

Table S1 Histopathological findings in lung sections from COVID-19 patients not mechanically ventilated or with MV shorter than 24 hours (5-21)

Publication: reference number, country, city, publication date, type of sample, number of patients	Sex, age, risk factors/comorbidities, outcome	Date histopathology lung after onset of symptoms	Clinical history	Ancillary methods	Main positive pathology lung findings	Findings in other organs
(20), China, Zeng <i>et al.</i> , <i>Histopathology</i> 2020 May, doi: 10.1111/his.14138. 10 April 2020, biopsy, 1 patient	F, 55 y, a benign pulmonary nodule, alive (n=1)	Day 0	Right lower lung lobe resection for pulmonary nodule. Afebrile, no respiratory symptoms. Confirmed to have preoperative SARS-CoV-2 infection	Anormal accumulation of CD4+ helper T lymphocytes and CD163+ M2 macrophages in the lung tissue	Lungs: Intra-cytoplasmic viral-like inclusion in pneumocytes and macrophages. Exudative inflammation (lymphocytes and monocytes) surrounding the visceral pleura. Widened alveolar septa, with obvious hyperemia, dilated capillaries. Multinucleated giant cells in alveolar spaces. No HM nor fibrin. Focal pneumocyte hyperplasia. Scattered large protein globules in alveolar spaces. Alveolar spaces were filled with a large amount of light red, homogeneous, proteinaceous fluid, admixed with variable numbers of red blood cells, lymphocytes and monocytes	
(16), USA, New York, Magro <i>et al.</i> , <i>Transl Res</i> 2020 Apr 15, doi: 10.1016/j.trsl.2020.04.007, 09 Apr 2020, limited autopsy, 1 patient	M, 62 y, CVD, DM, hepatitis C, chronic renal disease, death (n=1)	Day 0	Presented in extremis with severe hypoxemia and blood pressure of 180/100 mmHg. Placed on comfort measures and died a few hours after presentation	Chest X ray: bilateral opacities most prominent in the peri-hilar distribution. SARS-CoV-2 spike & envelope proteins demonstrated with IHC. Significant vascular deposits of C5b-9 and C4d seen with DAB technique, standard bacterial and fungal respiratory cultures: negative, no other potential pulmonary viral pathogens detected	Lungs: complement significant fibrin deposition within septal capillary lumens and walls accompanied by endothelial cell necrosis. Pattern of cutaneous and pulmonary pathology involving microvascular injury and thrombosis, consistent with activation of the alternative pathway and lectin pathway of complement. Permeation of the inter-alveolar septa by neutrophils amidst the damaged capillaries, along with intra-alveolar neutrophils	
(5), USA, Oklahoma, Barton <i>et al.</i> , <i>Am J Clin Pathol</i> 2020;153:725-33. doi: 10.1093/AJCP/AQAA062, 10 Apr 2020, full post-mortem examination, 2 patients	Case 1: M, 77 y, obesity, HT, deep venous thrombosis, splenectomy, liver cirrhosis, death (n=1)	Day 6	Case 1: fever and chills for 6 days, died while being transported for medical care, not seen by a physician, exhibited symptoms suspicious for COVID-19 at the time of death, no ante-mortem testing for COVID-19	Case 1: IHC: sparse infiltrate CD3-positive T-lymphocytes within the alveolar septa, only rare CD20-positive B-lymphocytes. CD8-positive T-cells slightly outnumbered CD4-positive T-cells. CD68 highlighted a few macrophages	Case 1: lungs: DAD with HM, interstitial lymphoid inflammation, thrombi in small pulmonary arteries, right pleural adhesions	Case 1: hypertensive heart disease with acute ischemia, coronary arteries and abdominal aorta atherosclerosis, arterio-nephrosclerosis, hepatic centrilobular steatosis, liver cirrhosis
	Case 2: M, 42 y, obesity, liver cirrhosis myotonic dystrophy, death (n=1)	Day 2	Case 2: abdominal pain, fever, shortness of breath, cough, survived only a few hours in hospital, at PM examination, evidence of intubation and chest compressions	Case 2: IHC: CD68 highlighted numerous macrophages within the areas of BN; chest CT scan: bilateral GGO; no antemortem testing for COVID-19	Case 2: lungs: acute BN with aspiration, no DAD, filling of peribronchiolar airspaces by neutrophils and histiocytes	Case 2: liver cirrhosis, right renal mass (oncocytoma), mild coronary arteries atherosclerosis, nephrosclerosis
(6) Germany, Hamburg, Wichmann <i>et al.</i> , <i>Annals Intern Medicine</i> 2020, doi: 10.7326/M20-2003, 6 May 2020, full autopsy, 7 patients (patients 1, 2, 5, 6, 8, 9, 10)	Case 1: M, 52 y, obesity	Not mentioned	Case 1: cardiopulmonary resuscitation, sudden cardiac death	PM RT-PCR: + in lungs in all patients (range, 1.2x10 ⁴ to 9x10 ⁹ copies/mL) and in the pharynx of 9 patients. Six patients showed moderate viremia (<4x10 ⁴ copies/mL). In 5 of these patients, viral RNA was also detected in other tissues (heart, liver, or kidney) in concentrations exceeding viremia. Patients without viremia showed no or a low viral load in the other tissues. Only 4 patients had detectable viral RNA in the brain and saphenous vein	3 cases with DAD: 3 activated pneumocytes; 3 HM; 1 fibroblasts; 1 granulocytic infiltration of the alveoli and bronchi, resembling bacterial focal bronchopneumonia; 2 lymphocytes, 1 fibrosis, 1 necrosis; additional findings: 1 congestion of small vessels; 2 thrombi; 1 focal neutrophils. 4 cases without DAD: 4 granulocytic infiltration of the alveoli and bronchi, resembling bacterial focal bronchopneumonia, 2 congestion of small vessels, 2 emphysema, 1 fibrosis	In the 7 cases: pharynx normal. In 1 patient with DAD: acute bronchitis & chronic bronchitis. Patients without DAD: 2 acute bronchitis, 1 chronic bronchitis. In 3 men: vein thrombosis & thrombosis in prostate. Case 1: atherosclerosis
	Case 2: M, 70 y, Parkinson disease, CAD, peripheral artery disease, CRD		Case 2: basic supportive care, respiratory failure, pneumonia			
	Case 5: M, 66 y, CAD		Case 5: cardiopulmonary resuscitation, sudden cardiac death			
	Case 6: F, 54 y, dementia, epilepsy, trisomy 21		Case 6: basic supportive care, respiratory failure, aspiration pneumonia			
	Case 8: M, 82 y, Parkinson disease, type 2 DM, CAD		Case 8/9/10: basic supportive care, respiratory failure, viral pneumonia			
	Case 9: f, 87 y, Lung cancer, CAD, CRD					
	Case 10: M, 84 y, type 2 DM, HT, ulcerative colitis					
	Death (n=7)					
