Supplemental Table S1. Search strategy

PubMed
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WHO Covid-19 database
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BioArxiv
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((biopsy OR autopsy OR pathology OR cytopathology OR histopathology) AND (2019ncov OR "2019 ncov" OR "sars cov 2"))
((biopsy OR autopsy OR pathology OR cytopathology OR histopathology) AND (COVID2019 OR "COVID 2019" OR sars2 OR "ncov 2019"))

MedArxive
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MEDLINE via OVID
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Limit 1 to COVID-19

PubMed Central (PMC)
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word] OR SARS-COV*[title word] OR SARSCOV*[title word] OR 2019ncov[title word] OR "2019 ncov"[title word] OR novel coronavirus*[title word] OR novel corona virus*[title word] OR ((coronavirus*[title word] OR corona virus*[title word] OR pneumonia virus*[title word] OR cov[title word] OR ncov[title word]) AND (outbreak[title word] OR wuhan[title word] OR "new"[title word])) OR covid19[title word] OR "covid 19"[title word] OR ((coronavirus*[title word] OR corona virus*[title word]) AND 2019[title word]) OR "sars cov 2"[title word] OR sars2[title word] OR new coronavirus*[title word] OR new corona virus*[title word] OR "ncov 2019"[title word] OR "sars coronavirus 2"[title word] OR "sars corona virus 2"[title word] OR "severe acute respiratory syndrome cov 2"[title word] OR "severe acute respiratory syndrome cov2"[title word]) AND ("2019/09/01"[PDAT] : "3000/12/31"[PDAT]))

Google Scholar

biopsy|autopsy|pathology|cytopathology|histopathology "COVID-19"|COVID19|2019ncov|"2019 ncov"|"sars cov 2"

biopsy|autopsy|pathology|cytopathology|histopathology COVID2019|"COVID 2019"|sars2|"ncov 2019"

covid19|"covid 19"|2019ncov|"2019 ncov|cov|coronavirus"|"2019 novel|new coronavirus|cov"| "wuhan coronavirus|cov|ncov|outbreak"| "wuhan*coronavirus|cov|ncov|outbreak"| "wuhan**coronavirus|cov|ncov|outbreak"|"coronavirus|cov|ncov*wuhan"

Embase

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Web of Science
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Academic Search Premier
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virus 2" OR "severe acute respiratory syndrome cov 2" OR "severe acute respiratory syndrome cov2" OR "COVID-19" OR "COVID19" OR "COVID2019" OR "COVID 2019" OR "severe acute respiratory syndrome coronavirus 2" OR "SARS-COV*" OR SARSCOV*)) OR (TX("Biopsy" OR "biopsy" OR "biopsies" OR "biopsie" OR "Autopsy" OR autops* OR obduct*) AND SU("COVID-19" OR "severe acute respiratory syndrome coronavirus 2" OR 2019ncov OR "2019 ncov" OR "novel coronavirus*" OR "novel corona virus*" OR covid19 OR "covid 19" OR "sars cov 2" OR sars2 OR "new coronavirus*" OR "new corona virus*" OR "ncov 2019" OR "sars coronavirus 2" OR "sars corona virus 2" OR "severe acute respiratory syndrome cov 2" OR "severe acute respiratory syndrome cov2" OR "COVID-19" OR "COVID19" OR "COVID2019" OR "COVID 2019" OR "severe acute respiratory syndrome coronavirus 2" OR "SARS-COV*" OR SARSCOV*)) OR (TX("Biopsy" OR "biopsy" OR "biopsies" OR "biopsie" OR "Autopsy" OR autops* OR obduct*) AND AB("COVID-19" OR "severe acute respiratory syndrome coronavirus 2" OR 2019ncov OR "2019 ncov" OR "novel coronavirus*" OR "novel corona virus*" OR covid19 OR "covid 19" OR "sars cov 2" OR sars2 OR "new coronavirus*" OR "new corona virus*" OR "ncov 2019" OR "sars coronavirus 2" OR "sars corona virus 2" OR "severe acute respiratory syndrome cov 2" OR "severe acute respiratory syndrome cov2" OR "COVID-19" OR "COVID19" OR "COVID2019" OR "COVID 2019" OR "severe acute respiratory syndrome coronavirus 2" OR "SARS-COV*" OR SARSCOV*)) OR (TI("Biopsy" OR "biopsy" OR "biopsies" OR "biopsie" OR "Autopsy" OR autops* OR obduct* OR "pathology" OR patholog* OR neuropathol* OR cytopathol* OR telepathol* OR histopathol*) AND TX("COVID-19" OR "severe acute respiratory syndrome coronavirus 2" OR 2019ncov OR "2019 ncov" OR "novel coronavirus*" OR "novel corona virus*" OR covid19 OR "covid 19" OR "sars cov 2" OR sars2 OR "new coronavirus*" OR "new corona virus*" OR "ncov 2019" OR "sars coronavirus 2" OR "sars corona virus 2" OR "severe acute respiratory syndrome cov 2" OR "severe acute respiratory syndrome cov2" OR "COVID-19" OR "COVID19" OR "COVID2019" OR "COVID 2019" OR "severe acute respiratory syndrome coronavirus 2" OR "SARS-COV*" OR SARSCOV*)) OR (KW("Biopsy" OR "biopsy" OR "biopsies" OR "biopsie" OR "Autopsy" OR autops* OR obduct*) AND TX("COVID-19" OR "severe acute respiratory syndrome coronavirus 2" OR 2019ncov OR "2019 ncov" OR "novel coronavirus*" OR "novel corona virus*" OR covid19 OR "covid 19" OR "sars cov 2" OR sars2 OR "new coronavirus*" OR "new corona virus*" OR "ncov 2019" OR "sars coronavirus 2" OR "sars corona virus 2" OR "severe acute respiratory syndrome cov 2" OR "severe acute respiratory syndrome cov2" OR "COVID-19" OR "COVID19" OR "COVID2019" OR "COVID 2019" OR "severe acute respiratory syndrome coronavirus 2" OR "SARS-COV*" OR SARSCOV*)) OR (SU("Biopsy" OR "biopsy" OR "biopsies" OR "biopsie" OR "Autopsy" OR autops* OR obduct*) AND TX("COVID-19" OR "severe acute respiratory syndrome coronavirus 2" OR 2019ncov OR "2019 ncov" OR "novel coronavirus*" OR "novel corona virus*" OR covid19 OR "covid 19" OR "sars cov 2" OR sars2 OR "new coronavirus*" OR "new corona virus*" OR "ncov 2019" OR "sars coronavirus 2" OR "sars corona virus 2" OR "severe acute respiratory syndrome cov 2" OR "severe acute respiratory syndrome cov2" OR "COVID-19" OR "COVID19" OR "COVID2019" OR "COVID 2019" OR "severe acute respiratory syndrome coronavirus 2" OR "SARS-COV*" OR SARSCOV*)) OR (AB("Biopsy" OR "biopsy" OR "biopsies" OR "biopsie" OR "Autopsy" OR autops* OR obduct*) AND TX("COVID-19" OR "severe acute respiratory syndrome coronavirus 2" OR 2019ncov OR "2019 ncov" OR "novel coronavirus*" OR "novel corona virus*" OR covid19 OR "covid 19" OR "sars cov 2" OR sars2 OR "new coronavirus*" OR "new corona virus*" OR "ncov 2019" OR "sars coronavirus 2" OR "sars corona virus 2" OR "severe acute respiratory syndrome cov 2" OR "severe acute respiratory syndrome cov2" OR "COVID-19" OR "COVID19" OR "COVID2019" OR "COVID 2019" OR "severe acute respiratory syndrome coronavirus 2" OR "SARS-COV*" OR SARSCOV*))

Emcare
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ScienceDirect
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Supplemental Table S2. Quality analysis of the included articles

Article	Number of patients included	Clinical data reported per case	Microscopic evaluation reported per case	Macroscopic evaluation reported per case	Quality assessment
Ahouach et al	1	yes	yes	no	Moderate
Barton et al	2	yes	yes	yes	High
Baud et al	1	yes	yes	no	Moderate
Bradley et al	12	yes	no	no	Low
Cai et al	7	yes	yes	no	Moderate
Carsana et al	38	no	no	no	Low
Chen et al *Chin Med J*	3	yes	yes	yes	High
Chen et al *Chin J Path*	3	yes	yes	yes	High
Chen et al *MedRxiv*	6	no	no	no	Low
Copin et al	6	no	no	no	Low
Diao et al	6	yes	yes	no	Moderate
Ding et al	1	yes	yes	no	Moderate
Fernandez-Nieto et al	1	yes	yes	no	Moderate
Fox et al	4	no	no	no	Low
Gianotti et al	3	yes	yes	no	Moderate
Karami et al	1	yes	yes	no	Moderate
Kissling et al	1	yes	yes	no	Moderate
Kolivras et al	1	yes	yes	no	Moderate
Konopka et al	1	yes	yes	yes	High
Kuang et al	1	yes	yes	no	Moderate
Lagana et al	1	yes	yes	no	Moderate

Larsen et al	1	yes	yes	no	Moderate
Liu et al	1	yes	no	yes	Low
Luo et al	1	yes	yes	yes	High
Magro et al	5	yes	yes	yes	High
Menter et al	21	yes	yes	yes	High
Pernazza et al	1	yes	yes	no	Moderate
Schweitzer er al	1	yes	yes	yes	High
Su et al	26	yes	yes	no	Moderate
Tavazzi et al	1	yes	yes	no	Moderate
Tian et al *J Thor Onc*	2	yes	yes	no	Moderate
Tian et al *Mod Pathol*	4	yes	yes	no	Moderate
Varga et al	3	yes	yes	no	Moderate
Wichmann et al	12	yes	yes	yes	High
Xiao et al	1	yes	yes	no	Moderate
Xu, Chang et al	10	no	no	no	Low
Xu, Kuang et al	1	yes	yes	no	Moderate
Xu et al *Lancet*	1	yes	yes	no	Moderate
Yao et al *Cell Res*	1	yes	yes	no	Moderate
Yao et al *Zhonghua*	3	yes	no	no	Low
Zeng et al	1	yes	yes	yes	High
Zhang et al	1	yes	yes	no	Moderate

Supplemental Table S4. Pulmonary and upper respiratory pathology reports (n=131 cases)

Article	Case number	Macroscopic findings	Microscopic findings (histology, and, when applicable, additional experiments)	Main pattern(s) of lung injury (epithelial, vascular, fibrotic)
Barton et al	1	Weight 1183g (right), 1269g (left). Parenchyma: diffusely edematous & firm, no focal lesions. Upper & lower airways: patent, no mucus plugging. Mucosa without gross abnormalities. Edematous right pleural adhesions. No effusions	Acute stage DAD: numerous hyaline membranes, no interstitial organization. Very patchy, sparse interstitial chronic inflammation (mainly of lymphocytes). Focal alveolar septal capillary congestion, edema fluid within airspaces. Mild chronic inflammation within bronchi & bronchioles, with prominent mucosal edema within the bronchial mucosa. No mucus plugging. No eosinophils or neutrophils. IHC: CD-3: sparse infiltrate within the alveolar septa. CD-20: rare. Slightly more CD8 than CD4. CD68: few.	Epithelial
	2	Weight 579g (right), 612g (left). Parenchyma red/tan mottled appearance in upper lobes, diffusely saturated dark red appearance in lower lobes. No adhesions / effusions	Foci of acute bronchopneumonia with rare aspirated food particles. Peribronchiolar airspaces filled with neutrophils, histiocytes. No DAD, mucus plugging or eosinophils. Focally, aspirated foreign material (bacteria, squamous cells, vegetable matter) in airways. IHC: Similar findings to case 1. CD68: numerous macrophages within the areas of bronchopneumonia.	Not classifiable (aspiration pneumonia, no certain COVID-associated lung disease)
Bradley et al	1-12*	n=5: heavy, edematous lungs. Weight average 1049g (right), 755g (left). Variable volume of pleural fluid: 0-450 mL per pleural space. n=2 sub-segmental pulmonary emboli. n=1 intraparenchymal hemorrhage. No pulmonary consolidation.	12 cases: enlarged, reactive type II pneumocytes with nucleomegaly and prominent nucleoli. 9 cases: changes of acute and/or organizing DAD (presence of intra-alveolar fibrin, hyaline membranes, loosely organizing connective tissue in alveolar septal walls) 6 cases: focal areas of acute bronchiolitis 2 cases: bronchopneumonia 1 case: small basophilic cytoplasmic inclusions and larger eosinophilic cytoplasmic inclusions Multinuclear cells in a subset of cases. No microthrombi in the pulmonary system. 10 cases: chronic (lymphocytic) tracheitis, 9 cases: edema of trachea, 4 cases acute tracheitis, 1 case fibrosis	n=12 Epithelial n=9 Vascular Caveat: n=2 bronchopneumonia

