National Board of Examinations - Journal of Medical Sciences
Volume 3, Issue 1, Pages 120–132, January 2025
DOI 10.61770/NBEJMS.2025.v03.i01.014

CASE SERIES

Different Clinical Presentations of Pulmonary Embolism: A Case Series

Santhosh K B,[1,*] Rakesh Nayaka K[1] and Rahul S R[2]

[1]*Assistant Professor, Department of Emergency Medicine, Subbaiah Institute of Medical Sciences and Research Centre, Shivamogga, Karnataka, India*
[2]*Senior Resident, Department of General Medicine, Subbaiah Institute of Medical Sciences and Research Centre, Shivamogga, Karnataka, India*

Accepted: 22-October-2024 / Published Online: 06-January-2025

Abstract

Pulmonary embolism can present with a wide spectrum of clinical symptoms ranging from asymptomatic to life threatening phase to emergency department. It is one of the medical challenges with respect to its diagnosis and treatment. We present a case series of five such cases of presented to a tertiary care centre Subbaiah Institute of Medical Sciences and Research Centre, Shivamogga, Karnataka, India. Clinical, electrocardiographic and point of care ultrasound of heart, lungs and bilateral lower limb two point compression ultrasound done at the bedside along with all the initial stabilization steps done and their outcome are described in this case series. Early high degree of clinical suspicion and appropriate treatment is crucial. Failing which can lead to mortality. This case series highlights the different clinical presentations of pulmonary embolism including periarrest states that can present to emergency departments.

Keywords: Pulmonary embolism, Deep vein thrombosis, Hypercoagulability

*Corresponding Author: Santhosh K B
Email: santhosh.achar15892@gmail.com

Introduction

Pulmonary embolism (PE) is the third most common cause of cardiovascular death after myocardial infarction (MI) and cerebrovascular accidents (CVA) [1]. Various conditions lead to the generation of PE. Virchow's triad of hypercoagulability, venous stasis, and vessel wall injury is the final mechanism for most of the risk factors for developing PE. Overall, major risk factors for thromboembolic events include recent immobilization, myocardial infarction, cerebrovascular accidents, recent surgery and trauma. Additional major risk factors include prior venous thromboembolism, advanced age, malignancy, known thrombophilia, and indwelling venous catheter [2]. Pulmonary embolism can present with varied clinical profile starting from asymptomatic to life threatening stages. Diagnosing and managing pulmonary embolism at their extreme conditions is very challenging in emergency departments (ED). Owing to their severity and time constrains, only our high degree of clinical suspicion and clinical scoring systems can guide in further stabilizing the patients. Here, we present five such cases of pulmonary embolism with different clinical profiles who presented to our tertiary care centre Subbaiah Institute of Medical Sciences and Research Centre, Shivamogga, Karnataka, India.

Case Presentation

Case 1

A 40 years old male patient was brought to ED with sudden onset dyspnoea, excessive sweating, severe pain in upper back and right joint for ten minutes. There was history of right lower limb swelling for three days. Patient was known case of tuberculosis of right hip with secondary arthritis of right hip for two years with total hip arthroplasty status for three weeks followed by bed rest since then. Patient had completed antitubercular therapy. He had no other comorbidities. On arrival to ED, Patient was conscious with tachypnea, tachycardia with normal blood pressure and hypoxia with cold extremities and sweating. Electrocardiogram (ECG) showed sinus tachycardia with S1Q3T3 (deep S wave in lead I, deep Q wave and inversion of T wave in lead III) pattern (Figure 1). Point of care ultrasound (POCUS) of heart showed dilated right atrium(RA) and right ventricle(RV) with strain with D-sign(bowing or flattening of the interventricular septum into the left ventricle) (Figure 2) with distended and non collapsible inferior venacava (IVC) and negative bilateral lower limb venous compression ultrasound. Blood samples for D-dimer and troponin i were sent and both of which found elevated. The initial clinical diagnosis of pulmonary embolism was made based on the presentation. Patient was initially started on supplemental oxygen, fluids resuscitation, unfractionated heparin and tramadol. Eventually patient’s sensorium got deteriorated and landed up in cardiac arrest. Cardiopulmonary resuscitation (CPR) was started along with injection adrenaline and airway was secured. Arterial blood gases showed high anion gap metabolic acidosis for which injection sodium bicarbonate was given. CPR was continued till next 2hours. Patient could not be revived inspite of achieving return of spontaneous circulation (ROSC) three times during resuscitation and was declared dead.

