American Journal of Psychiatric Rehabilitation Expert Opinion Article

Unusual Presentations of Pulmonary Embolism on CT Pulmonary Angiography (CTPA)

Dr. Nidarshan.J[1*], Dr.G. Murugan[2]

[1*]M.D Radiodiagnosis, Junior Resident, Sree Balaji Medical College and Hospital, Chennai, Tamil Nadu
[2]Professor and HOD, Department of Radiodiagnosis, Sree Balaji Medical College and Hospital, Chennai, TamilNadu

Abstract
Computed Tomography Pulmonary Angiography (CTPA) is the gold standard for diagnosing the life-threatening condition, pulmonary embolism (PE), and therapy is needed as quickly as possible. Its radiological appearance, however, can occasionally be different from classic patterns and therefore be a subject for a diagnostic challenge. This study aims to characterize atypical presentations of PE on CTPA, focusing on unique interpretative difficulties. Retrospective analysis was performed on 5 patients with confirmed PE, in whom there were unusual CTPA findings in all patients. PE mimicking pneumonia, mass-like pulmonary infarction, saddle embolism without right heart strain, isolated subsegmental embolism with minimal symptoms, and tumour embolism from renal cell carcinoma were included in the cases. Initial diagnoses were postponed in three cases due to misleading thoracic imaging appearances resembling other thorax lesions. Accurate diagnoses require a thorough correlation with clinical data and re-evaluation of CTPA images. Findings underscored the need for a continued high index of suspicion for the rare cases and integration of clinical context with imaging to distinguish the image from other pathologies. Therefore, the radiologists must be vigilant and use advanced imaging techniques to aid timely and effective management of PE. The present case series illustrates the necessity for additional education in the radiologic

Keywords: CT Pulmonary Angiography (CTPA), pulmonary embolism (PE), retrospective analysis, life-threatening condition

1. Introduction

Pulmonary embolism (PE) is a life-threatening thromboembolic disorder triggered by the obstruction of the pulmonary arterial system by thrombi, usually arising from veins located deep within the lower limbs. PE is a prominent factor in avoidable in-hospital mortality and affects approximately 1 in 1000 individuals globally annually (Danwang et al., 2017; Heit et al., 2016). Delays, if long, become complicated by right heart failure, hypoxia, or death (Konstantinides et al., 2019; Tritschler et al., 2018). CTPA has now become the diagnostic of choice because of its superb spatial resolution, widespread availability, and the ability to also assess alternative thoracic pathologies. CTPA is very effective (>90% sensitivity, >90% specificity) in CTPA, taking the place of ventilation perfusion scans in many clinical applications (Patel et al., 2020; Remy-Jardin et al., 2006).

Characteristic findings include central or segmental intraluminal filling defects, the 'polo mint' or 'railway track' signs, and secondary features such as right ventricular (RV) dilatation and reflux of contrast into the inferior vena cava (Siripornpitak et al., 2021; Ahuja et al., 2022).

Although the diagnostic yield of CTPA is high, the interpretation of CTPA can be problematic. They might not notice subtle or non-classical presentations (Kaptein et al., 2021), so being overly reliant on classical sign may be a problem. In real life, sites of atypical findings like isolated subsegmental emboli, pulmonary infarction imitating infectious consolidation, or peripheral thrombi appearing neoplastic (Skulec et al., 2023; Rotzinger et al., 2020) may cause the differential diagnoses and misinterpretation. Moreover, motion artifacts, poor contrast timing, or technical errors can also lead to the compromising of image quality, which also makes the diagnosis difficult (Chaosuwannakit et al., 2020). Missed or delayed diagnosis of PE is a very important clinical problem: missed PE leads to worse outcomes (e.g., longer hospital stays and higher mortality). In up to 6% of CTPA scans, misdiagnoses may occur especially as it occurs in emergency studies where time limitations and overlapping thoracic pathologies may lead to cognitive bias or interpretative error (Roshkovan & Litt, 2018; Kwok et al., 2022). Additionally, such patients with atypical presentation, including patients with underlying malignancy, pneumonia and those with reduced cardiopulmonary reserve are less likely to exhibit the classical clinical or radiological characteristics of PE (Kligerman et al., 2018; Yao et al., 2024).

There is a discrepancy between theoretical radiologic expectations and the actual diagnostic practice. While CTPA is very useful in showing the typical features of embolic occlusion, it is not without limitations. Most of the current training and

literature focus on the typical presentations, which may mask some of the atypical or unusual radiologic appearances that may be seen in clinical practice. The research gap thus relates to the absence of detailed case descriptions of atypical CTPA findings that may confuse even to experienced radiologists and emergency physicians. Some of the previous works have called for better training in the interpretation of CTPA and highlighted the importance of clinical correlation in reducing diagnostic errors (Khasin et al., 2023). There is still lack of extensive case reports and series that describe the diagnostic challenges and atypical imaging features of PE (Schonberger et al., 2020). This is not only a problem in clinical practice but also in medicolegal practice, as missed PE is one of the most common radiological malpractice claims (Revel et al., 2007; Thomas et al., 2024).

This paper will discuss atypical appearances of pulmonary embolism on CTPA that posed diagnostic difficulties. Every case is an excellent learning experience as it demonstrates how atypical imaging can mimic other thoracic processes like pneumonia, neoplasm, or infarction. These cases were chosen to cover a wide range of imaging characteristics from peripheral emboli with ground-glass appearance to tumour embolism with vessel occlusion. In this case series, these presentations are discussed to:

• Describe practical issues in the interpretation of CTPA images.
• Stress on the clinical and radiologic correlation in the cases of doubt.
• Increase the knowledge of the radiologist and clinician about non-classical signs of PE.
• Introduce less interpretational errors within the first diagnosis and set the right tone.
Case-based learning is an effective approach in teaching and learning of radiology. It enables practitioners to get a feel of the diagnostic reasoning process using narratives and illustrations.

This series intends to enrich this conceptual framework toward the implementation of an enhanced value of the vigilant and pattern recognition in the thoracic imaging. This work aims at filling a clinically relevant diagnostic gap, the underdiagnosis of atypical presentations of PE on CTPA.