(17), USA, Boston, Prilutskiy <i>et al.</i> , <i>American Journal of Clinical Pathology</i> , doi: 10.1093/ajcp/aqaa124, Posted May 12 2020, Published 18 July 2020, limited autopsy (chest, abdomen), 2 patients	Case 2: M, 91 y; case 3: M, 72 y. comorbidities not mentioned, death (n=2)	Case 2: day 8; case 3: day 6	Progressive dyspnea. Severe ARDS. High fever, hyperferritinemia and hypertriglyceridemia. Case 2: treatment: HCQ/DOX/AZ; case 3: treatment: CRO/AZ/sariluma	ICH CD 163 to detect haemophagocytosis. ICH for human herpesvirus-8 (HHV-8), cytomegalovirus (CMV), and Epstein-Barr virus (EBV) by <i>in situ</i> hybridization for EBV small RNA (EBER): negative in lymph nodes with haemophagocytosis	Lungs: cases 2/3: acute exudative phase of DAD. Mediastinal and pulmonary hilar lymph nodes grossly enlarged containing multifocal clusters of hemophagocytic histiocytes localised in the subcapsular sinuses. Lymphohagocytosis was the predominant form of haemophagocytosis. Case 2: probable HLH syndrome. Case 3: follicular and interfollicular hyperplasia	Spleen (case 3): focal hemophagocytosis (areas of red pulp hemorrhage with admixed phagocytic histiocytes) and white pulp depletion
(7), Belgium, Brussels, Rimmelink <i>et al.</i> , medRxiv 2020;2020.05.27.20114363, posted May 28, 2020, full autopsy, 6 patients (cases 2, 7, 8, 11, 13, 14)	Case 2: F, 91 y, HT, CAD, liver cirrhosis, CRD	Case 2: day 15	Case 2: acute kidney injury, hypoxic hepatitis, ARDS and development of respiratory failure leading to death	Case 2: CT scan: negative; SARS-CoV-2 PCR: positive	Case 2: lungs: early DAD, microthrombi, emphysema, focal lymphoplasmocytic infiltrate, atypical pneumocytes	Cases 13, 14: perivascular chronic inflammatory infiltrate
	Case 7: M, 56 y, no comorbidities	Case 7: day 7	Case 7: respiratory failure	Case 7: CT scan: bilateral consolidation; SARS-CoV-2 PCR: positive	Case 7: lungs: early DAD, late DAD, lung infarct, acute BN, bilateral invasive aspergillosis	
	Case 8: M, 66 y, HT, CAD, DM, cerebrovascular disease, renal failure	Case 8: day 14	Case 8: acute kidney injury, septic shock and multiple organ failure	Case 8: CT scan: emphysema; SARS-CoV-2 PCR: positive	Case 8: lungs: early DAD damage, acute BN, interstitial fibrosis, emphysema	
	Case 11: M, 76 y, DM, liver cirrhosis, cancer	Case 11: day 5	Case 11: ARDS, sudden death	Case 11: CT scan: bilateral consolidation; SARS-CoV-2 PCR: positive	Case 11: lungs: early DAD, hyperplasia of pneumocytes type-II, syncytial multinucleated giant cells	
	Case 13: M, 73 y, DM	Case 13: day 10	Case 13: ARDS and respiratory failure	Case 13: CT scan: ground glass opacity and bilateral consolidation; SARS-CoV-2 PCR: positive	Case 13: lungs: early DAD, acute BN	
	Case 14: F, 77 y, HT, DM	Case 14: day 9	Case 14: acute kidney injury, hypoxic hepatitis, ARDS and respiratory failure	Case 14: CT scan: ground glass opacity and bilateral consolidation; SARS-CoV-2 PCR: positive	Case 14: lungs: no specific abnormalities; bilateral alveolar edema, bilateral aspiration pneumonia	
	Death (n=6)					
(15), Brazil, Sao Paulo, Duarte-Neto <i>et al.</i> , <i>Histopathology</i> 2020 Aug, doi: 10.1111/his.14160, epub 2020 Jul 24, ultrasound-guided MIA, 3 patients (in total 10 patients, 3 without MV). The clinical & other information not individualized is described here jointly for the 10 patients	Description of the 10 patients (n=2): 69 [33–83]. HT (n=5), DM (n=5), chronic cardiopathy (n=5), COPD (n=3), CRD (n=1), cancer (n=1). Death (n=3)	3 patients without MV: day 3–10	Description of the 10 patients: fever, dyspnoea (n=9), cough (6), diarrhoea, nausea/vomiting (n=2), myalgia, running nose, sore throat (n=1)	Description of the 10 patients: IHC: paucity of CD20+ B cells in all cases and no signs of lymphoid aggregates formation. T cell markers: CD4 and CD8 varied from scarce, especially in the cases with exudative DAD, to moderate, forming small aggregates in the patients with fibroproliferative DAD. CD57+NK cells: sparse in all cases and did not vary according to DAD patterns. CD68+ macrophages present mostly in the alveolar spaces and within areas of tissue remodelling in fibroproliferative areas. Some multinucleated atypical giant-cells were CD68+ alveolar macrophages	Definition of Exudative DAD: intense and diffuse alveolar exudates with hyaline membranes, septal edema, and mild/moderate lymphocytic infiltration. Intense pleomorphic changes on alveolar epithelial cells and also in the airways (giant cells) suggestive of virus cytopathic effects, with diffuse epithelial desquamation. Definition of proliferative DAD: poorly organised fibrous tissue within alveolar septa and alveolar lumen and was more prevalent in patients with long periods of illness and hospitalisation. Lung description in 3 not-MV patients: 3 DAD (2 exudative & proliferative; 1 exudative); 3 CE, 1 alveolar squamous metaplasia, 1 septal lymphocytic inflammation, 1 alveolar SM, 3 arteriolar microthrombi, 3 alveolar megakaryocytes, 2 alveolar haemorrhage, 3 suppurative pneumonia	Organs studied in the 10 patients: liver, heart, kidneys, spleen, brain, skin, skeletal muscle, and testis. Main systemic findings associated with comorbidities, age, and sepsis, in addition to possible tissue damage due to the viral infection. Findings attributed to shock: centrilobular congestion in the liver (10 cases) and acute tubular lesion (n=8 kidney analysed). Findings possibly due to the viral infection: dermatitis-superficial perivascular mononuclear infiltrate (n=8), myositis (n=2), orchitis (in all 2 testicle samples), mild lymphomononuclear myocarditis (n=2), endothelial changes in small vessels (cell tumefaction, vessel wall edema and fibrinoid alteration), small thrombi less frequent in glomeruli (n=6), spleen, heart (n=2), dermis (n=3), testis (n=2), and liver sinusoids (n=1)
(18), USA, New York, Barnes <i>et al.</i> , <i>J Exp Med</i> 2020, doi: 10.1084/jem.20200652, accepted: 13 April 2020, lung (limited) autopsy, 1 patient	M, 64 y, DM, end-stage renal disease on hemodialysis, heart failure and hepatitis C on ledipasvir/sofosbuvir therapy. Death (n=1)	Day 0 (not well specified). Autopsy performed within 5 h after death	He declined medical intervention, not intubated, died in the emergency room 5 h after presentation, shortly after developing fever. No sepsis. Treatment: ledipasvir/sofosbuvir therapy	Premortem cultures negative	Extensive neutrophil infiltration in pulmonary capillaries, with acute capillaritis with fibrin deposition, and extravasation into the alveolar space. Neutrophilic mucositis of the trachea	–

Table S1 (continued)

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Publication: reference number, country, city, publication date, type of sample, number of patients	Sex, age, risk factors/ comorbidities, outcome	Date histopathology lung after onset of symptoms	Clinical history	Ancillary methods	Main positive pathology lung findings	Findings in other organs
