

Vinodini Wanigasekara¹, Sunali Devika Nanayakkara²

- 1. (vinodini34@gmail.com)
- 2. (sunalinanayakkara@gmail.com)

Corresponding author

S D Nanayakkara (sunalinanayakkara@gmail.com)

Case Report 7

Management Of Acute Pulmonary Embolism Following Chest Trauma In A Resource Limited Setting – A Case Report

KEY WORDS - Pulmonary embolism, Trauma, Frontal contusion, Cardiac arrest, Recourse limited setting

ABSTRACT

Introduction: Pulmonary embolism is a known complication among trauma patients and contribute to significant mortality and morbidity. In the setting of trauma, the signs and symptoms of VTE may be masked. Therefore, a high index of suspicion and timely intervention can improve outcome.

Presentation of case: A 57-year-old healthy female presented with a history of fall from a train, in to a gap between the train and the platform. She had sustained cerebral contusions in the frontal lobes and right sided lung contusion. She was resuscitated and managed in the trauma ICU and send to a ward.

She desaturated in the ward and followed by a cardiac arrest. Bed side 2D echocardiogram and CT pulmonary angiogram were suggestive of PE. Thrombolysis was followed by clot retrieval. Patient was discharged home on day 13 after cardiac arrest on warfarin therapy.

Discussion: Causes for PE are multi-factorial. Failure to initiate prophylaxis therapy for deep vein thrombosis in trauma victims is a major cause for PE. Management of PE in a haemodynamically unstable patient with a background history of trauma is challenging. Neuro-imaging revealed bi frontal cerebral contusions hence, the treatment of choice was clot retrieval. However, due to limited resources and logistics, medical thrombolysis was initiated with the involvement of a multidisciplinary team. Clot retrieval was initiated within 14 hours of diagnosis.

Conclusion: Clinical suspicion and early diagnosis of PE can improve outcomes. Management is challenging in a low & middle income countries, where resources are limited. In the presence of contraindications and failed thrombolysis, clot retrieval remains an important aspect in the management of PE.

INTRODUCTION

Acute PE is a major cause of mortality and morbidity. The exact prevalence of PE is not known but the incidence is reported at 50-75 per 100

000 in Australia and New Zealand, with a 30-day mortality rate ranging from 0.5% to more than 20%1. In recent studies overall mortality due to PE is reported to be as high as 18% to 65 %. Mortality is around

20% in patients who have received thrombolysis as treatment. Associated cardiogenic shock increases mortality to 25% -30%. Patients who had cardiopulmonary resuscitation mortality was as high as 65%2,3,4.

PE and VTE are widely reported among medical, surgical and trauma patients. Trauma is responsible for 12% of VTE occurring in the community5. According to a study conducted by Bahloul et al PE complicates 18% of ICU admissions due to trauma6. It was reported to be the third most common cause of death in patients who survived the first 24 hours 5.

Local prevalence data on PE are not available, despite a number of reported cases of PE with varied presentations in Sri Lanka.

This case report highlights the timely diagnosis and intervention of a trauma victim who suffered a cardiac arrest following PE but recovered successfully in a setting with limited resources.

CASE REPORT

A previously healthy, 57-year-old woman was admitted to National Hospital accident service following a rail way tract accident. While trying to get on to a train she had fallen in between train and the platform. She was conscious and rational on admission to hospital with a GCS of 15/15. Pulse rate was 104 beats per minute and and blood pressure was 110/69 mmHg. She complained of headache and difficulty in breathing. Non-contrast CT scan of the brain showed bi frontal cerebral contusions She had a right sided lung contusion, clavicular fracture and a haemothorax.. Following insertion of a right sided intercostal drain She remained comfortable and haemodynamically stable. She was transferred to the surgical ward for further care.

Patient developed desaturation with poor respiratory efforts and was transferred

to the trauma intensive care unit, where she received intermittent non-invasive ventilation and multi modal analgesia. Intermittent pneumatic compression devices were applied. Pharmacological DVT prophylaxis was not initiated due to cerebral contusions and haemothorax. After three days of supportive management in ICU she was re transferred to the ward. Intercostal tube was removed after one week of insertion and patient was mobilized.

