



(RESEARCH ARTICLE)



## Pulmonary embolism in critically ill ICU patients with COVID-19: Retrospective study in the intensive care unit at Hassan II University Hospital in Fez, Morocco

Aissam El Rhari \*, Mehdi Haloui, Abdelkarim Shimi, Brahim Bechri, Ali Derkaoui and Mohamed Khatouf

*Polyvalent Intensive Care Unit A1, Hassan II University Hospital, Fez, Morocco.*

World Journal of Advanced Research and Reviews, 2026, 30(01), 2560-2566

Publication history: Received on 16 March 2026; revised on 26 April 2026; accepted on 28 April 2026

Article DOI: <https://doi.org/10.30574/wjarr.2026.30.1.1069>

### Abstract

**Introduction:** Pulmonary embolism (PE) is the most frequent thromboembolic complication in COVID-19 patients in intensive care. It results from a hypercoagulable state linked to SARS-CoV-2, involving endothelial damage, systemic inflammation, and venous stasis. This study describes the clinical, radiological, and evolutionary characteristics of confirmed PE cases in a series of COVID-19 patients admitted to intensive care at Hassan II University Hospital in Fez.

**Methods:** This was a retrospective, descriptive, single-center study conducted in intensive care unit A1 between July 2020 and February 2021. Patients aged 16 years or older, hospitalized for respiratory distress due to confirmed SARS-CoV-2 infection, and presenting with pulmonary embolism (PE) confirmed by chest CT angiography, were included. Epidemiological, clinical, biological, therapeutic, and outcome data were analyzed.

**Results:** Among 480 admitted patients, 6 (1.25%) developed a confirmed pulmonary embolism (PE). The mean age was 68.8 years, with a male predominance (83.3%). The main comorbidities were diabetes and hypertension. The mean SpO<sub>2</sub> on admission was 80.3%. PEs were distributed as massive bilateral proximal (33%), lobar (33%), and segmental or subsegmental (33%). All patients received therapeutic anticoagulation without the need for thrombolysis. The mean length of stay was 5.6 days. Mechanical ventilation was required in one patient. The overall mortality rate was 33.3%.

**Conclusion:** Pulmonary embolism (PE) complicates a significant proportion of severe COVID-19 cases, with a high mortality rate. The frequency of severe forms underscores the importance of early preventive anticoagulation and systematic screening in intensive care units.

**Keywords:** COVID-19; SARS-CoV-2; Pulmonary embolism; Intensive care; Coagulopathy; Chest CT angiography

### 1. Introduction

Since the emergence of SARS-CoV-2 in late 2019, the COVID-19 pandemic has posed a considerable therapeutic challenge, particularly due to a clinical spectrum extending well beyond the respiratory sphere [1]. Among extrapulmonary complications, venous thromboembolic events—and especially pulmonary embolism (PE)—have emerged as a major clinical challenge in intensive care units (ICUs) [2].

Pulmonary embolism (PE) is the most frequently reported thromboembolic complication in severe COVID-19, with incidences ranging from 14.7% to 50% depending on the studies and screening protocols used [3, 4]. Its pathophysiology is specific, involving a multifactorial coagulopathy combining direct endothelial injury by the virus, platelet activation, cytokine storm, and the formation of neutrophil extracellular traps (NETs) [5, 6]. Some authors have

\* Corresponding author: Aissam El Rhari

suggested the hypothesis of pulmonary thrombosis in situ rather than a classic venous embolism, based on the frequent discrepancy between the high prevalence of PE and the low incidence of associated deep vein thrombosis (DVT) [7, 8].

Data on pulmonary embolism (PE) during COVID-19 primarily come from European and American centers. African data, and more specifically from the Maghreb region, remain almost nonexistent. In this context, we report a series of six confirmed cases of PE occurring among 480 COVID-19 patients hospitalized in intensive care at Hassan II University Hospital in Fez, Morocco, describing their epidemiological, clinical, paraclinical, therapeutic, and evolutionary characteristics, and comparing them with available international data.

---

## **2. Materials and methods**

### **2.1. Framework and type of study**

This retrospective, descriptive, single-center study was conducted in the Multipurpose Intensive Care Unit A1 (ICU-A1) of Hassan II University Hospital in Fez, a 1,500-bed university hospital and regional referral center. The study period covered July 2020 to February 2021, encompassing the first two waves of the COVID-19 epidemic in Morocco. The writing of this article followed the STROBE reporting guidelines for observational studies.

