Challenges in the diagnosis and management of post-covid-19 organizing pneumonia: A clinician's perspective

Kok Wei Poh[1], Shobhana Sivandan[2], Kwee Choy Koh[1]

Abstract

COVID-19 caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection can result in multiple complications such as long COVID syndrome, pulmonary fibrosis, and organizing pneumonia (OP). Although OP is a well-known complication of COVID-19, several challenges remain; from suspecting and confirming the diagnosis to its management. These challenges are aggravated further in patients who are critically ill and when surgical biopsy is not feasible. Post-COVID-19 OP is a subset of secondary organizing pneumonia that shares similar clinical and radiological characteristics and similar computerized tomography (CT) scan features with OP of various etiologies. In this review, we propose a clinical approach based on current available evidence for the management of COVID-19 patients with suspected OP. Typical CT findings such as consolidations, perilobular opacity, reversed halo sign and ground-glass opacities are highly suggestive of OP, but are not pathognomonic. Confirmation by histopathology should be done but when not possible, a trial of corticosteroid therapy may be considered. However, biopsy should be done if corticosteroid therapy fails or when there is clinical deterioration and worsening of hypoxia while on corticosteroid therapy especially if the onset of the symptoms is longer than two weeks.

Keywords: *COVID-19; SARS-CoV-2; organizing pneumonia; corticosteroid; computerized tomography scan*

Introduction

Coronavirus disease 2019 (COVID-19), caused by the severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) infection, is an ongoing pandemic with multiple complications including long COVID syndrome, pulmonary fibrosis, and organizing pneumonia (OP). The latter is of particular interest because of the challenges in its diagnosis and management, especially in the early course of the illness, for the clinicians managing these cases.

Defining organizing pneumonia

Organizing pneumonia is a process of pulmonary tissue repair, defined histopathologically by the presence of intra-alveolar plugs of granulation tissue (Masson bodies) consisting of fibroblasts and myofibroblasts mixed with the connective matrix. These granulation tissues extend from the alveoli into the lumen of distal bronchioles.[1–4] Various etiologies have been identified to cause organizing pneumonia (secondary organizing pneumonia). These secondary causes include viral infection, connective tissue disease, toxic fumes, and drugs.[2,5,6] When no specific etiologies are found, it is called cryptogenic organizing pneumonia (COP). However, several studies have shown no significant clinical or radiological differences between secondary and COP.[7–10] Hence, it is conceivable the two share similar pathological and clinical processes; regardless of whether a specific causative agent is found or not. When OP is linked to SARS-CoV-2 infection, it is known as post-COVID-19 OP. The definitive evidence for post-COVID-19 OP is limited as the majority of reported post-COVID-19 OP were not biopsy-confirmed but rather diagnosed based on clinical presentation and computerized tomography (CT) findings [Table I] and hence, should be interpreted cautiously.[11–23]

[1] Internal Medicine, IMU Clinical School, International Medical University, Jalan Dr Muthu, Bukit Rasah, 70300 Seremban, Negeri Sembilan, Malaysia.

[2] Radiology Department, Hospital Tuanku Ja'afar Seremban, Jalan Rasah, Bukit Rasah, 70300 Seremban, Negeri Sembilan, Malaysia.

Address for Correspondence:
Kok Wei Poh
Internal Medicine, IMU Clinical School, International Medical University, Jalan Dr Muthu, Bukit Rasah, 70300 Seremban, Negeri Sembilan, Malaysia.
Tel: +606-7677798 (Ext 235) Email: kokweipoh@imu.edu.my

Table I

Case reports & case series on post-COVID-19 OP. Abbreviations: GGO: Ground-glass opacity.

	DATE OF CT SCAN (Day of illness)	CT SCAN FINDINGS	BIOPSY PERFORMED	TREATMENT	TIMING OF TREATMENT (Day of illness)	RESPONSE TO TREATMENT
Okamori et al., July 2020	Day 13 (estimation)	Consolidation with reversed halo sign, traction bronchiectasis, and volume loss of the lower lobes.	No	1,000mg methylprednisolone daily for 3 days, followed by a daily dose of 80mg/day (1 mg/kg) prednisolone. Tapered and off within 18 days of admission (12 days of steroid).	Day 13 (estimation)	Improved, response rate unclear.
	Day 13 (estimation)	Bilateral Consolidations with band-like opacities and distributed in subpleural or peri-bronchial region. Traction bronchiectasis was also present.	No	50mg/day (0.75 mg/kg). Tapered and off within 18 days of admission (12 days of steroid).	Day 13 (estimation)	Improved, response rate unclear.
de Oliveira Filho et al., Feb 2021	Day 21	Peripheral areas of consolidation in both lower lobes.	No	Prednisone at 1 mg/kg. Tapering dose not reported.	Day 26	Improved, response rate unclear.
	Day 14	Patchy areas of subpleural consolidation, perilobular distribution with reticular opacities.	No	Prednisone at 1 mg/kg. Tapering dose not reported.	Day 23	Tapering off ventilatory support in less than 48 hours.
	Day 14	Consolidation and GGOs with perilobular distribution.	No	Prednisone at 1 mg/kg. Tapering dose not reported.	Day 23	De-escalating to low-flow nasal cannula from CPAP after three doses of steroid.

