

CLINICAL VIGNETTE

Management of Acute Sub-massive Pulmonary Embolism

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Introduction

Pulmonary embolism (PE) refers to obstruction of pulmonary artery or its branches by material, such as thrombus, that originated elsewhere in the body. Acute pulmonary embolism from venous thromboembolic event is common and sometimes fatal, accounting for approximately 100,000 annual deaths in the US.¹ PE has a wide spectrum of presenting clinical features and imposes high overall mortality.² We present a 47-year-old female with sub-massive, intermediate-high risk PE who underwent percutaneous, aspiration pulmonary thrombectomy.

Case Report

A 47-year-old female with no significant past medical history had sudden onset of lightheadedness and dyspnea on minimal exertion. She is a psychiatrist who had just completed a clinical session and was getting up when she developed these symptoms. She continued to have shortness of breath that prompted her to come to the emergency room. She had liposuction two weeks prior with onset of bilateral leg swelling and was given furosemide. Upon arrival in the emergency room, she was hypertensive with tachycardia and hypoxia (heart rate at 125 bpm, 87% O₂ saturation on room air). Electrocardiogram showed sinus tachycardia with S wave in lead I, Q wave and inverted T wave in lead III (Figure 1). These ECG findings has been associated with acute PE causing cor pulmonale.³ High sensitivity troponin was significantly elevated to 116 and 164 ng/L (normal < 10 ng/L). Arterial blood gas revealed pO₂ of 63mmHg (normal reference 85-100mmHg). CT pulmonary angiogram demonstrated pulmonary emboli in the right main pulmonary artery and bilateral segmental and subsegmental branches bilaterally (Figures 2). RV strain was also noted with RV/LV ratio > 1.1 (Figure 3). Echocardiogram demonstrated normal LV size and function but with dilated RV and RV systolic dysfunction. RV free wall was akinetic with sparing of apex, consistent with McConnell's sign. Patient was given intravenous heparin for systemic anticoagulation. She was deemed to have sub-massive, intermediate-high risk PE. To prevent progression of cardiovascular compromise with right ventricular dysfunction, treatment option with percutaneous, aspiration pulmonary thrombectomy in addition to systemic anticoagulation was discussed in detail with the patient. She agreed to proceed with thrombectomy to minimize future adverse sequelae of PE. Patient underwent right heart catheterization, with pre-thrombectomy mean PA pressure of 35mmHg. Fick's cardiac output was 3.99 L/min. Pulmonary

angiograms were completed using Trieriver24 Aspiration catheter from Inari Medical. Bilateral pulmonary thrombectomy was then performed using the same Trieriver24 Aspiration catheter (Figure 4). Throughout the procedure, blood aspirates were filtered through a FlowSaver© device from Inari Medical. Thrombi were excluded by the filter and blood returned to the patient to minimize blood loss. Post procedure pulmonary angiograms performed (Figure 5) showed and a significant reduction of mean PA pressure of 19mmHg. The patient reported immediate improvement in symptoms and avoided transfer to the intensive care unit. She was weaned off supplemental oxygen and transferred to a contracted facility for ongoing management.

Discussion

Venous thromboembolic disease is increasing with many predisposing risk factors.⁴ Increased incidence and prevalence of cancer, trauma, surgery, oral contraceptive medications, traveling, and sedentary lifestyle contributes to this rise.⁵⁻⁷ Deep vein thrombosis and pulmonary embolism are part of same spectrum of disease. However, pulmonary embolism poses a more serious mortality risk. PE, mortality risk is related to the embolic burden and effects on the right ventricle along with any other cardiopulmonary co-morbidities. Sudden increase in PA pressure can lead to RV dysfunction and RV ischemia; decreased RV contractility which can lead to cardiogenic shock and death.⁸⁻¹⁰ Diagnosis of PE remains challenging, as presenting symptoms are often nonspecific. Even with the diagnosis of PE, treatment has largely been systemic anticoagulation. Some PE cases have high mortality rates, and systemic anticoagulation may not prevent the progression of cardiogenic shock in certain patients. Also, inadequately treated PE's with high thrombus burden have long term consequences and co-morbidity risks. These include chronic thromboembolic pulmonary hypertension with exercise limitations.¹¹⁻¹² These sequelae can impact long-term quality of life, especially in young patients it is important to risk stratify each PE patient, based on their hemodynamic status and clinical features, determining appropriate therapy¹³. Contemporary management of PE includes systemic anticoagulation/thrombolysis, surgical embolectomy, or catheter-based procedure. Systemic thrombolysis poses significant risk for major bleeding with higher risk in subgroup of patients. Surgical embolectomy remains significantly invasive and is not without complications. Catheter-

