Lethal COVID-19: Radiological-Pathological Correlation of the Lungs

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Key Points
- Ground-glass opacities and consolidations in COVID-19 correlate with multiple pathologic processes, notably diffuse alveolar damage, capillary dilatation and congestion and microthrombosis.
- Acute bronchopneumonia is more frequently associated with bronchial wall thickening and consolidation.
- Vascular alterations such as vascular enlargement sign, capillary dilatation and congestion and microthrombosis are prominent findings in both CT and histopathology.

Summary statement
This study deepens our understanding of the pathophysiology of lethal COVID-19 by an in-depth pathological-radiological correlation analysis and confirms the presence of vascular alterations.
Abstract

Background
CT has emerged as an important diagnostic tool in COVID-19, but the underlying pathological changes behind CT findings are not yet fully elucidated.

Purpose
The purpose of this retrospective study was to correlate CT patterns of fatal cases of COVID-19 with post-mortem pathology observations.

Material and Methods
The study included 70 lung lobes of 14 patients who died from RT-PCR confirmed COVID-19. All patients underwent ante-mortem CT and autopsy between March 9 and April 30, 2020. Board-certified radiologists and pathologists performed lobe-wise correlations of pulmonary observations. In a consensus reading, 267 radiological and 257 histopathological observations of the lungs were recorded and systematically graded according to severity. These observations were matched and evaluated.

Results
Predominant CT observations were ground glass opacities (GGO; 59 of 70 lobes examined) and areas of consolidation (33/70). The histopathological observations were consistent with diffuse alveolar damage (70/70), capillary dilatation and congestion (70/70), often accompanied by microthrombi (27/70), superimposed acute bronchopneumonia (17/70) and leukocytoclastic vasculitis (7/70). Four patients had pulmonary emboli. Bronchial wall thickening on CT histologically corresponded with
acute bronchopneumonia. GGOs and consolidations corresponded with mixed histopathological observations including capillary dilation and congestion, interstitial edema, diffuse alveolar damage and microthrombosis. Vascular alterations were prominent observations in both CT and histopathology.

**Conclusion**

A significant proportion of GGO correlated with the pathologic processes of diffuse alveolar damage, capillary dilatation and congestion and microthrombosis. Our results confirm the presence and underline the importance of vascular alterations as a key pathophysiologial driver in lethal COVID-19.
## Abbreviations

| Abbreviation | Definition                                      |
|--------------|-------------------------------------------------|
| COVID-19     | Coronavirus disease 2019                        |
| CT           | Computed Tomography                             |
| CTA          | CT angiogram                                    |
| CTPA         | CT pulmonary angiogram                          |
| DAD          | Diffuse alveolar damage                         |
| GGO          | Ground-glass opacities                          |
| HU           | Hounsfield unit                                 |
| LLL          | Left lower lobe                                 |
| LUL          | Left upper lobe                                 |
| MinIP        | Minimum intensity projection                    |
| ML           | Middle lobe                                     |
| PE           | Pulmonary embolism                              |
| RLL          | Right lower lobe                                |
| RT-PCR       | Reverse transcription polymerase chain reaction |
| RUL          | Right upper lobe                                |
| SARS-CoV-2   | Severe acute respiratory syndrome coronavirus 2 |
Introduction

Coronavirus disease 2019 (COVID-19) caused a pandemic with more than 34'000'000 cases worldwide (1). It is caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV2) and has an estimated mortality rate of up to 5-10% (2). Given the high reproduction number of the virus, diagnostic tools for rapid diagnosis and evaluation are important to track and mitigate transmission. Computed tomography (CT) of the chest is sensitive for COVID-19 in high prevalence areas and may have a role in identifying other causes of respiratory failure (3). Frequently found observations include ground-glass opacities (GGO), consolidations, crazy-paving pattern, reticulations, thickened interlobular septa and air bronchogram. A better characterization of the main pathological drivers for each radiological pattern, which is currently lacking (4, 5), could provide a strong, rational foundation for treatment strategies. The first post-mortem examination of a COVID-19 patient (6) showed pulmonary edema and hyaline membrane formation in both lungs, which is the assumed histopathological correlate of GGO (7). Based on knowledge previously acquired from SARS cases, diffuse alveolar damage is suspected to represent the primary histological response that accompany acute lung injury (8). Furthermore, there is new evidence that associate thromboembolic complications in COVID-19 with coagulation activation, endothelial dysfunction, capillary congestion, and acute (exudative) diffuse alveolar damage (DAD) (9-11). Microthrombosis of lung capillary networks and thromboembolism are emerging as key pathophysiological drivers behind hypoxemia leading to mechanical ventilation (12, 13). Radiological imaging might reflect these vascular abnormalities by the vascular thickening sign (14) and perfusion abnormalities (15).
The purpose of this retrospective study is to gain knowledge on the pathologies underlying CT patterns in COVID-19 by means of radiological-pathological correlation analysis of a local COVID-19 autopsy series of patients who underwent ante-mortem chest CT.
Material and Methods

