Pulmonary Embolism or COVID-19 Pneumonia? A Case Report

Nahid Borna

Iran University of Medical Sciences (IUMS)

Maryam Niksolat

Iran University of Medical Sciences (IUMS)

Behnam Shariati

Iran University of Medical Sciences (IUMS)

Vahid Saeedi

Iran University of Medical Sciences (IUMS)

Leila Kamalzadeh (✉ lkamalzadeh@gmail.com)

Iran University of Medical Sciences (IUMS)

Case Report

Keywords: Venous Thromboembolism, Pulmonary Embolism, COVID-19, Delayed Diagnosis

Posted Date: February 24th, 2022

DOI: <https://doi.org/10.21203/rs.3.rs-1378278/v1>

License: This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Abstract

Background: Pulmonary embolism (PE) is the most serious clinical presentation of venous thromboembolism (VTE), a major cause of sudden death. In the current Coronavirus disease 2019 (COVID-19) pandemic, when a patient presents with respiratory symptoms, many serious possible non-Covid causes including PE may be considered the next priority after COVID-19 on the differential diagnosis list. Thus, the correct diagnosis may be delayed or missed, with grave consequences to the patient's outcome and safety.

Case Presentation: We herein present a case of a 63-year-old Iranian female who was admitted to our hospital showing symptoms of delirium superimposed on dementia. Soon after admission, she developed fever and respiratory symptoms. However, overestimating the likelihood of COVID-19 pneumonia and attributing the patient's symptoms to this disease led to delayed diagnosis and treatment of pulmonary embolism, resulting in the patient's death.

Conclusions: During the COVID-19 pandemic, a high index of suspicion is required for the timely diagnosis of PE, especially in patients with identifiable risk factors. This is specifically true for patients who are unable to express their symptoms due to neurocognitive disorders.

Introduction

Pulmonary embolism (PE), the most lethal presentation of venous thromboembolism (VTE), often arises from a deep venous thrombosis (DVT)(1). However, PE can originate de-novo without any evidence of DVT(2). PE is one of the most common cardiovascular diseases, occurring in 1–2 per 1,000 people annually(1). Major risk factors for PE include immobilization, smoking, obesity, malignancy, recent surgery, acute and chronic medical illness, recent trauma to lower extremities and pelvis, pregnancy, hormone replacement therapies, chronic obstructive pulmonary disease, and a previous history of PE or a known thrombophilia. Still, a third to a half of patients with PE have no identifiable provoking factors(1, 3). From age 45 years onwards, the lifetime risk of developing VTE is 8%(4). PE has a wide variety of clinical presentations ranging from a silent pulmonary embolus to a massive PE leading to right heart failure with shock(1). About 20% of patients with PE die before diagnosis or shortly thereafter, particularly if the embolism is associated with hemodynamic instability(5). Although the classic symptoms of PE are pleuritic chest pain, dyspnea, hemoptysis, tachypnea, and tachycardia, the diagnosis of PE can be challenging because signs and symptoms of PE are often nonspecific and overlap those of many other diseases(1). Notably, coronavirus disease 2019 (COVID-19) can also present with cardiopulmonary manifestations including chest pain, tachycardia, tachypnea, and dyspnea leading to pneumonia, bronchitis, and acute respiratory distress syndrome(6). Unfortunately, in the current pandemic situation, when a patient presents with respiratory symptoms, many serious possible non-Covid causes including PE may be considered the next priority after COVID-19 on the differential diagnosis list. Thus, the correct diagnosis may be delayed or missed, with grave consequences to the patient's outcome and safety. We

herein present a case of a 63-year-old female who was admitted to our hospital with symptoms mimicking COVID-19.

Case Presentation

In January 2022, at the height of the COVID-19 Omicron pandemic, a 63-year-old Iranian woman was referred to our hospital showing symptoms of confusion, disorientation, severe agitation, visual and auditory hallucinations, and impaired sleep. She had a 2-year history of dementia with progressive behavioral and psychological disturbances being treated with rivastigmine, memantine, risperidone, nortriptyline, and fluvoxamine; however, her family reported an acute deterioration in her cognitive state beginning 2 weeks before admission. Five days before admission, she was visited at a neurology clinic, where she was diagnosed with delirium superimposed on dementia, and her previous medications were discontinued and clonazepam (1 mg at bedtime), haloperidol (0.5 mg three times a day), and quetiapine (100 mg, twice a day) were administered. Nonetheless, her mental state continued to deteriorate. Past medical history was unremarkable except for chronic hypertension, for which she took amlodipine (5mg once daily) and losartan (25mg twice a day). There was no remarkable family history for psychiatric or medical illnesses.