(8), USA, Washington, Lacy <i>et al.</i> , <i>Am J Forensic Med Pathol</i> 2020, doi: 10.1097/PAF.0000000000000567, accepted April 9, 2020, full forensic autopsy, 1 patient	F, 58 y, type 2 DM, obesity, hyperlipidemia, mild intermittent asthma, chronic lower extremity swelling with ulceration	Day 7	Fever & respiratory, difficulty, self-quarantine, found dead in her bedroom after seen alive the previous night. COD: viral pneumonia due to COVID-19	PM Dacron-tipped swabs in viral transport media from the right and left main bronchi: + PCR for SARS-CoV-2 and – testing for influenza. Bacterial cultures from abnormal lung areas (with consolidation) swabbed with amies medium: + for methicillin-sensitive <i>Staphylococcus aureus</i> and <i>Streptococcus viridans</i> . Given the lack of acute histologic inflammation in the lungs, these bacterial culture results were interpreted as being most likely contaminants or post-mortem artifact	Lung: edema and dense amphophilic concretions along alveolar septae consistent with HM. Lung architecture preserved, and septae of normal thickness, but with mild mononuclear infiltrates. Prominent desquamating pneumocyte hyperplasia with focal multinucleated cells and bizarre forms. Acute alveolar haemorrhage and collections of reactive foamy alveolar macrophages were focally present, as were collections of alveolar fibrin	Heart: myocyte hypertrophy with interstitial and perivascular fibrous tissue but no acute ischemic changes or inflammatory infiltrates. Liver: mild steatosis and central lobular pallor and congestion, but no significant portal or lobular inflammation. Kidney: arteriolosclerosis, mesangial sclerosis and hypercellularity, and focal global glomerulosclerosis. A section of medulla had no inflammatory or ischemic changes. An incidental adrenal cortical nodule and a focus of papillary adenocarcinoma of the thyroid
(9), Switzerland, Zurich, Schweitzer <i>et al.</i> , <i>Forensic Imaging</i> 2020, doi: 10.1016/j.fri.2020.200378, available online 18 April 2020, forensic autopsy, 1 patient	M, 50 y, HIV. Death (n=1)	Week 5	Absence of fever, absence of dyspnoea and of thoracic pains. In self-quarantine. Found dead at home, a day after his nasopharyngeal swab was positive for SARS-CoV-2, three days after the sample had been taken as an outpatient. The evening of this positivity called the hospital for racing heart. He didn't attend the hospital. After history revision: some mild symptoms stretching over around five weeks	PM CT: features of a severe acute respiratory distress syndrome	Lung: distorted septal and alveolar architecture: congested vessels & edematous fluid. HM. Patchy lymphocytic infiltrates, in part binuclear and trinuclear	Acute liver dystrophy and acute tubular necrosis in the kidneys were found. Coronary artery atherosclerosis
(10), Switzerland, Geneva, Aguiar <i>et al.</i> , <i>Int J Leg Med</i> 2020, doi: 10.1007/s00414-020-02318-9, accepted: 19 May 2020, full forensic autopsy, 1 patient	F, 31 y, morbid obesity. Death (n=1)	A few days (not specified)	Found dead in her flat during confinement. Cough during the previous days. High fever (rectal temperature of 41.4 °C, 2 h after death). Only symptomatic treatment (ibuprofen & opioid antitussive sirup) found at the death scene. COVID-19 diagnosis made after death (tracheobronchial swab)	PM CT: diffuse bilateral GGO and panlobar consolidations and air bronchograms. ICH: CD3-positive T cells and megakaryocytes. PM tracheobronchial swab: + SARS-CoV-2; negative for RV. Bacteriology: mixed flora	Lung (summary: DAD and interstitial pneumonia). HP: edema, early-DAD (heterogeneous pattern, mostly affecting the central part of the lungs) with HM, and focal areas of intra-alveolar haemorrhages and bacterial proliferation. Alveolar deposit of fibrin as cotton wools, and moderate type II pneumocytes hyperplasia (mainly desquamated). Moderate intra-alveolar macrophages and only scant PMN and lymphocytes. In the interstitium: edema, vascular stasis, lympho-monocytic infiltrates. Within the alveolar septa and mainly into the capillaries, abundant PMN, indicating margination and diapedesis. Bronchi and bronchioles only minimal lymphocytic infiltrates within their walls	Other organs: chronic tracheitis. Liver: microabscesses. Not cardiac hypertrophy. Death related to COVID-19 pulmonary alterations and high fever
(11), Germany, Tübingen, Bösmüller <i>et al.</i> , <i>Virchows Archiv</i> , doi: 10.1007/s00428-020-02881-x, published online: 30 June 2020, full autopsy, 1 patient	Case 1: F, 78 y, obesity, HT, cardiac pacemaker due to atrioventricular block. Death (n=1)	Case 1: day 1. Autopsy 48 h after death	Case 1: 12 h period of symptoms: fever, cough, vomiting. Days from admission until death: 1 (home care)	Significant levels of SARS-CoV-2 RNA in the lungs (qRT-PCR), but not in the livers and hearts. qRT-PCR of cytokines in lung tissue revealed a massive increase of IL-1beta and IL-6 mRNA	Case 1: generalized edema, the lower lobes showed florid capillary endotheliitis with increased neutrophils, formation of MT in alveolar capillaries, and small pulmonary vessels, including septal veins. In addition, focal inflammatory exudate with neutrophils and sparse HM with incipient organizing changes but without hyperplasia of alveolar epithelium. COD: early pneumonitis with thrombotic micro-angiopathy resulting in inflammation-associated pulmonary edema and acute cardiac failure	Case 1: liver: moderate acute congestion and activation of Kupffer cells but lacked inflammatory infiltrates
(12), Germany, Hamburg, Fitzek <i>et al.</i> , <i>Rechtsmedizin</i> , doi: 10.1007/s00194-020-00401-4, published online: 25 May 2020, autopsy (embalmed body), 1 patient	M, 59 y, slightly obesity, probably HT, cardiac hypertrophy and marked, cor adiposum (only identified by the autopsy)	Day 6	Infection during a journey to Egypt. Sudden flickering of his eyes. Chills and dizziness, coughing. GI symptoms. Dizziness, coughing and general exhaustion. Decompensation of the CV system. No medical documentation available, antibiotics, invasive treatment: only oxygen administration	CT scan: bilateral moderate pleural effusions and global multifocal reticular consolidation; subpleural milky glass opacities with ground-glass density nodules. Artifacts not ruled out due to embalming. PM detection of SARS-CoV-2 in lung, pharyngeal mucosa and pharyngeal swab	Lung: ubiquitous HM, vascular compressions and microthrombi. DAD. Protein-rich edema with low-grade lymphocyte infiltration	Trachea: haemorrhagic tracheobronchitis. Heart: congestive cardiomyopathy (heart weight of 600 g) a cor adiposum. GI: moderate inflammatory cells in the intestinal wall
