ISSN: 2572-3235

Alami et al. Int J Radiol Imaging Technol 2023, 9:102
DOI: 10.23937/2572-3235.1510102
Volume 9 | Issue 1
Open Access

International Journal of
Radiology and Imaging Technology

RESEARCH ARTICLE

Pulmonary Embolism and COVID-19: A Diagnostic Dilemma – A German Single Centre Experience

M Alami[1], D Janati-Idrissi[1], T Lohmann[1], B Osswald[2], G Plehn[3] and K Luedtke[4]*

[1]*Clinic for Diagnostic and Interventional Radiology, Johanniter-Krankenhaus Rheinhausen, Germany*
[2]*Department of Cardiothoracic CIED Management, Johanniter Krankenhaus Rheinhausen, Germany*
[3]*Department of Cardiology, Johanniter Krankenhaus Rheinhausen, Germany*
[4]*Department of Anaesthesia and Intensive Care, Johanniter Krankenhaus Rheinhausen, Germany*

***Corresponding author:** *Mehdi Alami, Clinic for diagnostic and interventional Radiology, Johanniter Krankenhaus Rheinhausen GmbH, Kreuzacker 1-7, 47228 Duisburg - DE, Germany*

Abstract

Background/Objective: The high predisposition to pulmonary embolism (PE) in CoViD-19-patients increases with its seriousness, even under anticoagulation. Because neither clinical nor laboratory parameters seem to be specific in this case, the CTPA stays the gold standard for further diagnostic, with the risk however of overuse. Our study investigates the incidence of PE in CoViD-19-patients, who underwent aCTPA, and to assess the link of several clinical and laboratory parameters with its occurrence.

Methods: We led a retrospective monocentric study in Johanniter Hospital in Duisburg-Rheinhausen between April 1st, 2020, and October 26th, 2021, in patients CoViD-19 positive tested with a RT-PCR. Data about examined CoViD-19 patients with CTPA by PE suspicion or accidentally revealed PE were collected, as well as clinical and laboratory parameters too.

Results: among 330 CoViD-19-patients 52 (15.8%) underwent a CTPA. 9 Patients (17.3% of 52/2.7% of 330) showed a PE. By 4 patients (7.7% of 52/1.2% of 330) PE could not be excluded. Only the Caucasian race (OR – 6.25; 95% CI 1.140-34.290; p = 0.035) was found to be independently associated with PE.

Conclusion: Clinical and laboratory parameters are insufficient to clarify the occurrence of PE in patients with CoViD-19. Because of its seriousness and its relative higher incidence in CoViD-19-patients a lower barrier based on clinical appearance of the disease degree is required in the future to evocate the possibility of a PE and to include earliest possible the performance of a CTPA for evaluation.

Keywords

CoVid-19, Pulmonary embolism, Anticoagulation, Computer tomography pulmonary angiogram, Venous thromboembolism

Abbreviation

VTE: Venous Thromboembolism; PE: Pulmonary Embolism; CTPA: Computed Tomography Pulmonary Angiogram; RT-PCR: Reverse Transcriptase Polymerase Chain Reaction; DVT: Deep Vein Thrombosis; CrCl: Creatinine Clearance; RHS: Right Heart Strain; ICU: Intensive Care Unit; CRP: C-Reactive Protein; LMWH: Low-Molecular-Weight Heparin; UFH: Unfractionated Heparin

Introduction

The elevated morbidity and mortality of CoViD-19 [1] seems to be associated i.a. with a high predisposition to VTE [2,3], principally by severe and critical course [3-6], and especially to PE instead of other patients [2].

Since both the differentiation between CoViD-19-related symptoms and aggravation due to PE by clinical appearance is not easy and laboratory parameters fail to allow a clear differentiation, the use of CTPA remains ineluctable. CTPA is indeed the most common and practical imaging modality, regarding its availability, its high specificity and sensibility. It allows furthermore to state the ill severity and to confirm or to eliminate differential diagnosis.

Citation: Alami M, Janati-Idrissi D, Lohmann T, Osswald B, Plehn G, et al. (2023) Pulmonary Embolism and COVID-19: A Diagnostic Dilemma – A German Single Centre Experience. Int J Radiol Imaging Technol 8:102. doi.org/10.23937/2572-3235.1510102

Accepted: January 05, 2023; **Published:** January 07, 2023

The challenge is to avoid the overuse of the CTPA, because of the requirement of contrast media (induction/aggravation of renal disease), of a high level of radiation and of additional costs.

The aim of this study was to evaluate the detection rate in the CTPA of PE in CoViD-19-patients who were examined with a CTPA, either presenting a clinical and/or laboratory PE-suspicion, or for other indications, and to determine the risk of PE considering the clinical and laboratory criteria, in order to refine the diagnostic procedure.

