.

Pre-operatively, the patient was counselled on monitored anaesthesia care sedation and prone positioning and was instructed to communicate any discomfort. In the operating room, a large bore (18G) intravenous cannula was secured. She was positioned prone, as the plan was to access the popliteal vein. Monitors were then set up, which presented the following initial vital signs: blood pressure (BP) of 126/78 mmHg, heart rate (HR) of 101 bpm, and oxygen saturation (SpO_2) level of 98% on room air. Face mask oxygen (O_2) therapy at 6 L/min along with end-tidal carbon dioxide ($EtCO_2$) monitoring was initiated. To relieve anxiety, we intravenously administered 1 mg of midazolam and 50 mcg of fentanyl. Propofol was infused initially at 20 mcg/kg/min for the first 10 min. Once the access was secured, the infusion rate was tapered down to 12.5–20 mcg/kg/min to maintain a Ramsay scale score of II. The patient remained haemodynamically stable and was assessed intermittently for any discomfort, which she denied confidently. After inserting a 6 Fr sheath into the popliteal vein, venography confirmed occlusive DVT of the ilio-femoral-popliteal veins. Pharmaco-Mechanical-Thrombectomy was conducted and included intra-thrombus administration of 10 mg of Actilyse, and using a percutaneous thrombectomy device. Initially, the patient was comfortable during the course of the intervention. However, she suddenly complained of severe chest discomfort and difficulty of breathing. Her SpO_2 level started to decrease from 100% to 90%, and within seconds, both her SpO_2 and $EtCO_2$ levels were no longer recordable. The vascular access sites were immediately secured; the patient was positioned supine; and airway was initially supported with 100% O_2 therapy with continuous positive airway pressure. However, her SpO_2 level did not improve, and cyanosis was observed. Inhalational induction with 6% sevoflurane was initiated, and 100 mg succinylcholine was immediately administered. A cuffed endotracheal tube (ETT) was then inserted. After intubation, bilateral air entry was equal; however, the $EtCO_2$ level was 10 mmHg with regular waveforms on the graph. The ETT was re-confirmed to be within the trachea. MPE was strongly suspected. Thus, the vascular team introduced a pigtail catheter into the right ventricle (RV) and infused 100 mg of Actilyse as a bolus. A multidisciplinary team was called, as the patient was severely hypotensive, tachycardic, and hypoxic. Her BP dropped to 76/32 mmHg, HR to 132 bpm, SpO_2 level to 45% on 100% ventilation, positive end-expiratory pressure (PEEP) to 7 mmHg, and $EtCO_2$ level to 12 mmHg. Hypotension and hypoxia were managed with ephedrine, phenylephrine, fluids, hydrocortisone, and 100% O_2 therapy. Thereafter, her PEEP was gradually increased. All equipment malfunctioning was ruled out; invasive arterial and central venous lines were established; and laboratory

examination and arterial blood gas (ABG) analysis were performed.

ABG: pH: 7.20, PCO_2: 69.3, PO_2: 30.2, HCO_3: 26.8, and SO_2: 39.4

After 15 min of continuous resuscitative efforts the patient had cardiac arrest. High-quality cardiopulmonary resuscitation (CPR) was then initiated, and the patient achieved return of spontaneous circulation after five cycles of CPR with administration of two doses of epinephrine. At this point, she was still hypotensive, tachycardic, and hypoxic: Her BP was 76/23 mmHg; HR, 143 bpm; SpO_2 level, 51%; and $EtCO_2$ level, 11 mmHg.

Post-arrest ABG: pH: 6.95, PCO_2: 95.1, PO_2: 53.8, HCO_3: 20.6, and SO_2: 58.3

After cardiac arrest, transoesophageal echocardiography was performed by a cardiologist, which showed increased right atrial strain with RV dilation, confirming our suspicion of MPE. Despite all resuscitative efforts, the patient remained hypoxic due to MPE. Therefore, the cardiothoracic surgery team was called to the operating room. Extracorporeal membrane oxygenation (ECMO) was suggested and immediately started to bypass the heart and lungs to allow these organs to heal and prevent any hypoxia-induced organ injury. The events were discussed with her family. Meanwhile, inotropic support and anticoagulation with heparin were initiated according to the ECMO protocol. After the initiation of ECMO, her haemodynamics significantly improved: mean arterial pressure: 80 mmHg on adrenaline infusion, HR: 90 bpm, and SpO_2 level: 100%.