			EM: viral particles (~70-100 nm) in lung, trachea; individually or aggregates in cytoplasm or inside vesicles in type I and II pneumocytes. Type II pneumocytes: numerous autophagosomes (double membranes, presence of organelles) in cytoplasm. Some of autophagosomes contained viral aggregates. No conclusive aggregates of nucleoprotein or viral inclusions. Viral particles also present in tracheal epithelial cells and within the extracellular mucus in the tracheal lumen. RT-PCR: detectable levels of viral RNA in lungs, trachea	
Cai et al	1	NR	Typical malignant pathological features of the primary tumors. Wide range of lung interstitial inflammation with numerous infiltrating immune cells. Infiltrating cells: mainly plasma cells and macrophages. Rare lymphocytes. Thickened alveolar septum and fibrous connective tissue proliferation. Large number of macrophages and foam cells in the alveolar cavities. No evident pneumocyte hyperplasia. No obvious viral inclusion bodies and hyaline membrane.	Not classifiable
	2-7*	NR	Typical malignant pathological features of the primary tumors. No evident inflammation in the lung tissue away from the tumors.	Not classifiable
Carsana et al	1-38*	Heavy, congested and oedematous organs, with spotty involvement.	Features of DAD corresponding to exudative and early/intermediate proliferative phases of disease. Both phases often overlapped in different areas of the lungs, with plurifocal pattern of distribution. Scoring: focal (5-25%), present (25-50%), plurifocal (50-75%), diffuse (>75%) Exudative phase (counted if scored focal, present, plurifocal or diffuse): capillary congestion: 38/38 (13 diffuse); loss of pneumocytes: 38/38; interstitial and intraalveolar edema: 37/38 (19 focal); dilated alveolar ducts + collapsed alveoli: 36/38; increased megakaryocytes: 33/38 (25 focal); hyaline membranes: 32/38 (19 focal); alveolar hemorrhage: 32/38 (20 focal); granulocytes: 31/38; alveolar proteinosis: 28/38 (25 focal); collapsed alveoli: 28/38; localized DIC: 0/38 Platelet-fibrin thrombi in small arterial vessels: 33/38 (13 plurifocal) Proliferative phase (counted if scored focal, present, plurifocal or diffuse): type 2 pneumocyte hyperplasia: 38/38 (14 focal, 7 diffuse); interstitial myofibroblast reaction: 25/38 (18 focal); alveolar granulation tissue: 21/38 (13 focal); squamous metaplasia with atypia: 20/38 (12 focal); capillaries proliferation: 17 (14 focal); septal collagen deposition: 15 (13 focal); alveolar duct fibrosis: 12 (12 focal); complete alveolar occludent fibrosis: 11/38 (6 focal); ring alveolar occludent fibrosis: 15/38 (10 focal); alveolar buds: 10 (7 focal); organized alveoli + dilated alveolar ducts: 9 (6 focal); bronchiolitis obliterans: 0/38 Fibrotic phase: mural fibrosis: 12 focal, 10 present, 1 plurifocal, 1 diffuse; microcystic honeycombing: 9 focal, 6 ‘present’; 1 with fibrous microcysts; no pleural involvement or scars. n=31 interstitial inflammatory infiltrate (4 diffuse). n=23 alveolar inflammatory infiltrate (macrophages, 13 focal). n=13 alveolar multinucleated giant cells (9 focal, 2 diffuse)	n=38 Epithelial n=28 Vascular n=12 Fibrotic

			IHC: few CD45+ lymphocytes located in interstitial space; many CD68+ macrophages, mainly in the alveolar lumens. Increased number of CD61+ megakaryocytes in lung capillaries. EM: viral particles, with morphology typical of the family of Coronaviridae, localized along plasmalemmal membranes and within cytoplasmic vacuoles of pneumocytes (average diameter of 82nm and viral projection about 13nm in length).	
Chen et al, *Chin Med J*	1	Congestive and hemorrhagic necrosis. Consolidation, extreme pulmonary tissue edema. Congestion with intrapulmonary hematoma.	Parenchyma: Extensive pulmonary interstitial fibrosis with hyaline degeneration Intrapulmonary vessels: Occluded vessel lumen with microthrombosis	Vascular Fibrotic
	2	Congestive and hemorrhagic necrosis. Consolidation, extreme pulmonary tissue edema and congestion with intrapulmonary hematoma	Parenchyma: Extensive pulmonary interstitial fibrosis and alveolar hemorrhage Intrapulmonary vessels: Intravascular organized thrombosis and vasculitis	Vascular Fibrotic
	3	Consolidation, extreme pulmonary tissue edema and congestion with intrapulmonary hematoma	NR	Not classifiable
Copin et al	1	NR	Lymphocytic viral pneumonia. Infiltration of alveolar walls by numerous lymphocytes and edema. Type 2 pneumocyte hyperplasia with cytologic atypia. All 6 cases: fibroblastic bodies and fibroblasts surrounding intra-alveolar fibrin, as well as moderate interstitial T-cell lymphocytic, a plasma cells infiltrate, and type 2 pneumocyte hyperplasia with cytologic atypia. Vascular injury was also a prominent feature readily: endothelial injury with cytoplasmic vacuolization and cell detachment in small to medium-sized pulmonary arteries.	Epithelial
	2-6*	NR	Acute fibrinous and organizing pneumonia (AFOP): extensive intra-alveolar fibrin deposition called fibrin “balls”, rather than hyaline membranes. Organizing pneumonia consisting of intraluminal loose connective tissue within the alveolar ducts and bronchioles associated with the fibrinous acute injury. All 6 cases: fibroblastic bodies and fibroblasts surrounding intra-alveolar fibrin, as well as moderate interstitial T-cell lymphocytic, a plasma cells infiltrate, and type 2 pneumocyte hyperplasia with	Epithelial Vascular (AFOP) (Early) fibrotic

			cytologic atypia. Vascular injury was also a prominent feature readily: endothelial injury with cytoplasmic vacuolization and cell detachment in small to medium-sized pulmonary arteries.	
Ding et al	20	NR	Obvious inflammatory exudation, hyperplasia of alveolar epithelium, thickening of interstitial space and local formation of granulomatous nodule with proliferation of fibroblasts and lymphocytes in the nodule.	Epithelial Fibrotic (NSIP)
Fox et al	1-4*	Tracheae: normal caliber, mildly erythematous. Heavy lungs, left 680-1030g, right 800-1050g. No thrombi in pulmonary arteries at the hilum. Bronchi: thick, white mucous in the lungs (n=1), pink froth in the airways (n=3). Mild to moderate serosanguinous pleural effusions. Parenchyma: diffusely edematous and firm. Regions of dark-colored hemorrhage with focal demarcation (n=3). Frank hemorrhage upon cutting in areas identified as hemorrhagic on external surface. Cut surface: alternating areas of tan-grey consolidation with patchy areas of hemorrhage (3-6cm maximal diameter). Some cases: small, firm thrombi in sections of the peripheral parenchyma. Focal consolidation in 1 case (patient on immunosuppression), n=3 t	Bilateral DAD with a comparatively mild-to-moderate lymphocytic infiltrate (mixture CD4+ & CD8+ lymphocytes) located predominantly in the interstitial spaces and around larger bronchioles. CD4+ lymphocytes in aggregates around small vessels, some appeared to contain platelets and small thrombi. n=3 foci of hemorrhage. Desquamated type 2-pneumocytes with apparent viral cytopathic effect consisting of cytomegaly, and enlarged nuclei with bright, eosinophilic nucleoli within alveolar spaces. Largest of these cells frequently contained eccentric clearing of cytoplasm with small vesicles, likely representing viral inclusions. Scattered hyaline membranes, fibrin deposition, highlighted by trichrome stain. Notable thickened alveolar capillaries with surrounding edema. Fibrin thrombi within the capillaries and small vessels. Notably: CD61+ megakaryocytes present with significant nuclear hyperchromasia and atypia, located within alveolar capillaries, in association with, and actively producing platelets. Fibrin and platelets within small vessels appeared to aggregate inflammatory cells, with entrapment of numerous neutrophils. Focal acute inflammatory infiltrate only in patient on immunosuppression (possible consistent with secondary infection). Neutrophils in this case were partially degenerated and entrapped in fibers, possibly representing neutrophil extracellular traps; present in association with clusters of CD4+ mononuclear cells. n=3: no significant neutrophilic infiltrate within airways or the interstitium.	n=4 Epithelial n=4 Vascular

		no lobar infiltrate, abscess, or definitive gross inflammatory process.		
Karami et al	NA	NR	Alveolar spaces with focal hyaline membrane, pneumocyte proliferation, metaplastic changes. Viral cytopathic effect including multinucleation and nuclear atypia present. Most inflammatory cells present: mononuclear cells composed of lymphocytes and macrophages.	Epithelial
Konopka et al	NA	Heavy lungs with mucus plugging of the conducting airways and consolidation of the lung parenchyma. No evidence of hyperinflation / air-trapping	Paucicellular mucus plugs in the proximal airways, no tissue eosinophilia. Goblet cell metaplasia, mucus gland hyperplasia, and thickening of subepithelial basement membranes in cartilaginous and non-cartilaginous airways attested to the patient's history of asthma. Distal alveolated lung tissue: DAD, characterized by patchy, mild interstitial thickening by edema, focal pneumocyte hyperplasia, and scattered hyaline membranes. Rare fibrin thrombi within small vessels and a small muscular pulmonary artery consistent with endothelial injury, accompanied by a mild patchy fibrinous airspace exudate in which mononuclear inflammatory cells predominated with scattered neutrophils. Inflammatory infiltrate limited to distal airspaces, bronchi or bronchioles involvement.	Epithelial Vascular
Kuang et al	NA	NR	Wide range of pulmonary interstitial inflammation (also away from the tumor area): alveolar septum significantly wider/more broad, with fibrous connective tissue hyperplasia, mild fibrosis. Septum: mainly plasma cell infiltration, few lymphocytes, occasional neutrophils. Alveolar cavity: large number of macrophages, foam cells. Mild proliferation of alveolar epithelium, no obvious atypia, no obvious viral inclusions, no hyaline membranes. IHC: few CD3+, CD20+, and CD8+ lymphocytes in alveolar septum, mainly plasma cell infiltration. Many CD68+ macrophages and foam cells in the alveolar cavity. RT-PCR: 2019-nCoV positive	Fibrotic
Liu et al	1	Weight 700g (left), 1240g (right) after fixation. Big part of the left lung grayish white colored patches. Right lung is more congested, dark red colored patches and hemorrhages (more pronounced at the edge of the lung). Pleural effusions: not much, clear yellow liquid (no serous inflammation).	NR	Not classifiable

		Extensive pleural adhesions of the right lung, pleural thickening of right lung. Cut surface: large amount of grayish-white viscous fluid; fiber bands visible. Trachea: white foamy mucus. Right bronchus: gelatinous mucus attachment		
Luo et al	NA	Whole surface was bronzing, diffuse congestive appearance. Punctate haemorrhage and partly hemorrhagic necrosis (mainly in outer edge of the right lung). Bronchi: swollen mucosal surfaces covered with haemorrhagic exudation. Cut surfaces of the lung: severe congestive and haemorrhagic changes.	Extensive pulmonary interstitial fibrosis with partly hyaline degeneration, pulmonary hemorrhagic infarct. Small vessel hyperplasia, vessel wall thickening, and lumen stenosis and occlusion. Interstitial infiltration of inflammatory cells (lymphocytes, plasma cells and mononuclear cells). Alveolitis with atrophy, proliferation, desquamation and squamous metaplasia of alveolar epithelial cells (mainly type Ⅱ). Thickened alveolar septa. Massive fibrinous exudate, multinucleated giant cells and intracytoplasmic viral inclusion bodies present. Necrotizing bronchiolitis manifested necrosis of bronchiolar wall and epithelial cells were present in the lumen. Masson staining: pulmonary interstitial fibrosis. No bacterial and fungal infections found by additional staining. IHC: CD3, CD4, CD5, CD8, CD20, CD38, CD79a positive, mostly focally in lung interstitium and near blood vessels. CD31, TTF1, CK5/6, CK7, CK19, SMA, F VIII and Collagen IV positive.	Epithelial Vascular (possibly AFOP) Fibrotic
Magro et al	1	Lungs: congested and hemorrhagic appearance	Severe organizing hemorrhagic pneumonitis: significant fibrin deposition within septal capillary lumens and walls, with endothelial cell necrosis, consistent with a thrombotic necrotizing capillary injury syndrome. Pattern of septal capillary injury ranged from a pauci-inflammatory pattern to one characterized by permeation of the interalveolar septa by neutrophils amidst the damaged capillaries, along with intra-alveolar neutrophils. No viral cytopathic changes. No hyaline membranes reflective of DAD. No type II pneumocyte hyperplasia. Brunt of the lung injury was restricted to septal capillaries, without pneumocyte involvement. IHC: C4d deposition (extensive), C5b-9 deposition, granular C3d deposition (relatively few) in inter-alveolar septal capillaries. SARS-CoV-2 spike and envelope proteins expressed in respiratory epithelia and inter-alveolar sept. Co-localization of SARS-CoV-2 with septal vascular C4b and with C5b-9 resp.	Vascular

	2	Hemorrhagic and congested appearance	Extensive hemorrhagic pneumonitis. Congested inter-alveolar septa, with luminal and mural fibrin deposition within septal capillaries. Focal intra-alveolar collections of neutrophils and monocytes. No viral cytopathic changes. Striking red cell extravasation within the alveolar spaces, along with intra-alveolar fibrin deposition. Some focal hyaline membrane formation and type II pneumocyte hyperplasia in areas of hemorrhagic pneumonitis. Dominant pattern was septal capillary injury, not DAD. IHC: prominent deposition of C5b-9 within the microvasculature of the inter-alveolar septa, in larger caliber vessels of parenchyma, and in normal-appearing lung and tracheal soft tissues. C4d localized to inter-alveolar septa in regions of microvascular injury. MASP2: granular and punctate staining localized to the inter-alveolar septa.	Vascular
Menter et al	1	Parenchyma: heavy, firm, unevenly blue-ish red in colour with signs of severe congestion	Mucostasis, bilateral interstitial pneumonia with lymphocytic infiltrates, reactive pneumocytes, intraalveolar exudates with beginning alveolar damage, bronchopneumonia in left lower lobe	Epithelial
	2	Parenchyma: heavy, firm, unevenly blue-ish red in colour with signs of severe congestion	Mucostasis, alveolar damage in early exudative phase with subpleural interstitial oedema, reactive pneumocytes, fibrinous exudate	Epithelial Vascular
	3	Parenchyma: heavy, firm, unevenly blue-ish red in colour with signs of severe congestion	Mucosal nodularity of trachea due to erythroid extravasation and fibrooedema, interstitial pulmonal oedema and severe bacterial bronchopneumonia. Gram-positive cocci in alveolar space	Not classifiable (bronchopneumonia?)
	4	Parenchyma: heavy, firm, unevenly blue-ish red in colour with signs of severe congestion	Severe fibrinous erosive tracheitis, DAD in early exudative phase, subpleural intraalveolar and interstitial oedema	Epithelial
	5	Parenchyma: heavy, firm, unevenly blue-ish red in colour with signs of severe congestion	Fibrinous tracheitis, bilateral pleural effusions, severe bronchopneumonia with fibrinous exudates and reactive pneumocytes	Epithelial Vascular