In a single centre setting we assessed the results of CTPA in CoViD-19-patients and the association of several parameters with the risk of PE.

Materials and Methods

Study definition

We conducted a retrospective study in Johanniter-Krankenhaus-Rheinhausen - Duisburg (Germany), a medium-sized hospital (282 beds), evaluating all CTPA performed between April 1st 2020 and October 26th 2021 in admitted/examined patients, positive tested with RT-PCR for SARS-CoV-2 on nasopharyngeal swab, sputum, tracheobronchial secretion, oropharyngeal and on bronchoalveolar lavage. Excluded were patients who only were CoViD-19 positive tested, but neither admitted nor examined.

Analysis criteria

In order to compare our results and in the interests of reproductivity, we focused on the same criteria as prior studies and meta-analysis [7-10], an especially on the study of Filippi, et al. [2], that we considered as model. Patient records were analysed for information reported in Table 1, Table 2, Table 3, Table 4 and Table 5.

Patient management

1. Ward definition:

CoViD-19-Patients in our study are classified in three ward categories as they were tested (Table 6):

2. Care setting definition:

The patients were then immediately isolated and assigned to an adapted care intensity before the CT-scan. This assignment may have been revised after the CT-scan depending on the infect burden and/or on the location of the pulmonary embolism. These care assignment criteria were defined in Table 7.

The classical assignment in the different care categories was defined by the DIVI (German Interdisciplinary Association for Intensive Care and Emergency Medicine) in 2017 [11]. In an unprecedented situation such as the CoViD-19-pandemia, the RKI (Robert-Koch-Institute) published adapted WHO-Guidelines [12,13] to the CoViD-19-management, in which the assignment criteria to sort CoViD-19-patients were detailed. Although, we had to modify permanently our intern care assignment criteria, according to the pandemic situation, and took decision on a case-by-case basis, in order to avoid patients' overflow and to save resources for the serious to critical patients, especially at the beginning of the pandemic and during the different waves. In the same optic were the patients discharged as soon as possible.

Anticoagulation

The doses of heparin were defined as following (Table 8):

Imaging modalities:

1. CTA examination:

a) CT-indication:

With incidental findings as exception, the indication to CTA was decided in correlation with the symptomatic primarily, the clinical information, including the Wells-score, and the laboratory results. Some patients were immediately examined with a CT-scan after admission. We defined the admission day as d_0. Other patients underwent a CTA during the hospitalisation, mainly by worsening or delayed recovery.

b) CT-Protocol:

A 16-row scanner was used for the acquisition. The bolus was tracked in the truncus of the pulmonary artery after injection of 50 to 100 mL of non-ionic monomeric iodine contrast media, with a threshold of 160 to 250 HU.

Mediastinal and parenchymal windows were multiplanar reconstructed with slice-thickness of minimum 0.75 mm.

c) PE-Classification:

PE was classified as main pulmonary, lobar, segmental or subsegmental based on the location of the most proximal luminal defect.

d) CT-Infect burden evaluation:

Many scores were described in the literature to assess the severity of the infect burden in the CT-Scan, with different cut-off values [16-19].

We used the CT-Severity index score to visually assess the infect burden in the lung parenchyma on the CT-scan. We choose to distribute the patients in four categories depending on the estimated percentage of the affected lung volume (Table 9). Our cut-off value to define severe infect was 50%, as advocated by Ran, et al. and Fonseca, et al. [16,17]. However, without the help of AI remains our estimation unprecise.

2. Ultrasound:

An echocolour Doppler of the lower extremities was performed by patients with concomitant symptoms of DVT.

Table 1: Clinical and demographic features of the study population before CT-scan.