### **2.2. Inclusion and exclusion criteria**

All patients aged 16 years or older, hospitalized in the ICU-A1 for respiratory distress due to confirmed SARS-CoV-2 viral pneumonia (positive RT-PCR on nasopharyngeal swab and/or typical chest CT scan), and who developed a pulmonary embolism confirmed by chest CT angiography, were included. Patients under 16 years of age, those without evidence of COVID-19 infection, and patients without a pulmonary embolism confirmed by imaging were excluded.

### **2.3. Definition and confirmation of the diagnosis**

The diagnosis of PE was based on the demonstration of a pulmonary vascular filling defect on contrast-enhanced chest CT angiography, as interpreted by the radiologist. The topographic classification of PE was based on the CT angiographic location: proximal/massive (main pulmonary artery or lobes), lobar, or segmental/subsegmental.

### **2.4. Data Collection**

Data were collected retrospectively from medical records, imaging reports, laboratory results, and the Hosix computer system. The variables collected included: epidemiological data (age, sex, BMI, comorbidities, time from symptoms to resuscitation), clinical data at admission, paraclinical results (laboratory tests, chest CT scan, ECG), CT angiographic characteristics of PE, management modalities, associated organ failures, length of stay, and outcome (survival, death, cause of death).

### **2.5. Statistical Analysis**

This is a descriptive analysis. Continuous variables are expressed as means with standard deviations or medians with intervals. Categorical variables are presented as counts and percentages. The analyses were performed using Microsoft Excel 2013.

### **2.6. Ethical considerations**

The study was conducted in accordance with the principles of the Declaration of Helsinki. Anonymity and confidentiality of the data were preserved. The data were de-identified before analysis. The authors declare no conflict of interest.

---

## **3. Results**

### **3.1. Prevalence**

Of the 480 COVID-19 patients admitted to the ICU-A1 during the study period, 20 (4.16%) experienced a thromboembolic event. Among them, 6 patients (1.25% of the overall population and 30% of thromboembolic events) developed a pulmonary embolism confirmed by chest CT angiography.

### 3.2. Epidemiological and clinical characteristics

Table 1 summarizes the characteristics of the 6 patients. The mean age was 68.8 years (range 55–85 years). Five patients were male (83.3%). Comorbidities were present in 5 out of 6 patients (83.3%): type 2 diabetes (n=3, 50%), hypertension (n=2, 33%), smoking (n=2, 33%), history of stroke (n=1, 17%), and obesity (n=1, 17%).

The mean time between symptom onset and admission to the intensive care unit was 8 days. Respiratory distress was present in all patients upon admission. The mean SpO<sub>2</sub> on room air was 80.3% (70–93%). The mean respiratory rate was 27 breaths/min. Systolic blood pressure was preserved in all patients upon admission (mean 112 mmHg). The median Glasgow Coma Scale score was 14.

**Table 1** Clinical and epidemiological characteristics of the 6 patients with PE (N=6)

Characteristic	N or value	% or interval
Average age (years)	68.8	55–85
Male sex	5/6	83.3%
Present comorbidities	5/6	83.3%
Diabetes	3/6	50%
High blood pressure	2/6	33.3%
Smoking	2/6	33.3%
Average time to symptoms-ICU (days)	8	5–12
Mean SpO <sub>2</sub> on admission (%)	80.3	70–93
Average heart rate (cycles/min)	27	22–35
Glasgow median	14	12–15
positive RT-PCR	4/6	66.7%
Average parenchymal involvement (%)	52.5	25–>80
Elevated CRP (all patients)	6/6	100%
Lymphopenia	5/6	83.3%
Acute renal failure	3/6	50%

SpO<sub>2</sub>: pulse oxygen saturation; RR: respiratory rate; ICU: intensive care unit; RT-PCR: reverse-transcriptase polymerase chain reaction; CRP: C-reactive protein.

### 3.3. Paraclinical data

RT-PCR was positive in 4 of the 6 patients (66.7%). No COVID-19 serology was performed. A non-contrast chest CT scan was performed on all patients prior to CT angiography; the extent of parenchymal lesions averaged 52.5% (range 25% to >80%). CRP was elevated in all patients, with a mean value of 186 mg/L. Lymphopenia was observed in 5 patients (83.3%). D-dimer levels were measured in 2 patients and were positive in both cases. Acute renal failure was present in 3 patients (50%). Troponin levels were measured in 4 patients and were positive (elevated Troponin I) in 2 of them.