	6	Parenchyma: heavy, firm, unevenly blue-ish red in colour with signs of severe congestion	DAD in early exudative phase with fibrinous exudates, peribronchiolar metaplasia, interstitial haemorrhages, intraalveolar and interstitial oedema and beginning bronchopneumonia. Pre-existing interstitial fibrosis and pleural/mediastinal adhesions	Epithelial Vascular
	7	Parenchyma: heavy, firm, unevenly blue-ish red in colour with signs of severe congestion	DAD in early exudative phase, peribronchiolar metaplasia of left lower lobe, oedema, emphysema, beginning bronchopneumonia	Epithelial
	8	Parenchyma: heavy, firm, unevenly blue-ish red in colour with signs of severe congestion	Tracheitis and bronchitis. Bilateral bronchopneumonia with DAD in exudative stage, acute capillaritis and alveolitis.	Epithelial Vascular
	9	Parenchyma: heavy, firm, unevenly blue-ish red in colour with signs of severe congestion	DAD in exudative stage, acute bronchitis, metaplasia of alveolar epithelium, moderate emphysema, oedema, periphery pulmonary embolism of right middle lobe	Epithelial Vascular
	10	Parenchyma: heavy, firm, unevenly blue-ish red in colour with signs of severe congestion	DAD in exudative stage with capillary congestion and reactive pneumocytic changes, emphysema, atelectasis of left lower lobe, pulmonary artery sclerosis	Epithelial
	11	Parenchyma: heavy, firm, unevenly blue-ish red in colour with signs of severe congestion	DAD in exudative stage with capillary congestion and reactive pneumocytic changes, diffuse pulmonary haemorrhage and central / peripheral pulmonary embolisms	Epithelial Vascular
	12	Parenchyma: heavy, firm, unevenly blue-ish red in colour with signs of severe congestion	Mucopurulent bronchitis with capillary congestion, bronchopneumonia of left lower lobe, mononuclear and interstitial lymphoid infiltrate, pulmonary haemorrhage, mild emphysema, peribronchiolar metaplasia, anthracosis	Epithelial

	13	Parenchyma: heavy, firm, unevenly blue-ish red in colour with signs of severe congestion	DAD in exudative and concomitant proliferative phase, reactive pneumocyte changes, lymphocytic interstitial infiltrate, peribronchiolar metaplasia, residues of central and peripheral pulmonary embolisms, pulmonal artery sclerosis	Epithelial Vascular
	14	Parenchyma: heavy, firm, unevenly blue-ish red in colour with signs of severe congestion	Organising bronchopneumonia, prominent lymphoid interstitial infiltrate, oedema, ATTR amyloidosis of greater pulmonary vessels	Epithelial
	15	Parenchyma: heavy, firm, unevenly blue-ish red in colour with signs of severe congestion	DAD in exudative and concomitant proliferative phase, reactive pneumocyte changes, lymphocytic interstitial infiltrate, oedema	Epithelial
	16	Parenchyma: heavy, firm, unevenly blue-ish red in colour with signs of severe congestion	DAD in exudative and beginning concomitant proliferative phase, lymphocytic interstitial inflammation	Epithelial
	17	Parenchyma: heavy, firm, unevenly blue-ish red in colour with signs of severe congestion	DAD in proliferative phase with prominent interstitial lymphoid infiltrate, emphysema, acute pulmonary infarction in right lower lobe. Thrombotic angiopathy in periphery arteries of lungs (also in greater vessels kidneys and glomerular microangiopathy).	Epithelial Vascular
	18	Parenchyma: heavy, firm, unevenly blue-ish red in colour with signs of severe congestion	Organising pneumonia with focal bronchopneumonia, giant cells, peribronchiolar metaplasia, anthracosis	Epithelial
	19	Parenchyma: heavy, firm, unevenly blue-ish red in colour with signs of severe congestion	DAD in exudative phase, minimal lymphocytic interstitial infiltrate, oedema, emphysema, peribronchiolar metaplasia, anthracosis	Epithelial

	20	Parenchyma: heavy, firm, unevenly blue-ish red in colour with signs of severe congestion	DAD in exudative and proliferative phase, reactive pneumocyte changes, lymphocytic interstitial infiltrate, diffuse bronchopneumonia, giant cells, peribronchiolar metaplasia, silicoanthracosis, pulmonary artery sclerosis.	Epithelial
	21	Parenchyma: heavy, firm, unevenly blue-ish red in colour with signs of severe congestion	DAD in concomitant exudative and proliferative phase	Epithelial
Pernazza et al	NA	NA	Lung adenocarcinoma, with a predominant acinar pattern. Parenchyma surrounding the neoplasia: diffuse hemorrhages and clusters of alveolar macrophages, with occasional multinucleated cells. Diffuse pneumocyte loss and reactive hyperplasia, with focal pneumocytes showing nuclear inclusions. Scanty fibrin depositions on the alveolar surfaces, in the absence of hyaline membranes. Interstitial edema and mild inflammatory infiltrate, mainly composed of cytotoxic (CD8+) T lymphocytes. Diffuse aspects of neutrophil margination within small arterioles. Occasional fibrous plugs (organizing pneumonia). Interstitial changes were more marked in the subpleural area, with where mild fibrous thickening of alveolar septa. Areas of smoking-related interstitial fibrosis.	Epithelial Vascular (possibly AFOP) Fibrotic
Schweitzer et al	NA	Weight 1780 g. Extensive Tardieu spots and an increased consistency, with haemorrhagic foam or frothy fluid, in bronchi and trachea.	Congested blood vessels, some hyaline membranes, and patchy inflammation with lymphocytes (in part bi- and trinuclear).	Epithelial
Tian et al *J ThorOnc*	1	NR	Evident alveolar damage: alveolar edema, proteinaceous exudates. Prominent inspissated spherical secretions or globules. Patchy and mild inflammatory infiltration (no neutrophils). Focal fibrin clusters mixed with mononuclear inflammatory cells and multinucleated giant cells in airspaces. Patchy, severe pneumocyte hyperplasia and interstitial thickening. Suspected viral inclusions in some of these cells. Vascular congestion in lungs.	Epithelial Vascular (possibly AFOP)
	2	NR	Parenchyma: patchy, evident proteinaceous and fibrin exudates. Diffuse thickening of alveolar walls, consisting of proliferating interstitial fibroblasts and type II pneumocyte hyperplasia. Focal fibroblast plug and multinucleated giant cells in the airspaces. Some areas abundant alveolar macrophages along with type II pneumocyte hyperplasia.	Epithelial (Early) vascular (possibly AFOP) Fibrotic

Tian et al *Mod Pathol*	1	NA	Acute-phase DAD: hyaline membrane; focal sloughing of pneumocytes alternating with type II pneumocyte hyperplasia and syncytial giant cells formation; focal lymphocytic infiltration (changes of CLL).	Epithelial
	2	NA	Acute-phase DAD: mainly hyaline membranes formation. RT-PCR assay for SARS-CoV-2: positive.	Epithelial
	3	NA	Acute-phase DAD predominant: hyaline membrane; focal interstitial thickening, vascular congestion, mild inflammatory cellular infiltration.	Epithelial
	4	NA	Organizing-phase DAD: hyaline membrane; intra-alveolar hemorrhages, early organization, interstitial thickening, focal fibrinoid necrosis of small vessel wall, abundant intra-alveolar neutrophilic infiltration.	Epithelial Vascular Caveat: superinfection (abundant granulocytic infiltrate)
Varga et al	1	NR	Prominent endotheliitis with recruitment of inflammatory cells, as well as an unusual high amount of apoptotic bodies in many organs, especially in the pulmonary vessels but also in small bowel and heart. Strong accumulation of mononuclear cells was found in the lung, leading to congestion of most small lung vessels.	Vascular
	2	NR	Signs of ARDS and lymphocytic endotheliitis in lung (also heart, kidney, and liver)	Epithelial Vascular
Wichmann et al	1-12 **	Lungs: lung surface often displayed mild pleurisy and a distinct patchy pattern, with pale areas alternating with slightly protruding and firm, deep reddish blue hypercapillarized areas. On the cutting surfaces, this pattern was also visible. Consistency of the lung tissue:	Microthrombi were regularly found within the small lung arteries Chronic lymphocytic pharyngitis (n=6) RT-PCR positive in pharynx (n=9)	

		firm yet friable. In 8 cases, all parts of the lungs were affected by these changes. Pharynx: normal in all cases		
	1	Lungs: weight 1135g (right), 940g (left). Patchy aspect of lung surface. Massive pulmonary embolism (cause of death), with the thrombi deriving from the deep veins of the lower extremities. Cause of death: PE, pneumonia.	DAD: activated pneumocytes, fibroblasts, giant cells, sparse hyaline membranes, slight fibrosis. Additional findings: congestion of small vessels, thrombi. RT-PCR assay for SARS-CoV-2: positive.	Epithelial Vascular (Very early fibrotic)
	2	Lungs: weight 1220g (right), 1030g (left). Cause of death: pneumonia with bronchopneumonia.	DAD: activated pneumocytes, hyaline membranes, sparse lymphocytes. Additional findings: focal granulocytic infiltration, chronic bronchitis, acute bronchitis. RT-PCR assay for SARS-CoV-2: positive.	Epithelial
	3	Lungs: weight 1280g (right), 1445g (left). Massive pulmonary embolism (cause of death), with the thrombi deriving from the deep veins of the lower extremities. Status post VATS (resection of right upper lung lobe). Cause of death: PE, pneumonia.	DAD: squamous metaplasia, fibroblasts, activated pneumocytes, hyaline membranes. Additional findings: thrombi. RT-PCR assay for SARS-CoV-2: positive.	Epithelial Vascular
	4	Lungs: weight 1370g (right), 1100g (left). Massive pulmonary embolism (cause of death), with the thrombi deriving from the deep veins of	DAD: fibroblasts, activated pneumocytes, hyaline membranes, squamous metaplasia. Additional findings: hemorrhagic infarctions, thrombi. RT-PCR assay for SARS-CoV-2: positive.	Epithelial Vascular

		the lower extremities. Cause of death: PE, pneumonia.		
	5	Lungs: weight 955g (right), 845g (left). No pulmonary embolism. Cause of death: pneumonia.	DAD: activated pneumocytes, fibroblasts, hyaline membrane, necrosis, lymphocytes. Additional findings: surrounding small vessels, thrombi. RT-PCR assay for SARS-CoV-2: positive.	Epithelial Vascular
	6	Lungs: weight 275g (right), 275g (left). Focal purulent bronchopneumonia. Cause of death: pneumonia.	No DAD but extensive granulocytic infiltration of the alveoli and bronchi, resembling bacterial focal bronchopneumonia. Acute bronchitis. Congestion of small vessels. RT-PCR assay for SARS-CoV-2: positive.	Not classifiable
	7	Lungs: weight 690g (right), 655g (left). Focal purulent bronchopneumonia. Cause of death: pneumonia.	DAD: hyaline membranes, activated pneumocytes, squamous metaplasia. Additional findings: emphysema, congestion of small vessels. RT-PCR assay for SARS-CoV-2: positive.	Epithelial
	8	Lungs: weight 1160g (right), 940g (left). No pulmonary embolism. Cause of death: bronchopneumonia.	No DAD but extensive granulocytic infiltration of the alveoli and bronchi, resembling bacterial focal bronchopneumonia. Emphysema. RT-PCR assay for SARS-CoV-2: positive.	Not classifiable
	9	Lungs: weight 480g (right), 410g (left). Neuroendocrine tumor of the lungs. Cause of death: purulent bronchitis.	No DAD but extensive granulocytic infiltration of the alveoli and bronchi, resembling bacterial focal bronchopneumonia. Acute bronchitis. Bullous emphysema. Additional findings: neuroendocrine tumor composed of small cells. RT-PCR assay for SARS-CoV-2: positive.	Not classifiable
	10	Lungs: weight 730g (right), 630g (left). Cause of death: pneumonia, septic encephalopathy.	No DAD but extensive granulocytic infiltration of the alveoli and bronchi, resembling bacterial focal bronchopneumonia. Fibrosis. Emphysema, congestion of small vessels, chronic bronchitis. RT-PCR assay for SARS-CoV-2: positive.	Not classifiable
	11	Lungs: weight 1880g (right), 1540g (left). No pulmonary	DAD: hyaline membranes (sparse), giant cells, activated pneumocytes. Additional findings: emphysema, congestion of small vessels, granulocytic infiltration. RT-PCR assay for SARS-CoV-2: positive.	Epithelial

		embolism. Cause of death: pneumonia.		
	12	Lungs: weight 1580g (right), 1290g (left). Massive pulmonary embolism (cause of death), with the thrombi deriving from the deep veins of the lower extremities. Purulent tracheobronchitis. Cause of death: PE.	DAD: hyaline membranes, activated pneumocytes, fibrosis. Additional findings: lymphocytes, plasma cells, hemorrhagic infarctions, thrombi, congestion of small vessels, emphysema. RT-PCR assay for SARS-CoV-2: positive.	Epithelial Vascular Fibrotic
Xu et al *Lancet*	NA	NA	Bilateral DAD with cellular fibromyxoid exudates. Interstitial mononuclear inflammatory infiltrates (lymphocytes). Viral cytopathic-like changes: multinucleated syncytial cells with atypical enlarged pneumocytes characterised by large nuclei, amphophilic granular cytoplasm, prominent nucleoli in intra-alveolar spaces. No obvious intranuclear or intracytoplasmic viral inclusions. Right lung: evident desquamation of pneumocytes, hyaline membrane formation. Left lung: pulmonary oedema with hyaline membrane formation. Blood: reduced counts of CD4 and CD8 cells in peripheral blood. Hyperactivated status of CD4 and CD8 cells (high proportion of HLA-DR and CD38, increased concentration of proinflammatory CCR+ Th17 in CD4, high concentrations of cytotoxic granules in CD8).	Epithelial Fibrotic (Organizing pneumonia)
Xu, Kuang et al	A	NR	Interstitial pneumonia. Significantly widened alveolar septum with fibrous connective tissue hyperplasia and mild fibrosis, plasma cells and a few lymphocytes. In the alveolar cavity: a large amount of exudation and mononuclear cells	Epithelial Fibrotic
Yao et al *Cell Res*	NA	NR	Predominant diffuse alveolar damage: extensive desquamation of proliferative type II AE, exudative monocytes and macrophages. Some of alveolar walls: partially lined by low columnar type II AE and covered by the formation of hyaline membranes 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. IHC SARS-CoV-2 nucleocapsid: SARS-CoV-2 positive IHC: predominantly infiltrating CD68+ macrophages, CD20+ B cells, and CD8+ T cells. CD4+ T and CD38+ plasma cells barely detectable. EM: clear coronavirus particles in both bronchiolar epithelial cells marked by cilia and type II alveolar epithelial cells featured with lamellar body. Diameters of virus particles: 70–100 nm.	Epithelial Vascular