based therapies may be more appealing as a treatment option, as it minimizes procedural risk and can quickly relieve pulmonary vascular obstruction, without significant risk for bleeding. Recent technological advances in aspiration catheters include FDA approval of FlowTrieve catheter by Inari Medical.¹⁴ The efficacy and safety of this device was reported in the single-arm, prospective FLARE trial. This trial showed improved RV strain, as indicated by RV/LV ratio, in intermediate-risk PE patients 48 hours post-procedure.¹⁵ If adequate aspiration is performed, immediate improvement in hemodynamic measurements can be observed. With the emergence of new catheter-based therapies, it is important to accurately diagnose and risk stratify patients, so that such therapy can be offered to improve outcome in massive and sub-massive PE.

Conclusion

This patient was quickly diagnosed with sub-massive intermediate-high risk PE after surgery. Pulmonary Embolism Response Team (PERT) was notified and patient was placed on systemic anticoagulation. Catheter-based therapy with aspiration thrombectomy was offered to alleviate thrombus burden and RV strain. The procedure objective included unloading RV strain, and reduced risk for long-term consequences in an otherwise healthy, young patient. She underwent the procedure without complications with immediate improvement in hemodynamic measurements and resolution of symptoms. It is important to consider other treatment options in PE besides systemic anticoagulation/thrombolysis. Future randomized controlled studies will help guide the use of catheter-based therapy in addition to systemic anticoagulation in PE.

Figures

Figure 1: ECG showing sinus tachycardia with S wave in lead I, Q wave and inverted T wave in lead III

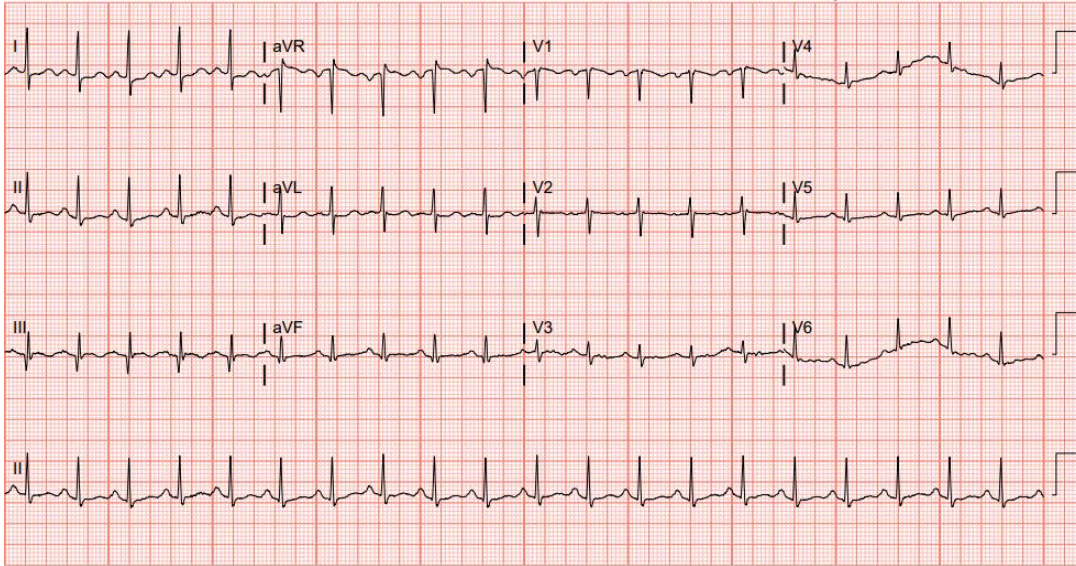


Figure 2: CT pulmonary angiogram showing pulmonary emboli in the right main pulmonary artery and bilateral segmental and subsegmental branches bilaterally