This retrospective, dual-center radiology and pathology study was approved by the local ethics committee, written informed consent was obtained from all subjects (ID *blinded for review*)

Study population

We retrospectively identified all lethal COVID-19 cases confirmed by reverse transcription polymerase chain reaction (RT-PCR) with both an antemortem CT and autopsy performed between March 9 and April 30, 2020 (n=14). In cases with multiple CTs, the last antemortem scan was selected for analysis. Complete autopsy was performed in 13 and partial autopsy of the upper respiratory tract, lungs and heart in one case. Autopsies were executed at (*blinded for review*) (n=10) and (*blinded for review*) (n=4). Main clinical symptoms and medication were summarized and evaluated.

Computed tomography

All patients underwent thin-section, 1-mm chest CT (scanners: Somatom Force [n=2; 2 x 192 slices], Somatom Definition Edge [n=2; 128 slices], Somatom Definition AS+ [n=7; 128 slices], Biograph 128 [n=1; 128 slices], all Siemens Healthineers; iCT 256 [n=2; 256 slices], Philips Healthcare). Peak kilovoltage was 80-120 kV (mean: 105; SD: 11), exposure 72-498 mAs (mean: 175; SD: 142), and slice thickness 1 mm (all cases). Image acquisition was conducted in supine position. Contrast agents were utilized in five cases (contrast agent: iopromide, Bayer), all other scans were without contrast. Fully anonymized, transverse chest CT series of this cohort in lung kernel
reconstruction are hosted on the cloud and are publicly available. The links can be found in supplement S1.

Two radiologists with 12 (*blinded for review*) and 12 (*blinded for review*) years of experience in thoracic radiology performed a consensus reading of the following CT patterns that are common in COVID-19 (16) (with * according to the Fleischner Society Glossary of Terms for Thoracic Imaging (17)): GGO*, reticulations*, crazy-paving*, consolidations*, bronchial wall thickening*, vascular thickening, atelectasis*, pleural effusion, pulmonary embolism, lymphadenopathy*, pulmonary congestion. Severity was graded lobe-wise using a 4 step-scale: none, mild (0-33 % of lobe involved), moderate (34-66 % of lobe involved), severe (67-100 % of lobe involved). Vascular thickening (vascular enlargement/vascular congestion) and pulmonary arterial enlargement (related to the corresponding bronchus) were rated by means of binary grading (negative/positive). Figures 1 A and B provide examples of vascular CT radiographic features examined in this study.

Gross findings

Our institute developed a COVID-19 optimized autopsy protocol in line with recently published recommendations (18). Thoracic organs, lungs, trachea and larynx were completely exenterated and perfused via the trachea with 4% refrigerated (+4°C) phosphate-buffered formalin (pH 7.4). The trachea was then closed with a clamp and specimens were left in formalin at room temperature for 72 hours before dissection. The lungs were subsequently cut into 0.5-1 cm parasagittal slices. At least two sections of the first 3 cm of subpleural parenchymal of each lobe as well as the trachea
were histologically analyzed. The observations were graded in a four-step scale according to severity (none, mild, moderate, severe; Table 1).

Microscopy

Tissue samples were processed using standard laboratory equipment and stained with standard histochemical methods (haematoxylin and eosin staining). The histopathological specimens were reviewed in consensus by two pathologists with 13 (*blinded for review*) and 20 (*blinded for review*) years of experience. The observations were graded in a four-step scale according to severity (none, mild, moderate, severe; Table 2). Figure 1 (C-H) provides an overview of all histopathological features examined in this study.

Radiological-pathological correlation and statistical analysis

To ensure the highest possible granularity despite retrospective study design and temporal and local variability of inflammatory processes, the rad-path correlation was performed at a lobe-wise level instead of a patient-wise level. The observations of each lobe were recorded separately and graded according to severity (either binary, positive/negative, or a four-step scale, using the terms none, mild, moderate, and severe) in close coordination between radiologists and pathologists. The correlation analysis examined whether radiological observations were associated with pathological patterns. In cases that displayed multiple radiological features within a lobe, features were independently compared between the corresponding gross specimen and the histopathological findings.
Continuous variables were analysed by means and ranges (demographics), means and interquartile ranges (IQR; laboratory values) and frequencies (number of lobes with a given feature). To test for differences in observations between patients with a short and long time interval between onset of symptoms and CT (imaging features), and between onset of symptoms and autopsy (histopathology features), patients were assigned to short interval and long interval cohorts according to the respective median. Patients having a shorter time interval than the median were assigned to the short interval group and patients having a longer time interval than the median were assigned to the long interval group. Chi-squared tests were conducted to assess differences between long / short interval groups regarding age (T-test) and sex (Chi-squared test). Analysis was conducted in R (19) and figures were produced using ggplot2 (20). P-values ≤ 0.05 were considered to be statistically significant.
Results

Clinical characteristics

Clinical features including comorbidities and symptoms are listed in Table 3. The mean interval from death to autopsy was 38.5 hours (range: 11.0-97.0 hours) and the mean interval between chest CT and death was 3.7 days (range: 0.0-17.0 days). The average hospitalization time before death was 5.5 days. The mean age in our collective was 76 years (range: 58-96 years); 29% of patients were female. There were no statistically significant differences in age and sex between patients with a short and long time interval between onset of symptoms and CT (age: p=0.19; sex: p=0.48) and death (age: p=0.27; sex: p=0.73), respectively.