PE	present (n = 9)	non-present (n = 39)	p-value	unclear (n = 4)
Male: females	3:6 (33%:67%)	20:19 (51%:49%)	0.176	4:0 (100%:0%)
Age (y)	69.4 [62.1-79.8]	67.7 [61.8-79.0]	0.355	67.8 [61.6- 72.0]
Non-Caucasian race	2 (22.2%)	14 (35.9%)	0.216	3 (75%)
Risk factor:				
Wells-Score ≥ 4	3 (33.3%)	15 (38.5%)	0.393	3 (75%)
Bed rest	5 (55.6%)	14 (35.9%)	0.213	3 (75%)
Previous VTE	2 (22.2%)	1 (2.6%)	0.112	0 (0%)
Active cancer	2 (22.2%)	6 (15.4%)	0.337	1 (25%)
Obesity	5 (55.6%)	25 (64.1%)	0.333	1 (25%)
CT-Indication:				
CT-datum after admission(d)	15.22 [2-6]	6 [0-5.5]	0.121	14 [4-22]
Respiratory worsening	6 (66.7%)	25 (64.1%)	0.446	3 (75%)
Low O_2 Saturation on ambient air	2 (22.2%)	14 (35.9%)	0.213	2 (50%)
Dyspnoea	3 (33.3%)	12 (30.7%)	0.445	2 (50%)
Tachypnoea	3 (33.3%)	4 (10.3%)	0.108	2 (50%)
Intubated	3 (33.3%)	9 (23.1%)	0.290	2 (50%)
Respiratory asymptomatic	3 (33.3%)	14 (35.9%)	0.446	0 (0%)
Chest Pain	0 (0.0%)	2 (5.2%)	0.080	0 (0%)
Infect focus	0 (0.0%)	3 (7.7%)	0.042	0 (0.0%)
Elevated D-Dimer	0 (0.0%)	3 (7.7%)	0.042	0 (0.0%)
Other CT-indications	3 (33.3%)	6 (15.4%)	0.167	0 (0%)
Setting:				
Intensive Care Unit	3 (33.3%)	8 (20.5%)	0.244	2 (50%)
Intermediate Care Unit	1 (11.1%)	7 (17.9%)	0.300	1 (25%)
Standard Care Unit	5 (55.6%)	24 (61.5%)	0.381	1 (25%)
Medication				
Steroids	5 (55.6%)	17 (43.6%)	0.274	3 (75%)
Antiaggregant				
Aspirin	1 (11.1%)	7 (17.9%)	0.300	1 (25%)
Other	0 (0%)	4 (10.3%)	0.022	0 (0%)
Heparin doses				
Regular prophylactic	0 (0%)	6 (15.4%)	0.006	1 (25%)
Duration (in days)	-	7 [2.25-9.75]		5
Therapeutic	5 (55.6%)	16 (41.0%)	0.233	3 (75%)
Duration (in days)	22.4 [2-25]	9.8[2-8]	0.188	17 [10-25]
DOAC	1 (11.1%)	4 (10.3%)	0.473	0 (0%)
D-Dimer (µg/L)				
< 500	0 (0%)	0 (0%)	0	0 (0%)
500-1500	1 (11.1%)	12 (30.8%)	0.081	2 (50%)
1500-4000	4 (44.4%)	12 (30.8%)	0.244	0 (0%)
> 4000	1 (11.1%)	6 (15.4%)	0.370	1 (25%)
Platelet < 150.000	2 (22.2%)	5 (12.8%)	0.281	0 (0%)
Gasometry				
PaO_2/FiO_2 ratio (mmHg)				
< 100	1 (11.1%)	3 (7.7%)	0.390	0 (0%)

100-200	1 (11.1%)	3 (7.7%)	0.390	0 (0%)
> 200	2 (22.2%)	12 (30.8%)	0.307	3 (75%)
pCO2 (mmHg)				
< 35	0 (0%)	7 (17.9%)	0.003	1 (25%)
Infect burden on CT-Scan				
Severe	4 (44.4%)	14 (35.9%)	0.333	2 (50%)
Moderate	0 (0.0%)	10 (25.6%)	< 0.05	2 (50%)
Mild	3 (33.3%)	13 (33.3%)	0.5	0 (0%)
No infiltrate	2 (22.2%)	2 (5.2%)	0.144	0 (0%)

Table 2: Multivariable regression analyses for the occurrence of pulmonary embolism.

		Odds Ratio	95%-CI	P-value
Caucasian		6.25	1.140-34.290	0.035
Previous TVT		10.86	0.863-136.602	0.065
Active cancer		1.57	0.261-9.470	0.635
D-Dimer	500-1500	0.28	0.032-2.506	0.259
	1500-4000	1.8	0.410-7.910	0.445
	> 4000	0.69	0.072-6.546	0.757
Wells Score ≥ 4		0.8	0.173-3.690	0.787

Table 3: Pulmonary embolism localisation.

	PE (n = 9/52)
Principal/lobar	5 (55.6%)
Segmental	0 (0%)
Subsegmental	4 (44.4%)

Table 4: In-hospital adverse events.

	Total (n = 52)	PE (n = 9)	Non-PE (n = 39)	Odd Ratio	95%-CI	p-value	Unclear (n = 4)
Death	9 (17.3%)	2 (22.2%)	6 (15.4%)	1.571	0.261-9.470	0.635	1 (25%)
Increase in oxygenation	41 (78.8%)	7 (77.8)	30 (76.9%)	1.05	0.184-5.977	0.960	4 (100%)
Increase in care intensity	28 (53.8%)	4 (44.4%)	21 (53.8%)	0.686	0.160-2.946	0.625	3 (75%)

Table 5: Findings in PE-Patients.