Kanaoka et al., Feb 2021	Day 26	GGOs and consolidations.	Yes	Prednisolone at 1 mg/kg/day (60mg) for a week and reduced to 0.5mg/kg/day for 6 weeks, followed by 15mg OD.	Day 35	From nasal prong oxygen to room air within 7 days.
	Day 43	GGOs and consolidations.	Yes	Prednisolone at 1 mg/kg/day (60mg) for a week and reduced to 0.5mg/kg/day for 4 weeks, followed by 20mg OD.	Day 51	Improved within 7 days with resolution of hypoxia on exertion.
Horii et al., November 2020	Day 17 (estimation)	Previous ground glass opacities progressed to lung consolidation without new GGOs.	No	Prednisolone ay 1 mg/kg/day (60mg). Then, tapering down gradually (approximately 10mg per week) to 30mg upon discharge (28 days of inpatient steroid). After discharge, prednisolone was tapered down to 20 mg for 20 days, 10 mg for 14 days and 5 mg for 14 days and discontinued thereafter.	Day 17 (estimation)	From 10 L/min oxygen requirement to 1L/min after 3 days.
Kim et al., October 2020	Day 20	Multiple patchy areas of consolidation, mainly in the sub-pleural zones of the right lower lobe with GGOs.	No	Methylprednisolone at 40 mg for 3 days, followed by prednisolone 30mg for 3 days and 20mg for 2 days.	Day 27	From 4L/min nasal cannula to 2L/min within 48 hours.

Simões JP et al., Jan 2021	Day 30	Diffuse patchy consolidations and GGOs with band opacities and perilobular distribution.	No	Prednisolone at 1mg/kg/day for 16 days and were tapered over 13 weeks	Day 30 (presumed)	Improvement within 36 hours and complete resolution of respiratory failure within 7 days.
	Day 30	Patchy consolidations and GGOs with peripheral and peribronchic distribution	No	Methylprednisolone 1mg/kg/day for 20 days and were tapered over 13 weeks	Day 30 (presumed)	Resolution of respiratory failure within 6 days.
Pogatchnik et al., August 2020	3 weeks (approximation)	Patchy GGOs distributed peripherally in the lungs and crazy paving patterns	Yes	Remdesivir; other medications not mentioned.	N/A	Clinically recovers and CT imaging 26 days later showed minimal residual opacities.
Giannakis et al., June 2021	Day 22	GGOS with crazy paving; “bullseye” and “reversed halo” sign.	No	Dexamethasone (unknown dose and duration)	N/A	Improved and discharged home.
Seo H et al., February 2021	Day 17	GGOs with consolidations	Yes	No steroid given	N/A	Improved and discharged home.
Funk et al., April 2021	4 weeks (approximation)	Patchy subpleural GGOs and linear consolidation	Yes	No steroid given	N/A	N/A

Tamura et al., December 2020	Day 19	Consolidation with traction bronchiectasis and volume loss.	No	Methylprednisolone 1000mg for 3 days followed by prednisolone 40mg (0.5mg/kg/day). Tapering regime not known.	Day 19	Extubated 3 days after initiation of therapy.
	N/A		No	Methylprednisolone 1000mg for 3 days followed by prednisolone 30mg (0.5mg/kg/day). Tapering regime not known.	Day 15	Extubated 6 days after initiation of therapy.
	N/A		No	Methylprednisolone 1000mg for 3 days	Day 29	Extubated the next day.
	N/A		No	Methylprednisolone 1000mg for 3 days followed by prednisolone 30mg (0.5mg/kg/day). Tapering regime not known.	Day 14	Extubated 15 days after initiation of therapy.
Cortés Colorado et al., June 2021	Day 11 (approximation)	GGOs, peribronchovascular consolidation, and bilateral pleural effusion.	Yes	Prednisolone 1mg/kg/day. Tapering regime not known.	N/A	Radiological improvement after 15 days of steroid. Clinical details are not available.

Ng et al., October 2021	Approximately 2 months	Bilateral GGOs with visible intralobular lines	No	Methylprednisolone 500 mg/day for 3 days, followed by intravenous dexamethasone 6 mg/day for 10 days. Then, oral prednisolone of 0.5 mg/kg/day for 4 weeks, followed by 20 mg/day for 4 weeks tapered to 10 mg/day for 2 weeks and 5 mg/day for 2 weeks	N/A	Improved over 2 weeks
	Approximately 2 months	Right middle lobe consolidative mass	Yes	Prednisolone at 0.5 mg/kg/day for 4 weeks, then 20 mg/day for 4 weeks, followed by 10 mg/day for another 2 weeks and tapered over 3 months.	N/A	Improved at outpatient review at 12 weeks. Inpatient details not reported.

Are post-COVID-19 OP and DAD the same?

Katzenstein et al. introduced the term "diffuse alveolar damage" (DAD) to describe the histopathological changes that occur in the lung after insults from different aetiologies.[24] Since then, this histopathological description was considered the hallmark finding for acute respiratory distress syndrome (ARDS), although several later studies showed not all ARDSs have histological findings of DAD.[25–32] Although DAD is not pathognomonic of COVID-19 pneumonia, the main pulmonary pathology in COVID-19 is DAD characterized by histopathologic evidence of oedema, endothelial and alveolar lining cell injury with the presence of hyaline membrane.[33–35]

DAD is divided into 3 phases, namely: early exudative phase, proliferative/organizing phase, and the fibrotic phase. The early (acute) exudative phase is characterized by edema, hyaline membranes, and interstitial acute inflammation. This is followed by the subacute (proliferative/organizing) phase with loose organizing fibrosis predominantly within alveolar septa and the presence of type II pneumocyte hyperplasia.[25,36–39] The proliferative phase typically occurs a week after the exudative phase and is replaced by the fibrotic phase 2-3 weeks later; although these phases tend to overlap.[40,41]

Thus, one important differential for OP is DAD in the proliferative or organizing phase, but such distinction is not always clear.[39,42,43] As they share similar histopathological descriptions, it is of interest to know if the organizing phase of DAD is a separate entity from OP or they are inter-related entities within the spectrum of the same pathology. Histologically, the proliferative phase of DAD is evident by the presence of myofibroblasts in both the intra-alveolar and interstitial space. Although the fibroblast-myofibroblast mixed matrix is also present in OP, the interstitial space is usually spared or only mildly inflamed, with relative preservation of the general architecture of lung parenchyma.[44,45] Interestingly, foci of OP may also be found in the DAD process, which makes differentiating OP from DAD difficult.[42,46]

A separate histopathological entity, known as acute fibrinous and organizing pneumonia (AFOP), is worth mentioning here. AFOP is diagnosed by histopathological evidence of intra-alveolar fibrin deposition, often described as "fibrin ball" in addition to the intraluminal loose connective tissue with the absence of hyaline membrane as seen in OP.[47] It is uncertain whether AFOP is an entity alongside DAD and OP or whether it results from tissue sampling issues.[3] A full discussion of AFOP is beyond the scope of this review. However, it is crucial to realize that it shares significant similarities with OP and DAD on CT scan; and responds somewhat well to corticosteroid therapy.[48–51] AFOP, once thought rare, has been reported fairly frequent in COVID-19 patients with ARDS.[33,41,52,53] Hence, a patient with COVID-19 may have pneumonia with or without ARDS from DAD, secondary OP, or AFOP; and all 3 are not easily differentiated from each other by CT as described later in this review.