2. Methodology

2.1 Study Design

This was a retrospective multicentric study from January 2021 to March 2025 across two tertiary care hospitals. The purpose was to assess atypical presentations of PE on CTPA. This case series presents five cases of PE with atypical imaging features in total, which are difficult to diagnose. Thus, the emphasis was made on the atypical patterns of embolization, such as peripheral, segmental, emboli mimicking pneumonia and tumour emboli which are difficult to diagnose.

2.2 Patient Selection

This analysis was conducted on patients they had CTPA confirmed pulmonary embolism but with non-classical images of the condition. Specifically, five cases of atypical imaging findings were discussed, including peripheral embolism that may be confused with pneumonia, mass-like pulmonary infarction, or tumour embolism. These cases were selected to demonstrate the potential sources of mistake that one can encounter when atypical CTPA findings are evident. In the study, patients in this condition were omitted if they had characteristic imaging features of PE or had not received definitive confirmation of PE.

2.3 Imaging Protocol

64-slice multi-detector CT (MDCT) scanners were used for CT Pulmonary Angiography with a non-ionic iodinated contrast agent administered intravenously at a rate of 4 mL/s. Imaging was done in axial, coronal and sagittal planes with a slice thickness of 1.0 mm and increment of 0.5 mm to improve visualization. In three of the five cases, 3D post processing was used to enhance the assessment of peripheral and subsegmental emboli that were difficult to visualize with standard axial imaging alone. Bolus tracking was used to optimize the contrast bolus to achieve proper enhancement of the pulmonary arteries.

2.4 Ethical Approval

Both participating hospitals obtained ethical approval from the Institutional Review Board (IRB). Since this was a retrospective review of anonymized data, informed consent was waived. Identifiers of the patients were removed for data protection regulations. The research followed Helsinki Declaration ethical standards and received necessary permission from the respective IRBs of both institutions for the retrospective analysis of CTPA data.

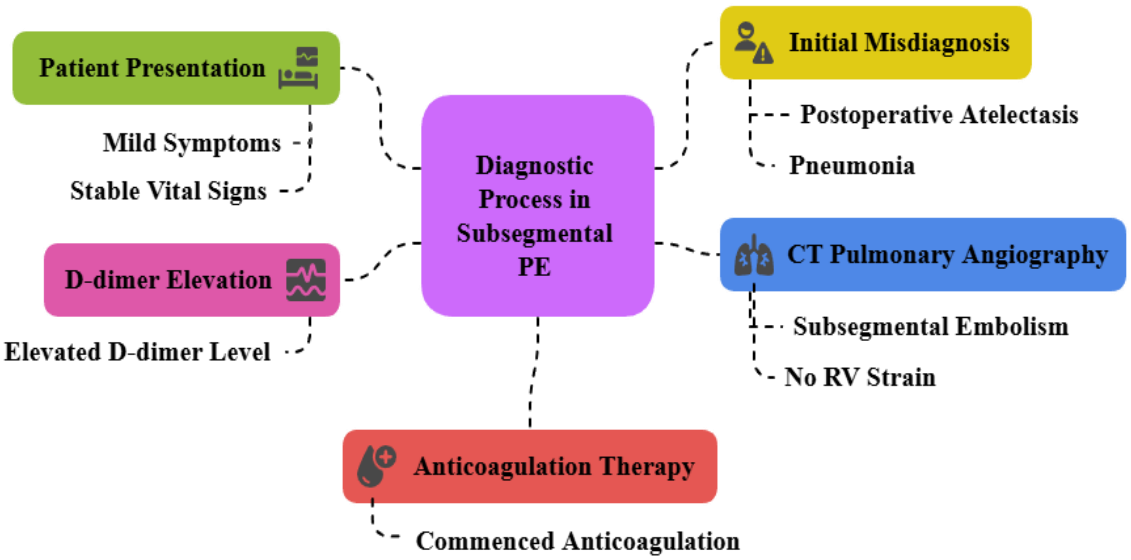

Figure 1: Diagnostic Process of Pulmonary Embolism

Figure 1 shows the algorithm of the diagnostic approach to subsegmental pulmonary embolism in postoperative patients. It starts with patient presentation, D-dimer level increase, and initial misdiagnosis and goes through confirmation by CTPA and ends with the commencement of anticoagulation therapy.

3. Case presentations

Case 1: Peripheral PE Mimicking Pneumonia with Ground-Glass Opacities

A 54-year-old man with hypertension and obesity, presented with symptoms of a pleural-type chest pain that has started two days prior to admission and dyspnoea. He had been diagnosed with an upper respiratory tract infection few days ago, but the symptoms did not subside. A long-distance truck driver, his probability of developing venous stasis and deep vein thrombosis (DVT) was high. The patient had no history of pulmonary embolism or any other severe respiratory diseases. At the time of presentation, he was hemodynamically stable with blood pressure of 135/85 mmHg, heart rate of 92 beats per minute and oxygen saturation of 92% on air. Routinely laboratory examinations indicated the presence of D-dimmer at a level of 2.8 µg/mL, which was a sign of the possible thromboembolic process. Ground troponin was not elevated, and on ECG there was normal sinus rhythm without any indication of right ventricular compromise. A chest X-ray was done which showed ground-glass opacities in the right lower lobe which was first diagnosed as pneumonia (Table 1).

Table 1: Laboratory and Diagnostic Test Findings in Case 1

Test	Normal Range	Results	Clinical Interpretation
D-dimer	< 0.5 µg/mL	2.8 µg/mL	Elevated, suggestive of thromboembolic event, but not specific to PE.
Troponin	< 0.04 ng/mL	Within normal limits	Excludes myocardial injury, not consistent with acute MI.
ECG	Normal sinus rhythm	Sinus rhythm, no signs of right heart strain	No evidence of acute right ventricular overload, ruling out massive PE.
Chest X-ray	Normal lung parenchyma	Ground-glass opacities in right lower lobe	Initially interpreted as pneumonia, but did not correlate with clinical symptoms.
CTPA	N/A	Peripheral segmental embolus with ground-glass opacities	Confirmed PE; embolus located in peripheral arteries; ground-glass opacities consistent with ischemic changes.
B-type natriuretic peptide (BNP)	< 100 pg/mL	Within normal limits	Rules out acute heart failure, supporting absence of heart failure as cause of symptoms.
Arterial Blood Gases (ABG)	pH: 7.35-7.45, PaO2: 80-100 mmHg	PaO2: 92 mmHg	Normal oxygenation, no significant hypoxemia, further supporting non-respiratory etiology for hypoxia.