The most prevalent clinical symptoms were cough (n=10), followed by fever (n=7) and dyspnea (n=3). All patients suffered from at least three comorbidities (three comorbidities: n=7, four comorbidities: n=6; more than four comorbidities: n=1; Table 3). Patients in our cohort were treated with hydroxychloroquine (n=11), iopinavir/ritonavir (n=7), tocilizumab (n=5), antibiotics (n=4), remdesivir (n=1), or did not receive COVID-19 specific mediation (n=3).

Computed Tomography

Lobe-wise analysis resulted in 267 radiological observations (Figure 2a): GGO (n=59; 14/14 (100%) patients), vascular thickening (n=36; 12/14 (86%) patients), pulmonary arterial enlargement (n=36; 12/14 (86%) patients), consolidations (n=33; 11/14 (79%) patients), bronchial wall thickening (n=32; 8/14 (57%) patients), reticulations (n=23; 8/14 (57%) patients), crazy paving (n=13; 4/14 (29%) patients), atelectasis (n=12; 7/14 patients).
(50%) patients), pleural effusion (n=9; 6/14 (43%) patients), lymphadenopathy (n=7; 7/14 (50%) patients), pulmonary embolism (n=5; 1/5 patients with contrast enhanced CT) and pulmonary congestion (n=2; 1/14 (7%) patients). Anonymized CT scans of all patients are hosted on our freely accessible e-learning server. Links are provided in Supplement S1.

Time interval between onset of symptoms and CT

The mean interval from first symptoms to CT was 5.7 days. The comparison of patients with a short (n=9) vs. long (n=5) interval from first symptoms to CT showed varying incidence of multiple radiological observations (Figure 2b). The differences were statistically significant for bronchial wall thickening (+12.9%; p<0.001), pulmonary arterial enlargement (+4.1%; p-value=0.05) and pulmonary embolism (+ 5.0%; p=0.003).

Gross pathology

The overall gross findings of all lobes in all patients are shown in Table 4. Overall presence per patient was heterogeneous and as follows: interstitial edema of interlobular septae (n=65; 13/14 (93%) patients), areas of consolidation (n=43; 10/14 (71%) patients), emphysema (n=13; 7/14 (50%) patients), haemorrhage (n=9; 5/14 (36%) patients) and pulmonary embolism (n=9; 4/14 (29%) patients). In all cases, lung parenchyma was heavy and firm and unevenly bluish-red with signs of severe congestion.
Histopathology

In the overall lobe-wise analysis of our examined collective, 257 pathological observations were recorded (Figure 3a). Observations included capillary dilatation and congestion (n=70; 14/14 (100%) patients), acute (exudative) phase DAD (n=54; 11/14 (79%) patients) and organizing (proliferative) phase DAD (n=26; 7/14 (50%) patients), interstitial edema (n=64; in 14/14 (100%) patients), superimposed acute bronchopneumonia (n=17; 7/14 (50%) patients), microthrombi (n=27; 7/14 (50%) patients) and leukocytoclastic vasculitis (n=7; 3/14 (21%) patients). Leukocytoclastic vasculitis showed the presence of neutrophils in the vessel wall and was primarily interpreted in the context of severe acute bronchopneumonia, no patient had evidence of generalized systemic vasculitis. Anonymized high-resolution scans of histology sections of each patient with representative lung observations are hosted on a local server and are available online. Links can be found in Supplement S1.

Time interval between symptom onset and death

The mean interval between symptom onset and death was 10.4 days. The comparison of patients with a short (n=7) vs. long (n=7) interval between symptom onset and death showed varying incidence of histopathological observations (Figure 3b). There were statistically significantly higher frequencies of acute (exudative) phase DAD (p=0.04) and interstitial edema (p=0.03) in the short group.