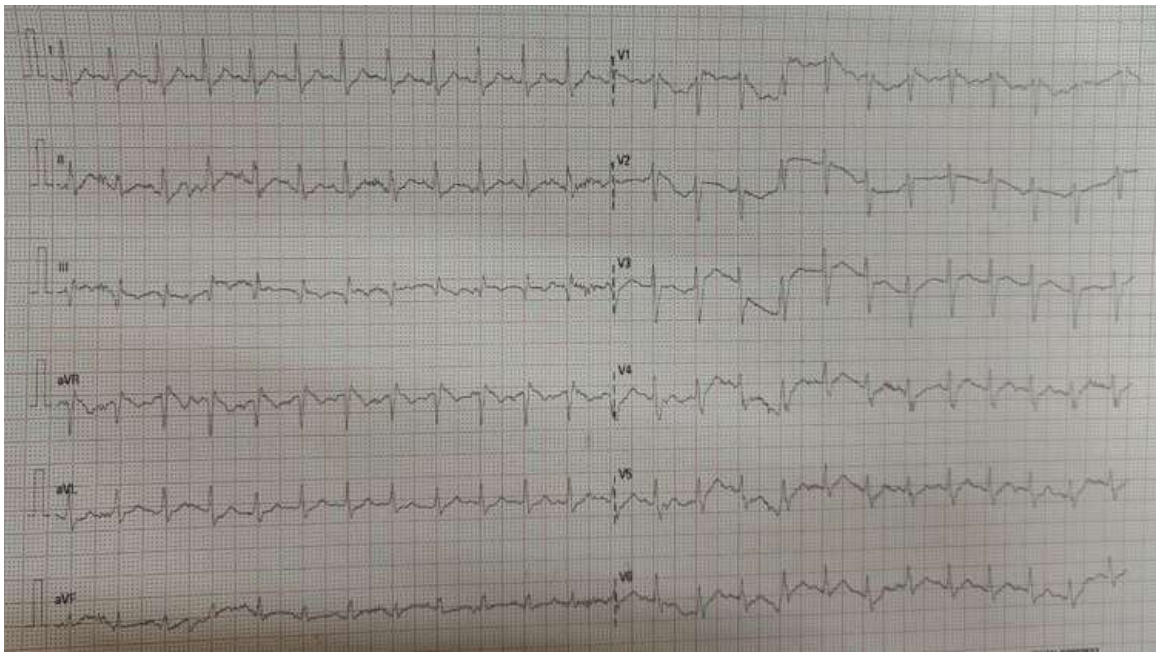

Figure 1. ECG showing sinus tachycardia and S1Q3T3 pattern

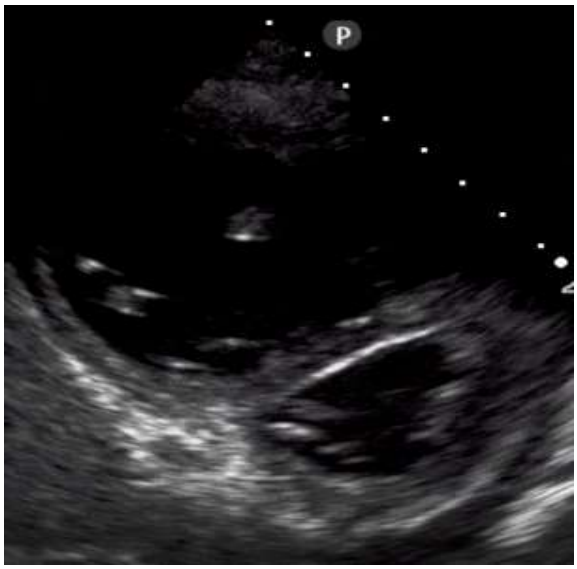

Figure 2. Cardiac POCUS showing D-sign (bowing of interventricular septum towards LV) in parasternal short axis view

Case 2

A 60 years old male presented to ED with vomiting and generalised weakness for 3 days. Patient was known case of metastatic carcinoma of tail of pancreas and had undergone exploratory laparotomy, palliative gastrojejunostomy and hepaticojejunostomy for duodenal obstruction 1 month before with stoma bag in situ containing bile stained fluid. Patient also had thrombosis of common femoral vein, superficial femoral vein, popliteal vein and proximal posterior tibial vein of right lower limb for which he was on tablet dabigatran 110 mg twice daily and tablet aspirin 150 mg once daily since 1

month. Patient was known smoker and alcoholic with no other comorbidities. At presentation to ED, he had tachycardia with normal oxygen saturation and blood pressure with mild pallor. Per abdomen was distended, mild tenderness in epigastrium and umbilical region with a sluggish bowel sounds. ECG showed sinus tachycardia (Figure 3). Ultrasound (US) of abdomen showed no new findings other than previous known pathology. Blood panel showed mild anemia with normal hematocrit with normal sodium, potassium and chloride levels. Patient was treated with fluids resuscitation, proton pump inhibitor, antiemetic, and intravenous anticoagulants. After two hours of arrival, patient developed sudden onset of breathlessness immediately followed by cardiac arrest. CPR was done according to advanced cardiac life support (ACLS) protocol for 30minutes along with securing the airway. POCUS while ongoing CPR showed dilated RV and IVC with a noncompressible right common femoral and popliteal veins (Figure 4 and 5). Unfractionated heparin was given while ongoing CPR. But the patient could not be revived and declared dead.