### 3.4. Angio-CT scan characteristics of pulmonary embolisms

Table 2 details the CT angiographic locations of the 6 PEs. Four cases (66.7%) involved proximal or lobar branches, and 2 cases (33.3%) were bilateral segmental/subsegmental. The precise case-by-case description was as follows:

**Table 2** Angiographic locations of the 6 cases of pulmonary embolism

Case	Angioscannographic localization	Topography	Evolution
1	massive bilateral proximal PE (main pulmonary arteries)	Proximal/Massive	Death
2	Proximal pulmonary embolism of the right pulmonary artery extending to the right middle and inferior segmental branches and subsegmental branches	Proximal	Survival
3	Bilateral PE involving the different branches of the right and left pulmonary arteries	Bilateral lobe	Survival
4	Bilateral PE of the left upper lobe and right lower lobe	Bilateral lobe	Survival
5	PE left lower lobar extending to segmental and subsegmental branches + right upper lobar segmental branch	Lobar/Segmental	Survival
6	bilateral lower lobar subsegmental PE	Bilateral sub-segment	Death*

\* Death occurred in both cases due to refractory hypoxia, not directly related to the topography of the PE but to overall respiratory failure.

### 3.5. Therapeutic management

All patients received therapeutic anticoagulation upon confirmation of pulmonary embolism (PE): low molecular weight heparin (LMWH; enoxaparin 0.1 mL/10 kg/12 h SC) in 5 patients (83.3%), and unfractionated heparin (UFH; 20 IU/kg/h IV infusion) in 1 patient (16.7%) due to severe renal impairment (creatinine clearance <15 mL/min). Antiplatelet therapy with aspirin 100 mg/day was administered in 4 patients (66.7%). No patient underwent systemic thrombolysis, mechanical thrombus fragmentation, or surgical embolectomy.

Regarding the management of underlying COVID-19: all patients received antibiotic therapy (azithromycin + 3rd generation cephalosporin ± quinolone) according to the national protocol; 5 patients received corticosteroid therapy (dexamethasone 6 mg/day or methylprednisolone 80 mg/day); 5 patients received hydroxychloroquine (200 mg twice daily). Oxygen support was required in all patients: high-concentration mask (n=4), non-invasive ventilation (n=4), orotracheal intubation (n=1; 16.6%).

### 3.6. Evolution and prognosis

The average length of stay was 5.6 days (range 2–16 days). Four patients (66.7%) recovered well: three were transferred to a general medicine ward and one was discharged directly. Two patients died (mortality rate 33.3%), both from refractory hypoxia in the context of global respiratory failure.

**Table 3** Comparison of our series with the main studies on PE during COVID-19 in intensive care

Study (Country)	Grillet et al. (France)	USA (Indy) Study	Lodigiani et al. (Italy)	Spanish study	Our series (Morocco)
Ref.	[9]	[3]	[4]	[10]	—
N total patients	135	328	61	30	480
Frequency EP (%)	24%	22%	40%	50%	1.25%*
Proximity / Massives (%)	31%	NR	19%	14%	33.3%
Intubation rate (%)	21%	65%	57%	26.7%	16.6%
Mortality (%)	13%	8.3%	15.4%	0%	33.3%
Thrombolysis (%)	NR	NR	NR	NR	0%
Systematic screening	Yes	Partial	Yes	Yes	No

\* The low prevalence in our study is probably related to the absence of systematic screening by CT angiography. NR: not reported.

## 4. Discussion

### 4.1. Prevalence and probable underestimation

The incidence of PE in our cohort (1.25%) appears much lower than the 22–50% reported in major European and American centers [3, 4, 9, 10]. This discrepancy is primarily explained by the diagnostic strategy used in our study: in the absence of systematic screening by chest CT angiography, only symptomatic PEs or those suspected due to clinical deterioration were confirmed. Studies using systematic screening report incidences two to ten times higher [3, 4].

Furthermore, the limited resources of Hassan II University Hospital, particularly the reduced availability of contrast-enhanced CT scanners in the ICU during epidemic peaks, likely contributed to this underestimation. A meta-analysis of 23 studies reported an overall incidence of PE of 14.7% (95% CI: 9.9–21.3%) in the medical ward and 23.4% (95% CI: 16.7–31.8%) in the ICU [11].

### 4.2. Patient profile and risk factors

The epidemiological profile of our patients with PE—mean age 68.8 years, male predominance (83%), high prevalence of diabetes (50%) and hypertension (33%)—corresponds to the characteristics of high-risk thrombotic populations described in the literature [4, 5, 12]. Advanced age is an independent risk factor for PE in COVID-19, due to the increased frequency of cardiovascular comorbidities and immobility [5].