Radiological-pathological correlation of pulmonary observations

When correlating the summed CT features with histopathology of all lobes, the areas of GGO (n=59 lobes / 14 patients) reflected the presence of multiple pathologic
processes (Figure 4). Figures 5 and 6 display typical observations in COVID-19, each depicted in gross pathology (left column), CT (middle column) and microscopy (right column). Acute (exudative) phase DAD was observed in 46 (see also Figure 5a-c, 6a, 6c), organizing (proliferative) phase DAD in 21 (see Figure 5b, 6a, 6c), acute bronchopneumonia in 8 (see Figure 5a, 6b), capillary dilatation and congestion in 57 (see Figure 5a-c, 6a-c), leukocytoclastic vasculitis in 6 (see Figure 6b), interstitial edema in 53 and microthrombi in 22 (see Figure 5a) of the 59 lobes presenting GGO. Consolidations on CT (n=33 lobes / 11 patients) were microscopically characterized by multiple pathologic processes of acute (exudative) phase DAD in 21, organizing (proliferative) phase DAD in 8, acute bronchopneumonia in 4, capillary dilatation and congestion in 31, leukocytoclastic vasculitis in 4, interstitial edema in 29 and microthrombi in 14 of the 33 lobes presenting consolidations. A sub analysis of lobes with pure GGO and consolidation in CT also showed heterogeneous histopathological patterns including exudative DAD, organizing (proliferative) DAD, acute bronchopneumonia and capillary dilatation and congestion. The comparison of lobes with pathologically defined acute bronchopneumonia vs. without revealed consolidations in 58.8% vs. 40.4% of lobes and bronchial wall thickening in 47.1% vs. 42.0% of lobes, respectively.

Patients with consolidations (n=11) on CT showed a shorter time to death (2.7 days) compared to those without (n=3; 7.3 days). When comparing histological observations in patients with and without consolidations, DAD was more frequent in cases without consolidations (+13.5%), whereas acute bronchopneumonia (+4.6%) and leukocytoclastic vasculitis (+3.6%) were more frequent in cases with consolidations.
Discussion

This is the first systematic morphological comparison of ante-mortem chest CT scans with post-mortem gross findings and histopathology in COVID-19. CT patterns of COVID-19 were characterized by GGO, consolidation, bronchial and vascular changes. Gross findings included severe interstitial edema and congestion resulting in increased parenchymal consistency, while histological examination revealed prominent capillary dilatation and congestion with or without microthrombosis, interstitial edema and DAD as the most consistent finding. Correlations of radiological and pathological observations showed that CT patterns reflect multiple pathologic processes of varying composition. All histological changes investigated were found in GGO as well as in consolidations. Consolidations and bronchial wall thickening were more frequently observed in presence of acute bronchopneumonia, and pulmonary arterial enlargement might be an indicator of microangiopathic change.

Our results showed predominantly bilateral areas of GGO and consolidations, consistent with previous reports on the occurrence of various pulmonary CT observations in COVID-19 (19-21). Radiological-pathological correlation showed that CT observations were characterized by multiple pathologic processes of DAD, interstitial edema and capillary dilatation and congestion. These changes are consistent with previously published studies without detailed CT correlation (10).
While most recent studies also described the radiological pattern of organization of pneumonia in patients with COVID-19 (4, 21), no histopathological features for fibrosis were found in our study. Interestingly, in a recent study using post-mortem transbronchial lung cryobiopsy from six patients with a median illness duration of 32 days, three showed late/fibrotic phase diffuse alveolar damage, one of them with honeycombing (22). In another study, for five patients, who died around 20 days after the beginning of symptoms, the histologic pattern was an acute fibrinous and organizing pneumonia (AFOP) (23). The various radiological patterns showed only minor differences in the frequency of underlying histopathological changes. The most important components for GGO were capillary dilatation and congestion (26.9%), interstitial edema (25.0%) and acute (exudative) DAD (21.7%). Consolidation showed similar histopathological patterns with slightly more microthrombosis (12.6% vs. 10.4%) and leukocytoclastic vasculitis (3.6% vs. 2.4%). Importantly, the observed CT patterns were not linked to a specific histopathological finding.

Furthermore, an increase in the frequencies of bronchial wall thickening, pulmonary arterial enlargement and pulmonary embolism in patients with longer symptom onset to CT was found. The increase in bronchial wall thickening and consolidation might be explained by superimposed bacterial superinfections and subsequent acute bronchopneumonia, which was evident in our collective. Figure 5a provides an example. Of note, while there was no evident temporal dynamics of microthrombosis, more pulmonary emboli were detected in patients with a longer interval between onset of symptoms and CTPA in our cohort. Surprisingly, the temporal dynamic of radiographic changes could not be clearly correlated with corresponding histopathological results. Further evaluation of the histopathological observations as
a function of disease duration showed an increase of acute (exudative) and organizing (proliferative) phase DAD, whereas no fibrotic changes were observed.