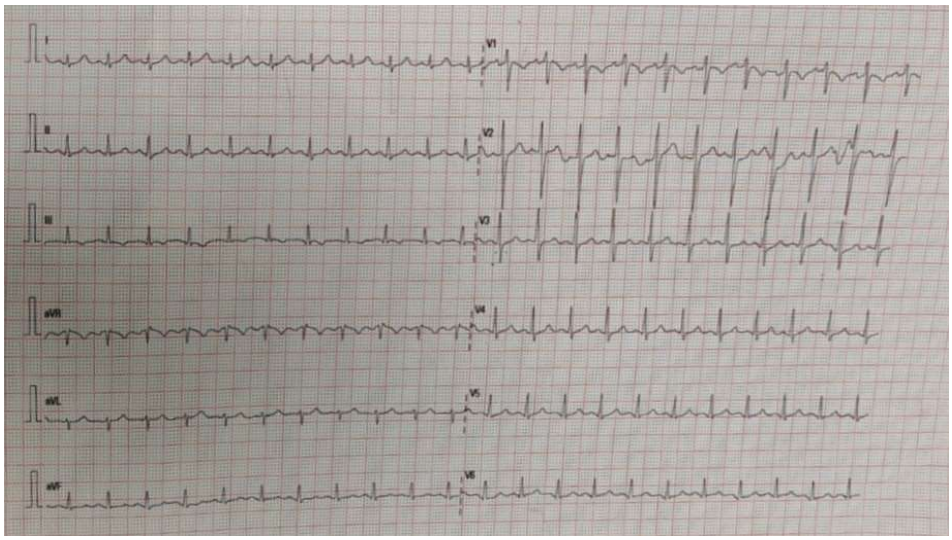

Figure 3. ECG showing sinus tachycardia

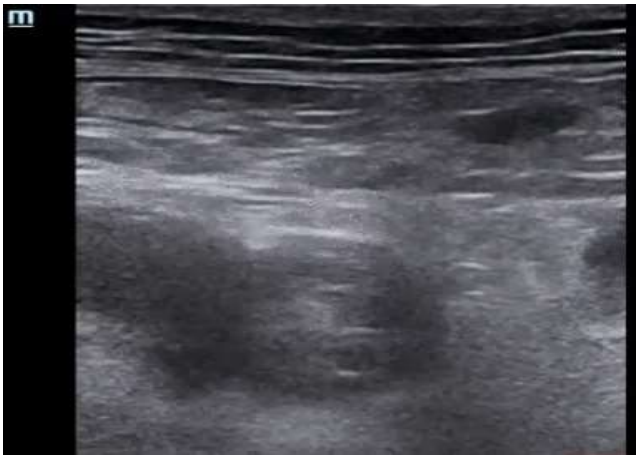

Figure 4. POCUS showing non compressible right common femoral vein with a thrombus inside

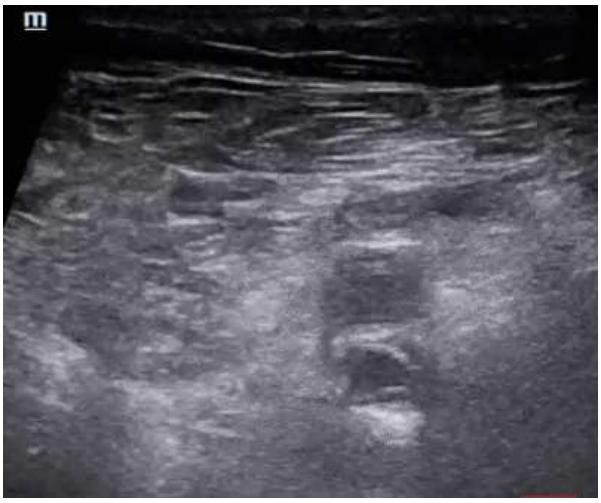

Figure 5. POCUS showing non compressible right popliteal vein

Case 3

A 44 years old female who was known case of systemic lupus erythematosus, antiphospholipid antibodies (APLA) positive status, hypothyroidism, type 2 diabetes mellitus, bilateral lower limbs deep vein thrombosis and pulmonary thromboembolism (PTE) for 6 years on home based oxygen therapy, tablet warfarin 5mg once daily, oral hypoglycemic agents and thyroxine supplements was brought to ED with complaints of exertional breathlessness and bilateral lower limb swelling for 1 month and fever for 3 days. At presentation, patient had tachycardia, tachypnoea and hypoxia of @ 80% @ room air which picked upto 95 % with 4 L/min of oxygen supplementation. Patient was put on noninvasive ventilation (NIV) support in view of increased work of breathing, but later got intubated. Electrocardiogram showed sinus tachycardia (Figure 6). POCUS of heart showed severe left ventricle (LV) dysfunction with ejection fraction (EF) of 35%, severe pulmonary arterial hypertension, dilated RA and RV with non collapsible IVC. POCUS of lungs showed showed bilateral A profile. Bilateral lower limbs US showed cellulitis with no evidence of current deep vein thrombosis (DVT). Bedside chest radiograph showed enlargement of right side of heart and large pulmonary arteries (Figure 7). Computed tomography of pulmonary angiogram (CTPA) was not done as the patient was unstable. On performing urinary catheterization, there was mild hematuria. International normalized ratio (INR) was 11.5, D-dimer was 400ng/mL and serum procalcitonin was 8.498 ng/ml. Antiplatelets and anticoagulants were withheld. Patient was started on injection vitamin K and 4 pints of fresh frozen plasma in view of high INR, broad spectrum antibiotics and other supportive therapies for the underlying conditions. After 4 hours, patient's consciousness was deteriorated and developed hypotension for which he was put on injection noradrenaline and vasopressin infusion. Eventually patient developed cardiac arrest. Code blue was announced and CPR was started according to ACLS protocol. Resuscitation was continued till next 20 minutes but couldn't revive. Later patient was declared dead.