			Digital PCR: positive SARS-CoV-2 virus nucleic acid.	
Yao et al *Zhong hua*	1-3*	NR	Varying degrees of damage to the alveolar structure. Minor serous exudation and fibrin exudation. Organization of exudates in some alveolar cavities. Hyaline membrane formation in some alveoli. Immune infiltrate in alveoli: mostly macrophages, monocytes. Some multinucleated giant cells. Few lymphocytes, eosinophils, and neutrophils. Alveolar epithelium: type II hyperplasia; focal desquamation, degeneration, shedding, necrosis. Septal blood vessels dilated, congested, edematous, with modest infiltration of monocytes and lymphocytes. Hyaline thrombi in a minority of microvessels. Focal hemorrhage in lung tissue. Pulmonary interstitial fibrosis. Bronchial epithelia: partly exfoliated. IHC: many CD68 positive cells. CD4 positive cells, no CD8 or CD20. CK and TTF-1 positive. Some alveolar type II epithelial cells positive for 2019-nCoV-spike protein S1 and nucleocapsid protein. EM: thin type II epithelium, swollen mitochondria, more lamellar bodies, rough ER, smooth Golgi apparatus. Some type II alveolar epithelial cells and bronchial mucosal epithelia contain coronavirus particles. RT-PCR: positive for 2019-nCoV nucleic acid.	n=3 Epithelial n=3 Vascular
Zeng et al	NA	Two fragments of lung tissues were obtained from the right lower lobectomy, measuring 12.5x5.7x3.0 cm and 11.9x9.0x3.5 cm. No area of consolidation or nodular lesions could be found on gross examination.	Viral pneumonia near the visceral pleura: exudative inflammation of monocytes and lymphocytes, centered small blood vessels, which further infiltrate the surrounding alveolar septa and spaces. Alveolar septa: widened, with obvious hyperemia, dilation of capillaries. Interstitial fibrosis was rarely present. Alveolar spaces: large number of monocytes, a few lymphocytes and variable numbers of red blood cells to form clumps. Multinucleated giant cells. No fibrinous exudate, hyaline membrane formation. Areas of serous exudation with pulmonary edema: some alveolar spaces were filled with a large amount of light red, homogenous proteinaceous fluid, admixed with variable numbers of red blood cells, lymphocytes and monocytes. Also scattered large protein globules in alveolar spaces. Intact bronchial structure. focal hyperplasia of type II pneumocytes (some mild cytologic atypia). Some enlarged pneumocytes abundant cytoplasm with a ground-glass appearance, and prominent eosinophilic nucleoli. Meningothelial-like nodules near the hilum with extensive thick-walled vessels. Only capillary dilation and congestion were observed around the nodules. Intracytoplasmic viral-like inclusions in a few type II pneumocyte-like cells or macrophage-like cells.	Epithelial

			IHC: Inflammatory cells infiltrated the blood vessels and alveolar wall were mainly composed of T lymphocytes (T helper cells mostly, scattered cytotoxic T cells) with a few B lymphocytes and plasma cells. Macrophages in alveolar spaces, CD163 (M2 macrophages) positive. PCK, TTF1: alveolar structure essentially intact. The enlarged cells with amphophilic granular cytoplasm and prominent nucleoli along the alveolar spaces recognized on H&E stain were PCK positive. RT-PCR: positive for SARS-CoV-2.	
Zhang et al	NA	NA	DAD (organizing phase); denudation of alveolar lining cells, with presence of reactive type II pneumocyte hyperplasia; intra-alveolar fibrinous exudates, interstitial loose fibrosis with chronic inflammatory infiltrates; intra-alveolar loose fibrous plugs. Intra-alveolar organizing fibrin in most foci. IHC: prominent SARS-CoV-2 Rp3 NP protein expression on alveolar epithelial cells, including damaged, desquamated cells within the alveolar space; minimal expression on blood vessels or in the interstitial areas between alveoli.	Epithelial Vascular (possibly AFOP) Fibrotic

*Data are aggregated and not described for each individual case.

** Row displays general findings that are applicable to all cases included in the study. Individual patient data are described in the rows below.

Abbreviations: NA - Not applicable, NR - not reported, PE - pulmonary embolism, EM - electron microscopy, IHC - immunohistochemistry, AFOP - acute fibrinous organizing pneumonia, ARDS - acute respiratory distress syndrome, DAD - diffuse alveolar damage, NSIP - nonspecific interstitial pneumonia, RT-PCR - reverse transcription polymerase chain reaction, CLL - chronic lymphocytic leukemia.

Supplemental Table S5. Cardiovascular pathology reports (n=61 cases)

Article	Case number	Macroscopic findings	Microscopic findings (histology, and, when applicable, additional experiments)
Barton et al	1	Weight: 402 g. CHD, marked 2 vessel. No adhesions, effusions, or thrombi	Thrombi within a few small pulmonary artery branches Acute myocardial ischemia Coronary artery atherosclerosis with marked two-vessel disease
	2	Heart weight: 372 g. No adhesions, effusions, or thrombi. Mild CHD	Mild coronary artery atherosclerosis
Bradley et al	1-12*	Varying degrees of atherosclerotic CHD (3/5) Cardiomegaly (>450g) (3/5)	The majority of cardiac findings were associated with prior injury or hypertensive changes. Myocyte hypertrophy (12/12), interstitial fibrosis (10/12), histologic evidence of remote infarct (3/12), foci of lymphocytic inflammation associated with acute myocyte necrosis, consistent with lymphocytic myocarditis (1/12)
Fox et al	1-3*	Weight: 430-550g. Mild to moderate serosanguinous pericardial effusions. Right ventricular dilatation (1/3). Myocardium free of significant lesions (3/3). No significant stenosis or thrombi in coronary arteries.	Scattered individual cell myocyte necrosis (3/3), no large/confluent areas of myocyte necrosis. In rare areas, lymphocytes were adjacent to, but not surrounding degenerating myocytes; no significant brisk lymphocytic inflammatory infiltrate with pattern of viral myocarditis. No obvious viral cytopathic effect by light microscopy
Liu et al	NA	Epicardium: mildly edematous Myocardium has a grayish-red color and a fish/meat-like texture.	NR
Menter et al	1-17, 19-21 (n=20)	SARS-CoV2-specific RT-qPCR: variable, predominantly low levels of RNA copy numbers.	

	1	Autopsy findings: Generalised atherosclerosis, bilateral pleural effusions, mild myocardial hypertrophy, coronary sclerosis, myocardial infarction scar, heart weight: 360g, 65. percentile. Peracute myocardial contraction band necroses (“sequelae of shock”)	NR
	2	Autopsy findings: Generalised atherosclerosis, normal heart weight, stenosis of RIVA and RCA, diffuse myocardial fibrosis, heart weight: 358g, 60. percentile	NR
	3	Autopsy findings: Eccentric myocardial hypertrophy	NR
	4	Autopsy findings: Cor bovinum maximum (780 g, >95. percentile), peracute posterior myocardial infarction, biological aortic valve implant, severe coronary sclerosis	NR
	5	Autopsy findings: Eccentric biventricular myocardial hypertrophy (425 g, 85. percentile), fibrosis of papillary muscles, valvular degeneration, coronary sclerosis, biatrial dilation, generalised atherosclerosis. Contraction band necrosis in myocardium (“sequelae of shock”).	NR
	6	Autopsy findings: Eccentric myocardial hypertrophy (430 g, 60. percentile), posterior myocardial fibrosis, valvular degeneration, coronary sclerosis and stenosis of RIVA, infrarenal atherosclerosis	NR
	7	Autopsy findings: Eccentric myocardial hypertrophy (440g, >95. percentile), dilation of left atrium with organising thrombi, posterior	NR

		myocardial infarction scar, fibrosis of papillary muscles, sclerosis of coronary arteries with stent in RVA, valvular degeneration, generalised atherosclerosis	
	8	Autopsy findings: Eccentric myocardial hypertrophy with biatrial dilation (510 g, 95. percentile), apical aneurysm of left ventricle with adherent thrombus, posterolateral myocardial infarction scar, fibrosis of papillary muscles, stenoses of RVA, RIVA, RCX, double bypass. S.p. left atrial appendage resection. aortic valve implant, generalized atherosclerosis, aortic aneurysm with prosthesis. Pericardial adhesions	NR
	9	Autopsy findings: Senile ATTR amyloidosis. Eccentric myocardial hypertrophy (480g, 90. percentile) with biatrial dilation, myocardial left-ventricular fibrosis, endocardial fibrosis, coronary sclerosis	NR
	10	Autopsy findings: Senile interstitial ATTR amyloidosis. Eccentric myocardial hypertrophy (right > left, 550g, > 95. percentile) with biatrial dilation, coronary sclerosis, atherosclerosis	NR
	11	Autopsy findings: Eccentric myocardial hypertrophy (left > right, 460g, >95. percentile).	NR
	12	Autopsy findings: Senile cardiac ATTR amyloidosis, eccentric hypertrophy (435g, 80. percentile), coronary sclerosis, infrarenal atherosclerosis	NR

	13	Autopsy findings: Eccentric hypertrophy (655g, >95. percentile) coronary sclerosis, patent foramen ovale, atherosclerosis	NR
	14	Autopsy findings: Cardiac vascular ATTR amyloidosis	NR
	15	Autopsy findings: Cardiac interstitial ATTR amyloidosis, myocardial fibrosis	NR
	16	Autopsy findings: Eccentric hypertrophy (550g, >95. percentile), coronary sclerosis, myocardial fibrosis, biatrial dilation, generalized atherosclerosis	NR
	17	Autopsy findings: Mild eccentric hypertrophy (450g, 85. percentile), biatrial dilation	NR
	19	Autopsy findings: Eccentric hypertrophy (485g, > 95. percentile), myocardial infarction scar, coronary sclerosis (stent in RIVA), patent foramen ovale, atherosclerosis	NR
	20	Autopsy findings: Heart weight 475g, 50. percentile, coronary artery sclerosis, generalised atherosclerosis	NR
	21	Autopsy findings: Senile cardiac ATTR amyloidosis Eccentric hypertrophy (570g, >95. percentile) with biatrial dilation, coronary sclerosis , pacemaker in right ventricle; severe generalised atherosclerosis	NR
Schweitzer et al	NA	Weight 340 g. Coronary artery atherosclerosis with marked two-vessel disease. No macroscopic signs of myocardial ischemia.	No relevant histological findings (such as contraction band necrosis, infarction, or inflammation) were noted.

Tavazzi et al	NA	NA	Low-grade interstitial and endocardial inflammation. Minimal, focal, and perivascular interstitial fibrosis. Cardiac myocytes: focal myofibrillar lysis, lipid droplets. No myocyte hypertrophy or nuclear changes. Large, vacuolated CD68-positive macrophages with membrane damage and cytoplasmic vacuoles. Single or small groups of viral particles with the morphology and size of coronaviruses. Small intramural vessels were free from vasculitis and thrombosis.
Tian *Mod Pathol*	1	NA	Focal mild edema, interstitial fibrosis, and myocardial hypertrophy; no inflammatory cellular infiltration. RT-PCR assay for SARS-COV-2: positive
	4	NA	Focal mild edema, interstitial fibrosis, and myocardial hypertrophy; no inflammatory cellular infiltration. RT-PCR assay for SARS-COV-2: negative
Varga et al	1	NR	Prominent endotheliitis with recruitment of inflammatory cells, as well as an unusual high amount of apoptotic bodies in many organs, especially in the pulmonary vessels but also in small bowel and heart.
	2	NR	Lymphocytic endotheliitis in lung, heart, kidney, and liver. Histology showed an acute posterior MI, but no signs of viral lymphocytic myocarditis.
Wichmann et al	1-12**	All cases except for case 6 presented with preexisting heart disease, including high-grade coronary artery sclerosis (7 of 12); myocardial scarring, indicating ischemic heart disease (6 of 12); and congestive cardiomyopathy.	RT-PCR positive in heart (n=5) RT-PCR positive in saphenous vein (n=4)
	1	Heart: weight 660g. Excentric hypertrophy of both ventricles. Arteries: atherosclerosis Veins: DVT involving both legs. Thrombosis of prostatic vein	NR
	2	Heart: weight 515g. CHD with stenting, status post MI, cardiac aneurysm, hypertrophy Arteries: atherosclerosis	NR

		Veins: normal	
	3	Heart: weight 510g. Biventricular hypertrophy, moderate CHD. Arteries: atherosclerosis Veins: DVT involving both legs, phlebosclerosis	Lymphocytic myocarditis: mononuclear infiltrations consisting of lymphocytes in the myocardium of the right ventricle.
	4	Heart: weight 605g. Left ventricular hypertrophy. Arteries: slight atherosclerosis Veins: DVT involving both legs.	NR
	5	Heart: weight 360g. CHD, status post MI. Arteries: slight atherosclerosis Veins: DVT involving both legs.	NR
	6	Heart: weight 250g. No abnormalities. Arteries: normal Veins: normal	NR
	7	Heart: weight 415g. CHD, moderate hypertrophy, calcification of the mitral ring, status post MI, pacemaker, lipomatosis cordis. Arteries: atherosclerosis Veins: normal	NR
	8	Heart: weight 575g. CHD, status post bypass surgery, status post MI, cardiac aneurysm, global hypertrophy. Arteries: atherosclerosis Veins: DVT involving both legs.	NR
	9	Heart: weight 355g. Left atrial dilatation, CHD, status post MI. Arteries: atherosclerosis Veins: normal	NR

	10	Heart: weight 390g. CHD, status post MI. Arteries: normal Veins: normal	NR
	11	Heart: weight 650g. CHD, status post aortic valve replacement, biventricular hypertrophy. Arteries: atherosclerosis Veins: DVT involving both legs.	NR
	12	Heart: weight 745g. CHD, hypertrophy. Arteries: atherosclerosis Veins: DVT involving both legs.	NR
Xu *Lancet*	NA	NA	Few interstitial mononuclear inflammatory infiltrates No other substantial damage in the heart tissue
Yao et al *Cell Res*	NA	NA	Neither coronavirus particles nor SARS-CoV-2 nucleocapsid were detected in the heart.
Yao et al *Zhonghua l*	1-3*	NA	Cardiomyocyte hypertrophy, necrosis of some cardiomyocytes, mild hyperemia, edema. Mild infiltration of lymphocytes, monocytes, neutrophils (IHC: mainly macrophage infiltration, few CD4+cells, no CD8- or CD20- positivity). One case had an old infarction. No 2019-nCoV virus component in myocardial tissue based on EM, IHC and RT-PCR.