The observation of enlarged pulmonary arteries in our series might be related to an increase of parenchymal and predominantly intravascular pressure (24), due to the severe COVID-19 pulmonary microangiopathy affecting the alveolar capillary network (25). In the context of recent reports of pathologically altered coagulation, these results may also indicate an increase in vascular incidents (10, 26). 30 of 388 patients in the study by Lodigiani et al. underwent CTPA confirming pulmonary embolism in 10 cases (9). Although higher, this positive rate of 33% seems in line with the observed rate in our collective of 20%; importantly, all our patients received anticoagulation. While most of the currently available literature relies on non-contrast CT (27), the need to assess vascular abnormalities is being recognized as an increasingly important factor (28, 29), both to distinguish COVID-19 pneumonia from other viral infections, and to exclude pulmonary embolism. Interestingly, the high incidence of microthrombosis and the low number of pulmonary emboli detected with CTPA suggests a possible underestimation of the vascular alterations associated with COVID-19 using imaging, especially in un-enhanced scans. Newly described signs such as “vascular thickening”, “pulmonary arterial enlargement”, or “vascular congestion” could reflect these alterations. Severe influenza pneumonia has previously been described to cause a hyper-inflammatory response with virally associated platelet activation, which can lead to pulmonary thrombosis with passive congestion. Similar to influenza pneumopathy, COVID-19 is able to limit compensatory ventilation responses by means of vascular leakage and alveolar edema, thus contributing to widespread haemorrhage (10, 30). According to a comparative autopsy study, alveolar capillary
microthrombi were 9 times as prevalent in patients with COVID-19 as in patients with influenza and extent of angiogenesis was 2.7 times higher than in influenza (25). Therefore, described vascular alterations seem to be specific for COVID-19.

Our study has several limitations. First, its retrospective design and the relatively small number of cases. For this reason, no complex statistical analyses have been performed. Second, the time intervals between symptoms, CT and autopsy varied. This is a factor that cannot be controlled in an observational study. Third, as all patients died from COVID-19, there is a bias towards more severe and rapidly progressive courses of disease. Fourth, the correlation analysis was performed on a per-lobe level. A finer granularity was not possible due to the specific COVID-19 autopsy protocol. For future studies, image-guided tissue sampling is desirable to further increase the resolution of analysis. However, as stated in the results, multiple pathologic processes were found also in lobes with pure GGO / consolidation patterns in CT. Finally, CT parameters and vendors differed due to the dual-center nature of this study. However, all scans were acquired with a high diagnostic quality clearly suitable for this correlation analysis as shown in supplementary materials.

The results of this study deepen our understanding of COVID-19 pathophysiology confirming the importance of vascular alterations. Our observations imply that both severe acute lung injury and vascular complications contribute to fatal outcomes. Therefore, therapy optimization must focus on both pathologies to improve clinical management of COVID-19.
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Table 1. Severity scoring system for gross findings.

|                      | mild             | moderate         | severe           |
|----------------------|------------------|------------------|------------------|
| Hemorrhage           | <5% of lobe      | 5-50% of lobe    | >50% of lobe     |
| Consolidation        | <10% of lobe     | 10-50% of lobe   | >50% of lobe     |
| Emphysema            | palpable         | visible          | bullae           |
| Interstitial edema   | <10% of lobe     | 10-50% of lobe   | >50% of lobe     |
| Pulmonary embolism   | peripheral       | central          |                  |
Table 2. Severity scoring system of histological findings.

| Condition                        | Mild                                      | Moderate                                               | Severe                                                   |
|----------------------------------|-------------------------------------------|-------------|------------------------------------------------|
| Acute (exudative) DAD            | <25% of parenchyma, hyaline membranes with pneumocyte denudation | 25-50% of parenchyma, and/or multinucleated cells       | >50% of parenchyma, and/or necrosis                       |
| Organizing (proliferative) DAD   | <25% of parenchyma, intraalveolar proliferation of fibroblasts | 25-50% of parenchyma                                  | >50% of parenchyma, thickening of interstitial space     |
| Bronchopneumonia                 | <10% of parenchyma                        | 10-50% of parenchyma                                  | >50% of parenchyma                                        |
| Capillary dilation and congestion| <10% of parenchyma                        | 10-50% of parenchyma                                  | >50% of parenchyma                                        |
| Vasculitis                       | Present                                   |                                                        |                                                            |
| Interstitial edema               | <10% of parenchyma                        | 10-50% of parenchyma                                  | >50% of parenchyma                                        |
Table 3. Clinical features including comorbidities and symptoms

| Clinical characteristics                  | mean       | range       |
|------------------------------------------|------------|-------------|
| Age                                      | 76 y       | 58 - 96 y   |
| Sex                                      | M 10 : F 4 | M 71.0% : F 29.0% |
| Time between CT and death                | 3.7 d      | 0 - 17 d    |
| Hospitalization                          | 5.5 d      | 0 - 17 d    |
| Post-mortem interval                     | 38.5 h     | 11 - 97 h   |
| Mechanical ventilation                   | 4 of 14 patients |
| Anticoagulation (prophylactic / therapeutic) | 8 : 6       |
| Comorbidities                            | n          | %           |
| Hypertension                             | 14         | 100.0%      |
| Cardiovascular disease                   | 7          | 50.0%       |
| (Pre-)obesity                            | 6          | 42.8%       |
| Diabetes mellitus, type 2                | 4          | 28.6%       |
| Initial clinical presentation            |            |             |
| Cough                                    | 10         | 71.4%       |
| Fever                                    | 7          | 50.0%       |
| Dyspnea                                  | 3          | 21.4%       |
| Laboratory values                        | median     | IQR         |
| Interleukin-6 (IL-6)                     | 781 ng/l   | 399 - 2668 ng/l |
| C-reactive protein (CRP)                 | 248 mg/l   | 136 - 319 mg/l |
| Ferritin                                 | 3086 µg/l  | 2259 - 4113 µg/l |
| Procalcitonin (PCT)                      | 1.19 µg/l  | 0.10 - 3.64 µg/l |
| Troponin T (cTnT)                        | 33 ng/l    | 18 - 55 ng/l |
| Lactate dehydrogenase (LDH)              | 602 pg/ml  | 360 - 776 pg/ml |