(21), Italy, Rome, Pernazza <i>et al.</i> , <i>Virchows Archives</i> 2020, doi: 10.1007/s00428-020-02829-1, 26 Apr 2020, biopsy (lung tumor excision), 1 patient	M, 61 y, smoking. Full remission of a MALT lymphoma. Lung adenocarcinoma. Alive (n=1)	Day 0	Asymptomatic, afebrile. Elective lobectomy for lung adenocarcinoma. After surgery: cough, dyspnea, fatigue, and high fever. Treatment: meropenem and Bactrim, antiviral drugs: acyclovir, darunavir, tocilizumab; HCLOR. Supplemental oxygen through a CPAP mask	Pharyngeal swab positive for SARS-CoV-2 on PCR (during life)	Lung: early changes in parenchyma surrounding the neoplasia: diffuse hemorrhages, clusters of alveolar macrophages, occasional multinucleated cells, loss and reactive pneumocyte hyperplasia, with nuclear inclusions. No HM or DAD. The interstitium showed edema & mild inflammatory infiltrate mainly composed of cytotoxic (CD8+) T lymphocytes. Occasional fibrous and mild fibrous thickening of subpleural alveolar septa. Scanty fibrin depositions on the alveolar surfaces	Smoking-related interstitial fibrosis
(19), China, Beijing, Xu <i>et al.</i> , <i>Lancet Respir Med</i> 2020, doi: 10.1016/S2213-2600(20)30076, 8 Apr 2020, post-mortem biopsies, 1 patient	M, 50 y, travel history to Wuhan	Day 14	Fever, chills, dried cough, fatigue, and shortness of breath 8 days before admission. He refused ventilator support in the ICU repeatedly because of claustrophobia. Therefore, he received high-flow nasal cannula (HFNC) oxygen therapy (60% concentration, flow rate 40 L/min). On day 13 of illness, symptoms had still not improved, but oxygen saturation remained above 95%. The day 14 of illness, his hypoxaemia and shortness of breath worsened. Despite receiving HFNC oxygen therapy (100% concentration, flow rate 40 L/min), oxygen saturation decreased to 60%, and he had sudden cardiac arrest with an. Rescue unsuccessful rescue. Treatment: interferon alfa-2b lopinavir + ritonavir, moxifloxacin, methylprednisolone	SARS-CoV-2 PCR +: day 9 of illness. Flow cytometry peripheral CD4 & CD8 T cells substantially reduced but hyperactivated. High proportions of HLA-DR (CD4 3-47%) and CD38 (CD8 39-4%) double-positive fractions. Increased concentration of highly pro-inflammatory CCR6+ Th17 in CD4 T cells and high cytotoxicity of CD8 T cells	Lungs: bilateral DAD (exudative phase), cellular fibromyxoid exudates, desquamation of pneumocytes & HM, indicating acute respiratory distress syndrome. Pulmonary edema. Interstitial lymphocytic infiltrates and multinucleated syncytial cells with atypical enlarged pneumocytes (with large nuclei, amphophilic granular cytoplasm, and prominent nucleoli) in the intra-alveolar spaces, showing viral cytopathic-like changes. No intranuclear or intracytoplasmic viral inclusions	Liver: moderate microvesicular steatosis and mild lobular and portal activity due to primary or secondary drug effect. Heart: few interstitial mononuclear inflammatory infiltrates
(13), China, Chongqing, Yao <i>et al.</i> , <i>Cell Research</i> 2020, doi: 10.1038/s41422-020-0318-5, published online: 28 April 2020, autopsy, 1 patient	F, 78 y, no comorbidities except contact with COVID-19 patient 2 days before admission. Death (n=1)	Day 16	Admitted to hospital due to falling-resulted trauma. Exposed to a COVID-19 patient 2 days before. Two days after admission, the patient showed pneumonia symptoms. Four days after the onset of illness: + PCR SARS-CoV-2 confirmation (NP swab). Day 5: chest CT scan: multiple patchy shadows in both lungs, implying pulmonary infection. From day 10th to 12th: 3 negative NP PCR tests. Day 15: chest CT: relief. Day 16 after the onset: When she was ready for discharge, she fell suddenly into fatal condition with cardiac arrest, and died. Leukopenia. Treatment: lopinavir, interferon alfa-1b, Ritonavir. From the 6 th day after the onset of symptoms: methylprednisolone & oxygen supplementation (nasal cannula, 5 L/min) (10 days of treatment)	Digital PCR on tissue sections (lung, liver, heart, intestine, and skin): only positive SARS-CoV-2 in the lung. IHC staining (monoclonal antibody against SARS-CoV-2 nucleocapsid): + only in lung. EM: coronavirus particles in both bronchiolar epithelial cells marked by cilia and type II alveolar epithelial cells (type II AE) featured with lamellar body. ICH staining showed that the cell types of infiltrated immune cells in alveolar space and septa were predominantly infiltrating CD68+ macrophages, CD20+ B cells, and CD8+ T cells	Predominant DAD (extensive desquamation of proliferative type II AE, exudation of fibrin, monocytes, and macrophages). Some of alveolar walls were partially lined by low columnar type II AE and covered by the formation of HM in alveolar space. Thickening of alveolar septa with scattered interstitial inflammatory infiltration and hyaline thrombus in microvessels, but no pulmonary edema	Chronic respiratory disease associated changes in the lung tissues
(14), Spain, Alicante, Muñoz-Quirós <i>et al.</i> , <i>Gaceta Intern Cienc For</i> 2021, forensic autopsy, 1 patient	F, 68 y, liver cirrhosis. Death (n=1)	Day 2	Found at home death. Her husband was COVID-19 +. She refused any analysis. Sudden death		Perivascular interstitial pneumonia with HM lining alveolar spaces, few septal capillary microthrombi capillaries with the presence of lymphocytes, and scarce polymorphonuclear and macrophages with foamy appearance. Areas of type II pneumocyte hyperplasia and areas of intra-alveolar fibrin organization. Bilateral pleural and septal fibrosis, with focal chronic inflammatory infiltrate and associated areas of alveolar hyperdistension. Left lung: areas of exudates of neutrophilic polynuclear cells overlapping alveolar spaces and bronchial lumens with a basal predominance. Diagnosis: interstitial pneumonia with bilateral diffuse alveolar damage and added left pulmonary acute bronchopneumonia	Heart: moderate anterior ventricular adipose infiltration. Liver cirrhosis with marked inflammatory activity

This table includes not mechanical ventilated patients as well as those under CPAP, HFNC, oxygen administration and oxygen supplementation. AE, alveolar epithelia; ARDS, acute respiratory distress, syndrome; AZ, azithromycin; BN, bronchopneumonia; CAD, coronary artery disease; CT, computer tomography, CPAP, continuous positive airway pressure; CRD, chronic renal disease; CRO, ceftriaxone; CVD, cardiovascular disease; DAB, diamino benzidine; DAD, diffuse alveolar damage; DM, diabetes mellitus; DOX, doxycycline; F, female; HM, hyaline membranes; HT, hypertension; IHC, immunohistochemistry; ICU, intensive care unit; GGO, ground-glass opacity; M, male; MIA, minimally invasive autopsy; MV, mechanical ventilation; PCR, polymerase chain reaction; RV, respiratory virus; Y, years.