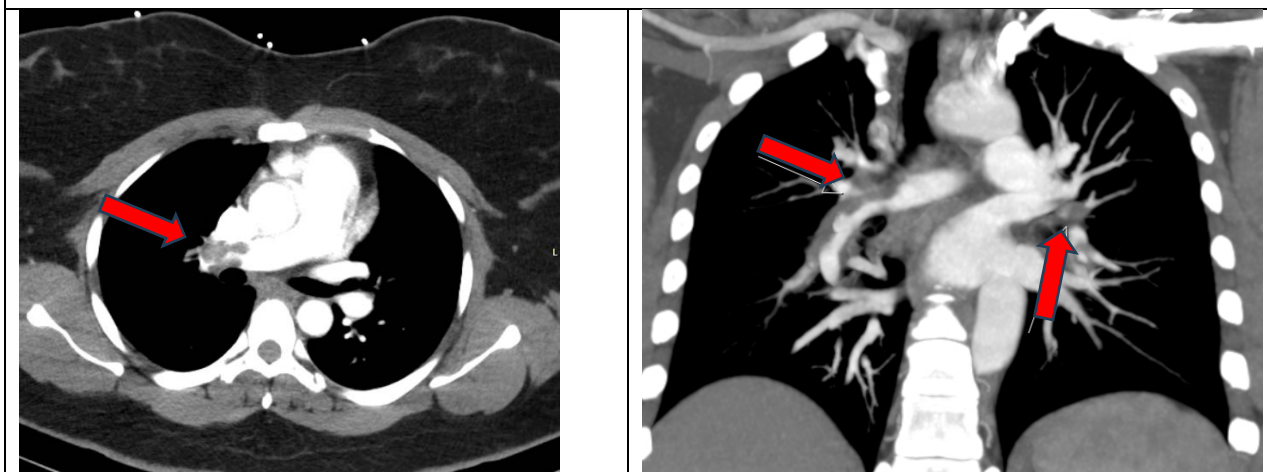


Figure 3: CT showing RV strain with RV/LV ratio > 1.1

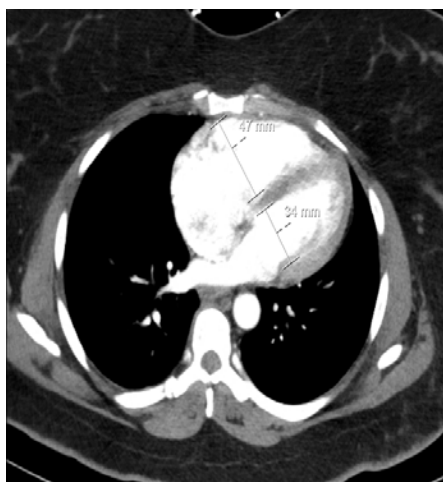


Figure 4: Pulmonary artery pressures and thrombi aspirated



Figure 5: Pulmonary angiograms showing improved flow in pulmonary arteries after aspiration thrombectomy



REFERENCES

1. **Horlander KT, Mannino DM, Leeper KV.** Pulmonary embolism mortality in the United States, 1979-1998: an analysis using multiple-cause mortality data. *Arch Intern Med.* 2003 Jul 28;163(14):1711-7. doi: 10.1001/archinte.163.14.1711. PMID: 12885687.
2. **Alotaibi GS, Wu C, Senthilselvan A, McMurtry MS.** Secular Trends in Incidence and Mortality of Acute Venous Thromboembolism: The AB-VTE Population-Based Study. *Am J Med.* 2016 Aug;129(8):879.e19-25. doi: 10.1016/j.amjmed.2016.01.041. Epub 2016 Feb 27. PMID: 26925811.
3. **Chan TC, Vilke GM, Pollack M, Brady WJ.** Electrocardiographic manifestations: pulmonary embolism. *J Emerg Med.* 2001 Oct;21(3):263-70. doi: 10.1016/s0736-4679(01)00389-4. PMID: 11604281.
4. **Haider A, Goldberg J.** National trends in pulmonary embolism management and outcomes: shifting paradigms. *J Am Coll Cardiol.* 2019;7(suppl 1):1901.
5. **Anderson FA Jr, Spencer FA.** Risk factors for venous thromboembolism. *Circulation.* 2003 Jun 17;107(23 Suppl 1):I9-16. doi: 10.1161/01.CIR.0000078469.07362.E6. PMID: 12814980.
6. **Rogers MA, Levine DA, Blumberg N, Flanders SA, Chopra V, Langa KM.** Triggers of hospitalization for venous thromboembolism. *Circulation.* 2012 May 1;125(17):2092-9. doi: 10.1161/CIRCULATIONAHA.111.084467. Epub 2012 Apr 3. PMID: 22474264; PMCID: PMC3342773.
7. **Chew HK, Wun T, Harvey D, Zhou H, White RH.** Incidence of venous thromboembolism and its effect on survival among patients with common cancers. *Arch Intern Med.* 2006 Feb 27;166(4):458-64. doi: 10.1001/archinte.166.4.458. PMID: 16505267.
8. **Piazza G, Goldhaber SZ.** The acutely decompensated right ventricle: pathways for diagnosis and management. *Chest.* 2005 Sep;128(3):1836-52. doi: 10.1378/chest.128.3.1836. PMID: 16162794.