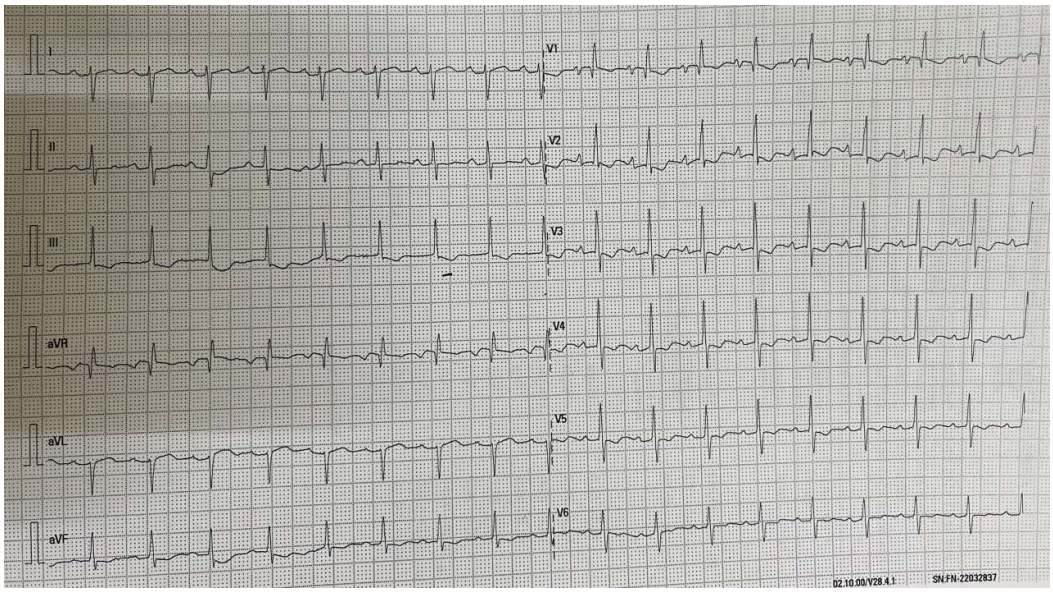

Figure 6. ECG showing sinus tachycardia

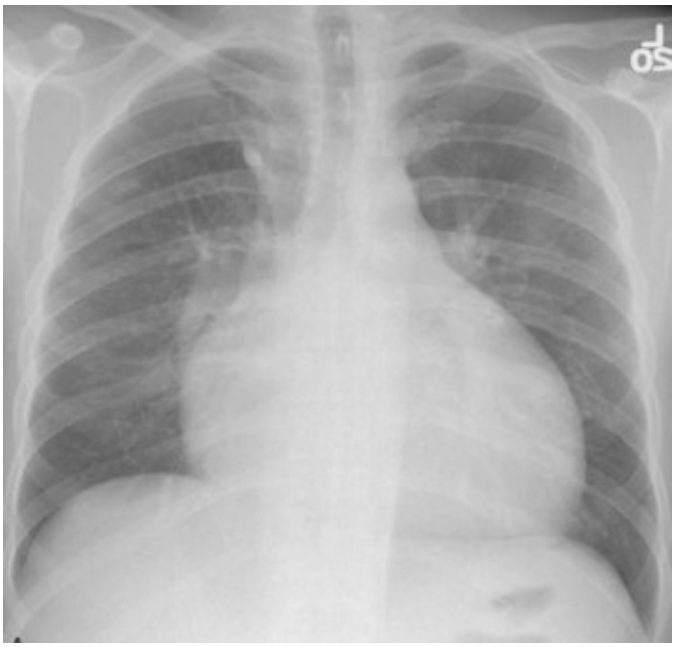

Figure 7. Bedside chest radiograph showing enlargement of right side of heart and large pulmonary arteries

Case 4

A 75 years old female with known case of hypertension and prior cardiac illness with no documents was presented to emergency department in drowsy state with a history of dyspnea from 1 day and progressive right leg swelling since 2 months. While examining the patient suddenly became unresponsive and went into cardiac arrest. Immediately code blue was announced and CPR started and resuscitation as per ACLS protocol after 5 minutes of CPR, ROSC achieved and endotracheal intubation was done and connected to ventilator. Post cardiac arrest care was initiated. ECG showed right axis

deviation, right bundle branch block with atrial fibrillation with fast ventricular rate (Figure 8). POCUS showed RA and RV dilated with global hypokinesia with normal left atrium (LA) (Figure 9). IVC was distended and non-collapsible. DVT scan of right leg showed no thrombus. Troponin I level was 25.9ng/L (>100ng/L to rule in). D-dimer level was 1500ng/mL. Since patient was unstable, CTPA was not done. Clinical examination, history, Well's score of 7.5 and cardiac POCUS were suggestive of pulmonary thromboembolism, hence injection streptokinase 2.5 lakh IU intravenous bolus was given. Again after 10 minutes, patient went into cardiac arrest. CPR was reinitiated and resuscitation was done for the next 15minutes. But the patient could not be revived and was declared dead.