Table S2 Histopathological findings in lung sections from COVID-19 patients on MV longer than 24 hours (6,7,11,15-17,22-28)

Publication: reference number, country, city, publication date type of sample, number of patients	Sex, age, risk factors/ comorbidities outcome	Date histopathology lung after onset of symptoms	Clinical history	Ancillary methods	Main positive pathology lung findings	Findings in other organs
(16), USA, New York, Magro <i>et al.</i> , <i>Transl Res</i> 2020 Apr 15. doi: 10.1016/j.trsl.2020.04.007, 09 Apr 2020, limited autopsies, 1 patient	Case 2: M, 73 y, smoker, obesity, pre-DM. Death (N=1)	Case 2: day 5	Respiratory failure (n=5) and purpuric skin rash (n=3). Case 2: evaluated at the ED by respiratory distress; fever, tachypnea, severely hypoxemic, required emergent endotracheal intubation. On day 4 th of MV developed thrombocytopenia and severe hypercapnia. MV 5 weeks	Staining for SARS-CoV-2 spike & envelope proteins. (IHC) assessment for the deposition of C5b-9, C3d, and C4d via DAB technique. Significant vascular deposits of C5b-9 and C4d. Co-localization of COVID-19 spike glycoproteins with C4d and C5b-9 in the inter-alveolar septa and the cutaneous microvasculature. MASP2 staining demonstrated granular and punctate staining localized to the inter-alveolar septa. No other potential pulmonary viral, bacterial nor fungal pathogens	Lungs case 2: pauci inflammatory hemorrhagic pneumonitis, septal capillary injury (activated complement deposits), septal capillary mural and luminal fibrin deposition and permeation of the inter-alveolar septa by neutrophils. Intra-alveolar fibrin deposition. Focal HM formation and type II pneumocyte hyperplasia	
(22), Switzerland, Zurich, Varga <i>et al.</i> , <i>Lancet</i> , doi: 10.1016/S0140-6736(20)30937-5, 17 Apr 2020, full autopsy, 2 patients	Case 1: M, 71 y, CAD, renal transplant recipient, HT. Death (n=1)	Day 8	Dyspnea, fever, tachycardia, hypotension, and confusion, HT. Treatment: MV (8 days), piperacillin/tazobactam & UFH	In all cases: IHC: caspase 3	Lungs: severe DAD, mononuclear cells and neutrophils in lungs. Thickened lung septa, including a large arterial vessel with mononuclear and neutrophilic infiltration. Other organs: Prominent endohepatitis with recruitment of inflammatory cells, apoptotic bodies in many organs, especially in the pulmonary vessels but also in small bowel and heart. Virus within endothelial cells	EM analysis revealed viral inclusion structures in endothelial cells of the transplanted kidney (glomerula capillary loops)
	Case 2: F, 58 y, type 2-DM, HT, obesity. Death (n=1)	Day 19 (16 days after admission)	Cough, fever, and dyspnea for 3 days at home. Admitted directly to ICU due to progressive respiratory failure. Developed (within the first week) multi-organ failure, requiring MV renal & replacement therapy. On day 16, mesenteric ischemia prompted removal of necrotic small intestine. Circulatory failure occurred in the setting of right heart failure consequent to an ST-segment elevation myocardial infarction, and cardiac arrest resulted in death (8 days after admission). Treatment: MV (lag >8). HCL, empiric antibiotic treatment UHF		Lung: DAD, mononuclear cells, lymphocytic endohepatitis (also in heart, kidney, and liver)	Liver: necrosis. Heart: myocardial infarction but no sign of lymphocytic myocarditis. Small intestine: endohepatitis and many apoptotic bodies of the submucosal vessels with only scattered fibrin thrombi
(28), China, Zhang <i>et al.</i> , <i>Ann Int Med</i> 2020;172:629-32, doi: 10.7326/M20-0533, transthoracic needle biopsy, 1 patient	M, 72 y, DM, HT. Death (n=1)	Not mentioned	Cough and fever. Rapidly progressive respiratory failure requiring endotracheal intubation and MV (1 week after presentation)	Transthoracic lung biopsy: IHC with antibody to the Rp3 NP protein of SARS-CoV-2 revealed prominent expression on alveolar epithelial cells. In contrast, viral protein expression was minimally detectable on blood vessels or in the interstitial areas between alveoli	Lung biopsy: DAD (organizing phase), reactive type II pneumocyte hyperplasia, intra-alveolar fibrinous exudates, loose interstitial fibrosis, and chronic inflammatory infiltrates. Intra-alveolar loose fibrous plugs of organizing pneumonia, with presence of intra-alveolar organizing fibrin in most foci	
(6), Germany, Hamburg, Wichmann <i>et al.</i> , <i>Annals Intern Medicine</i> 2020, doi: 10.7326/M20-2003, 6 May 2020, full autopsy, 5 patients with MV	Case 3: M, 71 y, HT, smoker, granulomatous pneumopathy	Not mentioned. PMI 4 days	Case 3: respiratory failure, pneumoniae		Lungs. 5 patients with DAD: 5 activated pneumocytes, 2 fibroblasts, 1 fibrosis, 5 HM, 1 GC, 3 with SM, 1 LC and 3 with thrombi. Additional findings: 2 haemorrhagic infarctions, 2 emphysema, 2 congestion of small vessels, granulocytic infiltration, 1 plasma cells	In the 4 cases: pharynx normal and veins thrombosis; in 2 of them: thrombosis in prostate. Left cardiac dilatation, calcification of the mitral ring, cardiac pacemaker, atherosclerosis
	Case 4: M, 63 y, type 2 DM, obesity, bronchial asthma		Case 4: cardiorespiratory failure, pulmonary emboli			
	Case 7: F, 75 y, atrial fibrillation, CAD, smoker		Case 7: respiratory failure, viral pneumonia			
	Case 11: M, 85 y, CAD, HT, bronchial asthma, atrial fibrillation		Case 11: cardiac arrest, respiratory failure			
(17), USA, Boston, Prilutskiy <i>et al.</i> , <i>Am J Clin Path</i> , doi: 10.1093/ajcp/aqaa124, posted May 12 2020, published 18 July 2020, limited autopsy (chest, abdomen), 2 patients	Comorbidities not mentioned. Case 1: M, 72 y; case 4: F, 64 y. Death (n=2)	Case 1: day 18; case 4: day 15	Progressive dyspnea. Severe ARDS. High fever, hyperferritinaemia and hypertriglyceridemia. Case 1: HCL, AZ, anakinra, intubation time: from 7 d to 18 (11 days on MV). Case 4: sarilumab, CRO. MV: from day 12 to 15	ICH CD 163 to detect haemophagocytosis. ICH for human herpesvirus-8 (HHV-8), cytomegalovirus, (CMV), and Epstein-Barr virus (EBV) by <i>in situ</i> hybridization for EBV small RNA (EBER): negative in lymph nodes with haemophagocytosis	Lung: (cases 1, 4): acute exudative phase of DAD. Case 1: mediastinal and pulmonary hilar lymph nodes grossly enlarged with multifocal clusters of hemophagocytic histiocytes. Marked distention of cortical and subcortical sinuses with focal necrosis as well as lymphocyte depletion. Lymphophagocytosis was the predominant form of haemophagocytosis. Definite HLH syndrome. Case 4: no haemophagocytosis	