Post-ECMO ABG: pH: 7.4, PCO_2: 18.7, PO_2: 562, HCO_3: 12.1, and SPO_2: 100%.

The patient was transferred to the intensive care unit (ICU) for further management. She received 2.5 L of isotonic crystalloids and 500 mL of colloids. The total resuscitation time was 90 min. Her urine output was 200 mL at the end of resuscitative efforts.

In the ICU, the patient remained haemodynamically stable. Forty eight hours later, she was weaned off from ECMO and extubated. She developed right-sided pneumonia for which she received broad-spectrum antibiotics along with stress ulcer prophylaxis, analgesia, and sedation according to the ICU protocol. Immediately after extubation, right-sided body weakness was noted. Brain magnetic resonance imaging revealed left cerebellar and bilateral cerebral non-ischaemic infarcts suggesting a thromboembolic event. No haemorrhagic transformation was seen on repeat brain computed tomography. Therefore, a cardiology review was sought to rule out right-to-left shunting. Echocardiography was performed with an agitated saline bubble study, which revealed a patent foramen ovale (PFO). The cardiologist recommended closure of the PFO. This was explained to the family, who opted to defer the procedure. The patient developed ICU psychosis, which was managed successfully with haloperidol. She was transferred to the ward after 11 days in the ICU and later discharged home on oral anticoagulation and graded compression stockings for the lower limbs 33 days after admission. Neurological deficits gradually improved, and the patient eventually fully recovered few months after discharge. Venous duplex ultrasound of her lower limbs on follow-up showed completely patent deep veins free of thrombus with normal flow patterns. Lower limb oedema also essentially subsided.

Discussion

In this report, we discussed a case of acute MPE during percutaneous PMT for acute massive ilio-femoral-popliteal venous thrombosis. The patient had a fifth metatarsal bone fracture and underwent ORIF. She later developed severe symptomatic right lower limb DVT. Percutaneous PMT was then planned under sedation, with $EtCO_2$ monitoring. In general, $EtCO_2$ monitoring does not show the $PaCO_2$ level when acute MPE occurs. The difference between the $PaCO_2$ and $EtCO_2$ levels could be representative of a dead space, which has been studied as a variable for monitoring the effect of thrombolysis.[5] In our patient, hypoxaemia and hypercarbia were evident because of increased dead space, low cardiac output, and ventilation–perfusion mismatch.[6] Although non-specific, chest pain and dyspnoea were highly suggestive of APE.[7] First, the present case was a provoked acute DVT based on the history of trauma, ORIF for a fifth metatarsal bone fracture, and application of a non-weight-bearing cast. Second, RV strain and dilation and computed tomography pulmonary angiography (CTPA) findings confirmed the diagnosis. CTPA revealed extensive PE manifesting as multiple filling defects in the right and left pulmonary arteries in addition to the major branches. Massive APE was suspected intra-operatively, as the patient was undergoing venous thrombectomy without

inferior vena caval filters.[8] Generally, the most common cause of PE remains to be DVT.[9] At our institution, symptomatic acute massive iliofemoral DVT have long been successfully treated with either open surgical thrombectomy or endovascular PMT without vena cava interruption. The morbidity is negligible with no mortalities. Data regarding whether an IVC filter should be inserted before such procedures are limited. Therefore, there is no consensus on the use of prophylactic IVC filters in such cases. Additional research is needed to optimize care for this patient population.

The treatment for acute MPE requires rapid and accurate assessment. ECMO is one of the most common modalities used for treating near-fatal MPE. Dolmatova et al. described the use of ECMO for MPE, with an overall mortality rate of 40%.[10] Conversely, thrombolytic treatment improves pulmonary perfusion more rapidly than does anticoagulation alone, but may also increase the risk of major bleeding, including intracranial haemorrhage.[11] To confirm the extent of APE, we performed CTPA after stabilising the haemodynamic status of our patient in the ICU.