Earlier studies had reported the proliferative phase of DAD to be steroid responsive.[54,55] However these studies included cases with somewhat preserved lung architecture histologically which might very well be OP after all. It is uncertain if DAD, in its entirety is steroid-responsive or whether the proliferative phase of DAD, which is histologically similar to OP,

determines its responsiveness to corticosteroid therapy. The heterogeneity of histopathological findings in ARDS could explain the mixed responsiveness to corticosteroid therapy.[56–59]

OP has a wide range of clinical features and severity. They often mimic other lung diseases and requires high clinical suspicion followed by histopathological confirmation for diagnosis.[8,38] Although OP usually has a subacute presentation with symptoms such as cough, dyspnea, fever, malaise and flu-like symptoms, a more severe and acute form has been reported.[60,61] Similarly, DAD which is often linked to ARDS, would also have various presentations depending on the underlying causes. Although, DAD are generally more severe and of acute onset.[44,62,63]

The understanding of the natural course and histopathological process of both OP and DAD is crucial to help us in diagnosing post-COVID-19 OP. Hence, a patient who had a subacute presentation of dyspnea and cough after recovering from COVID-19 weeks later should raise the suspicion of OP rather than DAD. However, difficulty remains in patients with COVID-19 who have progressively worsening respiratory illness within the first two weeks of infection; differentiating between worsening DAD from OP is challenging.

Can CT differentiate between DAD and OP?

Typical CT findings of ARDS include areas of ground-glass opacities (GGO), reticular opacities with or without tractional bronchiectasis, and areas of consolidation.[64–66] Similarly, CT findings of COVID-19 pneumonia demonstrates an initial phase of GGO followed by a later phase of crazy-paving pattern, consolidation, interstitial thickening and/or interlobular septal thickening although these patterns often overlap.[67–71]

Comparisons between CT findings of ARDS/DAD and histopathology findings are very limited before the COVID-19 pandemic, and these comparisons were done at a time when histopathology definition and understanding were different.[64,72] A study conducted by Ichikado et al. comparing CT and histopathology findings in hyperoxia-induced DAD in pigs, showed that the normal attenuation area, GGO and interlobular septal thickening correlated with the exudative phase while heterogeneous attenuation and traction bronchiolectasis correlated with the early proliferative phase of DAD respectively.[73]

Although the COVID-19 pandemic has sparked a surge in studies about DAD, these studies were entirely post-mortem; and CT-histopathology comparison studies are scarce.[53,74–77] Kianzad et al. compared the autopsy findings of COVID-19 patients who had chest CT 72-hours prior to death, showed that patchy GGO corresponded to the exudative phase of DAD while the presence of thickened interlobular septa corresponded to both exudative and proliferative phases of DAD.[78] CT findings of areas of consolidation generally correlated with the proliferative phase of DAD. In addition, well-demarcated consolidation areas correlated with AFOP. On the other hand, a CT-histology comparative study by Henkel et al. showed heterogeneous findings instead.[74] However, the patients in this study had wide-ranging intervals between CT imaging day and death.

Typical CT features of OP include the presence of consolidations, perilobular opacity, reversed halo sign (atoll sign) and ground-glass opacities (Figure I). However, these features are not pathognomonic, and they often overlapped with DAD of COVID-19 and other viral pneumonia.[79–81]