CT Pulmonary Angiography (CTPA) was done which confirmed the diagnosis of PE with a peripheral embolus in the right lower lobe, in a segmental pulmonary artery (Figure 2). The embolus was

associated with ground-glass opacities, which made the clinicians worried about pulmonary infarction or infection. However, the contrast dynamics revealed ischemic changes in the embolic area, which did not enhance, excluding infection. Also, no right ventricular dilatation or increased RV/LV ratio was observed, which would indicate hemodynamic compromise. Subsequently, dual-energy CT showed that the ground-glass opacities were ischemic, which was indicative of PE and not an infection.

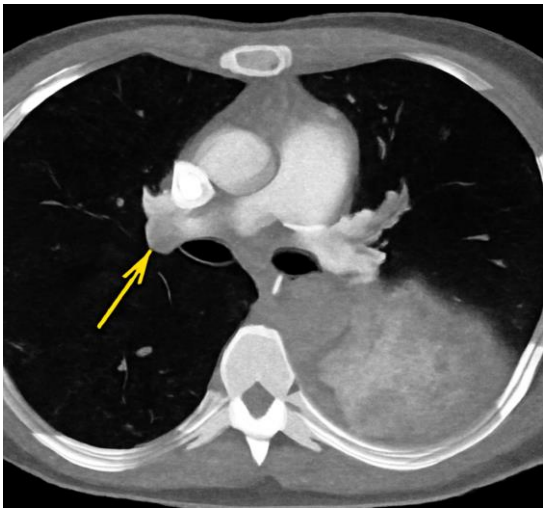

Figure 2: CT Pulmonary Angiography Showing Peripheral Pulmonary Embolism with Ground-Glass Opacities Mimicking Pneumonia

The initial misinterpretation of CTPA as pneumonia was due to the presence of ground-glass opacities that are characteristic of infections. However, the fact that the embolus was located peripherally, and no consolidation was present helped in ruling out pneumonia. Other differential diagnoses that were considered included pulmonary infarction and autoimmune diseases; however, the clinical signs and imaging findings were more suggestive of PE. This was supported by the patient's travel history of traveling long distances. The patient was initiated on unfractionated heparin and later switched to a DOAC after 48 hours. The subsequent imaging done at six weeks showed no more embolus and less ground-glass opacities indicating that the pulmonary infarction has improved. This case brings to light the importance of incorporating PE in differential diagnosis in instances of peripheral ground-glass opacities in such high-risk patients.

Case 2: Saddle Embolus with Hemodynamic Stability and Absent Right Heart Strain

This is a 48-year-old female, previous history of asthma and mild obesity presented in the emergency department with the clinical presentation of dyspnoea, tachypnoea, and sudden sharp chest pain on the right side while inspiring air. She had no previous history of DVT or PE, but she said that she was mostly confined to bed due to her knee operation which she had done few weeks ago. The patient also had a history of cardiovascular disease in the family history. She had a history of asthma which was controlled with inhaled corticosteroids; her vital signs on examination included blood pressure of 118/72 mmHg, pulse of 102 beats per minute, respiratory rate of 22 breaths per minute and oxygen saturation of 94% on air. Physical assessment revealed that the child was having increased breathing rate but no breathing difficulties, oxygen saturation or cyanosis and had wheeze suggesting asthma. Cardiovascular examination was unremarkable.

The first investigations included a D-dimer test which came out to be 4.5µg/mL, which indicated a thromboembolic event. The troponin levels were normal, and ECG showed sinus tachycardia without any signs of right-side heart stress. Chest X-ray was normal. Since the D-dimer was raised and the clinical index of suspicion was high, a Computed Tomography Pulmonary Angiography (CTPA) was carried out. The CTPA revealed a large saddle embolus in both main pulmonary arteries and extending into the left and right pulmonary arteries. The patient was not in any shock, right ventricle was not enlarged or strained, therefore no massive PE with hemodynamic compromise. Table 2 shows the laboratory and diagnostic tests of this case.

Table 2: Laboratory and Diagnostic Test Findings in Case 2

Test	Normal Range	Results	Clinical Interpretation
D-dimer	< 0.5 µg/mL	4.5 µg/mL	Elevated, strongly suggestive of thromboembolic event, but non-specific for PE.
Troponin	< 0.04 ng/mL	Normal	Excludes myocardial injury, ruling out acute myocardial infarction.
ECG	Normal sinus rhythm	Sinus tachycardia, no signs of right heart strain	No evidence of acute right heart strain, ruling out massive PE.
Chest X-ray	Normal lung parenchyma	Normal	No consolidation or pleural effusion, ruling out infection or other pulmonary pathology.
CTPA	N/A	Saddle embolus in both pulmonary arteries, no RV dilatation	Confirms PE with large saddle embolus, no hemodynamic collapse despite large embolus.
B-type natriuretic peptide (BNP)	< 100 pg/mL	Within normal limits	No indication of acute heart failure, supporting the diagnosis of PE rather than heart failure.
Arterial Blood Gases (ABG)	pH: 7.35-7.45, PaO2: 80-100 mmHg	PaO2: 94 mmHg	Normal oxygenation, ruling out significant hypoxia or respiratory failure.

The diagnostic challenge was the paradox of the patient's relative stability given the size of the embolus. Thus, initial misdiagnosis of PE severity occurred, and other conditions, like asthma exacerbation or costochondritis were considered instead. Heparin was started for anticoagulation and thrombolysis was not given due to no hemodynamic instability. The patient was then changed to a DOAC to achieve the target INR level. Follow-up imaging done after four weeks showed that the thrombus had decreased in size and so had the patient's symptoms. This case demonstrates that saddle emboli can be present without acute right heart strain or hemodynamic compromise even with large emboli. Clinicians should always consider PE in patients with such presentations, even if they do not have right heart strain or shock.