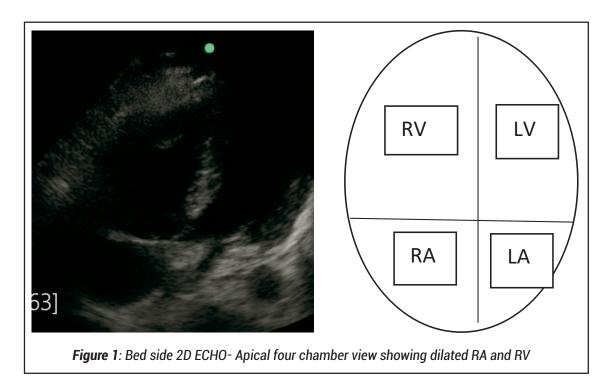
On the eighth day following the initial injury, she complained of sudden onset breathlessness on walking. Her respiratory rate was 40 breaths/min with oxygen saturation 75% on room air. She was given 15L of O2 via a non-rebreathing mask. Patient developed an asystolic cardiac arrest in the ward. Cardio pulmonary resuscitation was initiated as per the standard ALS guidelines. Return of spontaneous circulation was noted within six minutes of the cardiac arrest. She was intubated and an intercostal tube was re-inserted on the suspicion of a pneumothorax as the patient had reduced air entry over the right lung base.

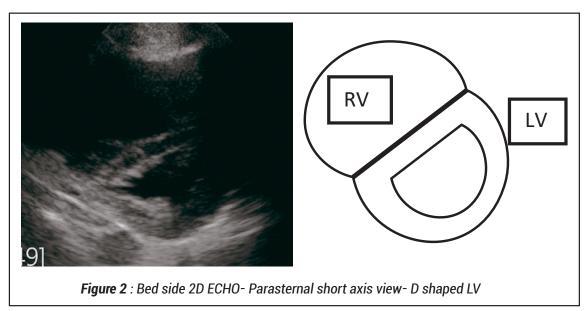
Patient was transferred back to trauma intensive care unit for post resuscitation care. On arrival she had GCS of 6/15 (Eye-1. Motor 4, Verbal-1). Bi lateral pupils were 3mm with equal reaction to light. Pulse rate was 174 beats/min, blood pressure was 100/55 mmHg on an infusion of noradrenalin. Bi lateral lung fields were clear. SpO2 was 90 % on 100% oxygen.

Initial arterial blood gas showed a PH of 7.188, PCO2 -26.2 mmHg, pO2-65.3 mmHg, Lactate 8.6 mmol/L, HCO3- 10 mmol/L and a base excess of (BE)- -18.4 mmol/L. Electrolytes were normal.

Electrocardiogram showed sinus tachycardia

A focused 2D ECHO was done in the ICU, which showed good LV contraction with significantly dilated RV and RA, flattening of the interventricular septum, and a dilated inferior vena cave with minimal respiratory variations.





Clinical presentation of sudden onset of shortness of breath followed by a cardiac arrest in a poorly mobilized patient with given 2D ECHO findings lead to a diagnosis of PE.

Patient underwent an urgent CTPA which showed a large pulmonary embolus with in the right upper segmental pulmonary artery and multiple large thrombi in the left lower segmental pulmonary arteries. Main pulmonary artery was dilated up to 3 cm in diameter. The RV was grossly dilated with the interventricular septum bulging in to the left ventricle.

Clot retrieval remained the treatment of choice. Medical thrombolysis could worsen the bi frontal contusions and the haemothorax. We opted for thrombolysis after considering the risk versus benefit with the multidisciplinary team. In a resource constraint setting organizing for clot retrieval was difficult. Patient was haemodynamically unstable for transfer to the radiology suite. The decision between the two treatment options was a challenge to the clinicians and posed a dilemma.

Intravenous alteplase 100 mg was given over two hours, followed by intravenous heparin

1000 IU per hour as an infusion, whilst monitoring APTT according to the standard protocol.

Despite improvement in oxygenation the vasopressor and inotrope requirement increased over the next few hours of initiation of thrombolysis. Her blood pressure was 94/65 mmHg and heart rate 130 beats per minute. She was on noradrenaline 0.5 mcg/kg/min, intravenous vasopressin 4U/hour and intravenous dobutamine 10 mcg/kg/min.

2D ECHO showed poor right heart functions.

Decision was made for clot retrieval via interventional radiological technique under general anaesthesia. Following retrieval haemodynamic parameters improved and cardiovascular support was tailed off gradually.

Unfractionated heparin infusion was converted to therapeutic doses of low molecular weight heparin.

Venous duplex scan of both lower limbs was negative for deep vein thrombosis.

Seven days after the cardiac arrest patient was transferred back to the ward on warfarin and enoxaparin as bridging therapy. She was discharged from hospital on day 13 following the cardiac arrest with no neurological sequalae.