	Case 12: M, 76 y, obesity	Death (n=5)	Case 12: pulmonary emboli	Treatment: catecholamine therapy 1 patient; AB + AC (n=1); AB (n=1); AC (n=1); 1 none. Lag of stay on MV: not mentioned. NIV (method not specified)		
(7), Belgium, Brussels, Rimmelink <i>et al.</i> , posted May 28, 2020, doi: 10.1101/2020.05.27.20114363, full autopsy, 11 patients	Case 1: M, 77 y, CAD, cerebrovascular disease, DM	Median time from admission to death was 13 days. Case 1: day 3	Case 1: ARDS, acute kidney injury, multiple organ failure, cardiogenic shock	8 patients with viral presence in all tested organs (lung, heart, spleen, liver, colon, kidney and brain). Case 1: CT thorax: negative; SARS-CoV-2 PCR: positive	Case 1: lung: early DAD, lung microthrombi, acute BN, atypical pneumocytes	No specific SARS-CoV-2 lesions were observed in any organ using RT-PCR, SARS-CoV-2 could be detected in all organs, even those without evident microscopic lesions. As some patients died outside the ICU without undergoing mechanical ventilation, we could not estimate lung compliance before death
	Case 3: M, 68 y, cancer, COPD	Case 3: day 15	Case 3: ARDS, acute kidney injury, respiratory failure	Case 3: CT thorax: GGO; SARS-CoV-2 PCR: positive	Case 3: lung: early DAD, lung microthrombi, emphysema	
	Case 4: F, 64 y, HT, cerebrovascular disease, cancer	Case 4: day 8	Case 4: ARDS, respiratory failure	Case 4: CT thorax: minor abnormalities; SARS-CoV-2 PCR: positive	Case 4: lung: early DAD, lung microthrombi, emphysema, atypical pneumocytes	
	Case 5: M, 56 y, COPD, cancer	Case 5: day 14	Case 5: ARDS, acute kidney injury, hypoxic hepatitis, multiple organ failure, mesenteric ischemia. ECMO	Case 5: CT thorax: GGO; SARS-CoV-2 PCR: positive	Case 5: lung: early DAD, lung microthrombi, lung infarct, acute BN	
	Case 6: M, 73 y, HT, CRD	Case 6: day 11	Case 6: ARDS, acute kidney injury, respiratory failure ECMO	Case 6: CT thorax: bilateral consolidation; SARS-CoV-2 PCR: positive	Case 6: lung: early DAD, late DAD, lung microthrombi, acute BN, atypical pneumocytes	
	Case 9: F, 49 y, HT, DM	Case 9: day 17	Case 9: ARDS, acute kidney injury, respiratory failure	Case 9: CT thorax: GGO; SARS-CoV-2 PCR: positive	Case 9: lung: early DAD, lung microthrombi, late DAD	
	Case 10: M, 63 y, HT, DM	Case 10: day 16	Case 10: ARDS, acute kidney injury, respiratory failure. ECMO	Case 10: CT thorax: GGO; bilateral consolidation, SARS-CoV-2 PCR: positive	Case 10: lung: early DAD, lung microthrombi, hyperplasia of pneumocytes type-II, syncytial multinucleated giant cells	
	Case 12: M, 75 y, HT, CAD, DM	Case 12: day 5	Case 12: ARDS, acute kidney injury, hypoxic hepatitis, multiple organ failure	Case 12: CT thorax: GGO; SARS-CoV-2 PCR: positive	Case 12: lung: early DAD, lung microthrombi, acute BN, late DAD, syncytial multinucleated giant cells	
	Case 15: M, 61 y, no comorbidities	Case 15: day 31	Case 15: ARDS, acute kidney injury, pulmonary embolism, multiple organ failure, septic shock	Case 15: CT thorax: GGO; lobar pneumonia, SARS-CoV-2 PCR: positive	Case 15: lung: early DAD, lung microthrombi, lung infarct, late DAD	
	Case 16: F, 70 y, HT, DM, liver tx	Case 16: day 19	Case 16: ARDS, acute kidney injury, pulmonary embolism, multiple organ failure, septic shock	Case 16: CT thorax: GGO; bilateral consolidation, SARS-CoV-2 PCR: positive	Case 16: lung: early DAD, lung microthrombi, late DAD, hyperplasia of pneumocytes type-II	
	Case 17: M, 53 y, HT, cerebrovascular disease	Case 17: day 13	Case 17: ARDS, acute kidney injury, pulmonary embolism, multiple organ failure, septic shock. ECMO	Case 17: CT thorax: GGO bilateral consolidation, lobar pneumonia; SARS-CoV-2 PCR: positive	Case 17: lung: acute BN, late DAD	
Death (n=11)						
(15), Brazil, Sao Paulo, Duarte-Neto <i>et al.</i> , <i>Histopathology</i> 2020 Aug. doi: 10.1111/his.14160, epub 2020 Jul 24, ultrasound-guided MIA, 7 patients MV (of a total of 10 patients)	Description of the 10 patients (n=2): 69 [33-83], HT (n=5), DM (n=5), chronic cardiopathy (n=5), COPD (n=3), CRD (n=1), cancer (n=1). Death (n=7)	7 patients: ICU with MV. Onset symptoms-death: day 5-16. Three patients died within 24 hours of hospitalisation	Information in Table 1	Information in Table 1	Definitions in Table 1. Lungs: 7 patients: 6 with exudative/proliferative DAD; 1 exudative DAD. 7 cytopathic effects, 5 alveolar squamous metaplasia, 7 septal lymphocytic inflammation, 5 arteriolar MT, 1 with a high density of alveolar megakaryocytes, 1 alveolar haemorrhage, 3 suppurative pneumonia. 5 alveolar SM	Information in Table 1

Table S2 (continued)

Table S2 (*continued*)

Publication: reference number, country, city, publication date type of sample, number of patients	Sex, age, risk factors/ comorbidities outcome	Date histopathology lung after onset of symptoms	Clinical history	Ancillary methods	Main positive pathology lung findings	Findings in other organs
(11), Germany, Tübingen, Bösmüller <i>et al.