*Data are aggregated and not described for each individual case.

** Row displays general findings that are applicable to all cases included in the study. Individual patient data are described in the rows below.

Abbreviations: NA - Not applicable, NR - not reported, CHD - coronary heart disease, MI - myocardial infarction, RT-PCR - reverse transcription polymerase chain reaction, EM - electron microscopy, IHC - immunohistochemistry.

Supplemental Table S6. Hepatobiliary pathology reports (n=55 cases)

Article	Case number	Macroscopic findings	Microscopic findings (histology, and, when applicable, additional experiments)
Barton et al	1	Liver weight: 2.232g. Hepatic centrilobular steatosis. Right upper quadrant adhesions. Cholecystectomy.	NR
	2	Liver weight: 1.683g. Hepatic cirrhosis, advanced. Cholecystectomy.	NR
Bradley et al	1-12*	Liver weight: 1.749g (range 1068-2425g; n=5) Congestion (5/5)	Chronic changes associated with pre-existing comorbidities. Liver inflammation was not prominent, although some patients displayed mild periportal lymphocytic inflammation. Congestion (9 out of 12 patients), steatosis (8/12), periportal lymphocytic inflammation (3/12), centrilobular necrosis (2/12), lobular neutrophilic inflammation (1/12)
Lagana et al	NA	NA	Acute cellular rejection: expansion of mixed inflammatory infiltrate, lymphocytic cholangitis and reactive changes of interlobular bile ducts, mild portal venulitis (overall rejection activity index 5). Moderate acute hepatitic pattern of injury: azonal pattern of clusters of apoptotic hepatocytes, singly dispersed apoptotic hepatocytes. Fragments of cytoplasmic debris were somewhat large, giving the impression of “crumbling” hepatocytes. Few scattered mitotic figures. Regions of Kupffer cell prominence with sinusoidal and central vein endotheliitis. Mild steatosis, generally macrovesicular (though small droplets) No viral inclusions were present. IHC for cytomegalovirus and adenovirus: negative
Menter et al	1	Autopsy findings: Macro- and microvesicular steatosis	
	2	Autopsy findings: Liver cirrhosis with macrovesicular steatosis and portal inflammation (NASH/ASH)	
	3	Autopsy findings: Hepatic congestion, periportal microvesicular steatosis, cholestasis	

	5	Autopsy findings: Macrovesicular steatosis. Perivenular haemorrhagic necroses in the liver (“sequelae of shock”).
	6	Autopsy findings: Shock necroses of liver (“sequelae of shock”)
	7	Autopsy findings: Macro- and microvesicular steatosis. Hepatic congestion (“sequelae of shock”)
	8	Autopsy findings: Macro- and microvesicular steatosis (20%)
	9	Autopsy findings: Haemorrhagic necrosis of liver (“sequelae of shock”)
	10	Autopsy findings: Hepatic congestion, macro- and microvesicular steatosis (15%)
	11	Autopsy findings: Hepatic congestion
	12	Autopsy findings: Macro- and microvesicular steatosis
	13	Autopsy findings: Steatohepatitis and micronodular liver cirrhosis
	16	Autopsy findings: Steatohepatitis
	17	Autopsy findings: No abnormalities
	19	Autopsy findings: Macrovesicular steatosis (30%). Cholecystolithiasis.
	20	Autopsy findings: Macrovesicular steatosis (5%). Hepatic congestion (“sequelae of shock”)
	21	Autopsy findings: Macro- and microvesicular steatosis (40%)

Schweitzer et al	NA	NR	Liver with micro- and macrovesicular steatosis and what appears to be the correlate for acute dystrophy; scant lymphocyte accumulations in portal triads but no infiltrates.
Tian *Mod Pathol*	1	NA	Nuclear glycogenation in hepatocyte; focal mild macrovesicular steatosis, neoplastic lymphocytic accumulation in portal tracts; ductopenia in some portal tracts. RT-PCR assay for SARS-COV-2: positive
	2	NA	Cirrhosis (pre-existing); specimen too limited for further evaluation
	3	NA	Mild zone 3 sinusoidal dilatation, patchy hepatic necrosis, mild increase in sinusoidal lymphocytes. RT-PCR assay for SARS-COV-2: negative
	4	NA	Mild zone 3 sinusoidal dilatation, patchy hepatic necrosis in periportal area and centrilobular area, Kupffer cells hyperplasia in focal sinusoids, mild increase in sinusoidal lymphocytes, scanty lymphocytes in the portal tracts RT-PCR assay for SARS-COV-2: negative
Varga et al	2	NR	Lymphocytic endotheliitis in lung, heart, kidney, and liver. Liver with massive centrilobular and parenchyma necrosis.
Wichmann et al	1-12**	NA	RT-PCR positive (n=5)
	1	Macroscopic: hepatomegaly. Weight 3880 g.	NR
	2	Macroscopic: shock liver. Weight 2030 g.	NR
	3	Macroscopic: shock liver. Weight 1930 g.	NR
	4	Macroscopic: shock liver. Weight 2180 g.	NR

	5	Macroscopic: normal. Weight 1645 g.	NR
	6	Liver macroscopic: normal. Weight 890 g. Cholecystolithiasis.	NR
	7	Liver macroscopic: normal. Weight 1380 g. Cholecystolithiasis.	NR
	8	Liver macroscopic: chronic congestion. Weight 1610 g. Status post cholecystectomy.	NR
	9	Macroscopic: fatty change. Weight 715 g.	NR
	10	Liver macroscopic: normal. Weight 945 g. Cholecystolithiasis.	NR
	11	Macroscopic: normal. Weight 1450 g.	NR
	12	Macroscopic: chronic congestion, fatty changes. Weight 2265 g.	NR
Xu *Lancet*	1	NA	Moderate microvesicular steatosis and mild lobular portal activity
Yao et al *Cell Res*	NA	NA	Neither coronavirus particles nor SARS-CoV-2 nucleocapsid were detected in the liver.
Yao et al *Zhonghua*	1-3*	NA	Hepatocellular degeneration, focal necrosis. Small bile plug in bile duct (unclear in how many cases)

*Data are aggregated and not described for each individual case.

** Row displays general findings that are applicable to all cases included in the study. Individual patient data are described in the rows below.

Abbreviations: NA - Not applicable, NR - not reported, IHC - immunohistochemistry, RT-PCR - reverse transcription polymerase chain reaction.

Supplemental Table S7. Renal pathology reports (n=82 cases)

Article	Case number	Macroscopic findings	Microscopic findings (histology, and, when applicable, additional experiments)
Bradley et al	1-12*	Hypertensive renal surface changes (5/5). Granular surface/scarring (4/5). Poor corticomedullary differentiation (1/5)	Prominent proximal tubular epithelial cell vacuolization consistent with acute tubular injury (1/5), acute tubular injury not analyzed (autolysis, 4/5) Chronic changes, including mild-moderate arterionephrosclerosis (9/12), moderate-severe arterionephrosclerosis (3/12), diabetic changes (3/12)). Amyloidosis (1/12). EM: viral particles in cytoplasm or vesicles in tubular epithelium (mostly proximal tubules and endothelial cells), rarely in podocytes.
Diao et al	1-6**	NR	Acute tubular necrosis (n=1 extremely severe, n=2 severe, n=3 moderate), luminal brush border sloughing and vacuole degeneration in different areas. Lymphocytic infiltration: n=2 severe infiltration in tubulointerstitium; n=3 moderate infiltration; n=1 absent n=3 viral infection associated-syncytia n=6 dilated capillary vessels in the glomeruli. No severe glomerular injury, although benign hypertensive glomerulosclerosis and autolysis were noted in three hypertensive patients IHC: cytoplasmic expression of viral NP antigen on kidney tubules and on inclusion bodies. Very low C5b-9 deposition on glomeruli and capillaries. EM (n=2): markedly swollen cell in infected renal tissues with expansion of mitochondria, lysosomes and dilation of rough - and smooth endoplasmic reticulum. Virus-like particles in both cases.
	1	NR	Severe tubular necrosis, severe intestinal infiltration, moderate autolysis, slightly benign glomerulosclerosis IHC: Viral NP antigen: severe; C5b-9 on tubules: moderate; CD68 moderate, no CD8, no CD56. RT-PCR of SARS-CoV-2: positive
	2	NR	Severe tubular necrosis, severe intestinal infiltration, slightly autolysis, slightly benign glomerulosclerosis IHC: Viral NP antigen: extremely severe; C5b-9 on tubules: extremely severe; CD68: extremely severe, CD8: moderate, CD56 slightly. RT-PCR of SARS-CoV-2: positive

	3	NR	Moderate tubular necrosis, moderate intestinal infiltration, slightly autolysis, no benign glomerulosclerosis IHC: Viral NP antigen: severe; C5b-9 on tubules: moderate; CD68: severe, CD8: moderate, CD56: slightly. RT-PCR of SARS-CoV-2: positive
	4	NR	Moderate tubular necrosis, moderate intestinal infiltration, slightly autolysis, slightly benign glomerulosclerosis IHC: Viral NP antigen: severe; C5b-9 on tubules: slightly; CD68: moderate, no CD8,CD56: no/slightly RT-PCR of SARS-CoV-2: positive
	5	NR	Moderate tubular necrosis, moderate intestinal infiltration, slightly autolysis, slightly benign glomerulosclerosis IHC: Viral NP antigen: severe; C5b-9 on tubules: moderate; CD68: moderate, CD8: no/slightly, no CD56. RT-PCR of SARS-CoV-2: positive
	6	NR	Extremely severe tubular necrosis, no intestinal infiltration, slightly autolysis, no benign glomerulosclerosis IHC: Viral NP antigen: severe; C5b-9 on tubules: slightly; CD68 no/slightly, no CD8, no CD56. RT-PCR of SARS-CoV-2: positive
Kissling et al	NA	NA	Two main features: severe collapsing focal segmental glomerulosclerosis (FSGS) and acute tubular necrosis without any significant interstitial inflammation. IF: no significant immune deposits (including anti-C5b-9 staining) EM: numerous spherical particles with typical appearance of viral inclusion body in vacuoles in podocyte cytoplasm RT-PCR for SARS-CoV-2 on kidney tissue and blood: negative
Larsen et al	NA	NA	Glomeruli: 14/24 globally sclerotic. Of intact glomeruli, often tuft collapse with overlying epithelial hypertrophy and hyperplasia in the Bowman space. Intact portions of glomeruli showed minimal mesangial expansion. No endocapillary hypercellularity or necrotizing lesions. Tubules: notable epithelial injury, most prominent proximally, and included reactive nuclei with mitotic figures, diffuse simplification with denudation of brush borders. Interstitial edema with inflammatory infiltrate of predominantly lymphocytes and plasma cells with scattered eosinophils. No tubulitis. Moderate interstitial fibrosis and tubular atrophy in the background. IF: negative for IgA, IgG, IgM, C3, C1q, kappa, lambda.

			EM: slightly thickened basement membranes that were slightly thickened. No immune-type electron-dense deposits. Severe foot process effacement, involving more than 90% of the glomerular basement membrane surface area. Occasional tubuloreticular inclusions in the glomerular endothelial cell cytoplasm. No definitive viral particles. A diagnosis of collapsing glomerulopathy was rendered. APOL1 genotyping: homozygous for the G1 risk allele (rs73885319). In situ analysis with RNAscope for SARS-CoV-2 RNA: negative
Liu et al	1	Granular solidified kidney (authors suspect this is related to the medical history)	NR
Menter et al	1-21**	NR	n=2 EM: prominent activation of podocytes and endothelial cells. The cytoplasm of podocytes contained multiple vesicles, some with attached ribosomes and double membranes. Occasionally, virus-like particles (70 -110 nm) with electron dense granules were detected within these vesicles. Sporadically, these particles were present in endothelial cells and proximal tubular epithelial cells. SARS-CoV2-specific RT-qPCR: variable, predominantly low levels of RNA copy numbers were detected.
	1	Renal congestion, arteriosclerosis, vascular scarring	Acute tubular damage in kidney (“sequelae of shock”)
	2	Glomerulosclerosis, interstitial fibrosis and tubular atrophy	Acute tubular damage in kidney (“sequelae of shock”)
	3	Granular atrophy	NR
	5	Hypertensive nephropathy and arteriosclerosis, vascular scarring	Acute tubular damage with osmotic nephrosis in kidney (“sequelae of shock”)
	6	Vascular scarring, diabetic glomerulopathy, diffuse/nodular glomerulosclerosis and severe arteriolosclerosis, pyelitis	Diffuse acute tubular damage in kidney (“sequelae of shock”)

	7	Arteriolosclerosis, vascular scarring	Diffuse acute tubular damage in kidney (“sequelae of shock”)
	8	Vascular scarring, nephrocalcinosis, arteriolosclerosis	NR
	9	Vascular scarring, nephrocalcinosis	Diffuse acute tubular damage in kidney (“sequelae of shock”)
	10	Interstitial fibrosis and tubular atrophy	NR
	11	No abnormalities	NR
	12	Autopsy findings: diabetic nephropathy, signs of renal shock	
	13	No abnormalities	NR
	16	Autopsy findings: signs of renal shock	
	17	Autopsy findings: Renal infarction, glomerular microangiopathy, arteriosclerosis. Thrombotic angiopathy in greater vessels kidneys (als in periphery arteries of lungs) and glomerular microangiopathy	
	19	Vascular scarring, fatty atrophy	NR
	20	Autopsy findings: Vascular scarring, Systemic thrombotic angiopathy glomerula of kidneys (also in lungs, left adrenal gland)	
	21	No abnormalities	NR
Schweitzer et al	NA	NR	Acute tubular necrosis
Su et al	1	NR	Tubule interstitium: severe acute tubular injury, mild to moderate arteriosclerosis
	2	NR	Tubule interstitium: moderate acute tubular injury, mild arteriosclerosis. EM: virus particles
	3	NR	Tubule interstitium: mild to moderate acute tubular injury, pigmented casts, mild arteriosclerosis. EM: virus particles, subendothelial lucent expansion.