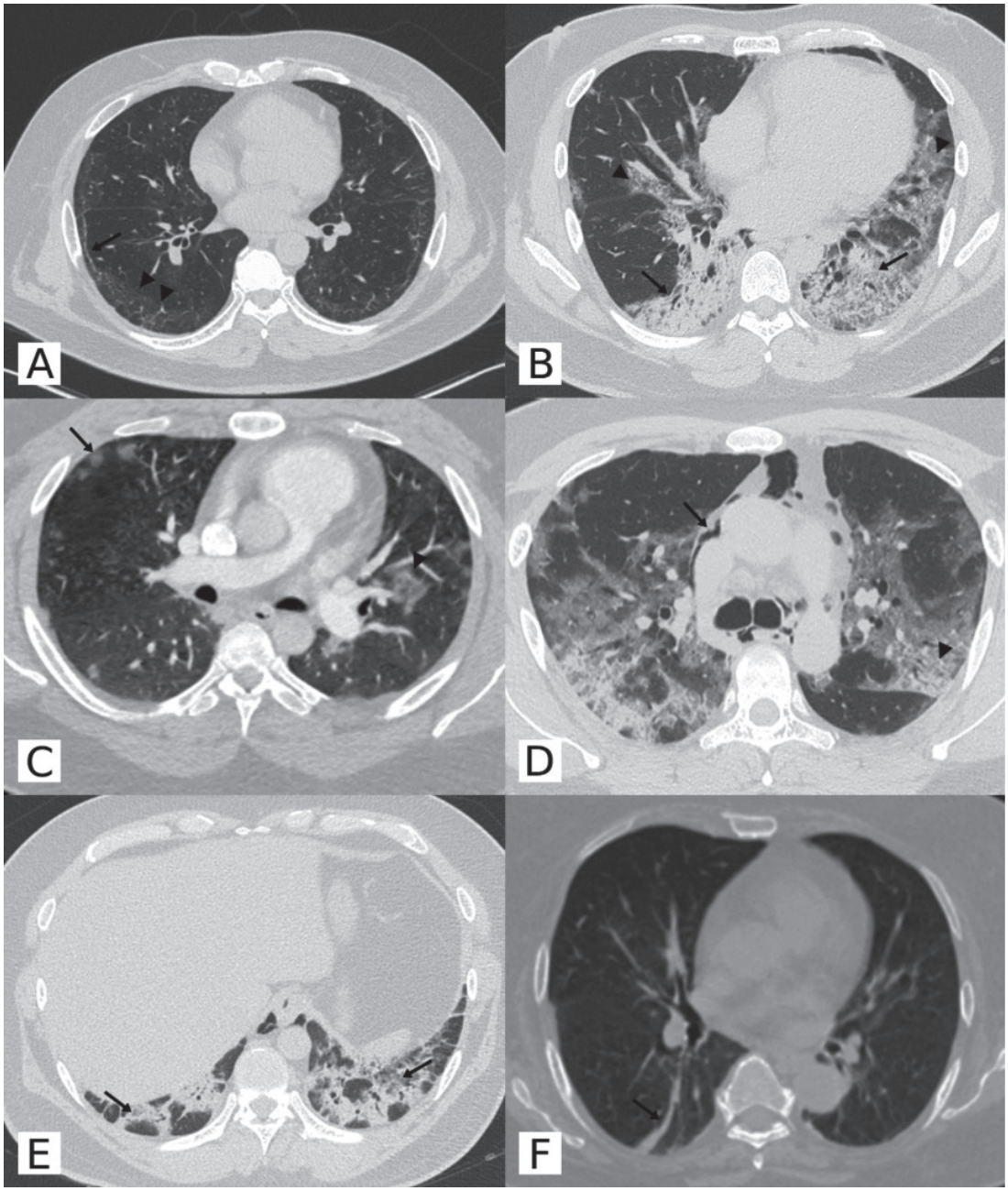

Figure I

(A) Mild ground-glass opacity (arrowhead) with mild reticular abnormality and subpleural bands (arrow). **(B)** Bilateral subpleural and bronchocentric consolidation (arrow) with bronchocentric ground-glass opacities (arrowhead). **(C)** Nodular ground-glass opacities (arrow) with atoll sign (arrowhead). **(D)** Pneumomediastinum (arrow) and crazy paving pattern (arrowhead). **(E)** Perilobular pattern (arrow) bilaterally with arcade-like or polygonal curvilinear opacities. **(F)** Band-like opacity (arrow).

Many studies attempted to correlate the temporal changes in CT features and the duration of symptoms of COVID-19.[67,68,71,82–84] An analysis of these studies together (Figure II) shows that GGO and consolidation are the most common features during the earlier course the disease becoming less common with time along with an increment of band opacities or reticulation. Similar correlative studies for COP showed the most frequent initial CT findings are GGO, consolidation, nodules and reticulation.[85,86] Unfortunately, the widely differing intervals between the time when CT scan was done and symptoms onset in these studies prevent direct comparison between COVID-19 secondary OP and cryptogenic OP.

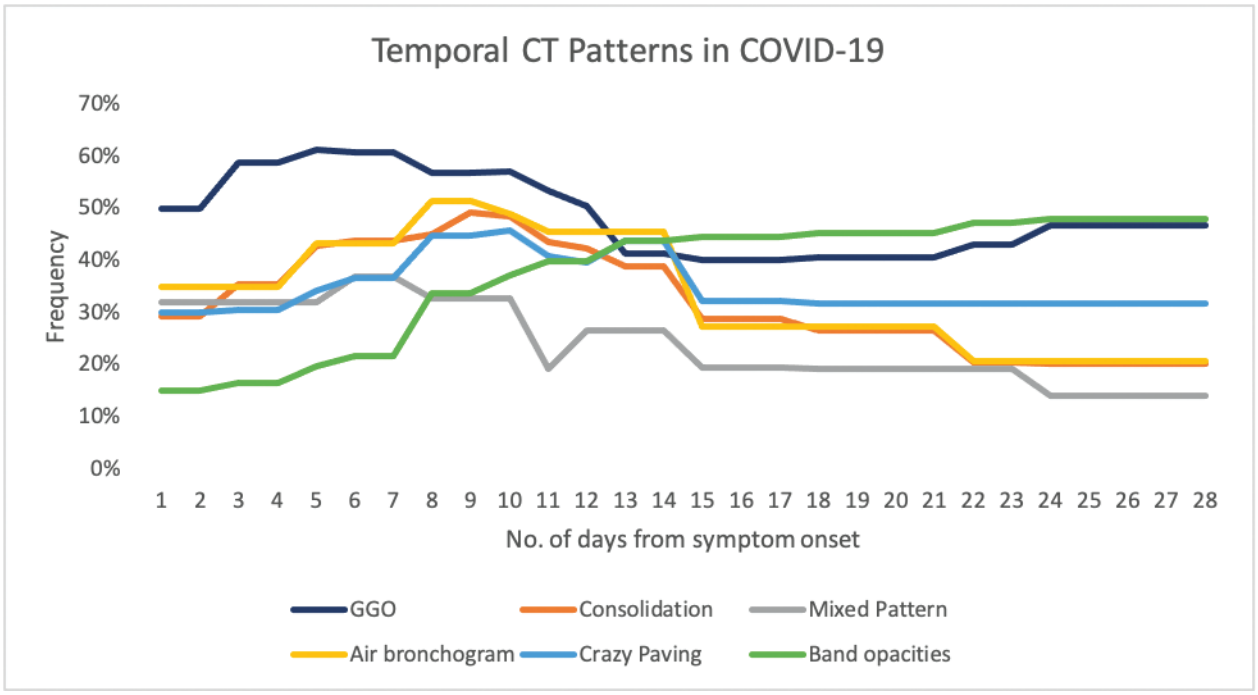

Figure II

Graph showing the temporal frequency of different CT findings (A combined analysis of multiple studies). Mixed pattern is referring to mixed GGOs with consolidations. Abbreviation: GGO: ground-glass opacity.