</i> , <i>Virchows Archiv</i> , doi: 10.1007/s00428-020-02881-x, published online: 30 June 2020, full autopsy, 3 patients (1 one of them on ECMO)	Case 2: M, 79 y, CAD, HT, type 2-DM, obesity and Parkinson's disease	Case 2: 3 weeks. Autopsy within 24 after death	Case 2: general weakness for 3 weeks, fever, & dry cough; worsening symptoms during the last 3 days before admission. Intubated due to respiratory failure. Neg. blood cultures for bacteria or fungi. Despite improvement in the next 2 days, then, IL-6 and D-dimer concentrations peaked, whereas thrombocytopenia worsened despite AC. Within 24 h, multi-organ failure & vasoplegic shock and death (4 days after peak of D-dimer concentration). Treatment: vasopressor, ORT & AC. 8 days MV (days in ICU). Days from admission until death: 9	EM: in both patients: viral particles in pulmonary endothelial cells; in patient 2: also in pneumocytes type 1. In patient 3: also in vacuoles within the interstitial space IL-1 beta and IL-6 mRNA were not increased in lung tissue in any patient. All patients: significant levels of SARS-CoV-2 RNA in the lungs (qRT-PCR), but not in the livers and hearts (in fresh samples-unfixed)	Case 2: edema. DAD with extensive intra-alveolar fibrin deposits with formation of HM, marked hyperplasia and desquamation of alveolar epithelium, and accumulation of macrophages with frequent multinuclear GC. Areas of organized DAD especially in lower lobes, with proliferation of fibroblasts and early collagen fibre deposits within the intra-alveolar exudate. Macroscopically visible thrombi mainly in small to medium-sized pulmonary vessels, both arteries and veins. Focally massive accumulation of leukocytes in medium-sized vessels, but florid neutrophilic capillaritis was absent	Case 2: liver: significant activation of macrophages with signs of, but no necrosis or inflammatory infiltrates
	Case 3: M, 72 y, CAD, HT, Merkel cell carcinoma under radiotherapy, obesity, polymyalgia rheumatica	Case 3: no specification of the lag from onset until death. Autopsy within 24 after death	Case 3: syncope, fever, cough, and emesis. Transferred to the ICU and intubated 4 days after admission with worsening respiratory symptoms and recurrent fever. Despite improving of oxygenation, the day 6 after ICU admission, acute hypercapnia & pulmonary superinfection with <i>Klebsiella oxytoca</i> . Treatment: meropenem therapy was started. Renal and liver failure, ORT (renal) was initiated, but he died 10 days after admission to ICU due to liver failure. Days from admission until death: 16; 11 days with MV		Case 3: macroscopically identifiable thrombi in pulmonary vessels. Advanced DAD, with extensive HM and intra-alveolar macrophage accumulations with multiple GC and pronounced, in part atypical hyperplasia of alveolar epithelium with focal squamous metaplasia and areas of organizing pneumonia. Neutrophils were infrequent arguing against bacterial superinfection	Case 3: no HP description of other organs
	Patient 4: M, 59 y, HT, intrinsic asthma, obesity	Case 4: day 14. Autopsy within 24 after death	Case 4: respiratory symptoms 2 weeks prior admission. Admitted to the ICU of a peripheral hospital and transferred to a big hospital 6 days later for his respiratory failure and started on ECMO. Dialysis. Two episodes with marked increase in D-dimer levels, the second 4 days before his death. 35 days in the ICU. 24 days on ECMO. He expired due to terminal ARDS and multi-organ failure 6 weeks since the beginning of his symptoms. Days at ICU: 35 (24 on ECMO). Days from admission until death: 35		Case 4: longstanding ARDS in organizing stage with extensive fibrinous exudates, diffuse thickening of alveolar septae, massive hyperplasia of alveolar and bronchial epithelium with focal squamous metaplasia, and typical concentrically layered plugs of loose connective tissue with concentrically arranged fibroblasts and central accumulation of inflammatory cells (lymphocytes) filling alveolar spaces	Case 4: cardiomegaly; signs of liver damage; intestinal mucositis and haemorrhage
(23), USA, Michigan, Ann Arbor, Konopka <i>Chest</i> 2020;158:e99-e101.28, doi: 10.1016/j.chest.2020.04.032, Published online 2020 Apr, autopsy, 1 patient	Death (n=3) M, 37 y, type-2 DM, asthma. Death (1)	Day 9	Admitted to hospital after 1-day history of fever, non-productive cough, and myalgias. On admission: CT chest with multifocal GGO. Progressive hypoxemia and ARDS. Treatment: Intubation and MV on hospital day 4. HCL, piperacillin/tazobactam, vancomycin, CRR. Lag MV: 6		Lung: DAD in distal alveolated lung tissue, with patchy mild interstitial thickening by edema, focal pneumocyte hyperplasia, and scattered HM. Rare fibrin thrombi within small vessels and a small muscular pulmonary artery (endothelial injury). This was accompanied by a mild patchy fibrinous airspace exudate in which mononuclear inflammatory cells predominated with scattered neutrophils. The inflammatory infiltrate was limited to distal airspaces without involvement of bronchi or bronchioles (probably early BN). Proximal airways: paucicellular mucus plugs, but without tissue eosinophilia	Findings probably due to the patient's history of asthma: Goblet cell metaplasia, mucus gland hyperplasia, and thickening of subepithelial basement membranes in cartilaginous and non-cartilaginous airways (mucus plugs) no evidence of hyperinflation/air trapping, the anticipated finding in patients who die of status asthmaticus
(26), USA, Texas, San Antonio, Yan <i>et al.</i> , <i>Arch Pathol Lab Med</i> 2020, doi: 10.5858/arpa.2020-0217-SA, "Minimalistic" (limited) autopsy (brain not extracted, except the heart, organs biopsied <i>in situ</i>), 1 patient	F, 44 y, obesity. Additional history: probable undiagnosed immunological disorder such as systemic lupus erythematosus. Death (n=1)	Day 13	One week history of fever, cough & dyspnoea. Invasive MV with endotracheal intubation due to worsening hypoxia, ARDS, severe multi-organ failure. Treatment: HCL, azithromycin, tozilizumab (one dose) (6 days of hospitalization and MV). Initial 12-lead ECG: sinus tachycardia along with slow R wave progression from V2 and V3; no ST-segment elevation. Transthoracic echocardiogram: severe septal, mid and mid-inferior hypokinesis; apical and infero-lateral wall motion was preserved. Mildly to moderately depressed left ventricular systolic function with an estimated left ventricular ejection fraction of 40–45%. Dx: reverse Takotsubo cardiomyopathy with clinical suspicion of viral myocarditis	Chest Rx: patchy bilateral airspace peripheral opacities that progressively worsened over the course of her admission. EM lung: structures consistent with viral capsids. Positive ANA result though with a very low titre of 1:40	Lung: extensive and markedly severe acute lung injury consistent with viral pneumonia (diffuse interstitial lymphocytic infiltrates and fibrinous exudates, DAD with HM, pulmonary infarction, severe edema, extensive desquamation of pneumocytes with intra-alveolar aggregation (resembling multinucleated giant cells), and pneumocyte morphological alterations suspicious for viral cytopathic, though marked reactive pneumocyte hyperplasia couldn't be entirely excluded. No microthrombi in lungs. Pulmonary blood vessels: extensive and widespread perivascular lymphocytic cuffing with a few foci of lymphocytic infiltration within vessel walls without fibrinoid necrosis, consistent with non-necrotizing lymphocytic vasculitis	Heart: myxoid edema, mild myocyte hypertrophy, and focal nuclear pyknosis. Notably, rare foci with few scattered CD45+ lymphocytes were identified in the left ventricular papillary muscle, though no definitive findings of acute or chronic myocyte necrosis. Kidney: unremarkable
