

A New Life, Unfortunately in A World Of Darkness

Azlan N^{1*}, Nathan JA², Latiff JY³ and Razak BT⁴

¹Department of Emergency Medicine, Faculty of Medicine, Universiti Kebangsaan Malaysia

*Corresponding author:

Nik Azlan,
Department of Emergency Medicine, Faculty
of Medicine, Universiti Kebangsaan Malaysia
Medical Centre, Malaysia,
E-mail: nikazlanmuhamad@hotmail.com

Received: 11 Feb 2021
Accepted: 01 Mar 2021
Published: 05 Mar 2021

Copyright:

©2021 Azlan N et al., This is an open access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and build upon your work non-commercially.

Citation:

Azlan N. A New Life, Unfortunately in A World Of Darkness. Ann Clin Med Case Rep. 2021; V6(1): 1-3

1. Abstract

Acute pulmonary embolism is a form of venous thromboembolism that is common and sometimes fatal. Submassive PE comprises of at least 20% of all acute cases of pulmonary embolisms. It has been shown to have an up to 5% in hospital mortality rate [1]. Thrombolytic use in submassive PE with signs of RV dysfunction or necrosis requires careful evaluation and still remains a dilemma for physicians due to its potential haemorrhagic complications [2]. We report a case of a patient who presented to us with a submassive pulmonary embolism who unfortunately developed intracranial bleeding post thrombolysis.

2. Introduction

Submassive pulmonary embolism has been defined as acute pulmonary embolism without systemic hypotension (SBP \geq 90 mmHg) but with either RV dysfunction or myocardial necrosis [3]. RV dysfunction shown by as at least one of the following -RV dysfunction on POCUS or RV dilation (RV:LV Diameter $>$ 0.9 on POCUS) or Elevated BNP ($>$ 500pg/mL) or new ECG Changes (Complete or Incomplete RBBB, Anteroseptal ST-Segment Elevation/Depression, or Anterolateral T-Wave Inversion). Myocardial necrosis is shown by an elevated Troponin I ($>$ 0.4ng/mL) or an elevated Troponin T ($>$ 0.1ng/mL) [3]. A Meta-analysis in 2014 showed that in patients who were haemodynamically stable, there was a 2.29% increase in short term mortality with those who had RV dysfunction [4].

3. Case Background

47 years old Indonesian lady who has underlying hypertension and a uterine mass under investigation. On the 19/05/18, he fainted at a bus station and was brought in to the emergency department via ambulance. She was unable to speak but nodded her head when asked if she had chest pain. GCS on arrival was 11/15 (E4V1M6), BP 108/81, HR 120, temp 36.5, spo2 60% under room air, ECG – Sinus tachycardia, RBBB, S1Q3T3. CTPA was done – multifocal bilateral lobar and segmental pulmonary ar-

tery embolisms. Bedside echo done – no RV thrombus, dilated RV, D shaped LV, Mc Connell sign present. D-dimer was $>$ 20 and Trop I - $>$ 4000. Her Spo2 remained 80 % only despite being on high flow mask. Her ABG showed type one respiratory failure with a Pao2 of 50% on the high flow mask. The patient was diagnosed as submassive pulmonary embolism and decision for thrombolysis was made. A CT scan of her brain was done prior to thrombolysis which showed no bleed. She was thrombolysed with IV Alteplase and her Spo2 improved to 100% after thrombolysis and she was haemodynamically stable with a GCS of E4V4M6. She was then transferred to CCU where she was stable overnight. The next morning, she complained of sudden onset of bilateral eyes blurring of vision. A repeated ct scan showed Acute intraparenchymal hemorrhages at bilateral occipital lobes with perilesional edema. She was diagnosed as bilateral eyes cortical blindness secondary to ICB post thrombolysis. She subsequently requested for AOR discharge to go back to her home country to continue treatment there. Upon discharge she was able to walk and did not require any oxygen support however she still remained blind.

4. Discussion

Thrombi most commonly arise from the deep veins of the lower limbs and pelvis from where they dislodge into the pulmonary arteries forming pulmonary emboli. This then causes abnormalities in the gas exchange and haemodynamic instability of the patient depending on the size of the thrombi, the patients existing cardiopulmonary status and compensatory neurohumoral adaptations [5]. Acute PE also leads to release of pulmonary vasoconstrictors which then lead to hypoxemia. Increase in Right ventricular afterload and dilatation due to the increase pulmonary vascular resistance eventually leads to RV failure and ischemia [6].

Systemic fibrinolysis has a role as a ‘medical embolectomy’ which reduces the thrombus burden, pulmonary vascular resistance and RV dysfunction. It also restores the pulmonary capillary blood flow and improves gas

exchange more expeditiously than anticoagulation alone. The use of it however remains limited due to the lack of conclusive RCTs and potential bleeding complications [7].

There has been many studies done with many controversies regarding the decision for thrombolysis of submassive PE or not. In a meta-analysis of 16 trials with 8 of them specifying inclusion of intermediate-risk pulmonary embolism, thrombolysis was associated with lower mortality but more major bleeding events [8].

In the large PEITHOS trial – whereby they studied >1000 patients across Europe and Israel, the clinical efficacy and safety of fibrinolytic therapy was investigated with a single bolus injection of tenecteplase, in addition to the standard anticoagulation therapy with heparin. The patients investigated and recruited were those with acute pulmonary embolism with an intermediate risk. It was concluded that in these patients, haemodynamic decompensation was reduced in those patients receiving fibrinolytic therapy as compared to conventional anticoagulation. There was however increased risk of major bleeding and stroke. Fibrinolytic treatment was shown to be associated with a 6.3% rate of major extracranial haemorrhage and a 2% rate of haemorrhagic stroke (Primarily in patients ≥ 75 years of age) [9].