Various studies on the time of appearance of CT features of COVID-19 OP have reported varying time intervals. Jeong et al. and Cereser et al. reported an astounding >90% of COVID-19 patients developed CT features of OP within a mean and median time of 5.5 ± 2.7 and five days, respectively, from symptoms onset.[87,88] However, these numbers may be an overestimation as the CT features of organizing phase of DAD are not easily differentiated from OP, as discussed earlier. In addition, different CT definitions for OP may have been used in these studies. In contrast, other studies have reported a wider range of approximately 7 to 21 days for CT features of post-COVID-19 OP being present from symptoms onset.[89–91] Autopsy studies

revealed a third of deceased COVID-19 patients had focal and diffuse OP between[5-34] days from symptoms onset with the majority of diffuse pattern occurring after more than ten days.[92]

We can conclude that CT findings alone is inadequate to diagnose post-COVID-19 OP. Studies comparing CT with histopathology findings in COVID-19 are needed to estimate the positive or negative predictive values of CT features in post-COVID-19 OP.

Is biopsy required for management of post-COVID-19 OP?

This is a tricky question. Although histopathology can confirm the diagnosis of OP, the heterogeneity of lung pathology in OP means highly selective and adequate biopsy sampling is required, which may not be feasible via the transbronchial approach. Hence surgically obtained lung biopsy is preferred over transbronchial biopsy but patients with ARDS or post-COVID-19 OP are often ill and may not be suitable for surgical biopsy.[93] Although there may be attempts to diagnose and treat OP without histopathology, for example in critically ill patients or whenever biopsy is not feasible; such an approach could lead to problems later if these patients do not respond to corticosteroid therapy.[2,94]

There are many success stories on the management of suspected post-COVID-19 OP without the need for biopsy [Table I]. The typical CT patterns and dramatic response to corticosteroid therapy lend credence to these 'clinically diagnosed' OP. An analysis of these cases suggests post-COVID-19 OP typically developed within 2-3 weeks after onset of first symptoms. However, it is important to know that OP may have developed earlier and was only diagnosed either by non-resolving or recurrence of respiratory failure after a period of improvement. A study by Rocha et al. on critically ill COVID-19 patients with CT features of OP without biopsy reported a very good response to high dose corticosteroid therapy.[95] These patients were at an average of 8.9 days from symptoms onset with a standard deviation (SD) of 3.9. However, there was no control group for comparison, and corticosteroid of OP dosage was started relatively late at an average of 13.0 ± SD 5.5 days from symptoms onset. The reported survival rate was 92%.

Since there are no randomized controlled trials for CT-only approach versus biopsy-diagnosed approach, it is impossible to predict which patient would fail to respond to corticosteroid therapy. Nevertheless, multiple cases of post-influenza OP have been reported, and these were often diagnosed solely on CT alone together with knowledge of disease progression. Since post-influenza OP and post-COVID-19 OP share similar disease progression, diagnosis by CT-only approach for the latter may be clinically feasible if the clinical scenario and timeline fit.[60,96,97] However, if there is an unsatisfactory response to corticosteroid, biopsy should be performed.

Management of post-COVID-19 OP

The mainstay of therapy for OP is corticosteroid. Typically in COP prednisolone is started at a dose of 0.75-1 mg/kg/day for 2-4 weeks and tapered gradually over 3-6 months.[38,98–100] Mild stable cases of COP can just be monitored carefully for spontaneous recovery or managed with macrolides.[101–103] Corticosteroid regimens in secondary OP varies as there are no large comparative trials for different treatment regimes. Evidences were largely based on case reports

and observational studies, with some clinicians using the treatment regime for COP while others initiate prednisolone at a lower dose of 0.5mg/kg/day.[6,20,21,60,96,97,104–107]

Similarly, there are no randomized studies yet for post-COVID-19 OP. Most of the evidences are based on previous experience and from case reports. Different treatment regimens were used with a shorter duration of tapering dose. In a prospective observational study by Myall et al., 30 out of 35 patients diagnosed with OP with CT scan 6 weeks after COVID-19 infection were started with prednisolone 0.5mg/kg/day and weaned over three weeks.[108] The remaining five patients were considered unsuitable for corticosteroid therapy either due to comorbidity or, limited or improving symptoms. The patients who received prednisolone in this study showed significant clinical and radiological improvements although it is unclear if any relapses occurred. It is important to note that this cohort of patients had baseline characteristics that were different from those reported in case reports/series. They were comparatively less severe and were diagnosed 6 weeks later. Whether this treatment regimen would work for more severe cases or those with earlier onset of OP is unknown.

Does routine corticosteroid therapy for COVID-19 pneumonia interfere with diagnosis of OP?

The landmark RECOVERY trial has defined the course of treatment for COVID-19 with the usage of dexamethasone 6 mg/day for ten days in patients requiring oxygen supplementation or mechanical ventilation.[109] An important sub-analysis from this study of patients with symptoms >7 days reported better response to dexamethasone than those with symptoms ≤7 days, with the median time of 13 days in ventilated patients. This raises the question of whether the better response rate in the former was due to the presence of OP rather than just simple pneumonia. Similar trends were found when we sub-analyzed the results from several other studies.[110–114] Studies that reported no benefit with corticosteroids either had a relatively earlier initiation of treatment from the onset of symptoms; or patients were given methylprednisolone pulse doses resulting in shorter treatment duration.[115–118]

There are no data or guidelines available on when to suspect OP for COVID-19 patients receiving dexamethasone therapy. Segala et al. reported a case series consisting of ten patients who developed post-COVID-19 OP diagnosed with CT scan after receiving the standard therapy of dexamethasone 6mg OD for ten days. In these patients, at least 20 days had transpired from symptoms onset before the diagnosis of post-COVID-19 OP. They were treated with methylprednisolone with gradual tapering of dosages every three days. Eight patients were successfully weaned off from oxygen supplementation and discharged within two weeks of treatment.[119]

In clinical practice, OP should be suspected when there is clinical deterioration and worsening hypoxia despite being on dexamethasone therapy, especially if the onset of the symptoms is more than two weeks. High-resolution CT scan is the preferred imaging, and other causes should be ruled out including ventilator-associated pneumonia. Lung biopsy is recommended when feasible and when patients failed to respond to standard OP therapy. A suggested approach is shown in Figure III.