On admission, she was confused, restless, incoherent, and disorientated to time, place, and person. Her vital signs included a temperature of 36.8°C, heart rate of 96 beats per minute (bpm), blood pressure of 120/70 mmHg, respirations of 18 breaths per minute, and oxygen saturation of 92% on room air. Her systemic physical examination was normal except for a body mass index (BMI) of 32. There were no focal neurological deficits but she did not follow commands.

Baseline laboratory investigations were significant for hypokalemia, elevated C-reactive protein (CRP), elevated erythrocyte sedimentation rate (ESR), increased creatine phosphokinase (CPK), increased lactate dehydrogenase (LDH), and a mild respiratory alkalosis (Table 1). Complete blood count (CBC) with differential, fasting plasma glucose (FPG), Hemoglobin A1c, lipid profile, coagulation profile, alkaline phosphatase, urea, creatinine, urinalysis as well as liver and thyroid function test results were in the normal range. Urine testing for drugs of abuse (methamphetamine, amphetamine, cannabis, methadone, and morphine) was negative. Cultures of the blood, urine, and sputum were obtained. Quetiapine, clonazepam, and haloperidol were discontinued and treatment with intravenous infusion of 15% potassium chloride (KCl) solution and unfractionated heparin as venous thromboembolism prophylaxis (5000IU two times a day) was begun.

Table 1
Laboratory data on admission

	Variable	Reference Range	On Presentation
Complete blood count	Hemoglobin (g/dl)	12–16	14.1
	Hematocrit (%)	36–46	40.7
	Red-cell count (per mm ³)	4500000–6300000	4980000
	Mean corpuscular volume (µm ³)	77–97	81.7
	White-cell count (per mm ³)	4500–11000	5500
	Differential count (%)		
	Mixed	0–10	4.6
	Segment	40–70	69.7
	Lymphocytes	22–42	23.7
	Platelet count (per mm ³)	140000–440000	141000
Electrolytes	Sodium (mmol/L)	136–145	143
	Potassium (mmol/L)	3.7–5.5	3.2*
	Phosphate (mg/dl)	2.5-5	3.2
	Calcium (mg/dl)	8.6–10.6	9.1
	Magnesium (mg/dl)	1.8–2.6	2.1
Liver Enzymes	Alanine aminotransferase (IU/L)	5–40	22
	Aspartate aminotransferase (IU/L)	5–40	39
	Erythrocyte sedimentation rate (mm/hr.)	< 20	23*
	C-reactive protein (mg/L)	< 6	24*
	Lactate dehydrogenase (U/L)	225–500	639*
	Creatine phosphokinase (IU/L)	24–195	923*

*Abnormal value

	Variable	Reference Range	On Presentation
Venous blood gases	pH	7.31–7.41	7.47*
	pCO ₂ (mm Hg)	35–40	34.7*
	pO ₂ (mm Hg)	41–51	153.2*
	Bicarbonate (mmol/L)	22–26	25.3*
	Base excess	-2+2	2.7*
*Abnormal value			

Throat swab samples were sent for severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) by real-time reverse-transcriptase–polymerase-chain-reaction assay (RT-PCR). High-resolution computed tomography (HRCT) of the lungs showed bilateral predominantly perihilar ground-glass opacities (Fig. 1). Brain magnetic resonance imaging (MRI) revealed moderate global atrophy and no acute abnormality that could explain the patient's current condition was found.

The next day, she developed a low-grade fever (37.7°C), tachycardia (103 beats/min), tachypnea (24 breaths/min), and shortness of breath. Oxygen saturation dropped to 90% on room air. No evidence of myocardial ischemia was discovered on electrocardiography. Initial nasopharyngeal swabs came back negative for SARS-CoV-2. Because of the high index of suspicion for COVID-19 despite the negative initial nasopharyngeal RT-PCR result, a second RT-PCR was done. Lumbar puncture was performed and empiric antimicrobial therapy with intravenous ampicillin (2 mg every 4hours), vancomycin (1mg every 12hours), ceftriaxone (2mg every 8hours), and acyclovir (500mg IV every 8hours) was started.