Listed values correspond to the highest (LDH, cTnT), latest (IL-6, ferritin, PCT, CRP) and, in case of administration of tocilizumab, last CRP value before administration.
|                          | n  | mild | moderate | severe |
|--------------------------|----|------|----------|--------|
| Interstitial edema       | 50 | 6    | 42       | 2      |
| Consolidation            | 43 | 33   | 5        | 5      |
| Emphysema                | 13 | 8    | 5        | 0      |
| Haemorrhage              | 9  | 7    | 2        | 0      |
| Pulmonary embolism       | 9  | 4    | 5        | 0      |
Figure 1: Overview of radiographic and histopathologic observations. (A) transverse CT image showing dilated peripheral focal pulmonary artery next to consolidation and GGO marked with an arrow (“vascular thickening”). (B) transverse CT image showing dilated segmental arteries marked with arrows (“pulmonary arterial enlargement”). (C) diffuse alveolar damage, exudative phase: hyaline membranes made up of fibrin covering surface of alveolae (arrows). There is only a sparse interstitial inflammatory infiltrate (hematoxylin & eosin [H&E], 100x). (D) diffuse alveolar damage, proliferative phase: within alveolae, there are fibrohistiocytic proliferations (arrows), hyaline membranes are already resorbed. Extensive capillary congestion is also found (asterisk) (H&E, 100x). (E) severe acute bronchopneumonia showing alveolae filled with aggregates of neutrophils and fibrin (asterisk). There are no hyaline membranes covering alveolar walls (H&E, 50x). (F) higher magnification of alveolar walls showing massive capillary congestion (H&E, 200x). (G) small pulmonary artery showing inflammatory infiltrates primarily consisting of neutrophils infiltrating vessel walls (H&E, 400x). (H) interstitial edema: lung parenchyma with massively widened interstitial spaces showing edema (asterisks), dilated capillaries (arrow) and lymphohistiocytic inflammatory infiltrates (arrowhead) (H&E, 100x).

2a Frequencies of lobe-wise analysis of radiological findings according to severity grading (in gray) and binary categories (present/absent) for the pulmonary arterial enlargement and vascular thickening sign (in blue). GGO = ground-glass opacities, P. = pulmonary.
2b Percentage of radiological findings according to the time interval (short interval = black, grey interval = long) between onset of symptoms and CT. P-values are provided if significant. Percentage defined as ratio of lobes with a given feature in relation to all lobes within the time interval category. GGO = ground-glass opacities, P. = pulmonary, ns = not significant.

3a Frequencies of lobe-wise analysis of microscopy findings according to severity grading (in grey) and binary categories (present/not present) for vasculitis (in blue). DAD = diffuse alveolar damage.
3b Percentage of histopathological findings according to the time interval (short interval = black, grey interval = long) between onset of symptoms and autopsy. Percentage defined as ratio of lobes with a given feature in relation to all lobes within the time interval category. P-values are provided if significant. DAD = diffuse alveolar damage, ns = not significant.

4 Frequencies of observed histopathological findings in lobes affected by consolidations and GGO. Consolidations were observed in 33 lobes / 11 patients. GGOs were observed in 59 lobes / 14 patients. GGO = ground-glass opacities.
5a Radiological-pathological correlation in patient 1, a 67-year-old female. Gross pathology findings revealed interlobular septal edema, congestion, thickened bronchial walls (arrow) and consolidation (arrowhead). The corresponding CT in sagittal plane discloses bronchial wall thickening (arrow) and dystelectasis in the posterior parts of the lower lobes (arrowhead). Microscopy disclosed bronchopneumonia in the left lower lobe (upper right), thickened bronchial walls (lower left), and microthrombosis (lower right, immunohistochemistry for fibrin, polyclonal antibody (Dako, Glostrup, Denmark, A0080)).