Case 3: Pulmonary Infarct Presenting as a Lung Mass (Mass-Like Consolidation)

A 62-year-old female with hypertension, diabetes, and COPD visits the clinic because she suddenly developed breathing problems, coughing, and blood in her sputum for three days. She had been bedridden for a long time after hip replacement surgery six weeks ago, but she had no history of DVT or PE in the past one week. She had a 20-year smoking history. On examination, the patient had the following vital signs: blood pressure 130/80 mmHg, pulse 100 bpm, respiratory rate 20 breaths per minute, and oxygen saturation of 90% on air. There were diminished breath sounds in the right lower lung with rales and wheeze in the same region. The laboratory investigations revealed a raised D-dimer level of 5.2 µg/mL, and a slightly raised leukocyte count of 12,000/µL (Table 3). Specifically, the troponin level was also within normal range indicating no acute myocardial injury had taken place. Chest X-ray examination revealed new infiltrate and consolidation in the right lower lobe which could be suggestive of lung cancer, pneumonia or pulmonary infarction. A CT Pulmonary Angiography (CTPA) was done to exclude PE. It showed a segmental embolus in the right lower lobe with infarction mimicking mass-like consolidation (Figure 3). The embolus was in the right lower segmental pulmonary artery, with alveolar haemorrhage and edema around it. The right ventricle appeared normal in size and function which indicated no pressure problems in the heart.

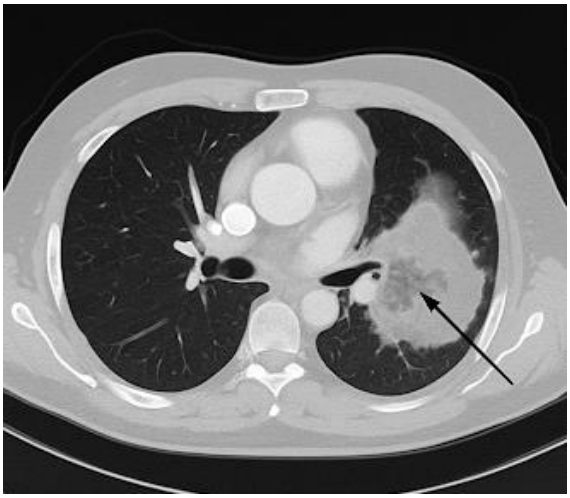

Figure 3: CTPA Showing Pulmonary Infarction Presenting as Mass-Like Consolidation in the Right Lower Lobe

Table 3: Laboratory and Diagnostic Test Findings in Case 3

Test/Diagnostic Feature	Normal Range	Results	Clinical Interpretation
D-dimer	< 0.5 µg/mL	5.2 µg/mL	Significantly elevated, indicating a thromboembolic event. Though non-specific, it suggests PE or pulmonary infarction.
Troponin	< 0.04 ng/mL	Normal	Excludes myocardial injury, ruling out acute myocardial infarction.
ECG	Normal sinus rhythm	Sinus tachycardia, no right heart strain	No evidence of acute right heart strain, which rules out massive PE.
Chest X-ray	Normal lung parenchyma	Mass-like consolidation in right lower lobe	Raised suspicion for pulmonary infarction or infection due to mass-like appearance.
CTPA	N/A	Segmental embolus with associated mass-like consolidation and alveolar haemorrhage in right lower lobe	Confirmed pulmonary embolism with infarction, presenting as mass-like consolidation.
B-type natriuretic peptide (BNP)	< 100 pg/mL	Normal	Rules out heart failure as a cause for symptoms, supporting PE diagnosis.
Arterial Blood Gases (ABG)	pH: 7.35-7.45, PaO2: 80-100 mmHg	PaO2: 94 mmHg	Normal oxygenation, confirming no significant hypoxia despite the infarction.
White Blood Cell Count (WBC)	4,000–10,000/µL	12,000/µL	Mild leucocytosis, possibly due to inflammation related to infarction.

The large lung tissue changes made doctors think about cancer or infection. Test results showed no signs of infection or cancer and ruled out other possible conditions because of the blocked blood flow. The medical team started the patient on heparin treatment then changed to DOAC when the patient reached stable condition. After four weeks of follow-up imaging showed the consolidation had shrunk while the patient experienced better symptoms. Patient no longer experienced bleeding from the lungs and continued receiving anticoagulation therapy for 3 to 6 months. The case shows that doctors should include pulmonary infarction in their list of possible diagnoses when they see mass-like lung consolidation on medical images. CTPA tests showed what caused the embolism and let doctors treat the patient without needing risky tests for possible cancer or infection.

Case 4: Subsegmental Embolism in Post-Operative Patient with Minimal Symptoms

A 60-year-old male who had knee arthroplasty surgery three days earlier came to the emergency department because of mild breathing problems and tiredness. He stayed in bed following surgery and only faced hypertension and obesity as his medical issues. The patient had smoked for 30 years and received enoxaparin treatment but did not experience pulmonary embolism or deep vein thrombosis before. Medical tests showed a stable patient with blood pressure at 135/85 mmHg, an 84-beat-per-minute pulse, 16 breathing cycles per minute, and 98% oxygen saturation while breathing regular air. On physical examination, there was some degree of discomfort around the surgical site and no features of pleural effusion or rales. Depending on the initial laboratory tests, the D-dimer was slightly increased at 1.8 µg/mL and less than normal range of <0.5 µg/mL suggesting a possibility of thromboembolic event. Elevated troponin levels were not suggestive of acute myocardial infarction as the troponin levels were within normal range. ECG showed sinus rhythm with no signs of right heart strain and chest X-ray was normal. However, due to the postoperative state, a CTPA was done to exclude PE despite the lack of significant signs (Table 4).

Table 4: Laboratory, Imaging, and Diagnostic Findings in Case 4

Test/Diagnostic Feature	Normal Range	Results	Clinical Interpretation
D-dimer	< 0.5 µg/mL	1.8 µg/mL	Elevated, suggests thromboembolic event, indicating potential for PE.
Troponin	< 0.04 ng/mL	Normal	Excludes myocardial infarction, confirming no acute cardiac involvement.
ECG	Normal sinus rhythm	Sinus tachycardia, no right heart strain	Normal, ruling out significant right heart strain typically seen in massive PE.

Chest X-ray	Normal lung parenchyma	Unremarkable, no signs of consolidation or pleural effusion	No indication of pneumonia or other pulmonary pathology, ruling out infection.
CTPA	N/A	Subsegmental embolism in right lower lobe	Confirms subsegmental PE, with no signs of right heart strain or infarction.
B-type natriuretic peptide (BNP)	< 100 pg/mL	Normal	No signs of acute heart failure, supporting the diagnosis of PE.
Arterial Blood Gases (ABG)	pH: 7.35-7.45, PaO2: 80-100 mmHg	PaO2: 94 mmHg	Normal oxygenation, confirming the absence of significant hypoxia.