	4	NR	Glomeruli: FSGS. Tubule interstitium: severe acute tubular injury, severe arteriosclerosis. EM: virus particles, subendothelial lucent expansion
	5	NR	Tubule interstitium: mild acute tubular injury, mild arteriosclerosis
	6	NR	Tubule interstitium: mild to moderate acute tubular injury, pigmented casts, mild arteriosclerosis
	7	NR	Tubule interstitium: severe acute tubular injury, mild to moderate arteriosclerosis
	8	NR	Glomeruli: focal segmental fibrin thrombus, FSGS. Tubule interstitium: moderate acute tubular injury, severe arteriosclerosis.
	9	NR	Tubule interstitium: moderate acute tubular injury, pigmented casts, moderate arteriosclerosis
	10	NR	Tubule interstitium: moderate acute tubular injury, moderate to severe arteriosclerosis
	11	NR	Glomeruli: focal segmental fibrin thrombus. Tubule interstitium: moderate to severe acute tubular injury, moderate to severe arteriosclerosis
	12	NR	Tubule interstitium: moderate to severe acute tubular injury, moderate arteriosclerosis. EM: virus particles, subendothelial lucent expansion
	13	NR	Tubule interstitium: mild to moderate acute tubular injury, mild arteriosclerosis
	14	NR	Glomeruli: diffuse segmental fibrin thrombus. Tubule interstitium: severe acute tubular injury, multiple focal bacteria in tubule interstitium, moderate arteriosclerosis.
	15	NR	Tubule interstitium: mild to moderate acute tubular injury, moderate to severe arteriosclerosis
	16	NR	Tubule interstitium: severe acute tubular injury, multiple focal bacteria in tubule interstitium, moderate to severe arteriosclerosis
	17	NR	Tubule interstitium: moderate acute tubular injury, moderate arteriosclerosis
	18	NR	Tubule interstitium: moderate acute tubular injury, mild arteriosclerosis
	19	NR	Tubule interstitium: mild acute tubular injury, moderate to severe arteriosclerosis

	20	NR	Tubule interstitium: moderate to severe acute tubular injury, moderate arteriosclerosis EM: virus particles. IF: no IgG, no IgA, no SARS-CoV nucleoprotein
	21	NR	Tubule interstitium: mild acute tubular injury, moderate to severe arteriosclerosis EM: dense deposits, subendothelial lucent expansion. IF: IgG expression, no IgA, no SARS-CoV nucleoprotein
	22	NR	Tubule interstitium: moderate acute tubular injury, mild to moderate arteriosclerosis. EM: virus particles, IF: SARS-CoV nucleoprotein expression, no IgG, no IgA.
	23	NR	Tubule interstitium: moderate acute tubular injury, moderate arteriosclerosis EM: no virus dense particles, no dense deposits, no subendothelial lucent expansion IF: SARS-CoV nucleoprotein expression, no IgG, no IgA
	24	NR	Tubule interstitium: Mild acute tubular injury, mild arteriosclerosis
	25	NR	Tubule interstitium: moderate to severe acute tubular injury, moderate to severe arteriosclerosis, EM: virus particles, dense deposits, subendothelial lucent expansion IF: IgA expression, no IgG, SARS-CoV nucleoprotein expression
	26	NR	Tubule interstitium: mild to moderate acute tubular injury, mild arteriosclerosis
Varga et al	1	NR	EM: viral inclusion bodies in a peritubular space and viral particles in endothelial cells of the glomerular capillary loops. Aggregates of viral particles appear with dense circular surface and lucid centre. Peritubular space consistent with capillary containing viral particles. Glomerular basement membrane with endothelial cell and a viral particle.
	2	NR	Lymphocytic endotheliitis in lung, heart, kidney, and liver.
Wichmann et al	1-12**	NA	RT-PCR positive in kidney (n=5)
	1	Macroscopic: shock kidneys. Weight 215 g (right), 305 g (left).	NR
	2	Macroscopic: normal. Weight 155 g (right), 1555 g (left).	NR

	3	Macroscopic: normal. Weight 240 g (right), 240 g (left).	NR
	4	Macroscopic: normal. Weight 2150 g (right), 210 g (left).	NR
	5	Macroscopic: normal. Weight 180 g (right), 165 g (left).	NR
	6	Macroscopic: old infarctions/chronic inflammation, nephrolithiasis. Weight 80 g (right), 90 g (left).	NR
	7	Macroscopic: normal. Weight 105 g (right), 120 g (left).	NR
	8	Macroscopic: cysts. Weight 145 g (right), 160 g (left).	NR
	9	Macroscopic: shrinkage (left kidney). Weight 100 g (right), 35 g (left).	NR
	10	Macroscopic: arteriosclerosis, atrophy, cysts. Weight 115 g (right), 145 g (left).	NR
	11	Macroscopic: normal. Weight 225 g (right), 240 g (left).	NR
	12	Macroscopic: multiple cysts. Weight 270 g (right), 220 g (left).	NR
Yao et al *Zhonghua*	1-3*	NA	Glomeruli: swollen endothelial cells, small amount of proteinous exudate Tubules: epithelium edematous with focal degeneration. In the lumen protein and pigment casts. Capillaries: transparent thrombus

*Data are aggregated and not described for each individual case.

** Row displays general findings that are applicable to all cases included in the study. Individual patient data are described in the rows below.

Abbreviations: NA - not applicable, NR - not reported, EM - electron microscopy, IHC - immunohistochemistry, RT-PCR - reverse transcription polymerase chain reaction, IF - immunofluorescence.

Supplemental Table S8. Brain pathology reports (n=33 cases)

Article	Case number	Macroscopic findings	Microscopic findings (histology, and, when applicable, additional experiments)
Barton et al	1	No macroscopic abnormalities	NR
	2	No macroscopic abnormalities	NR
Bradley et al	NR	1 case: Scattered punctate subarachnoid hemorrhages.	RT-PCR: no detectable levels of viral RNA
Liu et al	NA	Cerebral edema, light cortex atrophy as was expected with history of cerebral infarctions.	No specific manifestations of infection seen.
Menter et al	1-3,5-13,16,16,19-21 (n=17)	SARS-CoV2-specific RT-qPCR: variable, predominantly low levels of RNA copy numbers were detected. In the brain, copy numbers were generally low, although values in the olfactory bulb were higher than the brain stem.	
	1	Autopsy findings: Demyelising lesions in frontal, parietal and occipital semioval centre. Loss of myelin sheaths without inflammatory changes, indicative of chronic MS lesions; atrophy of optic nerve	
	2	Autopsy findings: No abnormalities	
	3	Autopsy findings: Hydrocephalus internus, Parkinson's disease with Lewy bodies in substantia nigra and locus coeruleus with moderate loss of dopaminergic neurons. Arteriosclerosis of perivascular space of basal ganglia, acute hypoxic ischemic encephalopathy of hippocampus	
	5	Autopsy findings: No abnormalities	
	6	Autopsy findings: No abnormalities	
	7	Autopsy findings: No abnormalities	
	8	Autopsy findings: Severe atherosclerotic changes of C. arteriosus, hydrocephalus internus, cerebral oedema	
	9	Autopsy findings: No abnormalities	
	10	Autopsy findings: No abnormalities	
	11	Autopsy findings: No abnormalities	

	12	Autopsy findings: No abnormalities	
	13	Autopsy findings: No abnormalities	
	16	Autopsy findings: No abnormalities	
	17	Autopsy findings: No abnormalities	
	19	Autopsy findings: No abnormalities	
	20	Autopsy findings: No abnormalities	
	21	Autopsy findings: No abnormalities	
Wichmann et al	1-12**	NR	RT-PCR positive in brain (n=4)
	1	Macroscopy: weight 1520 g	NR
	2	Macroscopy: weight 1430 g	NR
	3	Macroscopy: weight 1665 g	NR
	4	Macroscopy: weight 1435 g	NR
	5	Macroscopy: weight 1450 g	NR
	6	Macroscopy: weight 950 g, small brain.	NR
	7	Macroscopy: weight 1210 g	NR
	8	Macroscopy: weight 1170 g. Cerebral sclerosis.	NR
	9	Macroscopy: weight 1080 g	NR
	10	Macroscopy: weight 1350 g. Suspected septic encephalomalacia (brain dissection pending)	NR
	11	Macroscopy: weight 1400 g	NR
	12	Macroscopy: weight 1460 g. Cerebral sclerosis.	NR

*Data are aggregated and not described for each individual case.

** Row displays general findings that are applicable to all cases included in the study. Individual patient data are described in the rows below.
Abbreviations: NA - not applicable, NR - not reported, RT-PCR - reverse transcription polymerase chain reaction.

Supplemental Table S9. Pathological findings in other organs and tissues

Article	Study	Case number	Pathological findings
Gastrointestinal tract (n=32)	Barton et al	1	Gross examination: mouth, esophagus, stomach, bowel no abnormalities. Increased visceral adipose. No appendix present.
		2	Gross examination: mouth, esophagus, appendix no abnormalities. Stomach and bowel gaseous distension. Increased visceral adipose.
	Bradley et al	1-5*	Esophagus, stomach, intestines: RT-PCR showed no detectable levels of viral RNA EM: viral particles (~70-100 nm) in large intestines (both individually or in aggregates in the cytoplasm or inside vesicles)
	Liu et al	NA	Small intestine: segmentally dilated and narrowed. In rest of GI tract no abnormalities.
	Menter et al	9	Erosive gastritis
		12	Diverticulosis of sigmoid colon
		19	Diverticulosis
		20	Glycogenic anthracosis of oesophagus
	Varga et al	1	Prominent endotheliitis with recruitment of inflammatory cells, as well as an unusual high amount of apoptotic bodies in many organs, especially in the pulmonary vessels but also in small bowel and heart.
		2	Mucosal ischemic necrosis as well as prominent endotheliitis and many apoptotic bodies of the submucosal vessels with only scattered fibrin thrombi, almost identical to case 3.
		3	Ischemic mucosal necrosis and prominent endotheliitis of the submucosal vessels along with a large amount of apoptotic bodies
	Wichmann et al	1	Macroscopic: ischemic enterocolitis
		2	Macroscopic: normal
		3	Macroscopic: ischemic enterocolitis, diverticulosis

		4	Macroscopic: ischemic enterocolitis
		5	Macroscopic: status post abdominal surgery
		6	Macroscopic: adenoma of the duodenum
		7	Macroscopic: normal
		8	Macroscopic: diverticulosis of the small bowel
		9	Macroscopic: pseudomembranous colitis
		10	Macroscopic: normal
		11	Macroscopic: normal
		12	Macroscopic: normal
	Xiao et al	NA	Significant damage to mucous epithelium. Patchy infiltrate of lymphocytes in esophageal squamous epithelium. In lamina propria of stomach, duodenum, and rectum: numerous infiltrating plasma cells and lymphocytes with interstitial edema. IF of SARS-CoV-2 nucleoprotein: expression in cytoplasm of gastric, duodenal, and rectum glandular epithelial cell, but not in esophageal epithelium.
	Yao et al *Cell Res*	NA	Neither coronavirus particles nor SARS-CoV-2 nucleocapsid were detected in the intestine.
	Yao et al *Zhonghua*	1-3*	Partial epithelial degeneration, necrosis, shedding of gastric and intestinal mucosa, lamina propria and submucosa. No coronavirus (particles) detected in tissue apart from the lungs.
Pancreas (n=14)	Barton et al	1	No macroscopic abnormalities
		2	No macroscopic abnormalities
	Bradley et al	1-5*	Macroscopy: largely unremarkable
	Menter et al	5	Haemorrhagic fatty necroses of pancreas (“sequelae of shock”)
		9	Cystic lesion of pancreas (PanIN)

		19	Lipomatosis of pancreas
	Wichmann et al	10	Macroscopy: pancreatic fibrosis
	Yao et al *Zhonghua*	1-3*	Pancreas: small number of degenerated islet cells. No other abnormalities. No coronavirus (particles) detected in tissue apart from the lungs.
Genitourinary: bladder (n=5), prostate (n=17), testicle (n=17)	Bradley et al	1-5*	Bladder - RT-PCR SARS-CoV-2: no detectable levels of viral RNA
	Menter et al	1-17**	Testicles: SARS-CoV2-specific RT-qPCR: variable, predominantly low levels of RNA copy numbers.
		6	Prostate: Adenocarcinoma of prostate ypT3a, pN1, pM1b, V0, L1, Pn1
		12	Prostate: Benign prostatic hyperplasia
		19	Prostate: benign prostate hyperplasia
		20	Prostate: Benign prostatic hyperplasia
		21	Adenocarcinoma of prostate pT3, L0, V0, Pn1
	Wichmann et al	1-12**	Microthrombi occasionally within the prostate
		1	Prostate - Macroscopic: thrombosis
		2	Macroscopic: status post prostatectomy
		3	Prostate - Macroscopic: thrombosis, benign hypertrophy
		4	Prostate - Macroscopic: thrombosis
		5	Prostate - Macroscopic: thrombosis
		8	Prostate - Macroscopic: thrombosis, benign hypertrophy
		10	Prostate - Macroscopic: benign hypertrophy
		11	Macroscopic: status post prostatectomy