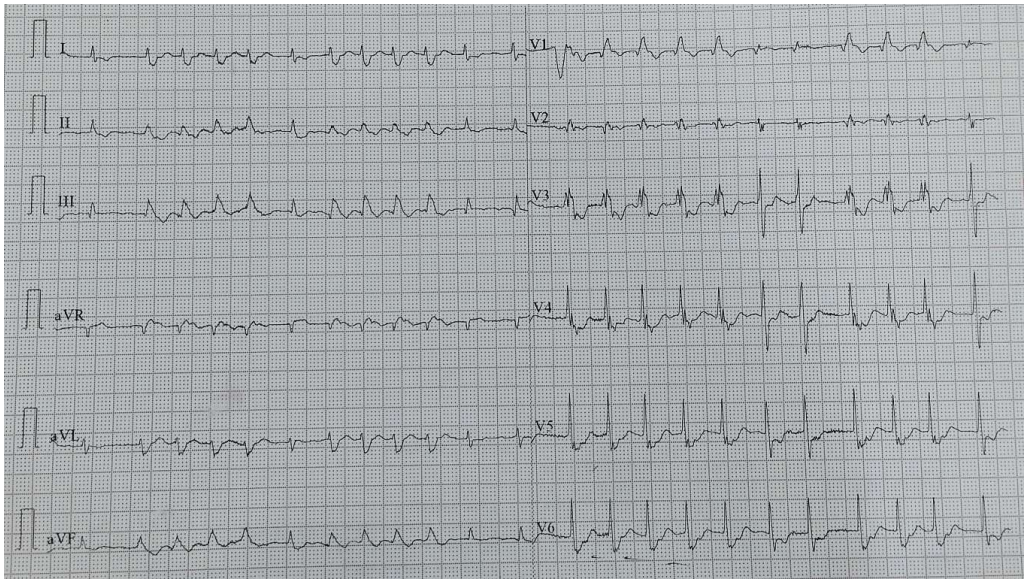

Figure 8. ECG showing right axis deviation, right bundle branch block and atrial fibrillation with fast ventricular rate

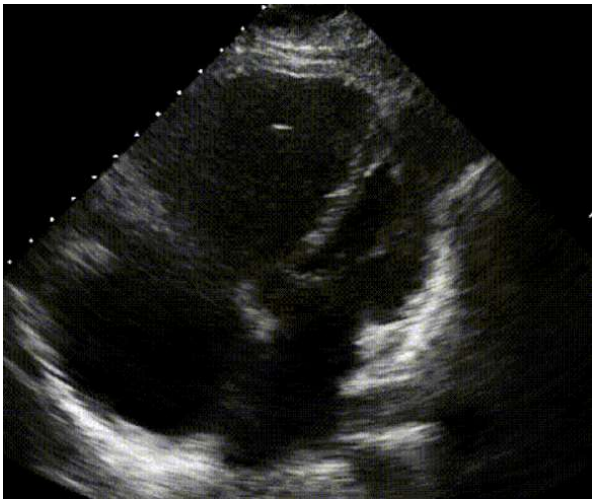

Figure 9. Cardiac POCUS showing dilated RA and RV in subcostal 4 chamber view

Case 5

A 61years old male with hypertension was brought to emergency department in gasping state with history of sudden onset of breathlessness since 4 hours and left leg swelling from 1 week. On examination, carotid pulse was absent. Immediately code blue was announced and CPR was started as per ACLS protocol and intubated after 2 cycles of CPR. POCUS during CPR showed dilated RV (Figure 10) and IVC. Left common femoral vein was non compressible (Figure 11). Unfractionated heparin 5000IU intravenous bolus was given. Effective resuscitation was continued till next 20 minutes. Despite adequate resuscitation and supportive care, the patient could not be revived and was declared dead.

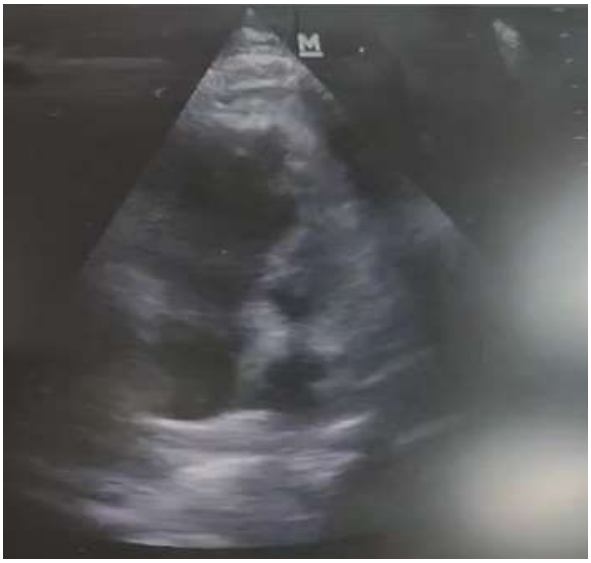

Figure 10. Cardiac POCUS showing dilated right ventricle with deviation of interventricular septum towards left ventricle in apical 4 chamber view