CTPA showed a subsegmental embolism in the right lower lobe without much pulmonary infarction or right ventricular (RV) strain. The embolus was visualized as a filling defect in a small peripheral pulmonary artery branch which could have been easily missed if high resolution imaging was not used (Figure 4). This was because the patient had very few complaints and normal vital signs, which resulted in the initial diagnosis of atelectasis or postoperative pneumonia. However, the D-dimer level and CTPA result supported subsegmental PE. Since the patient's clinical condition remained stable and there was no RV strain, thrombolysis was not needed, and the patient was started on anticoagulation with unfractionated heparin and then switched to DOACs. At the follow up after 2 weeks, the symptoms were much relieved and there was no dyspnoea or fatigue and the CTPA revealed that the embolism was partially resolved. This case underlines the importance of suspicion of subsegmental PE in postoperative patients even if there are no clinical signs. CTPA still holds the best practice in such circumstances.

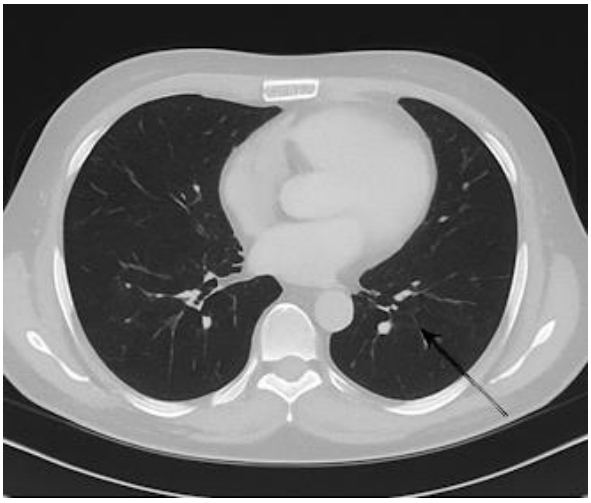

Figure 4: CTPA Showing Subsegmental Embolism in the Right Lower Lobe

Case 5: Tumour Embolism Secondary to Renal Cell Carcinoma Extension

A 65-year-old man with past medical history of RCC, which has been operated one year ago, left radical nephrectomy, was referred to our clinic with complains of dyspnoea, pleuritic chest pain and haemoptysis. He had a history of hypertension and smoking. The patient was stable after nephrectomy but had new complaints that required emergency care. He was on postoperative anticoagulation therapy with enoxaparin at that time. His vital signs were as follows: blood pressure 120/75 mmHg, pulse rate 100 beats per minute, respiratory rate 22 breaths per minute, and oxygen saturation 92% on room air. Evaluation of the chest X-ray revealed bilateral manners of opacities, which might be suggestive of pulmonary embolism or metastasis. The biochemical tests showed a raised D-dimer level of 6.5 μg/mL, and a slightly high leukocyte count of 12,500/μL (Table 5). Troponin was not elevated, thus eliminating myocardial infarction, but in ECG, sinus tachycardia was noted without signs of right heart strain.

Table 5: Laboratory, Imaging, and Diagnostic Findings in Case 5

Diagnostic Feature	Normal Range	Results	Clinical Interpretation
D-dimer	< 0.5 µg/mL	6.5 µg/mL	Strongly elevated, suggesting a thromboembolic event; non-specific, but strongly indicates embolic phenomenon.
Troponin	< 0.04 ng/mL	Normal	Excludes myocardial injury, confirming that the symptoms are not due to myocardial infarction.
ECG	Normal sinus rhythm	Sinus tachycardia, no signs of right heart strain	No acute right heart strain, ruling out a massive PE that would typically present with hemodynamic compromise.
Chest X-ray	Normal lung parenchyma	Diffuse opacities, no consolidation	Suggests a possible metastatic process or pulmonary infarction; warrants further investigation through imaging.
CTPA	N/A	Tumor thrombus extending from left renal vein into IVC and right pulmonary artery	Confirms tumor embolism due to RCC metastasis, obstructing pulmonary vasculature; no signs of classic PE.
B-type natriuretic peptide (BNP)	< 100 pg/mL	Normal	Excludes acute heart failure, reinforcing the primary diagnosis of tumour embolism rather than heart failure.
Arterial Blood Gases (ABG)	pH: 7.35-7.45, PaO2: 80-100 mmHg	PaO2: 92 mmHg	Normal oxygenation, confirming no significant hypoxia or respiratory failure despite the embolism.

CT Pulmonary Angiography (CTPA) done indicated the presence of a tumour thrombus in the left renal vein through to the IVC and in the right pulmonary artery. This finding confirmed the diagnosis of tumour embolism due to renal cell carcinoma. The embolus impacted the right pulmonary vasculature, and the diagnosis was in line with renal cell carcinoma metastasis. The diagnosis was made at the first instance difficult because the patient's presentation resembled that of pulmonary embolism. However, the CTPA findings of a tumour thrombus indicated that the embolic event was associated with RCC and its metastatic potential, which is a rare occurrence. This case demonstrates that non-thrombotic causes of embolism should be considered in patients with a history of cancer. The patient was prescribed low molecular weight heparin (LMWH) for anticoagulation. Since the patient developed tumour embolism, chemotherapy was started, and the oncologists considered the possibility of surgical removal of the embolic mass. The follow-up imaging done after 2 weeks revealed that the embolus was partly dissolved, and the patient's condition was much better. He then proceeded with targeted therapy for RCC. This case also underlines the importance of a multimodal approach in the treatment of tumour embolism and the value of CTPA in the diagnosis of this condition. It also reminds the clinician to consider tumour thrombi in patients with cancer who present with pulmonary symptoms even when the clinical signs of PE are not evident.