		12	Prostate - Macroscopic: thrombosis, benign hypertrophy, bladder: hemorrhagic cystitis.
Endocrine: thyroid, pituitary, adrenals (n=30)	Barton et al	1	Macroscopy: no abnormalities of thyroid, pituitary, adrenals
		2	Multinodular thyroid. Macroscopy: no abnormalities of pituitary, adrenals
	Bradley et al	1-5*	Macroscopy thyroid, pituitary, adrenals: largely unremarkable
	Menter et al	2	Adrenals: adrenal gland fatigue (“sequelae of shock”)
		3	Adrenals: adrenal gland fatigue (“sequelae of shock”)
		6	Adrenals: adrenal gland fatigue (“sequelae of shock”)
		10	Adrenals: adrenal gland fatigue (“sequelae of shock”)
		11	Adrenals: adrenal gland fatigue (“sequelae of shock”)
		16	Thyroid: Struma diffusa with lymphofollicular inflammation in line with Hashimoto’s disease
		20	Adrenals: Systemic thrombotic angiopathy in left adrenal gland (also lungs, glomerula of kidneys). Myelolipoma of right adrenal gland
		21	Adrenals: adrenal gland fatigue (“sequelae of shock”)
	Wichmann et al	1	Macroscopy: normal adrenal glands
		2	Macroscopy: normal adrenal glands
		3	Macroscopy: micronodular hyperplasia of adrenal glands
		4	Macroscopy: normal adrenal glands
		5	Macroscopy: normal adrenal glands
		6	Macroscopy: adenoma in adrenal glands
		7	Macroscopy: micronodular hyperplasia of adrenal glands
		8	Macroscopy: normal adrenal glands

		9	Macroscopy: normal adrenal glands
		10	Macroscopy: normal adrenal glands
		11	Macroscopy: micronodular hyperplasia of adrenal glands
		12	Macroscopy: micronodular hyperplasia of adrenal glands
	Yao et al *Zhonghua*	1-3*	Thyroid: no abnormalities
Lymph tissues: Spleen, bone marrow, lymph nodes (n=56)	Bradley et al	1-5*	1 case: mild splenomegaly (350g). For 5 cases weight ranged 154-350 (average 209g). 3 cases: evidence of splenic white pulp depletion Subcarinal lymph nodes: RT-PCR: detectable levels of viral RNA
	Chen et al *MedRxiv*	1-6*	Significantly lower total lymphocyte counts in spleen, also dominated by lymphocytes undergoing necrosis and apoptosis. Congested, hemorrhagic spleens, lacking lymphoid follicles. Spleen corpuscles: atrophic, with clear interstitial vessels and fibrous tissue hyperplasia in the splenic sinus. IHC: SARS-CoV-2 nucleocapsid protein cytoplasmic expression on spleen & lymph nodes (n=6). In spleen: nucleocapsid protein-positive cells primarily in red pulp and blood vessels, occasionally in white pulp. In lymph nodes: nucleocapsid protein-positive cells within marginal sinus of lymph nodes, capillaries, some in germinal centers. IF: nucleocapsid protein antigen is found in ACE2+ cells, CD68+ macrophages, and CD169+ macrophages in subcapsular sinus of lymph nodes. CD3+ T cells and B220+ B cells are negative.
	Liu et al	NA	Spleen macroscopy: no abnormalities
	Menter et al	1	Bone marrow: osteoporosis
		2	Bone marrow: Therapy-related MDS-EB2-F
		3	Spleen: Splenic hyperplasia of white pulp, atypic lymphocytes and plasmablasts in sinus Bone marrow: no abnormalities Lymph nodes: Reactive hilar lymph nodes with sinus ectasia and increased presence of reactive plasmablasts and capillary stasis
		4	Lymph nodes: Paratracheal lymph nodes with sinus ectasia and increased presence of plasmablasts
		5	Spleen: splenomegaly with reactive changes (“sequelae of shock”) Bone marrow: no abnormalities

		6	Spleen: splenomegaly with reactive changes (“sequelae of shock”) Bone marrow metastases of prostate carcinoma, reactive left shift myelopoeisis
		7	Bone marrow: Left shift myelopoeisis
		8	Bone marrow: no abnormalities
		9	Bone marrow: Waldenström’s macroglobulinaemia
		10	Spleen: Splenic hyaloserositis Bone marrow: no abnormalities
		11	Bone marrow: 1-5% of histiocytes with haemophagocytic activity, in line with HLH
		12	Spleen: acute reactive changes in spleen Bone marrow: no abnormalities
		13	Spleen: Acute reactive changes and splenomegaly (“sequelae of shock”) Bone marrow: Hypercellular with left shift myelopoeisis
		16	Spleen: Acute reactive changes in spleen (“sequelae of shock”) Bone marrow: Spondylosis
		17	Bone marrow: no abnormalities
		19	Bone marrow: no abnormalities
		20	Bone marrow: no abnormalities
		21	Spleen: runny and soft splenic parenchyma (“sequelae of shock”) Bone marrow: no abnormalities
	Wichmann et al	1	Macroscopic: weight 500g. Splenomegaly.
		2	Macroscopic: weight 355g. Enlarged.
		3	Macroscopic: weight 280g. Normal.
		4	Macroscopic: weight 240g. Normal.
		5	Macroscopic: weight 310g. Normal.

		6	Macroscopic: weight 90g. Normal.
		7	Macroscopic: weight 95g. Normal.
		8	Macroscopic: weight 260g. Chronic congestion.
		9	Macroscopic: weight 50g. Nonspecific acute splenitis.
		10	Macroscopic: weight 135g. Nonspecific acute splenitis.
		11	Macroscopic: weight 240g. Normal.
		12	Macroscopic: weight 360g. Chronic congestion.
	Xu, Chang et al	1-10*	Spleen: The cell composition of the spleen decreased, white pulp atrophied at different levels, meanwhile lymphoid follicles decreased or absent; in addition, the ratio of red pulp to white pulp increased with varying degrees. n=7 more neutrophil infiltration, n=5 scattered plasma cell infiltration. n=1 macrophage proliferation and hemophagocytic phenomena in a few cells. n=2 necrosis and lymphocytic apoptosis n=1 small artery thrombosis and spleen infarction n=1 fungal infection in 1 case IHC: T and B lymphocyte components of the spleen in all cases decreased in varying degrees. n=8: CD20+B cells accumulation in the lymphoid sheath around the splenic artery. In n=2: CD20 and CD21 → number of white pulp almost normal, and splenic nodules were atrophic. CD3+, CD4+ and CD8+ T cells were decreased. n=9 CD68+ macrophages: no significant changes in the distribution and quantity. n=1 more CD68+ cells in the medullary sinuses (related to fungal infection). Few CD56+ cells. EBV negative by in situ hybridization. RT-PCR of nucleic acid of 2019-nCov virus: 1/10 positive, 39 years old. 9/10 negative. EM: 2/10: Coronavirus particles in the cytoplasm of macrophage
	Yao et al *Cell Res*	NA	Neither coronavirus particles nor SARS-CoV-2 nucleocapsid were detected in the bone marrow.
	Yao et al *Zhonghua*	1-3*	Spleen: significantly reduced numbers of lymphocytes, cell degeneration and necrosis.
Skin (n=16)	Ahouach et al	NA	Skin biopsy showed slight spongiosis, basal cell vacuolation and mild perivascular lymphocytic infiltrate. PCR on whole-skin biopsy specimen was negative for SARS-CoV-2.

	Fernandez-Nieto et al	NA	A perivascular infiltrate of lymphocytes, some eosinophils and upper dermal edema.
	Gianotti et al	1	Superficial perivascular dermatitis with slight lymphocytic exocytosis. Mid dermis: small thrombus in a vessel. Patchy distribution of swollen thrombosed vessels with neutrophils, eosinophils and nuclear debris in the dermis.
		2	Superficial and deep perivascular dermatitis with cuffs of lymphocytes surrounding blood vessels in a vasculitic pattern. In the mid dermis extravasated red blood cells from damaged vessels.
		3	Superficial perivascular vesicular dermatitis. Focal acantholytic suprabasal clefts, dyskeratotic and ballooning herpes-like keratinocytes. Patchy band-like infiltration with occasional necrotic keratinocytes and minimal lymphocytic satellitosis. In dermis: swollen vessels, dense lymphocyte infiltration mixed with rare eosinophils. Within the epidermis a nest of Langerhans cells.
	Kolivras et al	NA	Superficial and deep lichenoid, perivascular and peri-eccrine infiltrate of lymphocytes with occasional plasma cells. Vacuolar alteration along the basal layer of the epidermis with scattered singly necrotic (apoptotic) keratinocytes which were occasionally present in the superficial layers of the epidermis. Smudged basement membrane zone. Papillary dermal fibrin confined near the ulcer edge. No pallor (edema) of the papillary dermis. Dense and lichenoid infiltrate in the papillary and superficial reticular dermis; deeper dermis tightly-cuffed, perivascular and peri-eccrine distribution. Some nuclear debris, no neutrophils. Plump endothelial cells in venules surrounded by the lymphoplasmacytic infiltrate. No intraluminal fibrin thrombi, no fibrin within venule walls. Direct-IF: negative. Diagnosis: COVID-19 infection-induced chilblains.
	Magro et al	1	(Lung: complement-mediated microvascular injury) In order to explore possible generalized complement activation in this patient, a sample of clinically-appearing normal skin was also found to have significant vascular deposits of C5b-9 within dermal capillaries. IF: septal capillary co-localization of C4d with SARS-CoV-2 glycoprotein and of C5b-9 with SARS-CoV-2 protein.
		2	Septal capillary co-localization of C4d with SARS-CoV-2 glycoprotein
		3	Striking thrombogenic vasculopathy accompanied by extensive necrosis of the epidermis and adnexal structures, including the eccrine coil. Significant degree of interstitial and perivascular neutrophilia with prominent leukocytoclasia. IHC: striking, extensive deposition of C5b-9 within the microvasculature.
		4	Superficial vascular ectasia. Occlusive arterial thrombus within the deeper reticular dermis, no inflammation. Extensive vascular deposits of C5b-9, C3d, and C4d throughout the dermis, with marked deposition in occluded artery. Biopsy of normal-appearing deltoid skin: conspicuous microvascular deposits of C5b-9
		5	Modest perivascular lymphocytic infiltrate in the superficial dermis along with deeper seated small thrombi within rare venules of the deep dermis, in the absence of a clear vasculitis. Significant vascular deposits of C5b-9 and

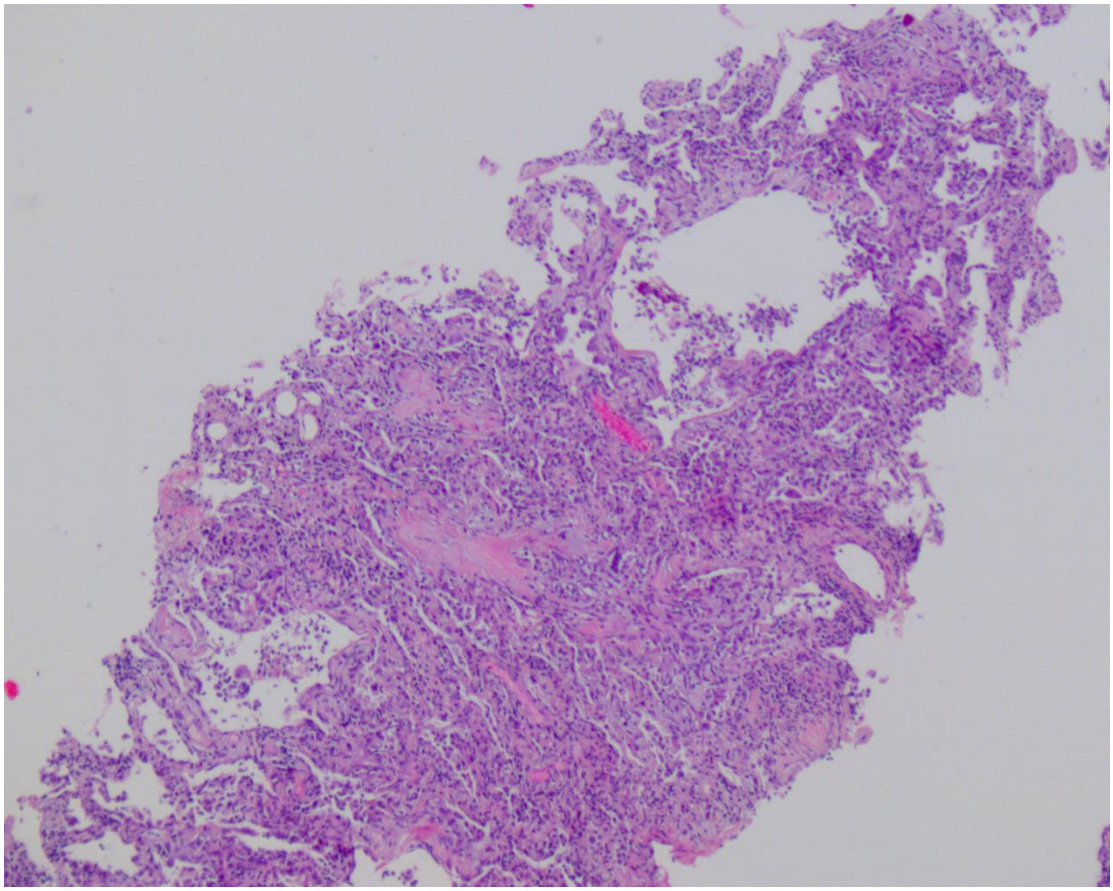
			C4d were observed. Normal deltoid skin: microvascular deposits of C5b-9 throughout the dermis
	Menter et al	12	Multiple cutaneous haemorrhages
	Yao et al *Cell Res*	NA	Neither coronavirus particles nor SARS-CoV-2 nucleocapsid were detected in the skin.
	Yao et al *Zhonghua*	1-3*	Normal appearance. No coronavirus (particles) detected in tissue apart from the lungs.
Muscles (n=2)	Menter et al	11	Hemorrhage of psoas muscle and sternocleidomastoid
		12	haemorrhage of psoas muscle
Placenta (n=4)	Baud et al	NA	Mixed inflammatory infiltrates composed of neutrophils and monocytes in the subchorial space and unspecific increased intervillous fibrin deposition (Figure). Funisitis (inflammation of the umbilical cord connective tissue suggesting fetal inflammatory response) was also present. Gram and periodic acid–Schiff staining of the placenta, PCR, and culture did not identify any bacterial or fungal infections. RT-PCR of SARS-CoV-2 was positive in placental submembrane and cotyledon, but not in fetal samples, maternal blood, and vaginal swab. Fetal autopsy showed no malformations, and fetal lung, liver, and thymus biopsies were negative for SARS-CoV-2.
	Chen et al, *Chin J Path*	1	Gross examination of complete placenta: grey-white nodule. Microscopic: nodule is chorionic hemangioma. Increased fibrin deposition in villi interstitium and around vili. No villitis, chorioamnionitis. No viral inclusions. No thrombosis in umbilical cord blood vessels, no clear multinucleated red blood cells in chorionic blood vessels. RT-PCR of SARS-CoV-2: negative
		2	Multifocal infarction of placental tissue, wich microscopic villous space collapse, fibrin deposition. Increased fibrin deposition in villi interstitium and around villi. No villitis, chorioamnionitis. Remaining tissue (fetal membranes, umbilical cord) not clearly abnormal. No viral inclusions. No thrombosis in umbilical cord blood vessels, no clear multinucleated red blood cells in chorionic blood vessels. RT-PCR of SARS-CoV-2: negative
		3	No villitis, chorioamnionitis. No viral inclusions. No thrombosis in umbilical cord blood vessels, no clear multinucleated red blood cells in chorionic blood vessels. RT-PCR of SARS-CoV-2: negative