Age	Sex	Death	localisation	Infiltrate and relevant finding	Symptomatic	Heparin doses (duration)	Antiaggrega-tion	RHS	D-Dimer before CT scan	DVT	Oncological	Corticoid	Obesity	Previous VTE
86	F	Y	Bilateral central	Severe	Respiratory insufficiency	Therapeutic (16d)	None		1500-4000	N	N	Y	N	N
62	M	N	Bilateral central lobar	None	None	None	None		> 4000	N	N	N	N	N
88	F	N	Small subsegmental	None	None	None	Apixaban			N	Y	N	Y	N
56	F	N	Small subsegmental	Severe	Dyspnoea	Therapeutic (1d)	None		1500-4000	N	N	N	Y	N
77	M	Y	Small subsegmental	Severe	Respiratory insufficiency	Therapeutic (25d)	None		1500-4000	N	N	Y	N	N

75	M	N	Central unilateral	Mild	None	None	Aspirin			N	Y	N	N	N
68	F	N	Small subsegmental	Mild, pericardiac effusion	Dyspnoea	None	None		500-1500	N	N	N	Y	N
55	F	N	Bilateral central	Severe	Respiratory insufficiency	Therapeutic doses (2d)	None		> 4000	N	N	N	Y	Y
65	F	N	Central unilateral	Mild, emphysema	Respiratory insufficiency	Therapeutic (67d)	None	Y	1500 -4000	Y	N	Y	Y	Y

Table 6: Care ward definition.

Emergency ward	Patients who presented on the emergency department with: - Influenzal symptomatic; - Impaired breathing; - Chest pain; - Suspicion of PE (e.g., from other physicians addressed); - Other symptoms.
Hospital ward	Patients who were already hospitalised in our institution for: - CoViD-19 (e.g., transferred from other institutions); - For elective therapy; - Other reasons.
Ambulant ward	Out-Patients, e.g., - For elective therapy; - For scheduled examination; - For follow-up.

Patients with hemodynamic instability underwent an echocardiography before the CT-scan.

Statistical Analyse

Normally distributed variables were communicated as mean ± standard deviation. Non-normal distributed parameters were reported as median and interquartile range.

Categorical data were expressed as quantity (total number) and percentage.

The Student t-test was used to compare continuous normally distributed variables, whereas chi-squared test or Fisher exact test was used to compare categorical variables.

To assess the individual and independent association of PE with clinical and laboratory parameters, univariable and multivariable logistic regression analyses were set and presented as odds ratio (OR) with 95% confidence interval (CI).

No relevant collinearity was found among the covariates by the variance inflation factors.

As a candidate for the multivariate analysis a parsimonious model was employed including variables with $p < 0.05$ by the univariate test.

In order to evaluate the in-hospital mortality risk in patients with vs. those without PE, multivariable logistic regression analyses were applied and the results were communicated as odds ratio (OR) with 95% confidence intervals (CI).

Results

330 patients admitted/examined in our institution were tested positive for SARS-CoV-2 with RT-PCR. The mean age was 68.4 and the male sex rate was 54%.

In 52 patients (15.8% of 330) a CTPA was performed. The PE was verified in 9 CoViD-19-patients (17.3%).

In 4 patients (7.7% of 52) the CTPA was non-conclusive as a widespread thromboembolic burden couldn't be excluded at the subsegmental level of PE. Because of respiratory artefacts, beam hardening, insufficient contrasting and/or by pronounced consolidations, their CTPA showed multiple defects in the vascular lumen, without any proven evidence of a complete abort of the distal contrasting.

We summarized our results in the Table 1, Table 2, Table 3, Table 4 and Table 5.

A low pCO_2 (< 35 mmHg) before the CT scan was found in none of our PE-patients, but in 17.9% of the non-PE-patients ($p < 0.003$).

No further significant differences concerning gender,

Table 7: Care setting definition.

Care Unit	*Disease Severity*	*Criteria*
Standard Care unit	Asymptomatic to moderate III	- No concrete risk of a life-threatening or organ-threatening disorder.
		- No highly elaborate care.
		- No constant monitoring or support of vital functions required. o Exception: ▪ Oxygenotherapy < 10 L/min to reach a minimal SaO_2 > 90%
		Mainly were hospitalised in this category patients with pre-existing conditions, that could predispose to an imminent worsening, e.g., - Moderate infect burden; - Central PE; - Age > 60 years; - Diabetes; - Adiposity; - Hypertension; - COPD or other lung diseases; - Impaired immune system; - Active oncologic diseases; - Other underlying diseases;
Intermediate Care Unit	Severe III	- Potentially severe instability of physiological parameters, e.g., o SaO_2 < 90% under oxygenotherapy < 10 L/min. o Tachypnoea > 30/min,
		- Equipment-based monitoring required,
		- Organ support but no organ replacement [14,15],
Intensive Care Unit	Critical III	- Life-threatening disorders of one or more body systems, e.g., o Neurologic impairment; o Cardiovascular function; o Respiratory function; o Renal function.