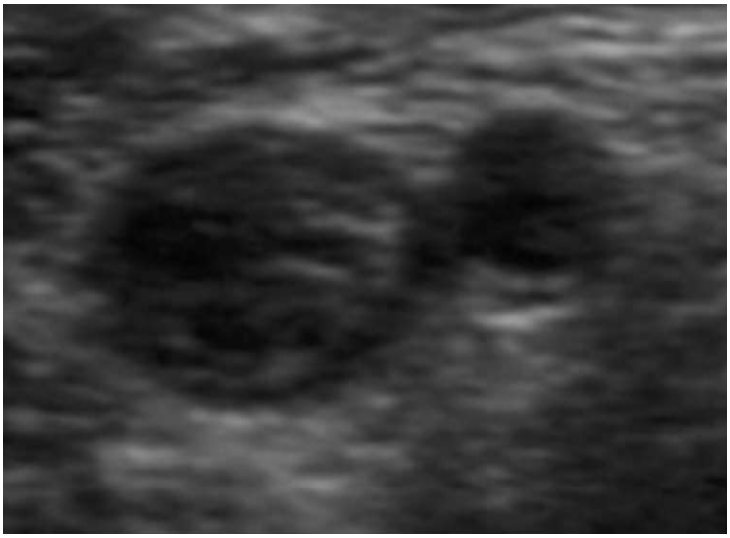

Figure 11. POCUS showing non compressible left common femoral vein

Discussion

Pulmonary embolism (PE) occurs when a thrombus originates elsewhere and enters the pulmonary circulation. In deep vein thrombosis, a thrombus develops within the deep veins, most commonly in the lower extremities. One or more risk factors were present in all 5 cases of the present study such as recent surgery, immobility, active malignancy, prior venous thromboembolism and deep vein thrombosis. Case 3 also had foci of sepsis which is a thrombogenic state, which could suggest it to be septic thromboembolism. Other risk factors for developing pulmonary embolism include advanced age, obesity, pregnancy, trauma, smoking, congestive heart failure, stroke, infections and associated inflammation and oral contraceptive pills. Pathophysiology typically involves the risk factors finally landed up in Virchow's triad i.e., stasis, vascular endothelial injury and hypercoagulable states and leading to pulmonary embolism.

Symptoms and signs were typical in all 5 patients. All patients had dyspnoea. Case 2 had only vomiting and generalized weakness with no respiratory involvement at initial presentation but later developed dyspnoea. Case 1, 2 and 3 had tachycardia and only case 1 and 3 had hypoxia. All cases except case 3 had unilateral lower limb swelling but only case 2 and 3 were previously known to be having deep vein thrombosis in lower limb. Case 3 had bilateral lower limb DVT as a consequence of APLA syndrome. Considering all risk factors, symptoms and signs, the modified Well's scores of all the cases in order were, 9, 11.5, 6, 7.5 and 6 respectively. All of them were >4 and are likely to have pulmonary embolism. Parameters of the modified Well's score system in pulmonary embolism have been depited in Table 1 [3].

Table 1: Modified Well's score for pulmonary embolism

Factor	Score*
Clinical symptoms of DVT	3
Other diagnoses less likely than pulmonary embolism	3
Heart rate >100 beats per min	1.5
Immobilization for 3 or more days or surgery in the previous four weeks	1.5
Previous history of DVT/PE	1.5
Hemoptysis	1
Malignancy	1

*Risk score interpretation (probability of pulmonary embolism): PE likely:>4.0, PE unlikely:≤4.0

Cases 1, 2 and 3 had sinus tachycardia, which is the most common ECG finding in PE. Case 1 had S1Q3T3 pattern as well which is pathognomonic of PE. Case 4 had right axis deviation, right bundle branch and atrial fibrillation with fast ventricular rate. For case 5, we couldn't get a 12 lead ECG as the patient was brought in cardiac arrest and CPR was started immediately.

POCUS was done for all 5 cases to assess the heart, lungs and both lower limbs for detecting deep vein thrombosis. Two point compression ultrasound, where

femoral vein and popliteal veins was used to assess for DVT. RV was dilated in all 5 cases. IVC was distended and non collapsible in all 5 cases. Case 1 had classical D-sign. Case 3 and 4 had LV hypokinesia also. Bilateral lower extremity compression ultrasound was positive in only case 2 and 5. In other cases it was negative. There could be a proximal DVT, distal DVT or DVT in a superficial vein which are not assessed in two point compression ultrasound [4]. At the same time, as in case 1, there were few cases reported as delayed fat embolism in patients who have undergone total hip replacement. So, this could create a dilemma whether its thromboembolism or delayed fat embolism. Other possible explanations for PE without DVT include complete embolization of lower extremity DVT, venous thromboembolism from uncommon sites (hepatic, renal, ovarian [5], neck, or upper extremity veins) [6,7], false-positive diagnosis of PE, false-negative venous leg ultrasound, isolated PE (I-PE) or de novo PE (DNPE) [8-10], and complete resolution of lower extremity DVT due to anticoagulation therapy in the short time interval between the diagnosis of PE and venous leg ultrasound [11].