5b Radiological-pathological correlation in patient 2, a 66-year-old man. Gross findings document interlobular septal edema and segmental hemorrhage in the anterobasal segment of the left lower lobe (arrow), while CT shows peripherally pronounced GGO in the left lower lobe (arrow). Inserted miniP reveals some focal bronchial dilatation in association to ground glass opacities. Microscopy reveals hyaline membranes as remnants of acute exudative (arrowhead) and intraalveolar fibroblastic proliferations as signs of proliferative DAD (arrow). Furthermore, capillaries show extensive congestion (asterisk).
5c Radiological-pathological correlation in patient 3, a 73 year old male. Gross findings include interstitial edema, congestion and chronic pulmonary embolism in the left upper lobe that was not disclosed by imaging due to un-enhanced acquisition (insert). CT discloses extensive GGO in transition to consolidation in the left lower lobe (arrow) as well as lingula. Microscopy reveals hyaline membranes as a correlate of acute exudative DAD predominantly in the left lower lobe (arrow).

6a Radiological-pathological correlation in patient 4, a 81-year-old female. Gross findings include congestion, interlobular septal edema and emphysema (arrow). CT reveals consolidation (arrow) in the posterior parts of the left lower lobe and crazy paving (arrowhead) in the apical upper lobe. Microscopy revealed both hyaline membranes as correlates of acute exudative (arrowhead) and intraalveolar fibrohistiocytic proliferates as signs of proliferative (arrow) DAD.
In press

6b Radiological-pathological correlation in patient 6, a 71-year-old male. Gross findings reveal congestion and severe consolidation (arrow). CT shows obstruction and consolidation in the posterior parts of the upper and lower right lobe (arrow). Microscopy showed extensive bronchopneumonia, dilated capillaries (arrow), vasculitis (insert at lower right), and microthrombosis (not shown). Next generation sequencing identified bacterial species such as Staphylococcus aureus and several strains of streptococcal species.

6c Radiological-pathological correlation in patient 9, a 58-year-old male. Gross findings show congestion, interlobular septal edema and multiple thromboembolisms (insert at lower left). CT reveals subtotal consolidation of the left lower lobe (arrow) and bilateral pulmonary embolisms (not shown). Microscopy shows hyaline membranes as correlates of acute exudative (arrow) and fibroblastic proliferations as signs of proliferative (arrowhead) DAD as well as alveolar hemorrhage (far right).

Supplementary Materials

S1. Links to original histology sections and chest CTs of all patients.
Anonymized, zoomable high-resolution scans of the histology sections of each patient as well as the corresponding transverse chest CT series in lung kernel reconstruction can be found following the links provided below.

Abbreviations: LLL = left lower lobe, LUL = left upper lobe, ML = middle lobe, RLL = right lower lobe, RUL = right upper lobe

**Patient 1**

RUL https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=42991&layerId=10001&x=222&y=0&scale=3.552912019826518&angle=0&vw=1540&vh=868

ML https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=44132&layerId=10001&x=155&y=0&scale=3.7454468838228476&angle=0&vw=1540&vh=868

RLL https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=42996&layerId=10001&x=176&y=0&scale=3.677387857775277&angle=0&vw=1540&vh=868

LUL https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43001&layerId=10001&x=174&y=0&scale=3.643679951463077&angle=0&vw=1540&vh=868

LLL https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43006&layerId=10001&x=257&y=0&scale=3.462909861613265&angle=0&vw=1540&vh=868

CT https://www.rapmed.net/#/publications/1/36927fad-e0f3-4d07-a0f7-4ff8e1e4f177

**Patient 2**

RUL https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43141&layerId=10001&x=193&y=0&scale=4.0836174181514115&angle=0&vw=1540&vh=868
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LUL https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43156&layerId=10001&x=360&y=0&scale=1.909856636985595&angle=0&vw=1540&vh=868

LLL https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43161&layerId=10001&x=371&y=0&scale=2.358921007654009&angle=0&vw=1540&vh=868

CT https://www.rapmed.net/#/publications/1/df8916bf-ba79-43e6-b889-0337ac37d0bf
Patient 3

RUL  https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43186&layerId=10001&x=237&y=0&scale=2.2200375654397954&angle=0&vw=1540&vh=868

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RLL  https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=44082&layerId=10001&x=197&y=0&scale=2.412629476015722&angle=0&vw=1540&vh=868

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CT  https://www.rapmed.net/#/publications/1/af7aca8c-4a8f-4283-9db9-b928e165b6f7
Patient 4

RUL  https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43231&layerId=10001&x=376&y=0&scale=1.745271756204838&angle=0&vw=1540&vh=868

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RLL  https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=44087&layerId=10001&x=309&y=0&scale=2.1033859318648655&angle=0&vw=1540&vh=868

LUL  https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43241&layerId=10001&x=130&y=0&scale=4.283568998255405&angle=0&vw=1540&vh=868

LLL  https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43246&layerId=10001&x=307&y=0&scale=2.1629661355163385&angle=0&vw=1540&vh=868

CT  https://www.rapmed.net/#/publications/1/b5b8a9c3-2635-4f3e-8a91-c5b4a547a501
Patient 5

**RUL**  https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43316&layerId=10001&x=303&y=0&scale=1.8485292160255557&angle=0&vw=1540&vh=868