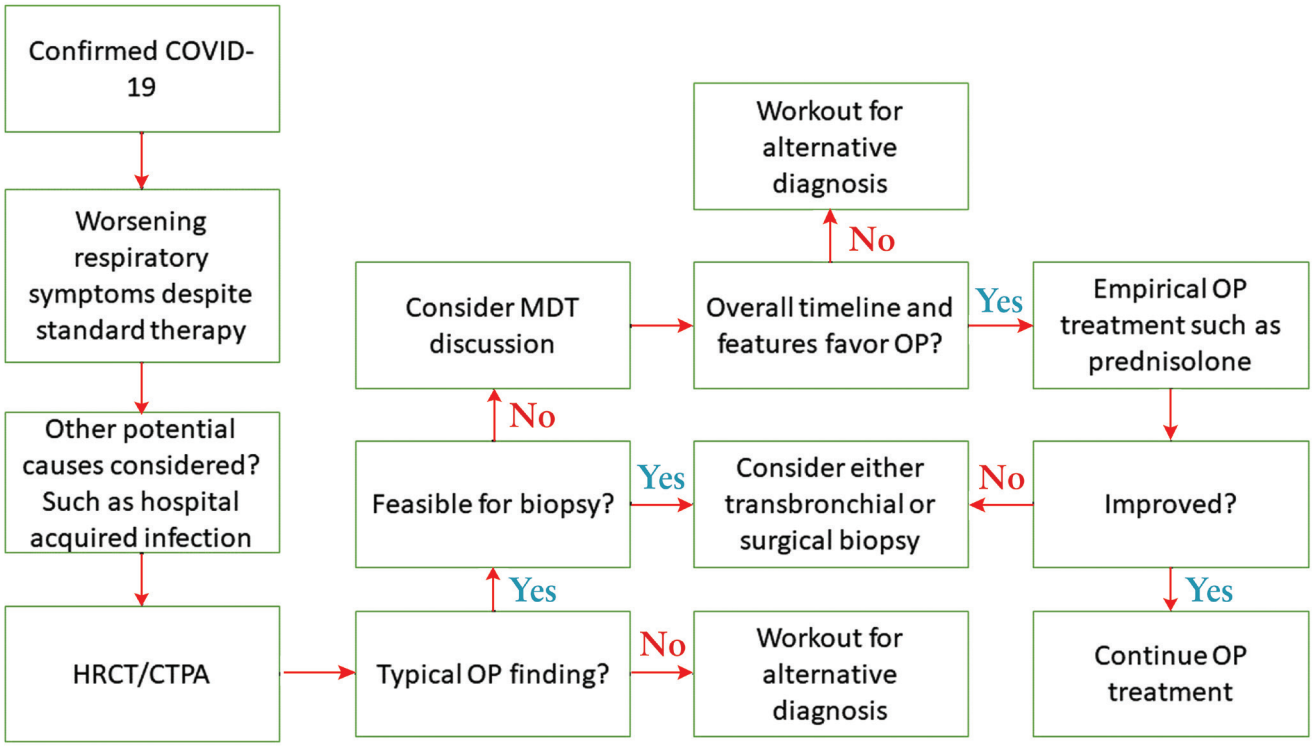

Figure III

Flow diagram on suggested approach for post-COVID-19 OP.

Abbreviations: **HRCT:** high-resolution computed tomography. **CTPA:** computed tomography pulmonary angiography. **OP:** organizing pneumonia. **MDT:** multidisciplinary team.

Conclusion

In summary, post-COVID-19 OP should not be diagnosed from CT alone without clinical context because DAD can show similar CT features. However, OP should be suspected when there is no clinical improvement or worsening hypoxia especially after 1-2 weeks from the onset of symptoms. When lung biopsy is not feasible, a trial of prednisolone 0.5-1.0 mg/kg od (or equivalent) is justifiable. If there is no clinical improvement, biopsy should be considered, preferably with a multidisciplinary team involving infectious disease physician, respiratory physician, radiologist, and pathologist.

Conflict of interest

The authors declared that they each have no conflict of interest.

Funding

None

Acknowledgement

We would like to thank the medical, emergency and radiology department for managing COVID-19 in Hospital Tuanku Ja'afar Seremban. We also would like to thank Dr Muhammad Hafidz Bin Azmi for identifying patients with post-COVID-19 OP. In addition, we would like to express our gratitude to every healthcare worker who has been working around the clock combating this pandemic.