D-dimer levels were elevated in cases 1, 2 and 4 and normal in case 3. It was not done in case 5 as the duration of resuscitation very short to send samples. Although CTPA having a gold standard value and highest diagnostic sensitivity and specificity for PE, it was not done in all 5 cases as all of them were hemodynamically unstable. One prospective study in the ED found that the triple POCUS exam (cardiac, lung, and leg veins ultrasound for DVT) in patients with a Well's score of 5 or more and positive D-dimer has a sensitivity of 90% and specificity of 86.2% for PE [12]. With the use of pretest probability, Well's score, ECG and POCUS of heart, lungs and bilateral lower extremity compression ultrasound, we can effectively suspect the hemodynamically unstable probable pulmonary embolism at the bedside.

Troponin I was elevated in case 1 and normal in case 4. However, troponin I being one of the cardiac biomarkers, can be elevated in pulmonary embolism also indicating the myocardial injury. INR was elevated in case 3. Based on the results of a study [13], and subsequent experience with other prospective clinical studies, the recommended therapeutic range is an INR of 2.0 to 3.0. An INR of 3.0 to 4.0 has been recommended for patients with antiphospholipid antibodies [14-16], although there is some disagreement on this issue [17]. Serum procalcitonin was done in case 3 which was found to be elevated. It indicates the presence of bacterial sepsis and guide the antibiotic approach.

Managing a case of massive pulmonary embolism is very challenging at ED. Early high degree of suspicion of the condition is crucial. While case 1 and 3 typically presented with symptoms of pulmonary embolism, for case 2 we did not have any clue that patient ends up in embolism. Case 4 and 5 presented to ED in periarrest condition. Although, there is only supportive therapy that can be given for fat embolism, we can still consider full anticoagulation until patient is not bleeding taking thromboembolism into account and this was considered in case 1. Case 1 was also treated with sodium bicarbonate in view of severe metabolic acidosis. Except case 3, all other patients received unfractionated heparin as full dose of anticoagulation. Case 3 was

initially treated with NIV support and early broad spectrum antibiotics as per sepsis guidelines. At the same time, while this patient was already on warfarin therapy and had high INR with mild hematuria at the presentation, the antiplatelets and anticoagulants were stopped and instead started on vitamin K and fresh frozen plasma to correct INR. Pulmonary embolism could still be the cause for the patient's breathlessness even with supratherapeatic INR values. The same had been reported as a case report in a study [18]. This is also supported by a study in which there was no clinically relevant difference in the INR values of patients who did or did not develop pulmonary embolism [19]. In case 4, with the background of history, examination and POCUS findings, fibrinolysis was done using streptokinase. Thrombolysis during ongoing CPR in presumed or confirmed PE has been supported in literature [20-22]. Periarrest patients like case 4 and 5 with suspected pulmonary embolism is not uncommon to receive in emergency departments. The effective use of bedside ultrasound (POCUS) of heart, lungs and DVT scan of limbs can be helpful. All 5 cases landed up in cardiac arrest and all of them received effective CPR according to ACLS guidelines and endotracheal intubation was also done in all patients. With the varied risk factors and comorbidities taken into account that led to pulmonary embolism, mortality will increase. Unfortunately, all 5 patients were died in spite of effective treatment done.

Conclusion

Pulmonary embolism has a wide range of clinical presentations ranging from asymptomatic to life threatening conditions. With the use of pretest probability, modified Well's score, electrocardiogram and point of care ultrasound, we can effectively diagnose the hemodynamically unstable probable pulmonary embolism at the bedside. Early high degree of suspicion and appropriate treatment is crucial. Failing which can lead to mortality. This case series highlights the different clinical presentations of pulmonary embolism including periarrest states that can present to emergency departments.

Statements and Declarations

Conflicts of interest

The authors declare that they do not have conflict of interest.

Funding

No funding was received for conducting this study.

Ethical Clearance

All ethical issues addressed by authors.

References

1. Weitz JI. Pulmonary embolism. In: Goldman L, Schafer AI, editors. Goldman's Cecil Medicine. Philadelphia, PA: Elsevier; 2011.
2. Tarbox AK, Swaroop M. Pulmonary embolism. Int J Crit Illn Inj Sci. 2013;3(1):69-72. doi: 10.4103/2229-5151.109427.
3. Vyas V, Sankari A, Goyal A. Acute Pulmonary Embolism. [Updated 2024 Feb 28]. In: StatPearls. Treasure Island (FL): StatPearls Publishing; 2024 Jan. Available from: https://www.ncbi.nlm.nih.gov/books/NBK560551/
4. Zitek T, Baydoun J, Yepez S, Forred W, Slattery DE. Mistakes and

Pitfalls Associated with Two-Point Compression Ultrasound for Deep Vein Thrombosis. West J Emerg Med. 2016;17(2):201-8. doi: 10.5811/westjem.2016.1.29335.