(27), China, Jiangsu, Chen <i>et al.</i> , <i>Chin Med J</i> , 10.1097/CM9.0000000000000839, biopsy of lung explant (patient alive), 2 patients (from a total of 3 patients)*. *, a third case not included here as HP findings not detailed in the paper	Case 1 (also described at Luo WR): M, 66 y, HT. Case 2: M, 58 y, HBV infection. Alive (case 2), death (case 1)	Case 1: day 42; case 2: day 37 (until LT)	Post-COVID-19 patients with pulmonary fibrosis-related ARDS leading to an irreversible pulmonary injury undergoing LT. Pre-LT chest imaging confirmed pulmonary consolidation with fibrotic change. Anti-viral drugs. Tracheostomies previously performed time under MV pre-LT: 27 & 22 days. ECMO days pre-LT: 15 & 7. Case 1: high fever and cough after coming back from Wuhan on January 4, 2020. He developed respiratory failure and septic shock during the treatment and was done with transplant. Case 2: survival	Case 1: virus-negative in: nasopharynx, BAL, and sputum. Case 2: nasopharynx BAL, sputum, and serum. Explanted lung virology. Mild positive in both patients	Case 1: parenchyma: extensive pulmonary interstitial fibrosis with hyaline degeneration. Intrapulmonary vessels: occluded vessel lumen with microthrombosis (vascular and fibrotic patterns). Interstitial infiltration of inflammatory cells including lymphocytes, plasma cells and mononuclear cells (focal monocytes). Alveolitis with edema, proliferation, atrophy, desquamation and squamous metaplasia of epithelial cells (mainly type II). Atrophy, vacuolar degeneration, proliferation, multinucleate giant cells and intracytoplasmic viral inclusion bodies. Case 2: parenchyma: extensive pulmonary interstitial fibrosis and alveolar haemorrhage. Intrapulmonary vessels: Intravascular organized thrombosis and vasculitis (vascular & fibrotic patterns)	
(24), China, Beijing, Shao <i>et al.</i> , <i>Human Pathol</i> 2020, doi: 10.1016/j.humpath.2020.04.015, available online 11 May 2020, autopsy, 1 patient, 65 y, no comorbidity, visited Wuhan 8 days ago. Death (n=1)	Day 21	Fever (38.6 °C) and dry cough since 4 days. On day 9, after admission, the chest computed tomography scan showed diffuse GGO in the patient's bilateral lungs. On day 11, worsening of respiratory symptoms & diagnosis of type I respiratory failure, coinciding with kidney injury, and type II respiratory failure occurred, coupled with multiorgan failure including the heart and liver. Day 12: intubation & ventilation support (9 days on MV). He died on day 21 with the diagnosis of septic shock. Treatment: methyl prednisolone, biapenem	On admission: NSP swab + SARS-CoV-2 tests were negative since day 13. IHC in lung for SARS-CoV-2 N protein negative. PAS, GMS, and CMV IHC staining was negative, excluding secondary fungi or CMV infection	Lung: DAD in the early organizing phase with focal HM, intra-alveolar edema, reactive type II pneumocyte hyperplasia, and focal intra-alveolar fibrosis. Extensive acute alveolitis with numerous intra-alveolar neutrophils, lymphocyte, and macrophage infiltrations. Multinucleated giant cells. Microthrombi in the dilated pulmonary capillaries. Purulent discharge in most of the areas of the alveolar spaces (probably due to a secondary bacterial infection, but, unfortunately, respiratory and blood cultures for bacteria and other organisms were not performed		
(25), Spain, Valencia, Navarro Conde <i>et al.</i> , <i>Rev Esp Patol</i> 2020;53:188-92, doi: 10.1016/j.patol.2020.04.002, accepted: 29 April 2020, available online 11 May 2020, autopsy, 1 patient	M, 69 y, non-invasive urothelial carcinoma of the bladder. Death (n=1)	Undetermined time with symptoms (but with a previous visit to an ED) previous admission. 2 days from admission to death	Fever (38 °C), dyspnoea, cough and hypoxia without acidosis. Previous diagnosis: common cold & discharged with symptomatic treatment. Antibiotic treatment with levofloxacin and ceftriaxone. Non-invasive MV up to 90% FiO ₂ since admission. Poor evolution, he was transferred to the ICU 2 days after admission, where he died four hours later in shock with renal failure	Chest-X-ray: bilateral interstitial infiltrate with a ground glass appearance involving the inferior lobes. CT angiogram: no pulmonary thromboembolism. COVID-19 diagnosis confirmed by RT-PCR assay on a throat swab sample taken during the patient's admission. The positive result was reported fifteen days subsequent to autopsy. PCR respiratory viruses: negative. IHC neg for: herpes simplex virus, cytomegalovirus and Epstein-Barr virus	Lung: pathological changes in 80% of the parenchyma. Large areas of DAD: edema and intra-alveolar haemorrhage, desquamation of type II pneumocytes and HM & thrombi in the medium sized vessels. Abundant intra-alveolar macrophages and occasional multinucleated giant cells. Vesicular nuclei with prominent nucleoli suggestive of viral cytopathic involvement in both pneumocytes and macrophages. Cells with large, hyperchromatic nuclei, similar to smudge cells described in adenovirus related pneumonitis. No intra-alveolar organized fibrin similar to that found in acute pneumonia was seen. 30% of the pulmonary parenchyma findings were consistent with the proliferative phase of DAD. Pneumocytic hyperplasia and myofibroblastic proliferations contributed to widening of alveolar septa. No collagen fibrosis; squamous pneumocytic metaplasia was evident. The inflammatory component consisted of a mild lymphoid infiltrate with abundant macrophages. Areas of emphysema	