The Moderate Pulmonary Embolism Treated with Thrombolysis trial (MOPETT Trial 2013) done is a prospective, controlled, randomized, single-center open study. It recruited 121 adult patients with symptomatic ‘moderate’ PE [10]. The intervention group of patients in this study were given half of the usual dose for thrombolysis. There was evidence of long term reduction in the incidence of pulmonary hypertension as compared to those who were given only anticoagulation. There were no cases of bleeding reported [10]. It was however noted that the definition of submassive PE in this trial was not standardized and the use of the term ‘moderate’ PE and its definitions may be biased.

The American College of Chest Physicians (ACCP) limits the use of fibrinolysis in submassive PE to patients with a high risk of haemodynamic collapse with a low risk of bleeding. (Grade 2c) [11]. A Scientific Statement from The American Heart association attributes that fibrinolysis may be considered for patient with submassive PE which is considered to have evidence of adverse outcomes and a low risk of bleeding (Class IIb , level of evidence C) [3]. The European Society of Cardiology (ESC) states that fibrinolysis can be considered in patients with submassive PE (Class IIb , level of evidence B) [12].

5. Conclusion

Thrombolytic use in massive PE is warranted, but patients with submassive PE require case-by-case analysis with shared decision making with close monitoring of the patients. The risks and benefits, primarily improved long-term outcomes, should be considered [1]. Catheter-directed treatment Half-dose thrombolytics demonstrate improvements with decreased risk of bleeding and improved long-term functional outcomes. Further studies that assess risk stratification, functional outcomes, and treatment protocols are needed.

Reference

1. Chatterjee S, Chakraborty A, Weinberg I, Kadakia M, Wilensky RL, Sardar P, et al. Thrombolysis for Pulmonary Embolism

and Risk of All-Cause Mortality, Major Bleeding, and Intracranial Hemorrhage. *Jama*.2014; 311(23): 2414. doi:10.1001/jama.2014.5990

2. Cho JH, Kutti Sridharan G, Kim SH, Kaw R, Abburi T, Irfan A & Kocheril AG. Right ventricular dysfunction as an echocardiographic prognostic factor in hemodynamically stable patients with acute pulmonary embolism: a meta-analysis. *BMC Cardiovascular Disorders*. 2014; 14(1): 64. doi:10.1186/1471-2261-14-64
3. Jaff MR, McMurtry MS, Archer SL, Cushman M, Goldenberg N, Goldhaber SZ, Jenkins JS, et al. Management of massive and submassive pulmonary embolism, iliofemoral deep vein thrombosis, and chronic thromboembolic pulmonary hypertension: A scientific statement from the American heart association. *Circulation*. 2011; 123(16): 1788-1830. doi:10.1161/CIR.0b013e318214914f
4. Kearon C, Akl EA, Comerota AJ, Prandoni P, Bounameaux H, Goldhaber SZ, Nelson ME, et al. Antithrombotic therapy for VTE disease: Antithrombotic therapy and prevention of thrombosis, 9th ed: American College of Chest Physicians evidence-based clinical practice guidelines. *Chest*. 2012; 141(2 SUPPL.); e419S-e496S. doi:10.1378/chest.11-2301
5. Kilinc G, Dogan OT, Berk S, Epozturk K, Ozsahin SL & Akkurt I. Significance of serum cardiac troponin I levels in pulmonary embolism. *Journal of Thoracic Disease*. 2012; 4(6): 588-593. doi:10.3978/j.issn.2072-1439.2012.10.13
6. Long B & Koyfman A. Current Controversies in Thrombolytic Use in Acute Pulmonary Embolism. *Journal of Emergency Medicine*. 2016; 51(1): 37-44. doi:10.1016/j.jemermed.2016.02.024
7. Meyer G, Vicaut E, Danays T, Agnelli G, Becattini C, Beyer-Westendorf J. et al. Fibrinolysis for Patients with Intermediate-Risk Pulmonary Embolism. *New England Journal of Medicine*. 2014; 370(15): 1402-1411. doi:10.1056/NEJMoa1302097
8. Piazza G. CLINICIAN ' S CORNER Submassive Pulmonary Embolism. 2013; 309(2): 171-180.
9. Rali PM, Gerard J Criner. Submassive Pulmonary Embolism. 2018. doi:10.1164/rccm.201711-2302CI
10. Sharifi M, Bay C, Skrocki L, Rahimi F & Mehdipour M. Moderate pulmonary embolism treated with thrombolysis (from the “mOPETT” Trial). *American Journal of Cardiology*. 2013; 111(2): 273-277. doi:10.1016/j.amjcard.2012.09.027
11. Sinha S K, Sachan M, Goel A, Singh K, Mishra V, Jha M J, et al. Efficacy and Safety of Thrombolytic Therapy in Acute Submassive Pulmonary Embolism: Follow-Up Study. *Journal of Clinical Medicine Research*. 2017; 9(2): 163-169. doi:10.14740/jocmr2829w
12. Torbicki A, Perrier A, Konstantinides S, Agnelli G, Galiè N, Pruszczyk P, et al. Guidelines on the diagnosis and management of acute pulmonary embolism. *European Heart Journal*. 2008; 29(18): 2276-2315. doi:10.1093/eurheartj/ehn310