5. Verde F., Johnson P.T. One not to miss: Ovarian vein thrombosis causing pulmonary embolism with literature review. J. Radiol. Case Rep. 2012;6:23–28. doi: 10.3941/jrcr.v6i9.1062.
6. Levy M.M., Albuquerque F., Pfeifer J.D. Low incidence of pulmonary embolism associated with upper-extremity deep venous thrombosis. Ann. Vasc. Surg. 2012;26:964–972. doi: 10.1016/j.avsg.2011.12.016.
7. Levy M.M., Bach C., Fisher-Snowden R., Pfeifer J.D. Upper extremity deep venous thrombosis: Reassessing the risk for subsequent pulmonary embolism. Ann. Vasc. Surg. 2011;25:442–447. doi: 10.1016/j.avsg.2011.01.005.
8. van Gent J.-M., Zander A.L., Olson E.J., Shackford S.R., Dunne C.E., Sise C.B., Badiee J., Schechter M.S., Sise M.J. Pulmonary embolism without deep venous thrombosis: De novo or missed deep venous thrombosis? J. Trauma Acute Care Surg. 2014;76:1270–1274. doi: 10.1097/TA.0000000000000233.
9. Van Gent JM, Zander AL, Olson EJ, Shackford SR, Dunne CE, Sise CB, Badiee J, Schechter MS, Sise MJ. Pulmonary embolism without deep venous thrombosis: De novo or missed deep venous thrombosis? J Trauma Acute Care Surg. 2014 May;76(5):1270-4. doi: 10.1097/TA.0000000000000233.
10. Schwartz T, Hingorani A, Ascher E, Marks N, Shiferson A, Jung D, Jimenez R, Jacob T. Pulmonary embolism without deep venous thrombosis. Ann Vasc Surg. 2012 Oct;26(7):973-6. doi: 10.1016/j.avsg.2012.01.014.
11. Becher M, Heller T, Schwarzenböck S, Kröger JC, Weber MA, Meinel FG. Negative Venous Leg Ultrasound in Acute Pulmonary Embolism: Prevalence, Clinical Characteristics and Predictors. Diagnostics (Basel). 202217;12(2):520. doi: 10.3390/diagnostics12020520.
12. Thoracic ultrasound for diagnosing pulmonary embolism: a prospective multicenter study of 352 patients. Mathis G, Blank W, Reissig A, Lechleitner P, Reuss J, Schuler A, Beckh S. https://doi.org/10.1378/chest.128.3.1531. Chest. 2005;128:1531–1538.
13. Hull R, Hirsh J, Jay RM, Carter C, England C, Gent M, Turpie AGG, McLoughlin D, Dodd P, Thomas M, Raskob G, Ockelford P. Different intensities of oral anticoagulant therapy in the treatment of proximal-vein thrombosis. N Engl J Med. 1982;307:1676-1681.
14. Khamashta MA, Cuadrado MJ, Mujic F, Taub NA, Hunt BJ, Hughes GRV. The management of thrombosis in the antiphospholipid-antibody syndrome. N Engl J Med. 1995;332:993-997.
15. Violi F, Ferro D, Valesini G. Thrombosis in the antiphospholipid antibody syndrome. N Engl J Med. 1995;333:665.
16. Derksen RHWM, de Groot PG, Kater L, Nieuwenhuis HK. Patients with antiphospholipid antibodies and venous thrombosis should receive

long term anticoagulant treatment. Ann Rheum Dis. 1993;52:689-692.

17. Ginsberg JS, Wells PS, Brill-Edwards P, Donovan D, Moffatt K, Johnston M, Stevens P, Hirsh J. Antiphospholipid antibodies and venous thromboembolism. Blood. 1995;86:3685-3691.
18. Rivera-Morales MD, Wu JC, Dub L, Ganti L. Rare Presentation of Deep Vein Thrombosis and Submassive Pulmonary Emboli Due to Hypercoagulable State With Supratherapeutic Anticoagulation. Cureus. 2021;13(8):e17300. doi: 10.7759/cureus.17300.
19. Hansen P, Zmistowski B, Restrepo C, Parvizi J, Rothman RH. Does international normalized ratio level predict pulmonary embolism? Clin Orthop Relat Res. 2012 Feb;470(2):547-54. doi: 10.1007/ s11999-011-2007-7.
20. Feltes J, Popova M, Hussein Y, Pierce A, Yamane D. Thrombolytics in Cardiac Arrest from Pulmonary Embolism: A Systematic Review and Meta Analysis. Journal of Intensive Care Medicine. 2024;39(5):477-483. doi:10.1177/ 08850666231214754
21. Rech, Megan A. Horng, Michelle Holzhausen, Jenna M. Van Berkel, Megan A. Sokol, Sarah S, Peppard, Sarah, Hammond, Drayton A. International Survey of Thrombolytic Use for Treatment of Cardiac Arrest Due to Massive Pulmonary Embolism. Critical Care Explorations 2020;2(6):e0132. DOI: 10.1097/CCE.0000000000000132
22. Liu W, Liu LN, Lin CW, Wang HY, Wang XD. Successful Recovery After Prolonged Cardiopulmonary Resuscitation and Rescue Thrombolytics in a Patient with Cardiac Arrest Secondary to Presumed Massive Pulmonary Embolism. Risk Manag Healthc Policy. 2021;14:4253-4256 https://doi.org/10.2147/RMHP.S317 205