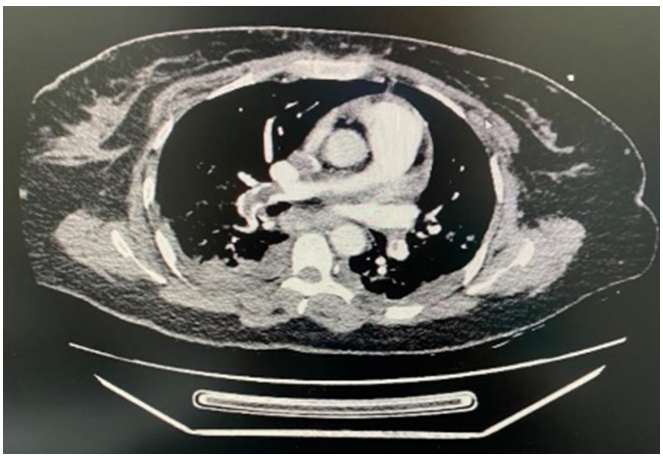

Fig. 1: Axial CTPA showing bilateral pulmonary trunk thrombus

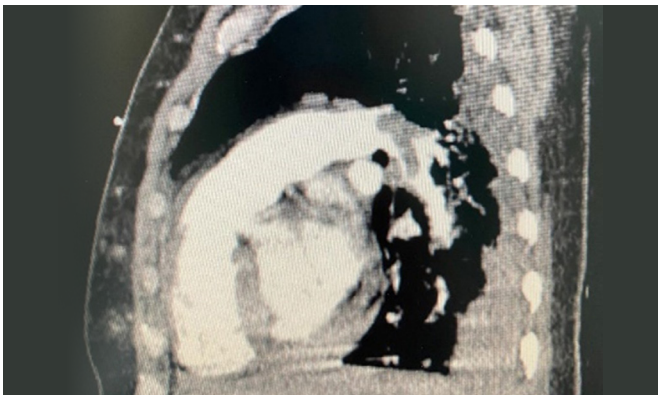

Fig. 2: Sagittal CTPA showing bilateral pulmonary embolus.

The patient also developed paradoxical embolism (PDE) through the PFO. PDE represents less than 2% of all arterial emboli, which makes it a rare event.[12,13] A PFO is an opening in the septum secundum, which allows blood to flow from the right to the left side of the heart during prenatal life. By the first year, the foramen closes permanently. PDE should be suspected when arterial embolism occurs in the absence of proximal arterial or cardiac sources.[13] Interestingly, in our patient, the presence of PFO may have lessened the effect of MPE as part of the thrombus passed from the right side to the left side of the heart.

In the ICU, we monitored the inflammatory markers of the patient, including the lactate level. The plasma lactate level is a sensitive marker of tissue hypoxia, which is associated with adverse outcomes in critically ill patients.[14,15]

The patient was finally transferred from the ICU to the ward and later discharged home. Follow-up was conducted in our outpatient clinic.

Conclusion

Acute MPE is a rare but potentially lethal intra-operative event. Immediate diagnosis may be challenging for the anesthetists. Patients with extensive DVT are at risk of developing PE especially during interventions. Therefore, pre-intervention insertion of a temporary IVC filter is prudent in these cases. In our patient, high index of suspicion, immediate bolus administration of an intra-cardiac thrombolytic agent and the use of ECMO were life-saving factors. The clinical progression was eventful owing to the presence of a previously undiagnosed PFO with showering of thrombus from the right to the left side of the heart and into the cerebral circulation, causing stroke. Teamwork in the operating room, and the availability of proper resources, personnel, and expertise are of utmost importance in the appropriate management of critical events in the operating theatre.

Availability of data and scientific materials

All data and materials described in the manuscript are freely available to any scientist wishing to use them for non-commercial purposes. Raw data are not available, as all are in the patient electronic medical record. Nevertheless, all data underlying the results are included in the case report.