Table 8: Heparin doses.

Doses	LMWH: Enoxaparin	UFH
	Adapted to body weight	*Used in impaired renal function*
Regular prophylactic	40 mg sc 1×/d	7500UI 2*/d
	20 mg sc 1 ×/d when ♀ < 50 kg bw ♂ < 60 kg bw	
	60 mg sc 1 ×/d when BMI > 50 kg/m^2	
Therapeutic	Max 0.8 mg/Kg/bw 2x/d + Surveillance of anti-Xa	In a perfusor, with an aPTT therapeutic range between 60-80 sec.

Table 9: CT-Severity index score.

Infect burden on CT-Scan	Percentage of affected lung
None	-
Mild	1-25%
Moderate	25-50%
Severe to critical	> 50%

age, nutritional status, in-hospital setting, or several clinical and laboratory parameters could be determined.

The thrombus was in 55.6% of cases central, i.e. in the main and/or the lobar pulmonary arteries, and 44.4% in the subsegmental arteries. There was no correlation between the location/burden of the thrombus and the symptomatic. None of the PE was restricted to the segmental arteries (Table 2).

Discussion

In our study we observed an incidence of PE in 17.3%, which is higher compared to other publications [7-9], but lower to a recent meta-analysis [10]. It remains higher than the one observed in general ward in non-CoViD-19-patient [20-23]. The proportion of severely to critically affected CoViD-19-patients (38.5% of our cohort) may contribute to the higher incidence. Studies found a proportional increase of PE-rate to the severity of CoViD-19 [2,9,10].

Most of the CTPA was performed in CoVid-19-patients who presented unspecific clinical symptomatic. They may occur due to the infection itself or to a PE, principally by worsening respiratory pattern such as hypocapnia, dyspnoea or tachypnoea. A further hint was a positive preclinical score result. Surprisingly, PE-patients were less likely to have a high (> 4) Wells-score of PE (33.3% versus 38.5%), and the PE was found fortuitously in three asymptomatic patients who underwent CTPA for other indications. No statistically significant correlation was found between the development of the PE and clinical presentation, or between PE and clinical complications, such as intensification of care or mortality, which is consistent with the finding of Filippi, et al. [2].

Primarily in elderly, the SARS-CoV-2 infection involves the whole organism, which alters significantly the haemostatics [24] and may explain the advanced age our cohort. Compared to other studies [3,25], age and sex have no influence on the risk of PE in our patients. From all of the investigated parameters, only the Caucasian race turned out as a statistically significant positive correlation ($p = 0.04$). This may be a reflection of the well-known correlation to different mutations (such as Factor V Leiden and prothrombin gene G20210A) but out of the available clinical data, a further differentiation of our patients was not possible.

This risk may further increase through some medications like steroids [26].

Most reports found the frequent involvement of the peripheral arteries without signs of DVT, which favours the local origin of the thrombosis [2,3,6,10,27,28]. 44.4% of the PE in our study were subsegmental.

A study showed that DVT does not necessarily lead to thromboembolism, and remains “sleeping” without clinical manifestations [3]. The COVID-19-induced DVT are able to cause complications and are resistant to anticoagulant therapy [3], as shown in one of our patients.

As found in the literature, the majority of our CoViD-19-patients presented elevated D-dimer and CRP [29]. In most of PE-Patients the D-dimer level varied in-between 1500 and 4000 ng/mL (44.4%). Thus, the sensibility of the D-dimer as a standalone parameter is argued. Whereas some authors rely on D-Dimer analysis [9], sometimes with different cut-off levels [3,10], others doubt whether D-dimer is rather a marker of thromboembolic complications in a clinical scenario dominated by (severe) infectious/inflammatory disorder [2]. In fact, D-dimer increases proportionally with the severity of CoViD-19, independent of the presence of PE [2].