Figure 3 - Filling defects in bi lateral segmental pulmonary arteries- CTPA image



Figure 4 - Filling defects in bi lateral segmental pulmonary arteries- image acquired during clot retrieval



Figure 3 - Filling defects in left sided segmental pulmonary arteries- image acquired during clot retrieval

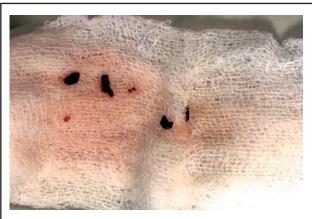


Figure 3 - Parts of thrombus retrieved

DISCUSSION

DVT and PE are known complications in trauma patients. Incidence of PE among trauma patients vary considerably, ranging from 0.35% to 24%5. Contrary to popular belief PE occur more commonly during days 5 to 7 after trauma many studies have shown increasing evidence of early onset PE. A descriptive study conducted by Menakar J et al reported 37% of all cases of PE were diagnosed within day 1 to 4, 18 % during day 5 to 7, 23% in day 8 to 14 and rest over day 14 to 20 after trauma7. Transient hypercoagulability that occurs in the first few days following injury leads to early on set PE5. Some studies have demonstrated that tissue factors and thrombin generation increase after trauma and levels of natural anti coagulants are diminished. (e.g.: anti thrombin III, protein C and S)8

Fractures of the lower limbs, obesity and age are known risk factors to develop PE5. In trauma victims' spinal injuries, traumatic brain injuries, severe chest trauma and high injury severity score were identified as predictive risk factors 5,9.

These injuries lead to prolong periods of immobilization and also hinder the use of anti-coagulants prophylactically within the first 48 hours after injury5,9. Traditional risk factors such as active cancer, prior history of DVT or PE, major venous repair, mechanical ventilation for more than four days, central line placements

are also relevant to this cohort of patients 10. Our patient had chest trauma, immobilization and delayed initiation of anti-coagulants as risk factors for developing PE.

Treatment of PE in a trauma patient pose challenges to the clinician. Management decisions are guided according to relative or absolute contraindications for thrombolysis or anti coagulation and weighing benefit vs harm. If there are contraindications for thrombolysis in a trauma patient, pulmonary interventional thrombolysis and thrombus aspirations techniques should be considered. There is a place for ECMO selected cases 11.

When compared to an ideal setting we faced many challenges during the management of this patient. This patient suffered major trauma just eight days prior to the cardiac arrest secondary to PE. Major trauma, or head injury in the previous three weeks is considered an absolute contra indication to thrombolysis according to the European society of cardiology guidelines on management of PE 12. Due to non-availability of appropriate size catheters for clot retrieval in our institution, we resorted to thrombolysis.

Other options discussed in the literature for management of PE include catheter directed thrombolysis, catheter embolectomy, surgical embolectomy and or mechanical circulatory support devices 13. catheter embolectomy was the next available option available in our setup.

Within 14 hours after ICU admission, we offered clot retrieval for the patient with smaller size catheter. By using smaller catheters they can disturb the clot facilitating clot lysis as well as perfusion through a disturbed clot.

Lower limb duplex scan was negative in our patient. Becher M et al in a recent study studied 168 patients with PE and 46.4% of patients had negative duplex scan for lower limb DVT. Most of these patients had peripherally located PE, and mostly diagnosed with V/P-SPECT rather than CTPA 14.

A recent article introduces a new concept i.e., PE and in situ PT to be two different clinical variants. This debate has arisen as some patients with PE were persistently negative for DVT as in the case of our patient. In a prospective cohort study done in the United States of America, it was found that independent risk factors for PT include shock on admission, major chest injury with an Abbreviated Injury Score of three or more and major venous injury15,16. In this trial pulmonary thrombosis was defined as pulmonary clots on chest CTPA without concomitant lower extremity DVT. Despite the new terminology, the management of these patients remain the same15.

CONCLUSION

Acute PE is a major cause of morbidity and mortality following cardiac arrest.

A bed side echocardiogram helped to identify RA and RV dilatation, so that management was initiated without undue delay. Despite the presence of contraindications to initiate thrombolysis in a trauma victim this patient was successfully treated with thrombolysis followed by clot retrieval with the best possible available devices in our institution.

Early detection, immediate management and involvement of the multidisciplinary team lead to the successful outcome.

ABBREVIATIONS—Pulmonary embolism -PE, Deep vein thrombosis- DVT, Venous thrombo embolism – VTE, Intensive care unit-ICU, Advanced life support ALS, Pulmonary thrombosis -PT, Computerized tomography pulmonary angiogram- CTPA, Right ventricle -RV, Right atria- RA, left ventricle-LV, Left atria- LA. Two-dimensional echo cardiogram-2D ECHO, Activated partial thromboplastin time, APTT, Ventilation/perfusion single photon emission computed tomography V/P SPECT, Extra corporal membrane oxygenation- ECMO.

CONSENT:

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

SOURCES OF FUNDING: None.

ACKNOWLEDGMENT: Cardiology team at the National Hospital of Sri Lanka, Interventional radiology team of National Hospital of Sri Lanka, Anaesthesia team of National Hospital of Sri Lanka.