Over the two following days, the patient's condition continued to deteriorate. On the fourth hospital day, she was intermittently confused and increasingly lethargic. The temperature was 38.1°C, the pulse rate was 130 bpm, the respirations were 26, the blood pressure was 90/60 mm Hg, and the oxygen saturation was 88% while she was breathing ambient air. The repeated RT-PCR test for SARS-CoV-2 came back negative. Cultures of the blood, urine, and sputum showed negative results. Analysis of the cerebrospinal fluid (CSF) was normal. Arterial blood gas test revealed a compensated metabolic acidosis (PH 7.27, pCO₂ 25.2 mmHg, and HCO₃ 11.6meq/L. Troponin level was 673.5(ng/ml) (normal < 19 ng/ml). D-dimer test was not performed because of the low clinical suspicion of PE. Transthoracic echocardiography revealed a hypokinetic and enlarged right ventricle suggestive of massive PE. While the patient was prepared for transfer to the intensive care unit (ICU), she developed a sudden cardiac arrest, and though resuscitation was started immediately, subsequently died.

Discussion

As the coronavirus pandemic is exerting pressure on health systems all over the world, physicians must stay tuned and have a high index of suspicion for COVID-19; however, too much focus on this disease can hinder timely diagnosis and prompt treatment of serious non-COVID conditions. Like COVID-19, pulmonary embolism may present with fever and cardiorespiratory symptoms such as a drop in blood pressure, tachycardia, dyspnea, tachypnea, and hypoxia. In the current case, overestimating the likelihood of COVID-19 pneumonia and attributing the patient's symptoms to this disease led to delayed diagnosis and treatment of pulmonary embolism. Other factors might additionally have led to a late diagnosis of PE in this patient. Having dementia, being unable to express her symptoms, and attributing symptoms of delirium to underlying dementia may have resulted in a delay in seeking medical attention by her family. Meanwhile, perhaps receiving thromboprophylaxis and lack of an apparent DVT led the physicians to consider the patient to be low-risk for PE, aggravating the delay in diagnosis.

This patient had several risk factors for developing PE that were overlooked by the physicians, including old age, being overweight, having hypertension, and prolonged limited mobility owing to dementia. Recent studies have also linked the current use of antipsychotics with the development of PE(7). As mentioned earlier, the patient had been using risperidone for two years before admission which may have increased the risk of PE. Interestingly, a recent study has shown that patients with dementia who have signs and risk factors of a PE are much less likely to be tested for PE than patients without dementia who had the same signs and risk factors. A possible explanation for this might be that patients with dementia might be challenging to assess because they may not be capable of conveying information about their medical condition(8). There is also the possibility that physicians underrate the risk factors that they cannot directly verify with their patients(9).

In the same vein, Bozorgmehr et al(10). and Overhoff et al.(11) reported cases of pulmonary thromboembolism and pulmonary infarction mimicking COVID-19 symptoms. All reported cases presented with fever, respiratory symptoms, and Chest CT findings typical for COVID-19. Nonetheless, in further investigations, COVID-19 was ruled out for them. Other case reports were published where PE presented atypically, mimicking bacterial pneumonia with symptoms of shortness of breath, cough, chest pain, and fever, but failed to respond to conventional therapy for pneumonia(12–14).

Conclusions

Taken together, during the COVID-19 pandemic, a high index of suspicion is required for the timely diagnosis of PE, especially in patients with identifiable risk factors. This is specifically true for patients who are unable to express their symptoms due to neurocognitive disorders.

Abbreviations

BMI: Body mass index

CBC: Complete blood count

COVID-19: Coronavirus disease 2019

CPK: Creatine phosphokinase

CRP: C-reactive protein

CSF: Cerebrospinal fluid

DVT: Deep venous thrombosis

ESR: Erythrocyte sedimentation rate

FPG: Fasting plasma glucose

HRCT: High-resolution computed tomography

ICU: Intensive care unit

KCL: Potassium chloride

LDH: Lactate dehydrogenase

MRI: Magnetic resonance imaging

PE: Pulmonary embolism

RT-PCR: Real-time reverse-transcriptase–polymerase-chain-reaction assay

SARS-CoV-2: Severe acute respiratory syndrome coronavirus 2

VTE: Venous thromboembolism

Declarations

Ethics approval and consent for publication

The authors state that they have followed the principles outlined in the Declaration of Helsinki. Written informed consent was obtained from the legal guardian for publication of this case report and any accompanying images.