Furthermore, in spite of prophylactic/therapeutic anticoagulation, many reports underlined the high incidence of thrombotic COVID-19-complications such as microvascular thrombosis, stroke and venous thromboembolic disease [2,9,27,30], especially PE [3,31-36], most notably in severely ill CoViD-19-patients with pneumonia admitted to the ICU [37], in comparison to other diseases [10]. This was the reason why many studies support the use of higher doses of anticoagulants, while some advocate an immediate onset of therapeutic anticoagulation in CoViD-19-patients, at least in those admitted to the ICU [25,26,38,39]. The results of a randomised clinical trial reported no clear advantage of intermediate over preventive doses of enoxaparin in severe ill patients [40]. Our findings may be concordant with these observations, since our coagulated PE-patients received exclusively therapeutical heparin doses. It remains to determine the heparin dosage with the best benefit-to-risk ratio [2]. Only in two patients a central PE was resistant to a long-term intensive anticoagulation and to a thrombolytic treatment, which is consistent with the finding of Kerbikov, et al. [3] and of Nahum, et al. [5].

The potential effect of antiplatelet drugs on the prevention of the inflammatory activation of the thrombocytes is still controversial [41-44]. Because of the small number of concerned patients, we could not debate on this subject.

Some authors suspect a synergic effect of CoViD-19 and active cancer [45-47] since the cancer and some oncological therapy itself are thrombogenic. In our cohort no relevant difference was found between the PE- and non-PE-population concerning the prevalence of an active cancer (22.2% vs. 15.4%, $p = 0.337$).

The benefit of the cardiac echography was underlined in a recent study [48]. We could not verify this observation, as the echocardiography was performed in only one PE-patient and only in 3 non-PE-patients.

Since the literature is controversial and our results do not indicate a certain pattern, the impact of PE on patient prognosis remains unclear [9,27]. Table 4 indicates the lack of influence of PE on death from any cause, on increase of oxygenation modality or on increase of care intensity.

Study Limitations

The limits of our study are related once to the retrospective design of the study but even more important to the limited number of patients in a single centre setting, which implies numerous possible biases that are not accounted for. Nevertheless, the findings about the lack of a flow-chart like decision-making are important information.

Finally, because of the high risk of exposure at the time, thromboembolic events may have been underreported due to the inability to pursue the appropriate imaging tests.

Conclusion

Aim of our study was to find a couple or certain parameters indicating a high incidence for PE in CoViD-19-patients, who presented clinical and/or laboratory criteria, which could indicate a VTE. We failed to show any relevant parameter able to predict the prevalence of PE. Most of them are unspecific, principally by a constellation dominated by infection and respiratory compromise.

Many studies support local inflammation being the onset of PE in CoViD-19-patients since the majority of lesions affects the peripheral level. This level is associated with a limited detection by CTPA. This may contribute to an underestimation of the proportion of PE in CoViD-19-patients. Nevertheless, if the PE extends to the subsegmental level, CTPA represents a valuable imaging technique, which enables a tailored therapy in critical ill CoViD-19-patients.

Since we aspire constantly to optimise patient care and to adapt to the still changing SARS-CoV-19, we could try to reconsider our PE-diagnostic strategy by refining our clinical evaluation and by using other diagnostic means, such as echocardiography in haemodynamic unstable patients, as recommended in the guidelines of the ESC, to avoid the overuse of the CTPA.

Acknowledgements

Special thanks to Dr. Welskop and his team preparation and providing the test results.

Statements and Declarations

Funding

No funds, grants, or other support was received.

Conflicts of interest/Competing interests

All authors have no relevant financial or non-financial interests to disclose.

Ethics approval

According to § 15 of the professional regulations in North Rhine-Westphalia (Germany), an ethics consultation is not required in anonymised retrospective data collections studies.

Consent to participate

Not applicable.

Consent for publication

Not applicable.

Availability of data and material

Available.

Code availability

Not applicable.

Authors' contributions

All authors have contributed equally.

References

1. Chen N, Zhou M, Dong X, et al. (2020) Epidemiological and clinical characteristics of 99 cases of 2019 novel coronavirus pneumonia in Wuhan, China: A descriptive study. Lancet 395: 507-513.
2. Filippi L, Sartori M, Facci M, et al. (2021) Pulmonary embolism in patients with COVID-19 pneumonia: When we have to search for it? Thrombosis Research 206: 29-32.
3. Kerbikov O, Orekhov P, Borskaya E, et al. (2021) High incidence of venous thrombosis in patients with moderate-to-severe COVID-19. Int J Hematol 113: 344-347.
4. Wichmann D, Sperhake JP, Lütgehetmann M, et al. (2020) Autopsy findings and venous thromboembolism in patients with COVID-19: A prospective cohort study. Ann Intern Med 173: 268-277.
5. Nahum J, Morichau-Beauchant T, Daviaud F, et al. (2020) Venous thrombosis among critically ill patients with coronavirus disease 2019 (COVID-19). JAMA Netw Open 3: e2010478.
6. Klok FA, Kruip MJHA, van der Meer NJM, et al. (2020) Incidence of thrombotic complications in critically ill ICU patients with COVID-19. Thromb Res 191: 145-147.
7. Fauvel C, Weizman O, Trimaille A, et al. (2020) Pulmonary embolism in COVID-19 patients: A french multicentre cohort study. Eur Heart J 41: 3058-3068.
8. Suh YJ, Hong H, Ohana M, et al. (2021) Pulmonary embolism and deep vein thrombosis in COVID-19: A systematic review and meta-analysis. Radiology 298: E70-E80.
9. Adams HJA, Kwee TC, Yakar D, et al. (2020) Systematic review and meta-analysis on the value of chest CT in the diagnosis of coronavirus disease (COVID-19): Sol scientiae, Illustra Nos. AJR Am J Roentgenol 215: 1342-1350.
10. Kwee RM, Adams HJA, Kwee TC (2021) Pulmonary