Consent for publication

Written informed consent for publication of this case report was obtained from the patient.

Acknowledgements

We thank the anaesthesia, vascular, cardiology, cardiothoracic, and ICU teams for their help in the resuscitative efforts.

References

1. Cohen AT, Agnelli G, Anderson FA, Arcelus JI, Bergqvist D, Brecht JG, et al. Venous thromboembolism (VTE) in Europe. The number of VTE events and associated morbidity and mortality. Thromb Haemost. 2007;98(4):756–64.
2. Crane SD, Lee JB, Morris FP. / Editor: Jason M Kendall / Reviewer: Eugene Henry / Codes: CAP6, HAP6 / Published: 11/02/2019 / Review Date: 11/02/2022 :https://www.rcemlearning.co.uk/reference/pulmonary-embolism/#1571052980266-59bc3533-cddf
3. Ongen G, Yılmaz A, Cirak AK, Ersoy CY, Erden F, Altıntaş F, et al. Venous thromboembolism risk and thromboprophylaxis among hospitalized patients: data from the Turkish arm of the ENDORSE study. PMID: 20829272 DOI: 10.1177/1076029610382103
4. Janata K, Holzer M, Domanovits H, Müllner M, Bankier A, Kurtaran A, et al. Mortality of patients with pulmonary embolism. https://pubmed.ncbi.nlm.nih.gov/12416281/
5. Goldhaber SZ, Elliott CG. Acute pulmonary embolism: part I: epidemiology, pathophysiology, and diagnosis. Circulation. 2003;108(22):2726–9.
6. Wells PS, Ginsberg JS, Anderson DR, Kearon C, Gent M, Turpie AG, et al. Use of a clinical model for safe management of patients with suspected pulmonary embolism. Ann Intern Med. 1998;129(12):997–1005. DOI: 10.7326/0003-4819-129-12-199812150-00002
7. Konstantinides SV, Torbicki A, Agnelli G, Danchin N, Fitzmaurice D, Galie N, et al. 2014 ESC guidelines on the diagnosis and management of acute pulmonary embolism. Eur Heart J. 2014;35(43):3033–69. 3069a
8. Kaufman JA, Barnes GD, Chaer RA, Cuschieri J, Eberhardt RT, Johnson MS, et al. Society of Interventional Radiology clinical practice guideline for inferior vena cava filters in the treatment of patients with venous thromboembolic disease: developed in collaboration with the American College of Cardiology, American College of Chest Physicians, American College of Surgeons Committee on Trauma, American Heart Association, Society for Vascular Surgery, and Society for Vascular Medicine. DOI: 10.1016/j.jvir.2020.06.014
9. Essien EO, Rali P, Mathai SC. Pulmonary embolism. PMID: 30955521 DOI: 10.1016/j.mcna.2018.12.013
10. Yusuff H, Zochios V, Vuylsteke A. Extracorporeal membrane oxygenation in acute massive pulmonary embolism: a systematic review. Perfusion. 2015;30(8):611–6. DOI: 10.1177/0267659115583377.
11. Zuo Z, Yue J, Dong BR, Wu T, Liu GJ, Hao Q. Thrombolytic therapy for pulmonary embolism. DOI: 10.1002/14651858.CD004437.pub6
12. https://www.heart.org/en/health-topics/congenital-heart-defects/about-congenital-heart-defects/patent-foramen-ovale-pfo
13. Guo S, Ingram R, Missri J. Paradoxical embolism, deep vein thrombosis, pulmonary embolism in patient with patent foramen ovale. J Med Case Rep. 2007;1:104.
14. Bakker J, Nijsten MW, Jansen TC. Clinical use of lactate monitoring in critically ill patients. 2013 May 10. DOI: 10.1186/2110-5820-3-12
15. Nichol A, Bailey M, Egi M, Pettila V, French C, Hart GK, et al. Dynamic lactate indices as predictors of outcome in critically ill patients. Crit Care. 2011;15:R242. DOI: 10.1186/cc10497