Availability of data and materials

Not applicable.

Competing interests

The authors declare that they have no competing interests.

Funding

None.

Authors' contributions

NB and BS have made substantial contributions to the conception of the work. NB and LK have drafted the work. MN and VS have substantively revised the manuscript. All co-authors have approved the submitted version and have participated sufficiently in the work to take responsibility for the content.

Acknowledgments

The authors would like to express their gratitude to the authorities of Rasool Akram Medical Complex Clinical Research Development Center (RCRDC) for their technical and editorial assistance.

References

1. Morrone D, Morrone V. Acute Pulmonary Embolism: Focus on the Clinical Picture. *Korean Circ J*. 2018 May;48(5):365–81.
2. Van Gent J-M, Zander AL, Olson EJ, Shackford SR, Dunne CE, Sise CB, et al. Pulmonary embolism without deep venous thrombosis: De novo or missed deep venous thrombosis? *J Trauma Acute Care Surg*. 2014;76(5):1270–4.
3. Doherty S. Pulmonary embolism: An update. *Aust J Gen Pract*. 2017 Sep 18;46:816–20.
4. Raskob GE, Angchaisuksiri P, Blanco AN, Buller H, Gallus A, Hunt BJ, et al. Thrombosis. *Arterioscler Thromb Vasc Biol*. 2014 Nov 1;34(11):2363–71.
5. Di Nisio M, van Es N, Büller HR. Deep vein thrombosis and pulmonary embolism. *Lancet (London, England)*. 2016;388(10063):3060–73.
6. Suh YJ, Hong H, Ohana M, Bompard F, Revel M-P, Valle C, et al. Pulmonary Embolism and Deep Vein Thrombosis in COVID-19: A Systematic Review and Meta-Analysis. *Radiology*. 2020/12/15. 2021 Feb;298(2):E70–80.
7. Liu Y, Xu J, Fang K, Xu Y, Gao J, Zhou C, et al. Current antipsychotic agent use and risk of venous thromboembolism and pulmonary embolism: a systematic review and meta-analysis of observational studies. *Ther Adv Psychopharmacol*. 2021 Jan 1;11:2045125320982720.
8. Kamalzadeh L, Moghaddamnia M, Malakouti SK, Rashedi V, Bahrampour S, Sharifi N, et al. Prevalence of Dementia Among Older Patients: A Hospital-Based Study in Iran. *Am J Alzheimers Dis Other Dement*. 2019;34(7–8):500–6.
9. Ly DP, Shekelle PG. How physicians evaluate patients with dementia who present with shortness of breath. *J Am Geriatr Soc*. 2022 Jan 1;70(1):119–25.
10. Bozorgmehr R, Pishgahi M, Tajabadi Z, Aryafar M. Pulmonary Thromboembolism and Infarction Mimicking COVID-19 Symptoms; Report of three Cases. *Arch Acad Emerg Med*. 2020 Nov

2;8(1):e88–e88.

11. Overhoff D, Walter T, Gruettner J, Janssen S, Riffel J, Hoffmann U, et al. Acute pulmonary embolism mimicking COVID - 19 pneumonia. *Int J Infect Dis.* 2020/05/18. 2020 Jul;96:475–6.
12. Payus AO, Rajah R, Febriany DC, Mustafa N. Pulmonary Embolism Masquerading as Severe Pneumonia: A Case Report. *Open access Maced J Med Sci.* 2019 Feb 10;7(3):396–9.
13. Saad M, Shaikh DH, Adrish M. A rare case report of a saddle pulmonary embolism presenting with high grade fevers, responsive to anticoagulation. *Medicine (Baltimore).* 2018 Mar;97(9):e0002–e0002.
14. Hayatu U, Augie I, Akintomide F. A, Muhammed E. H, Aminu M. S, Maiyaki S. A, et al. Pulmonary Embolism Mimicking Pneumonia, Acute Heart Failure, and Myocardial Infarction: A Case Report. *Clin Med Rev Case Reports.* 2020;7(5).

Figures

Figure 1

Initial High-resolution CT-Scan; axial lung window: red arrows indicate bilateral predominantly perihilar ground glass opacities