embolism in patients with COVID-19 and value of D-dimer assessment: A meta-analysis. Eur Radiol 31: 8168-8186.

11. Waydhas C, Herting E, Kluge S, et al. (2018) Intermediate care units. Med Klin Intensivmed Notfmed 113: 33-44.

12. Kluge S, Janssens U, Welte T, et al. (2020) S3-Leitlinie - Empfehlungen zur stationären Therapie von Patienten mit COVID-19, ver. 03.2020.

13. WHO (2020) Corticosteroids for COVID-19. Living guidance, 2 September 2020, WHO/2019-nCoV/Corticosteroids/2020.1.

14. Nasraway SA, Cohen IL, Dennis RC, et al. (1998) Guidelines on admission and discharge for adult intermediate care units. American College of Critical Care Medicine of the Society of Critical Care Medicine. Crit Care Med 26: 607-610.

15. Stacy KM (2011) Progressive care units: Different but the same. Crit Care Nurse 31: 77-83.

16. Yang R, Li X, Liu H, et al. (2020) Chest CT severity score: An imaging tool for assessing severe COVID-19. Radiol Cardiothorac Imaging 2: e200047.

17. Fonseca EKUN, Assunção Júnior AN, Araujo-Filho JAB, et al. (2021) Lung lesion burden found on chest CT as a prognostic marker in hospitalized patients with high clinical suspicion of COVID-19 pneumonia: A Brazilian experience. Clinics (Sao Paulo) 76: e3503.

18. Grodecki K, Lin A, Cadet S, et al. (2020) Quantitative burden of COVID-19 pneumonia on chest CT predicts adverse outcomes: A post-hoc analysis of a prospective international registry. Radiol Cardiothorac Imaging 2: e200389.

19. Almasi Nokiani A, Shahnazari R, Abbasi MA, et al. (2022) CT severity score in COVID-19 patients, assessment of performance in triage and outcome prediction: A comparative study of different methods. Egypt J Radiol Nucl Med 53: 116.

20. Kline JA, Kabrhel C (2015) Emergency evaluation for pulmonary embolism, Part 2: Diagnostic approach. J Emerg Med 49: 104-117.

21. Kabrhel C, Mark Courtney D, Camargo CA Jr, et al. (2009) Potential impact of adjusting the threshold of the quantitative D-dimer based on pretest probability of acute pulmonary embolism. Acad Emerg Med 16: 325-332.

22. Righini M, Robert-Ebadi H, Le Gal G (2017) Diagnosis of acute pulmonary embolism. J Thromb Haemost 15: 1251-1261.

23. Kline JA, Garrett JS, Sarmiento EJ (2020) Over-Testing for suspected pulmonary embolism in American emergency departments: The continuing epidemic. Circ Cardiovasc Qual Outcomes 13: e005753.

24. Spiezia L, Campello E, Cola M, et al. (2021) More severe hypercoagulable state in acute COVID-19 pneumonia as compared to other pneumonia. Mayo Clin Proc Innov Qual Outcomes 4: 696-702.

25. The European Society for Cardiology (2020) ESC Guidance for the Diagnosis and Management of CV Disease during the COVID-19 Pandemic.

26. Baumann Kreuziger L, Lee AYY, Garcia D, et al. (2021) COVID-19 and VTE/Anticoagulation: Frequently Asked Questions. (Version 9.0; Last Updated February 25, 2021).

27. Deshpande C (2020) Thromboembolic findings in COVID-19 autopsies: Pulmonary thrombosis or Embolism? Ann Intern Med 173: 394-395.

28. Fox SE, Akmatbekov A, Harbert JL, et al. (2020) Pulmonary and cardiac pathology in African American patients with COVID-19: An autopsy series from New Orleans. Lancet Respir Med 8: 681-686.

29. Tang N, Li D, Wang X, et al. (2020) Abnormal coagulation parameters are associated with poor prognosis in patients with novel coronavirus pneumonia. J Thromb Haemost 18: 844-847.