9. **Sibbald WJ, Driedger AA.** Right ventricular function in acute disease states: pathophysiologic considerations. *Crit Care Med.* 1983 May;11(5):339-45. doi: 10.1097/00003246-198305000-00004. PMID: 6340962.
10. **Santamore WP, Dell'Italia LJ.** Ventricular interdependence: significant left ventricular contributions to right ventricular systolic function. *Prog Cardiovasc Dis.* 1998 Jan-Feb;40(4):289-308. doi: 10.1016/s0033-0620(98)80049-2. PMID: 9449956.
11. **Pengo V, Lensing AW, Prins MH, Marchiori A, Davidson BL, Tiozzo F, Albanese P, Biasiolo A, Pegoraro C, Iliceto S, Prandoni P; Thromboembolic Pulmonary Hypertension Study Group.** Incidence of chronic thromboembolic pulmonary hypertension after pulmonary embolism. *N Engl J Med.* 2004 May 27; 350(22):2257-64. doi: 10.1056/NEJMoa032274. PMID: 15163775.
12. **Kahn SR, Hirsch AM, Akaberi A, Hernandez P, Anderson DR, Wells PS, Rodger MA, Solymoss S, Kovacs MJ, Rudski L, Shimony A, Dennie C, Rush C, Geerts WH, Aaron SD, Granton JT.** Functional and Exercise Limitations After a First Episode of Pulmonary Embolism: Results of the ELOPE Prospective Cohort Study. *Chest.* 2017 May;151(5):1058-1068. doi: 10.1016/j.chest.2016.11.030. Epub 2016 Dec 6. PMID: 27932051.
13. **Konstantinides SV, Torbicki A, Agnelli G, Danchin N, Fitzmaurice D, Galiè N, Gibbs JS, Huisman MV, Humbert M, Kucher N, Lang I, Lankeit M, Lekakis J, Maack C, Mayer E, Meneveau N, Perrier A, Pruszczyk P, Rasmussen LH, Schindler TH, Svitil P, Vonk Noordegraaf A, Zamorano JL, Zompatori M; Task Force for the Diagnosis and Management of Acute Pulmonary Embolism of the European Society of Cardiology (ESC).** 2014 ESC guidelines on the diagnosis and management of acute pulmonary embolism. *Eur Heart J.* 2014 Nov 14;35(43):3033-69, 3069a-3069k. doi: 10.1093/eurheartj/ehu283. Epub 2014 Aug 29. Erratum in: *Eur Heart J.* 2015 Oct 14;36(39):2666. Erratum in: *Eur Heart J.* 2015 Oct 14;36(39):2642. PMID: 25173341.
14. Inari Medical. Available at: <https://www.inarimedical.com/flowtriever>.
15. **Tu T, Toma C, Tapon VF, Adams C, Jaber WA, Silver M, Khandhar S, Amin R, Weinberg M, Engelhardt T, Hunter M, Holmes D, Hoots G, Hamdalla H, Maholic RL, Lilly SM, Ouriel K, Rosenfield K; FLARE Investigators.** A Prospective, Single-Arm, Multicenter Trial of Catheter-Directed Mechanical Thrombectomy for Intermediate-Risk Acute Pulmonary Embolism: The FLARE Study. *JACC Cardiovasc Interv.* 2019 May 13;12(9):859-869. doi: 10.1016/j.jcin.2018.12.022. PMID: 31072507.