REFERRENCES

- 1. Hepburn-Brown M, Darvall J, Hammerschlag G. Acute PE: a concise review of diagnosis and management. Internal medicine journal. 2019 Jan;49(1):15-27.
- 2. Bělohlávek J, Dytrych V, Linhart A. PE, part I: Epidemiology, risk factors and risk stratification, pathophysiology, clinical presentation, diagnosis and nonthrombotic PE. Experimental & Clinical Cardiology. 2013;18(2):129.
- 3. Torbicki A, Perrier A, Konstantidines S, et al. Guidelines on the diagnosis and management of acute PE. Eur Heart J. 2008;29:2276–315.
- 4. Widimský J, Malý J, Eliáš P, et al. Doporučení pro diagnostiku a léčbu akutní plicní embolie. Vnitř. Lék. 2008;54:1S25–1S72.
- 5. Bahloul M, Dlela M, Bouchaala K, Kallel H, Hamida CB, Chelly H, Bouaziz M. Post-traumatic PE: incidence, physiopathology, risk factors of early occurrence, and impact outcome. A narrative review. American Journal of Cardiovascular Disease. 2020;10(4):432.

THE SRI LANKAN JOURNAL OF ORTHOPEADIC SURGERY

- 6. Bahloul M, Dlela M, Bouchaala K, Triki A, Chelly H, Hamida CB, Haddar S, Bouaziz M. Early post-traumatic PE in intensive care unit: incidence, risks factors, and impact outcome. American Journal of Cardiovascular Disease. 2020;10(3):207.
- 7. Menaker J, Stein DM, Scalea TM. Incidence of early PE after injury. Journal of Trauma and Acute Care Surgery. 2007 Sep 1;63(3):620-4.
- 8. Bahloul M, Chaari A, Dammak H, Medhioub F, Abid L, Ksibi H, Haddar S, Kallel H, Chelly H, Hamida CB, Bouaziz M. Post-traumatic PE in the intensive care unit. Annals of Thoracic Medicine. 2011 Oct;6(4):199.
- 9. Bahloul M, Dlela M, Bouaziz NK, Turki O, Chelly H, Bouaziz M. Early post-traumatic pulmonary-embolism in patients requiring ICU admission: more complicated than we think!. Journal of Thoracic Disease. 2018 Nov;10(Suppl 33):S3850
- 10. Van Gent JM, Calvo RY, Zander AL, Olson EJ, Sise CB, Sise MJ, Shackford SR. Risk factors for deep vein thrombosis and PE after traumatic injury: A competing risks analysis. Journal of trauma and acute care surgery. 2017 Dec 1;83(6):1154-60.
- 11. Mi YH, Xu MY. Trauma-induced pulmonary thromboembolism: What's update?. Chinese Journal of Traumatology. 2022 Mar 1;25(02):67-76.
- 12. Konstantinides SV, Meyer G, Becattini C, Bueno H, Geersing GJ, Harjola VP, Huisman MV, Humbert M, Jennings CS, Jiménez D, Kucher N. 2019 ESC Guidelines for the diagnosis and management of acute PE developed in collaboration with the European Respiratory Society (ERS) The Task Force for the diagnosis and management of acute PE of the European Society of Cardiology (ESC). European heart journal. 2020 Jan 21;41(4):543-603.
- 13. Rivera-Lebron B, McDaniel M, Ahrar K, Alrifai A, Dudzinski DM, Fanola C, Blais D, Janicke D, Melamed R, Mohrien K, Rozycki E. Diagnosis, treatment and follow up of acute PE: consensus practice from the PERT Consortium. Clinical and Applied Thrombosis/Hemostasis. 2019 Jun 10;25:1076029619853037.
- 14. Becher M, Heller T, Schwarzenböck S, Kröger JC, Weber MA, Meinel FG. Negative Venous Leg Ultrasound in Acute PE: Prevalence, Clinical Characteristics and Predictors. Diagnostics. 2022 Feb 17;12(2):520.
- 15. Knudson MM, Moore EE, Kornblith LZ, Shui AM, Brakenridge S, Bruns BR, Cipolle MD, Costantini TW, Crookes BA, Haut ER, Kerwin AJ. Challenging Traditional Paradigms in Posttraumatic Pulmonary Thromboembolism. JAMA surgery. 2022 Feb 1;157(2):e216356-.
- 16. Van Gent JM, Zander AL, Olson EJ, Shackford SR, Dunne CE, Sise CB, Badiee J, Schechter MS, Sise MJ. PE without deep venous thrombosis: de novo or missed deep venous thrombosis?. Journal of Trauma and Acute Care Surgery. 2014 May 1;76(5):1270-4..