30. McFadyen JD, Stevens H, Peter K (2020) The emerging threat of (Micro) thrombosis in COVID-19 and its therapeutic implications. Circ Res 127: 571-587.

31. Daughety MM, Morgan A, Frost E, et al. (2020) COVID-19 associated coagulopathy: Thrombosis, hemorrhage and mortality rates with an escalated-dose thromboprophylaxis strategy. Thromb Res 196: 483-485.

32. Dolhnikoff M, Duarte-Neto AN, de Almeida Monteiro RA, et al. (2020) Pathological evidence of pulmonary thrombotic phenomena in severe COVID-19. J Thromb Haemost 18: 1517-1519.

33. Ackermann M, Verleden SE, Kuehnel M, et al. (2020) Pulmonary vascular endothelialitis, thrombosis, and angiogenesis in Covid-19. N Engl J Med 383: 120-128.

34. Zhang L, Feng X, Zhang D, et al. (2020) Deep vein thrombosis in hospitalized patients with COVID-19 in Wuhan, China: Prevalence, risk factors, and outcome. Circulation 142: 114-128.

35. Paolisso P, Bergamaschi L, D'Angelo EC, et al. (2020) Preliminary experience with low molecular weight heparin strategy in COVID-19 patients. Front Pharmacol 11: 1124.

36. Ferguson J, Volk S, Vondracek T, et al. (2020) Empiric therapeutic anticoagulation and mortality in critically ill patients with respiratory failure from SARS-CoV-2: A retrospective cohort study. J Clin Pharmacol 60: 1411-1415.

37. Moores LK, Tritschler T, Brosnahan S, et al. (2020) Prevention, diagnosis, and treatment of VTE in patients with coronavirus disease 2019: CHEST guideline and expert panel report. Chest 158: 1143-1163.

38. Long B, Brady WJ, Koyfman A, et al. (2020) Cardiovascular complications in COVID-19. Am J Emerg Med 38: 1504-1507.

39. Spyropoulos AC, Levy JH, Ageno W, et al. The subcommittee on perioperative and critical care thrombosis and haemostasis of the scientific SC of the IS on T and H (2020) Scientific and standardization committee communication: clinical guidance on the diagnosis, prevention, and treatment of venous thromboembolism in hospitalized patients with COVID-19. J Thromb Haemost 18: 1859-1865.

40. Bikdeli B, Talasaz AH, Rashidi F, et al. (2020) Intermediate versus standard-dose prophylactic anticoagulation and statin therapy versus placebo in critically-ill patients with COVID-19: Rationale and design of the INSPIRATION/INSPIRATION-S studies. Thromb Res 196: 382-394.

41. Canzano P, Brambilla M, Porro B, et al. (2021) Platelet and endothelial activation as potential mechanisms behind the thrombotic complications of COVID-19 patients. JACC Basic Transl Sci 6: 202-218.

42. Connors JM, Brooks MM, Sciurba FC, et al. (2021) Effect of antithrombotic therapy on clinical outcomes in outpatients with clinically stable symptomatic COVID-19: The ACTIV-4B Randomized Clinical Trial. JAMA 326: 1703-1712.

43. DiNicolantonio JJ, Barroso-Aranda J (2020) Harnessing adenosine A2A receptors as a strategy for suppressing the lung inflammation and thrombotic complications of COVID-19: Potential of pentoxifylline and dipyridamole. Med Hypotheses 143: 110051.

44. D'Souza R, Malhamé I, Teshler L, et al. (2020) A critical review of the pathophysiology of thrombotic complications and clinical practice recommendations for thromboprophylaxis in pregnant patients with COVID-19. Acta Obstet Gynecol Scand 99: 1110-1120.

45. Pertejo A, Martinez Recio S, Jimenez-Bou D, et al. (2021) 1582P Thromboembolic disease in COVID-19 cancer patients: Impact on overall survival and prognostic factors. Presented at: European Society of Medical Oncology (ESMO) Congress 2021.

46. Ananthula A, Tran D, Thomas K, et al. (2021) Association and prevalence of venous thromboembolism in cancer patients with COVID-19: A single healthcare system experience. Journal of Clinical Oncology 39: 2627-2627.

47. Li J-Y, Wang H-F, Yin P, et al. (2021) Clinical characteristics and risk factors for symptomatic venous thromboembolism in hospitalized COVID-19 patients: A multicenter retrospective study. Thrombo-COVID-19 Collaborative. J Thromb Haemost 19: 1038-1048.

48. Satoskar MA, Metkus T, Soleimanifard A, et al. (2022) Improving risk prediction for pulmonary embolism in COVID-19 patients using echocardiography. Pulm Circ 12: e12036.