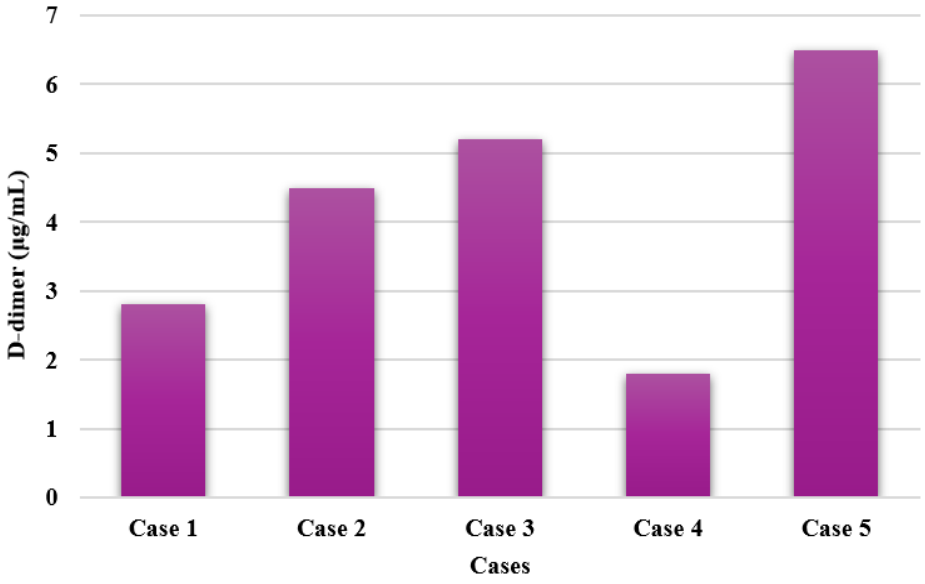

Figure 5: D-dimer levels about cases in diagnosing Pulmonary Embolism (PE)

Figure 5, represents the observation on cases of D-dimer (µg/mL) computed to measure the variation among the cases identified. Most of the time, elevated levels indicate the occurrence of a thromboembolic event, although D-dimer is not specific for PE. The graph shows the relationship between D-dimer level and the clinical probability of PE in the clinical practice.

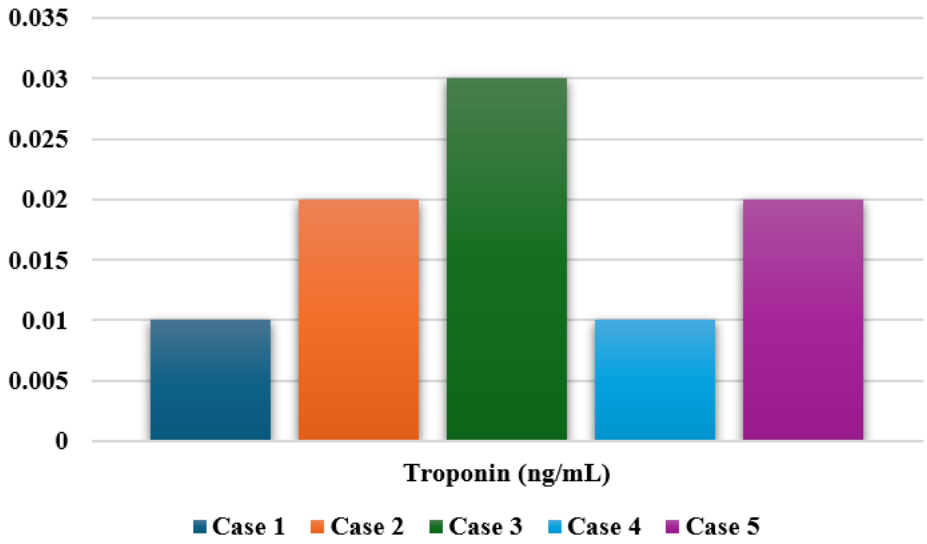

Figure 6: Troponin Levels Across Cases in the Exclusion of Myocardial Injury

Figure 6 shows the Troponin levels (ng/mL) for each case and all the values are within the normal range. The normal Troponin levels eliminate myocardial injury and therefore acute myocardial infarction. The graph also supports the fact that Troponin testing was useful in distinguishing between PE and other possible causes such as heart attack.

4. Discussion

Pulmonary embolism (PE) is associated with considerable clinical problems since its symptoms are multifaceted and unclear often mimicking various other processes, including infections, tumours, and infarctions. CTPA findings that are atypical include peripheral emboli or mass-like consolidations, which are seen in Case 1 and Case 3, respectively; these findings are like those seen in infectious pneumonia or pulmonary infarction (De Jong et al., 2024; Matusov & Tapson, 2021). On the other hand, the common manifestations of PE are central emboli with features of right heart dysfunction and vascular occlusion. Nevertheless, in Case 2, saddle embolism, the size of the embolism was massive, but the patient had few complaints and no signs of right heart failure, which means that even large emboli may not cause hemodynamic collapse (Ajah, 2024). These variations highlight the need to consider the whole range of clinical and imaging features to arrive at the correct diagnosis. Some of the conditions that may mimic PE include infection, malignancy, or pulmonary infarction, which leads to diagnostic errors. This is well illustrated in case 3 where a pulmonary infarct was mimicking a lung mass. This mass-like consolidation seen on CTPA was initially considered as malignancy, which is why it is crucial to distinguish between these possible diagnoses (Bellouki et al., 2024). Another complicating factor for this diagnosis is the presence of motion artefact or suboptimal bowel preparation where normally an embolic finding might be seen (Stals et al., 2021).

Multi-planar reconstructions are very useful in overcoming these challenges since they provide high-resolution imaging. CTPA's ability to display the embolism in axial, coronal, and sagittal planes, as well as the 3D reconstructions, improves the diagnostic accuracy of the test and provides a better assessment of the size, location, and vascular changes of the embolism (Teng et al., 2018). CTPA has many drawbacks. Motion artifacts, especially in the patients who are not cooperative or those with severe respiratory problems, can hide the findings, thus resulting in false negatives. Also, poor contrast opacification may lead to false positive results where vascular structures are depicted as emboli (Khandait et al., 2023; Mudalsha et al., 2011). The radiological mistakes in Case 5 highlight why there is a need to differentiate between embolic and tumour-related causes when diagnosing patients with renal cell carcinoma (Tan et al., 2023). In evaluating patients with PE, differential diagnosis should comprise many diseases that may mimic PE on imaging, such as infections, tumours, pulmonary embolism, and autoimmune disorders. Every case in this series highlighted the importance of ruling out other causes and especially in patients with comorbidities such as RCC or post-surgical status (Majidi et al., 2023). Furthermore, the comparison with the

literature review and case reports and retrospective studies, it can be concluded that even though PE is still a leading cause of sudden death, the diagnostic difficulties associated with atypical presentations have been described earlier (De Jong et al., 2024; Erythropoulou-Kaltsidou et al., 2020).