Diabetes, present in 50% of our PE patients, is particularly common in the Moroccan population and promotes atherosclerosis and hypercoagulability [13]. Male predominance is consistently observed in the literature on COVID-19 TEE and is partially explained by differential expression levels of ACE2 receptors and more intense immune responses in men [5].

### 4.3. Specific features of CT angiography

The locations in our series are marked by a predominance of proximal and massive forms (33.3%), a rate higher than that generally reported in systematic cohorts (14–19%) [4, 10]. This higher proportion of proximal forms in our study may be explained by a detection bias: only clinically significant PEs, i.e., those that led to significant clinical deterioration justifying urgent imaging, were detected in our non-systematic screening cohort.

The question of pulmonary thrombosis in situ versus classic embolism is important. Several authors have noted that the filling defects observed on CT angiography in COVID-19 suggestive of PE frequently appear incomplete and non-occlusive, resembling local pulmonary thrombi more than emboli [7, 8]. This hypothesis is reinforced by the absence of associated DVT in most PE patients in our series—no DVT was found among the 6 PE cases—which is consistent with published data where only 20% of COVID-19 PEs have associated DVT [7].

### 4.4. Pathophysiology of PE in COVID-19

PE in COVID-19 results from a specific multifactorial coagulopathy, distinct from that observed in other severe viral infections [5, 6]. Three main mechanisms are involved:

- Direct endothelial injury: SARS-CoV-2 binds to ACE2 receptors expressed on the pulmonary vascular endothelium, resulting in local procoagulant activation [14]. The presence of viral elements in endothelial cells of different organs has been demonstrated by Varga et al. [14].
- Systemic hypercoagulability: the cytokine storm (IL-6, TNF- $\alpha$ , complement) leads to activation of the coagulation cascade, an increase in fibrinogen and D-dimers, and inhibition of fibrinolysis [5, 6]. Neutrophil extracellular traps (NETs) play a central role in pulmonary immunothrombosis [15].
- Venous stasis: prolonged bed rest, sedation and immobilization in intensive care are classic contributing factors, amplifying the already high thrombotic risk [5].

### 4.5. Management and absence of thrombolysis

Therapeutic anticoagulation with LMWH (and UFH in cases of renal insufficiency) was initiated in all our patients upon diagnostic confirmation. LMWH remains the standard treatment for confirmed PE in the ICU, associated with a reduction in mortality in COVID-19 patients at high thrombotic risk [16].

The absence of thrombolysis in our series is noteworthy, particularly in the two cases of massive/proximal PE. The indications for systemic thrombolysis in COVID-19 PE remain limited to massive forms with hemodynamic instability. The lack of use of this therapy in our study can be explained by: the bleeding risk associated with previously administered anticoagulants; the clinical instability of the patients, limiting the expected benefit; and the constraints specific to intensive care units in a pandemic context [17].

#### 4.6. Mortality and prognosis

The mortality rate of 33.3% (2/6) in our PE series is higher than the 0–15% reported in large European cohorts [4, 9] but comparable to other series in resource-limited settings. This difference is multifactorial: our patients were older, presented later (with advanced respiratory distress at admission), had not undergone thrombolysis or interventional embolectomy, and was subjected to detection bias (only the most severe PEs were diagnosed). Notably, deaths occurred due to global refractory hypoxia rather than being directly attributable to the PE location.

#### 4.7. Strengths and limitations

To our knowledge, this series represents one of the first descriptions focused on COVID-19 PE in the intensive care unit in a Moroccan center, with precise case-by-case CT angiographic characterization and systematic comparison to the international literature. Its main limitations are: the retrospective design, the very small sample size (n=6), the lack of systematic screening (leading to a major underestimation of the true incidence), the absence of D-dimer testing in the majority of patients (an important diagnostic decision factor), and the impossibility of performing comparative statistical analyses due to the limited sample size.

---

### 5. Conclusion

Pulmonary embolism complicated 1.25% of COVID-19 hospitalizations in intensive care in our Moroccan cohort, with a mortality rate of 33.3%. Proximal and massive forms accounted for one-third of cases, highlighting the severity of COVID-19 coagulopathy. The lack of systematic screening most likely led to a significant underestimation of the true prevalence.