**ML**  https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=44092&layerId=10001&x=237.38000000000034&y=-96.60000000000036&scale=2.4970437237638845&angle=0&vw=1540&vh=868

**RLL**  https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43321&layerId=10001&x=257&y=0&scale=3.4142511427803175&angle=0&vw=1540&vh=868

**LUL**  https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43326&layerId=10001&x=332&y=0&scale=2.0007203054094935&angle=0&vw=1540&vh=868

**LLL**  https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=44097&layerId=10001&x=301&y=0&scale=3.3936543087320072&angle=0&vw=1540&vh=868

**CT**  https://www.rapmed.net/#/publications/1/2dbea93a-23bb-424d-8f25-ba773030fe81
Patient 6

RUL https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43276&layerId=10001&x=360&y=0&scale=2.0380071905495636&angle=0&vw=1540&vh=868

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CT https://www.rapmed.net/#/publications/1/3a99d3c5-ce74-4c18-b580-3d26bfc455b
Patient 7

RUL  https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43101&layerId=10001&x=243.13194534000036&y=3.7954978399973242&scale=3.8367206778243527&angle=0&vw=1540&vh=868

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LUL  https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43111&layerId=10001&x=278&y=0&scale=3.5586874009064555&angle=0&vw=1540&vh=868

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CT  https://www.rapmed.net/#/publications/1/c1e0a57e-cbb2-43a9-b08a-237d2b90968a
Patient 8

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ML https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43066&layerId=10001&x=294&y=0&scale=1.933504459430063&angle=0&vw=1540&vh=868

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Patient 9

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LUL  https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43406&layerId=10001&x=325&y=0&scale=2.0649382027692593&angle=0&vw=1540&vh=868

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Patient 10

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ML   https://ictvsidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43361&layerId=10001&x=50&y=0&scale=5.200219983384233&angle=0&vw=1540&vh=868

RLL  https://ictvsidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43366&layerId=10001&x=423&y=0&scale=1.833036324364811&angle=0&vw=1540&vh=868

LUL  https://ictvsidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43371&layerId=10001&x=351&y=0&scale=1.7678348382990571&angle=0&vw=1540&vh=868

LLL  https://ictvsidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43376&layerId=10001&x=503.6324670929184&y=32.54261062503866&scale=2.044381604860057&angle=0&vw=1540&vh=868

CT   https://www.rapmed.net/#/publications/1/cd8b65bb-87d4-4f70-9e04-ab54825dc79e
Patient 11

RUL  https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43757&layerId=10001&x=326&y=0&scale=2.069939450395901&angle=0&vw=1540&vh=868

ML

RLL  https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43752&layerId=10001&x=250&y=0&scale=3.887609783407397&angle=0&vw=1540&vh=868

LUL  https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43762&layerId=10001&x=269&y=0&scale=3.844262791401756&angle=0&vw=1540&vh=868

LLL  https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43767&layerId=10001&x=291&y=0&scale=1.8867962978687271&angle=0&vw=1540&vh=868

CT  https://www.rapmed.net/#/publications/1/fb61e029-0509-412a-85ee-cc17591b56e8
Patient 12

RUL

ML

RLL  https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43451&layerId=10001&x=128&y=0&scale=4.465460245370417&angle=0&vw=1540&vh=868

LUL  https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43451&layerId=10001&x=128&y=0&scale=4.465460245370417&angle=0&vw=1540&vh=868

LLL  https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43461&layerId=10001&x=274&y=0&scale=2.38006897881365&angle=0&vw=1540&vh=868

CT  https://www.rapmed.net/##publications/1/5377f057-3929-4c76-8191-c8cbce85b574
Patient 13

RUL https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43577&layerId=10001&x=75&y=0&scale=4.657569850552306&angle=0&vw=1540&vh=868

ML https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43582&layerId=10001&x=370&y=0&scale=2.150198852365424&angle=0&vw=1540&vh=868

RLL https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43587&layerId=10001&x=290&y=0&scale=2.0610501516514707&angle=0&vw=1540&vh=868

LUL https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=43577&layerId=10001&x=75&y=0&scale=4.657569850552306&angle=0&vw=1540&vh=868

LLL

CT https://www.rapmed.net/#/publications/1/1144d7b9-ef98-474b-b5d6-9d03d0356610
Patient 14

RUL  https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=44012&layerId=1&x=-203.22031778412042&y=-103.65175717648753&scale=3.6296485420715933&angle=0&vw=1540&vh=868

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LLL  https://ictvslidewp01.usb.ch/OlyViaWeb/Html5Viewer?dbId=798403c7-6d3e-4d97-a412-5bc0acd1705f&recordId=44024&layerId=1&x=-911.4121398571151&y=-105.25892966720812&scale=6.586314360049753&angle=0&vw=1540&vh=868

CT  https://www.rapmed.net/#/publications/1/8ae9e64f-1d14-4099-ba95-d68b3a5f5053