Some of the studies have revealed that delayed diagnosis due to wrong interpretation of CTPA is related with increased morbidity and mortality, which underlines the importance of accurate and timely interpretation of the images (Wijesuriya et al., 2013). These days advanced medical experts use dual-energy CT scans and perfusion imaging methods as additional tests to diagnose PE when standard tools are not enough. Studies show that dual-energy CT produces better image quality and finds pulmonary embolism better than standard imaging methods so it can help CTPA in hard-to-diagnose cases (Tan et al., 2023). The new technology lets doctors see how blood flows through the lungs and checks blood vessel health which helps them diagnose subsegmental embolism and tumour blockages. Each of these types of missed diagnosis of PE has clinical relevance, especially increased mortality, further morbidity, and potential for medicolegal consequences. This is why it is crucial to enhance the practice of radiology and to use more extensive diagnostic methods. The future recommendations for radiology practice include further advancement of the multi-planar reconstruction, improvement of the CTPA protocol, and the application of other imaging techniques like the dual-energy CT to minimize the errors and improve the accuracy of the diagnosis (Righini et al., 2017).

5. Conclusion

This case series emphasize on how practitioners can easily overlook signs of CTPA in the early stages by emphasizing with the case scenarios of patients with PE. While the conventional symptoms are fully described, this series outlines the problems caused by atypical symptoms such as peripheral embolism, pulmonary infarction and tumour embolism that are alleged to have signs resembling other pulmonary diseases. It was seen that misdiagnosis and inaccurate diagnosis is truly associated clinical history, imaging, and follow-up. It is crucial to encourage the continuing education of healthcare professionals, especially radiologists and clinicians, to be able to identify PE, even in atypical cases. Clinician, radiologist, and oncologist cooperation improves the diagnostic approach, especially in cases of pulmonary infarction or tumour embolism. Further studies should be directed towards optimizing the techniques like dual-energy CT and advanced perfusion imaging for better identification of subsegmental emboli and other uncommon forms of embolism. Hence, further revising guidelines for clinical diagnosis in the diagnosis of PE taking into consideration, newer diagnostic technologies and a broader approach for early detection of pulmonary embolism in high-risk cases or atypical patients shall prove useful in minimizing diagnostic errors and improving patient outcomes.

References

1. Ahuja, J., Palacio, D., Jo, N., Strange, C. D., Shroff, G. S., Truong, M. T., & Wu, C. C. (2022). Pitfalls in the imaging of pulmonary embolism. *Seminars in Ultrasound, CT, and MRI, 43*(3), 221–229. https://doi.org/10.1053/j.sult.2022.01.004
2. Ajah, O. N. (2024). Pulmonary embolism and right ventricular dysfunction: Mechanism and management. *Cureus, 16*(9), e70561. https://doi.org/10.7759/cureus.70561
3. Bellouki, O., Soufiani, I., Boualaoui, I., Ibrahimi, A., El Sayegh, H., & Nouini, Y. (2024). Renal cell carcinoma with massive cavo-atrial tumor thrombus leading to pulmonary embolism: Case study and literature review. *International Journal of Surgery Case Reports, 117*, 109577. https://doi.org/10.1016/j.ijscr.2024.109577
4. Chaosuwannakit, N., Soontrapa, W., Makarawate, P., & Sawanyawisuth, K. (2020). Importance of computed tomography pulmonary angiography for predicting 30-day mortality in acute pulmonary embolism patients. *European Journal of Radiology Open, 8*, 100340. https://doi.org/10.1016/j.ejro.2021.100340
5. Danwang, C., Temgoua, M. N., Agbor, V. N., Tankeu, A. T., & Noubiap, J. J. (2017). Epidemiology of venous thromboembolism in Africa: A systematic review. *Journal of Thrombosis and Haemostasis, 15*(9), 1770–1781. https://doi.org/10.1111/jth.13769
6. De Jong, C., Kroft, L., Van Mens, T., Huisman, M., Stöger, J., & Klok, F. (2024). Modern imaging of acute pulmonary embolism. *Thrombosis Research, 238*, 105-116. https://doi.org/10.1016/j.thromres.2024.04.016
7. Erythropoulou-Kaltsidou, A., Alkagiet, S., & Tziomalos, K. (2020). New guidelines for the diagnosis and management of pulmonary embolism: Key changes. *World Journal of Cardiology, 12*(5), 161. https://doi.org/10.4330/wjc.v12.i5.161
8. Heit, J. A., Spencer, F. A., & White, R. H. (2016). The epidemiology of venous thromboembolism. *Journal of Thrombosis and Thrombolysis, 41*(1), 3–14. https://doi.org/10.1007/s11239-015-1311-6
9. Kaptein, F., Kroft, L., Hammerschlag, G., Ninaber, M., Bauer, M., Huisman, M., & Klok, F. (2021). Pulmonary infarction in acute pulmonary embolism. *Thrombosis Research, 202*, 162-169.