REFERENCES

1. Colby T V. Pathologic Aspects of Bronchiolitis Obliterans Organizing Pneumonia. Chest. 1992;102(1):38–43. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0012369216348826
2. Cordier J-F. Rare diseases bullet 8: Organising pneumonia. Thorax. 2000;55(4):318–28. Available from: https://thorax.bmj.com/lookup/doi/10.1136/thorax.55.4.318
3. Travis WD, Costabel U, Hansell DM, King TE, Lynch DA, Nicholson AG, et al. An Official American Thoracic Society/European Respiratory Society Statement: Update of the International Multidisciplinary Classification of the Idiopathic Interstitial Pneumonias. Am J Respir Crit Care Med. 2013;188(6):733–48. Available from: http://www.atsjournals.org/doi/abs/10.1164/rccm.201308-1483ST
4. Baque-Juston M, Pellegrin A, Leroy S, Marquette CH, Padovani B. Organizing pneumonia: what is it? A conceptual approach and pictorial review. Diagn Interv Imaging. 2014;95(9):771–7. Available from: http://dx.doi.org/10.1016/j.diii.2014.01.004
5. Drakopanagiotakis F, Polychronopoulos V, Judson MA. Organizing pneumonia. Am J Med Sci. 2008;335(1):34–9. Available from: http://dx.doi.org/10.1097/MAJ.0b013e31815d829d
6. Asai N, Yokoi T, Nishiyama N, Koizumi Y, Sakanashi D, Kato H, et al. Secondary organizing pneumonia following viral pneumonia caused by severe influenza B: a case report and literature reviews. BMC Infect Dis. 2017;17(1):572. Available from: http://bmcinfectdis.biomedcentral.com/articles/10.1186/s12879-017-2677-1
7. Baha A, Yıldırım F, Köktürk N, Galata Z, Akyürek N, Demirci NY, et al. Cryptogenic and Secondary Organizing Pneumonia: Clinical Presentation, Radiological and Laboratory Findings, Treatment, and Prognosis in 56 Cases. Turkish Thorac J. 2018;19(4):201–8. Available from: https://turkthoracj.org/en/cryptogenic-and-secondary-organizing-pneumonia-clinical-presentation-radiological-and-laboratory-findings-treatment-and-prognosis-in-56-cases-131025
8. Drakopanagiotakis F, Paschalaki K, Abu-Hijleh M, Aswad B, Karagianidis N, Kastanakis E, et al. Cryptogenic and Secondary Organizing Pneumonia. Chest. 2011;139(4):893–900. Available from: http://dx.doi.org/10.1378/chest.10-0883
9. Yılmaz S, Akıncı Özyürek B, Erdo an Y, Cirit Koçer B, Demirağ F, Dadalı Y, et al. Retrospective evaluation of patients with organizing pneumonia: is cryptogenic organizing pneumonia different from secondary organizing pneumonia? Tuberk Toraks. 2017;65(1):1–8. Available from: http://tuberktoraks.org/managete/fu_folder/2017-01/2017-65-1-001-008.pdf
10. Lohr RH, Boland BJ, Douglas WW, Dockrell DH, Colby T V, Swensen SJ, et al. Organizing Pneumonia: Features and Prognosis of Cryptogenic, Secondary, and Focal Variants. Arch Intern Med. 1997;157(12):1323–9. Available from: http://archinte.jamanetwork.com/article.aspx?doi=10.1001/archinte.1997.00440330057006
11. Okamori S, Lee H, Kondo Y, Akiyama Y, Kabata H, Kaneko Y, et al. Coronavirus disease 2019-associated rapidly progressive organizing pneumonia with fibrotic feature. Medicine (Baltimore). 2020;99(35):e21804. Available from: https://journals.lww.com/10.1097/MD.0000000000021804
12. de Oliveira Filho CM, Vieceli T, de Fraga Bassotto C, da Rosa Barbato JP, Garcia TS, Scheffel RS. Organizing pneumonia: A late phase complication of COVID-19 responding dramatically to corticosteroids. Brazilian J Infect Dis. 2021;25(1):6–9.
13. Funk G-C, Nell C, Pokieser W, Thaler B, Rainer G, Valipour A. Organizing pneumonia following Covid19 pneumonia. Wien Klin Wochenschr. 2021; Available from: https://link.springer.com/10.1007/s00508-021-01852-9
14. Cortés Colorado JM, Cardona Ardila LF, Aguirre Vásquez N, Gómez Calderón KC, Lozano Álvarez SL, Carrillo Bayona JA. Organizing pneumonia associated with SARS-CoV-2 infection. Radiol Case Reports. 2021;16(9):2634–9.
15. Ng BH, Ban AY-L, Nik Abeed NN, Faisal M. Organising pneumonia manifesting as a late-phase complication of COVID-19. BMJ Case Rep. 2021;14(10):e246119. Available from: https://casereports.bmj.com/lookup/doi/10.1136/bcr-2021-246119
16. Horii H, Kamada K, Nakakubo S, Yamashita Y, Nakamura J, Nasuhara Y, et al. Rapidly progressive organizing pneumonia associated with COVID-19. Respir Med Case Reports. 2020;31:101295. Available from: https://doi.org/10.1016/j.rmcr.2020.101295

17. Kim T, Son E, Jeon D, Lee SJ, Lim S, Cho WH. Effectiveness of Steroid Treatment for SARS-CoV-2 Pneumonia With Cryptogenic Organizing Pneumonia-Like Reaction: A Case Report. Disaster Med Public Health Prep. 2020;1–4. Available from: https://www.cambridge.org/core/product/identifier/S1935789320004152/type/journal_article

18. Simões JP, Alves Ferreira AR, Almeida PM, Trigueiros F, Braz A, Inácio JR, et al. Organizing pneumonia and COVID-19: A report of two cases. Respir Med Case Reports. 2021;32:101359. Available from: https://linkinghub.elsevier.com/retrieve/pii/S2213007121000216

19. Giannakis A, Móré D, Mangold DL, von Stackelberg O, Rubtsov R, Heussel CP, et al. Simultaneous presence of the "bullseye" and "reversed halo" sign at CT of COVID-19 pneumonia: A case report. Radiol Case Reports. 2021;16(9):2442–6.

20. Tamura K, Nishioka S, Tamura N, Saito Z, Kuwano K. Successful treatment with methyl-prednisolone pulses for the late phase of COVID-19 with respiratory failure: A single-center case series. Respir Med Case Reports. 2020;31:101318.

21. Kanaoka K, Minami S, Ihara S, Tanaka T, Yasuoka H, Komuta K. Secondary organizing pneumonia after coronavirus disease 2019: Two cases. Respir Med Case Reports. 2021;32:101356. Available from: https://doi.org/10.1016/j.rmcr.2021.101356

22. Pogatchnik BP, Swenson KE, Sharifi H, Bedi H, Berry GJ, Guo HH. Radiology–Pathology Correlation Demonstrating Organizing Pneumonia in a Patient Who Recovered from COVID-19. Am J Respir Crit Care Med. 2020;202(4):598–9. Available from: https://www.atsjournals.org/doi/10.1164/rccm.202004-1278IM

23. Seo H, Jung J, Kim MJ, Jang SJ, Kim S-H. Radiologically Suspected Organizing Pneumonia in a Patient Recovering from COVID-19: A Case Report. Infect Chemother. 2021;53(1):191–6.