These results support: (1) systematic prophylactic anticoagulation at an appropriate dose upon admission to intensive care for all COVID-19 patients; (2) systematic D-dimer testing as a marker for monitoring coagulopathy; and (3) a lower threshold for performing chest CT angiography in the presence of any unexplained clinical deterioration, even in the absence of classic clinical signs of pulmonary embolism. Prospective multicenter studies in Morocco and the Maghreb are needed to establish screening recommendations adapted to the local context.

---

### Compliance with ethical standards

#### *Disclosure of conflict of interest*

No conflict of interest to be disclosed.

#### *Statement of informed consent*

Informed consent was obtained from all individual participants included in the study.

---

### References

- [1] World Health Organization. COVID-19 Weekly Epidemiological Update. Geneva: WHO; September 2021.
- [2] Ministry of Health of Morocco. Epidemiological situation of COVID-19 in Morocco. Available at: <https://www.sante.gov.ma>. Accessed October 2021.
- [3] Trigonis RA, Holt DB, Yuan R, et al. Incidence of venous thromboembolism in critically ill coronavirus disease 2019 patients receiving prophylactic anticoagulation. *Crit Care Med.* 2021;49(5):e470–e479.
- [4] Lodigiani C, Iapichino G, Carenzo L, et al. Venous and arterial thromboembolic complications in COVID-19 patients admitted to an academic hospital in Milan, Italy. *Thromb Res.* 2020;191:9–14. doi:10.1016/j.thromres.2020.04.024.

- [5] Klok FA, Kruip MJHA, van der Meer NJM, et al. Incidence of thrombotic complications in critically ill ICU patients with COVID-19. *Thromb Res.* 2020;191:145–147. doi:10.1016/j.thromres.2020.04.013.
- [6] Helms J, Tacquard C, Severac F, et al. ; CRICS TRIGGERSEP Group. High risk of thrombosis in patients with severe SARS-CoV-2 infection: a multicenter prospective cohort study. *Intensive Care Med.* 2020;46(6):1089–1098. doi:10.1007/s00134-020-06062-x.
- [7] Wichmann D, Sperhake JP, Lütgehetmann M, et al. Autopsy findings and venous thromboembolism in patients with COVID-19. *Ann Intern Med.* 2020;173(4):268–277. doi:10.7326/M20-2003.
- [8] Lax SF, Skok K, Zechner P, et al. Pulmonary arterial thrombosis in COVID-19 with fatal outcome: results from a prospective, single-center, clinicopathologic case series. *Ann Intern Med.* 2020;173(5):350–361. doi:10.7326/M20-2566.
- [9] Grillet F, Behr J, Calame P, Aubry S, Delabrousse E. Acute pulmonary embolism associated with COVID-19 pneumonia detected with pulmonary CT angiography. *Radiology.* 2020;296(3):E186–E188. doi:10.1148/radiol.2020201544.
- [10] Poissy J, Goutay J, Caplan M, et al. Pulmonary embolism in COVID-19 patients: awareness of an increased prevalence. *Traffic.* 2020;142(2):184–186. doi:10.1161/CIRCULATIONAHA.120.047430.
- [11] Suh YJ, Hong H, Ohana M, et al. Pulmonary embolism and deep vein thrombosis in COVID-19: a systematic review and meta-analysis. *Radiology.* 2021;298(2):E70–E80. doi:10.1148/radiol.2020203557.
- [12] Fernández-Capitán C, Barba R, Díaz-Pedroche MC, et al. Presenting characteristics, treatment patterns, and outcomes among patients with venous thromboembolism during hospitalization for COVID-19. *Semin Thromb Hemost.* 2021;47(4):351–361.
- [13] Ministry of Health of Morocco. National survey on cardiovascular disease risk factors 2017. Rabat: MS Morocco; 2017.
- [14] Varga Z, Flammer AJ, Steiger P, et al. Endothelial cell infection and endotheliitis in COVID-19. *Lancet.* 2020;395(10234):1417–1418. doi:10.1016/S0140-6736(20)30937-5.
- [15] Nakazawa D, Ishizu A. Immunothrombosis in severe COVID-19. *EBioMedicine.* 2020;59:102942. doi:10.1016/j.ebiom.2020.102942.
- [16] Tang N, Bai H, Chen X, Gong J, Li D, Sun Z. Anticoagulant treatment is associated with decreased mortality in severe coronavirus disease 2019 patients with coagulopathy. *J Thromb Haemost.* 2020;18(5):1094–1099. doi:10.1111/jth.14817.
- [17] Tapson VF. Thrombolytic therapy for acute pulmonary embolism. *Semin Thromb Hemost.* 2013;39(4):452–458. doi:10.1055/s-0033-1334145.