https://doi.org/10.1016/j.thromres.2021.03.022

10. Khandait, H., Harkut, P., Khandait, V., & Bang, V. (2023). Acute pulmonary embolism: Diagnosis and management. *Indian Heart Journal, 75*(5), 335-342. https://doi.org/10.1016/j.ihj.2023.05.007
11. Khasin, M., Gur, I., Evgrafov, E. V., Toledano, K., & Zalts, R. (2023). Clinical presentations of acute pulmonary embolism: A retrospective cohort study. *Medicine, 102*(28), e34224. https://doi.org/10.1097/MD.0000000000034224
12. Kligerman, S. J., Mitchell, J. W., Sechrist, J. W., Meeks, A. K., Galvin, J. R., & White, C. S. (2018). Radiologist performance in the detection of pulmonary embolism. *Journal of Thoracic Imaging, 33*(6), 350–357. https://doi.org/10.1097/rti.0000000000000361
13. Konstantinides, S. V., Meyer, G., Becattini, C., Bueno, H., Geersing, G., Harjola, V., Huisman, M. V., Humbert, M., Jennings, C. S., Jiménez, D., Kucher, N., Lang, I. M., Lankeit, M., Lorusso, R., Mazzolai, L., Meneveau, N., Áinle, F. N., Prandoni, P., Pruszczyk, P., . . . Pepke-Zaba, J. (2019). 2019 ESC Guidelines for the diagnosis and management of acute pulmonary embolism developed in collaboration with the European Respiratory Society (ERS). *European Heart Journal, 41*(4), 543–603. https://doi.org/10.1093/eurheartj/ehz405
14. Kwok, C. S., Wong, C. W., Lovatt, S., Myint, P. K., & Loke, Y. K. (2022). Misdiagnosis of pulmonary embolism and missed pulmonary embolism: A systematic review of the literature. *Health Sciences Review, 3*, 100022. https://doi.org/10.1016/j.hsr.2022.100022
15. Majidi, A., Mahmoodi, S., Baratloo, A., & Mirbaha, S. (2016). Atypical presentation of massive pulmonary embolism, a case report. *Emergency, 2*(1), 46. https://pmc.ncbi.nlm.nih.gov/articles/PMC4614616/
16. Matusov, Y., & Tapson, V. F. (2021). Radiologic mimics of pulmonary embolism. *Postgraduate Medicine, 133*(sup1), 64–70. https://doi.org/10.1080/00325481.2021.1931370
17. Mudalsha, R., Jacob, M., Pandit, A., & Jora, C. (2011). Extensive tumor thrombus in a case of carcinoma lung detected by F18-FDG-PET/CT. *Indian Journal of Nuclear Medicine: IJNM: The Official Journal of the Society of Nuclear Medicine, India, 26*(2), 117. https://doi.org/10.4103/0972-3919.90269
18. Patel, H., Sun, H., Hussain, A. N., & Vakde, T. (2020). Advances in the diagnosis of venous thromboembolism: A literature review. *Diagnostics, 10*(6), 365. https://doi.org/10.3390/diagnostics10060365
19. Remy-Jardin, M., Bahepar, J., Lafitte, J., Dequiedt, P., Ertzbischoff, O., Bruzzi, J., Delannoy-Deken, V., Duhamel, A., & Remy, J. (2006). Multi-detector row CT angiography of pulmonary circulation with gadolinium-based contrast agents: Prospective evaluation in 60 patients. *Radiology, 238*(3), 1022–1035. https://doi.org/10.1148/radiol.2382042100
20. Revel, M., Triki, R., Chatellier, G., Couchon, S., Haddad, N., Hernigou, A., Danel, C., & Frija, G. (2007). Is it possible to recognize pulmonary infarction on multisection CT images? *Radiology, 244*(3), 875–882. https://doi.org/10.1148/radiol.2443060846
21. Righini, M., Robert-Ebadi, H., & Le Gal, G. (2017). Diagnosis of acute pulmonary embolism. *Journal of Thrombosis and Haemostasis, 15*(7), 1251–1261. https://doi.org/10.1111/jth.13694
22. Roshkovan, L., & Litt, H. (2018). State-of-the-art imaging for the evaluation of pulmonary embolism. *Current Treatment Options in Cardiovascular Medicine, 20*(9). https://doi.org/10.1007/s11936-018-0671-6
23. Rotzinger, D. C., Knebel, F., Jouannic, M., Adler, G., & Qanadli, S. D. (2020). CT pulmonary angiography for risk stratification of patients with nonmassive acute pulmonary embolism. *Radiology: Cardiothoracic Imaging, 2*(4), e190188. https://doi.org/10.1148/ryct.2020190188
24. Schonberger, M., Lefere, P., & Dachman, A. H. (2020). Pearls and pitfalls of interpretation in CT colonography. *Canadian Association of Radiologists Journal, 71*(2), 140–148. https://doi.org/10.1177/0846537119892881
25. Siripornpitak, S., Kunjaru, U., Sriprachyakul, A., Promphan, W., & Katanyuwong, P. (2021). Correlating computed tomographic angiography of pulmonary circulation with clinical course and disease burden in patients with tetralogy of Fallot and pulmonary atresia. *European Journal of Radiology Open, 8*, 100363. https://doi.org/10.1016/j.ejro.2021.100363
26. Skulec, R., Parizek, T., David, M., Matousek, V., & Cerny, V. (2021). Lung point sign in ultrasound diagnostics of pneumothorax: Imitations and variants. *Emergency Medicine International, 2021*, 6897946. https://doi.org/10.1155/2021/6897946
27. Stals, M., Kaptein, F., Kroft, L., Klok, F., & Huisman, M. (2021). Challenges in the diagnostic approach of suspected pulmonary embolism in COVID-19 patients. *Postgraduate Medicine, 133*(sup1), 36–41. https://doi.org/10.1080/00325481.2021.1920723

28. Tan, J. E., Vishnu, S., & Singh, D. (2023). Pulmonary tumor embolism in renal cell carcinoma detected by hybrid CT and F18-PSMA PET. *Radiology Case Reports, 18*(11), 4222. https://doi.org/10.1016/j.radcr.2023.08.090
29. Teng, E., Bennett, L., Morelli, T., & Banerjee, A. (2018). An unusual presentation of pulmonary embolism leading to infarction, cavitation, abscess formation, and bronchopleural fistulation. *BMJ Case Reports, 2018*, bcr2017222859. https://doi.org/10.1136/bcr-2017-222859
30. Thomas, S. E., Weinberg, I., Schainfeld, R. M., Rosenfield, K., & Parmar, G. M. (2024). Diagnosis of pulmonary embolism: A review of evidence-based approaches. *Journal of Clinical Medicine, 13*(13), 3722. https://doi.org/10.3390/jcm13133722
31. Tritschler, T., Kraaijpoel, N., Le Gal, G., & Wells, P. S. (2018). Venous thromboembolism: Advances in diagnosis and treatment. *JAMA, 320*(15), 1583–1594. https://doi.org/10.1001/jama.2018.14346
32. Wijesuriya, S., Chandratreya, L., & Medford, A. R. (2013). Chronic pulmonary emboli and radiologic mimics on CT pulmonary angiography. *CHEST Journal, 143*(5), 1460–1471. https://doi.org/10.1378/chest.12-1384
33. Yao, D., Cao, W., & Liu, X. (2024). Clinical manifestations and misdiagnosis factors of pulmonary embolism patients seeking treatment in cardiology. *Medicine, 103*(49), e40821. https://doi.org/10.1097/MD.0000000000040821