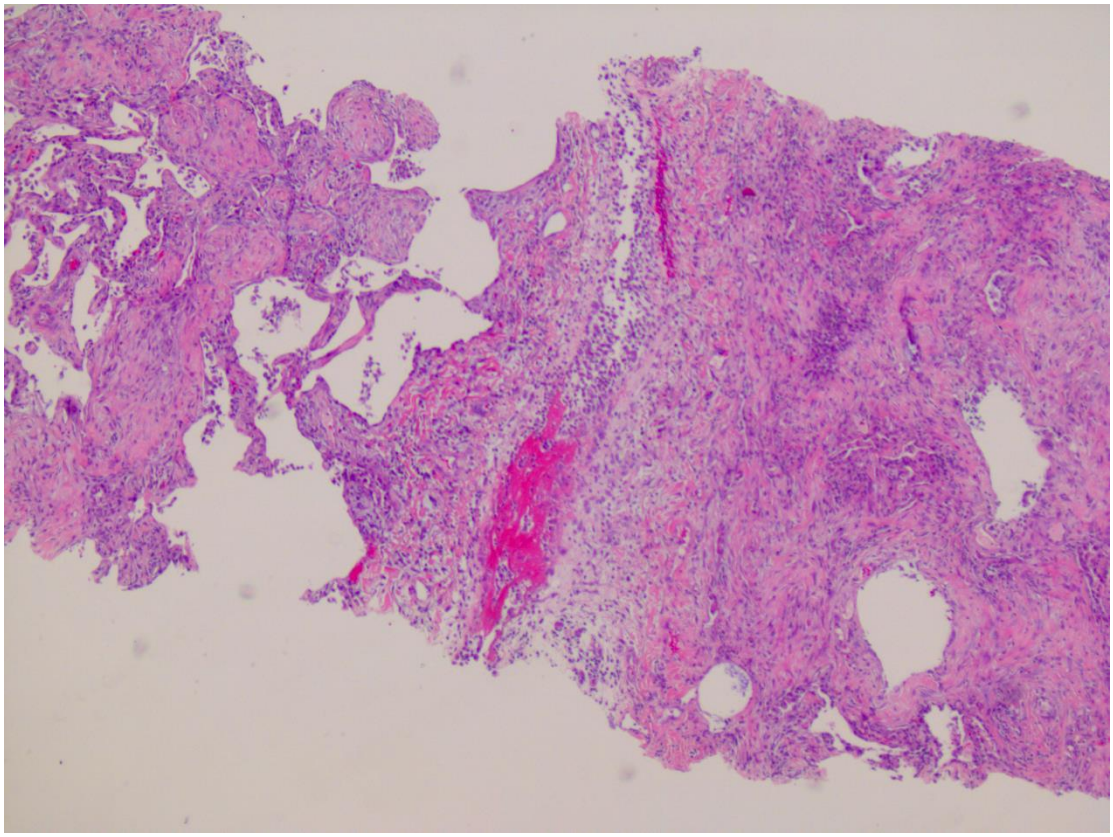
*Data are aggregated and not described for each individual case.

** Row displays general findings that are applicable to all cases included in the study. Individual patient data are described in the rows below.

Abbreviations: NA - not applicable, NR - not reported, RT-PCR - reverse transcription polymerase chain reaction, EM - electron microscopy, (Direct-)IF - (direct-)immunofluorescence, IHC - immunohistochemistry.

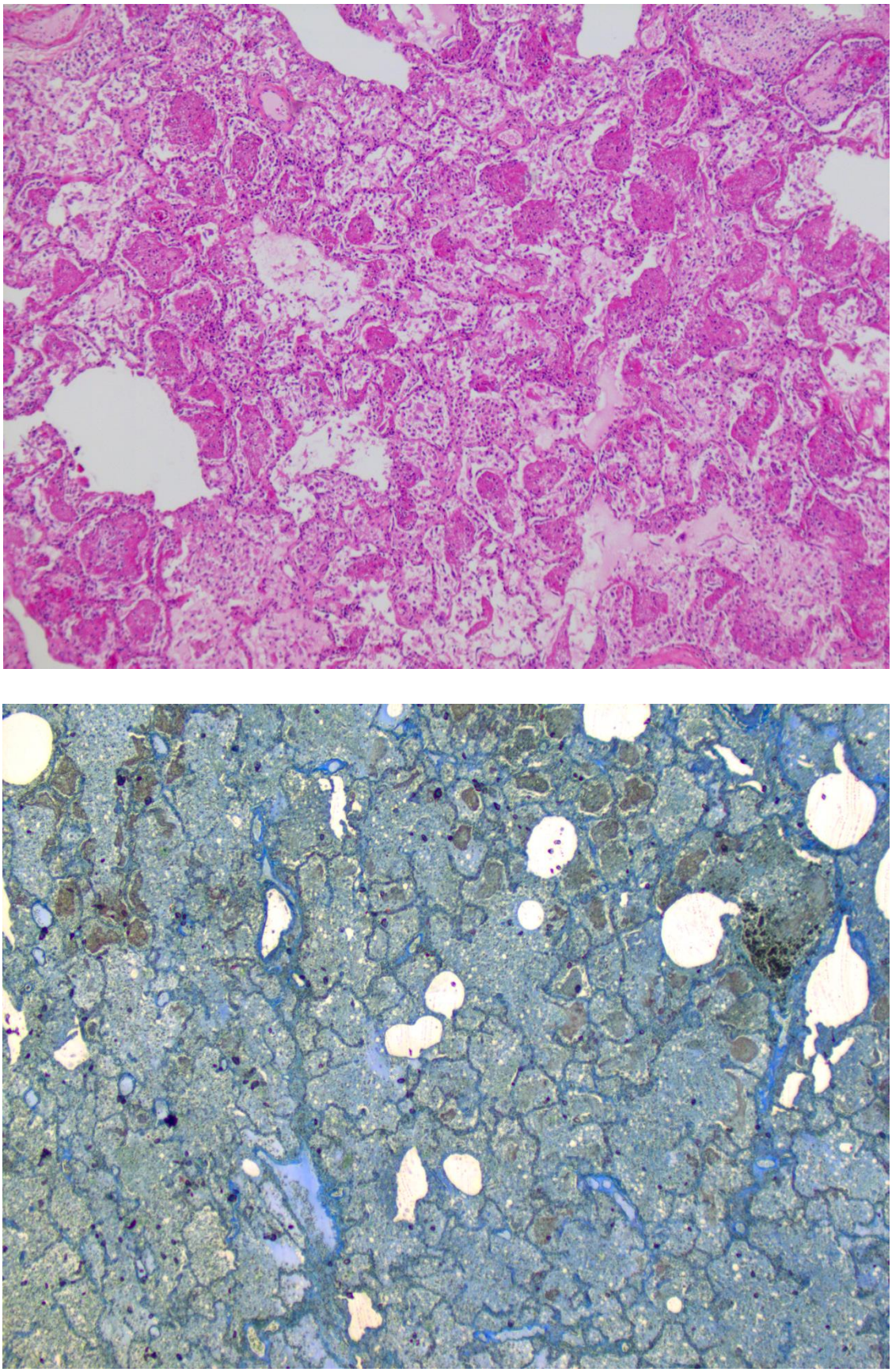
Supplemental Figure S1. Low-power images of two samples with the epithelial pattern of lung injury





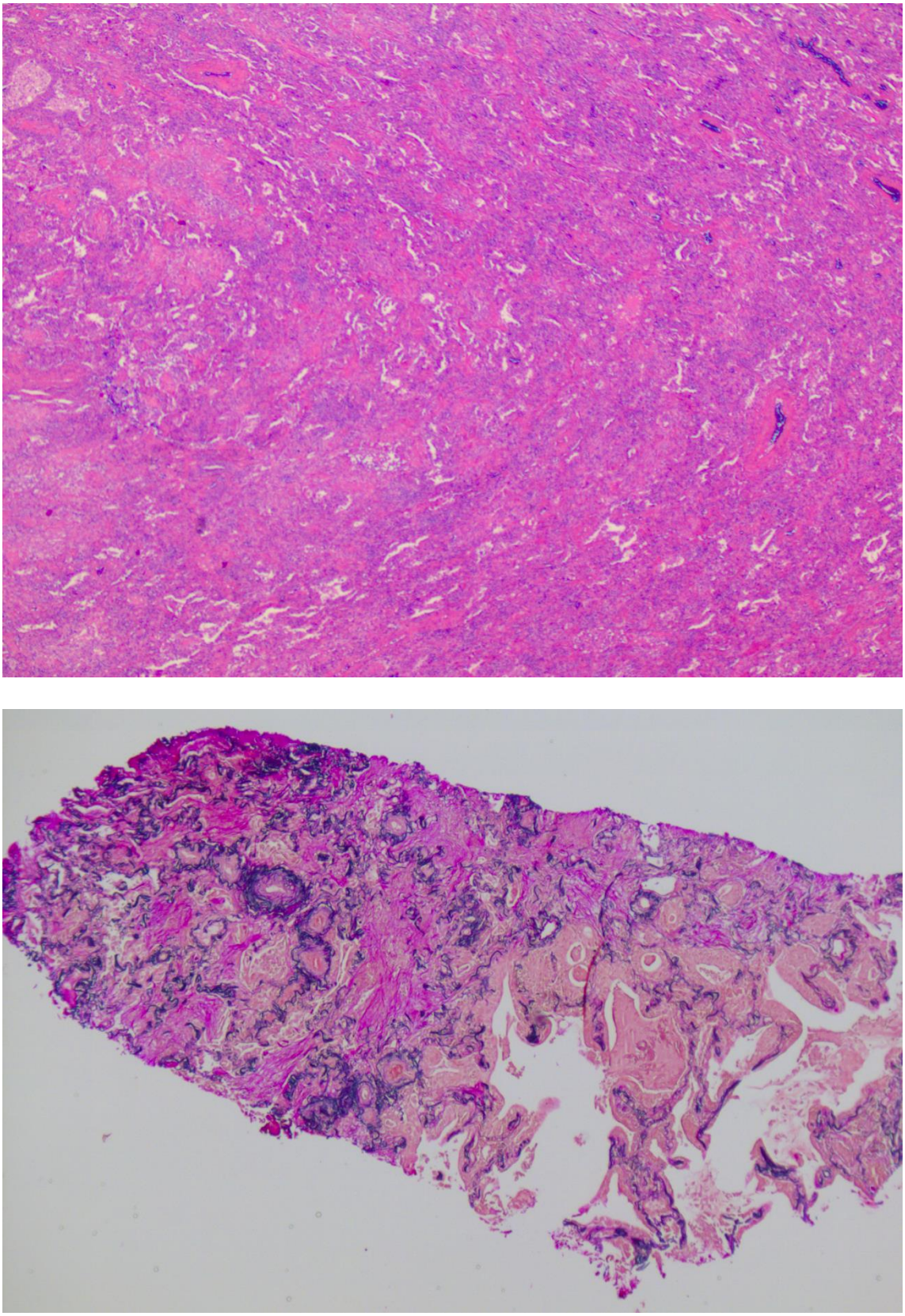
Images of these same samples, at higher magnification, were used for the top panels of Figure 2A.

Supplemental Figure S2. Low-power images of two samples with the vascular pattern of lung injury



Upper image: hematoxylin and eosin stain, lower image: fibrin-Lendrum (MSB) stain. Images of these same samples, at higher magnification, were used for the middle panels of Figure 2A.

Supplemental Figure S3. Low-power images of two samples with the fibrotic pattern of lung injury



Upper image: hematoxylin and eosin stain, lower image: Verhoeff-van Gieson stain. Images of these same samples, at higher magnification, were used for the bottom panels of Figure 2A.