24. Katzenstein ALA, Bloor CM, Leibow AA. Diffuse alveolar damage--the role of oxygen, shock, and related factors. A review. Am J Pathol. 1976;85(1):209–28.

25. Cardinal-Fernández P, Lorente JA, Ballén-Barragán A, Matute-Bello G. Acute Respiratory Distress Syndrome and Diffuse Alveolar Damage. New Insights on a Complex Relationship. Ann Am Thorac Soc. 2017;14(6):844–50. Available from: http://www.atsjournals.org/doi/10.1513/AnnalsATS.201609-728PS

26. Thompson BT, Guérin C, Esteban A. Should ARDS be renamed diffuse alveolar damage? Intensive Care Med. 2016;42(5):653–5.

27. Esteban A, Fernández-Segoviano P, Frutos-Vivar F, Aramburu JA, Nájera L, Ferguson ND, et al. Comparison of clinical criteria for the acute respiratory distress syndrome with autopsy findings. Ann Intern Med. 2004;141(6):440–5. Available from: http://www.ncbi.nlm.nih.gov/pubmed/15381517

28. Guerin C, Bayle F, Leray V, Debord S, Stoian A, Yonis H, et al. Open lung biopsy in nonresolving ARDS frequently identifies diffuse alveolar damage regardless of the severity stage and may have implications for patient management. Intensive Care Med. 2015;41(2):222–30. Available from: http://link.springer.com/10.1007/s00134-014-3583-2

29. Park J, Lee YJ, Lee J, Park SS, Cho Y-J, Lee S-M, et al. Histopathologic heterogeneity of acute respiratory distress syndrome revealed by surgical lung biopsy and its clinical implications. Korean J Intern Med. 2018;33(3):532–40. Available from: http://kjim.org/journal/view.php?doi=10.3904/kjim.2016.346

30. Thille AW, Esteban A, Fernández-Segoviano P, Rodriguez JM, Aramburu JA, Peñuelas O, et al. Comparison of the berlin definition for acute respiratory distress syndrome with autopsy. Am J Respir Crit Care Med. 2013;187(7):761–7.

31. de Hemptinne Q, Remmelink M, Brimioulle S, Salmon I, Vincent J-L. ARDS: a clinicopathological confrontation. Chest. 2009;135(4):944–9. Available from: http://www.ncbi.nlm.nih.gov/pubmed/19118274

32. Cardinal-Fernández P, Bajwa EK, Dominguez-Calvo A, Menéndez JM, Papazian L, Thompson BT. The Presence of Diffuse Alveolar Damage on Open Lung Biopsy Is Associated With Mortality in Patients With Acute Respiratory Distress Syndrome. Chest. 2016;149(5):1155–64.

33. Konopka KE, Nguyen T, Jentzen JM, Rayes O, Schmidt CJ, Wilson AM, et al. Diffuse alveolar damage (DAD) resulting from coronavirus disease 2019 Infection is Morphologically Indistinguishable from Other Causes of DAD. Histopathology. 2020;77(4):570–8. Available from: https://onlinelibrary.wiley.com/doi/10.1111/his.14180

34. Vasquez-Bonilla WO, Orozco R, Argueta V, Sierra M, Zambrano LI, Muñoz-Lara F, et al. A review of the main histopathological findings in coronavirus disease 2019. Hum Pathol. 2020;105(January):74–83.

35. Mikhaleva LM, Cherniaev AL, Samsonova M V., Zayratyants O V., Kakturskiy L V., Vasyukova OA, et al. Pathological Features in 100 Deceased Patients With COVID-19 in Correlation With Clinical and Laboratory Data. Pathol Oncol Res. 2021;27(August):1609900.

36. Castro CY. ARDS and Diffuse Alveolar Damage: A Pathologist's Perspective. Semin Thorac Cardiovasc Surg. 2006;18(1):13–9.

37. Kobashi Y, Manabe T. The fibrosing process in so-called organized diffuse alveolar damage. Virchows Arch A Pathol Anat Histopathol. 1993;422(1):47–52.

38. Lazor R. Organizing Pneumonias. In: Cottin V, Cordier J-F, Richeldi L, editors. Orphan Lung Diseases: A Clinical Guide to Rare Lung Disease. London: Springer London; 2015. p. 363–78. Available from: https://doi.org/10.1007/978-1-4471-2401-6_24

39. Butt Y, Kurdowska A, Allen TC. Acute Lung Injury: A Clinical and Molecular Review. Arch Pathol Lab Med. 2016;140(4):345–50. Available from: http://meridian.allenpress.com/aplm/article/140/4/345/65203/Acute-Lung-Injury-A-Clinical-and-Molecular-Review

40. Thille AW, Esteban A, Fernández-Segoviano P, Rodriguez J-M, Aramburu J-A, Vargas-Errázuriz P, et al. Chronology of histological lesions in acute respiratory distress syndrome with diffuse alveolar damage: a prospective cohort study of clinical autopsies. Lancet Respir Med. 2013;1(5):395–401.

41. Merdji H, Mayeur S, Schenck M, Oulehri W, Clere-Jehl R, Cunat S, et al. Histopathological features in fatal COVID-19 acute respiratory distress syndrome. Med intensiva. 2021;45(5):261–70. Available from: http://www.ncbi.nlm.nih.gov/pubmed/34059216

42. Mandal R V., Mark EJ, Kradin RL. Organizing pneumonia and pulmonary lymphatic architecture in diffuse alveolar damage. Hum Pathol. 2008;39(8):1234–8. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0046817708000117

43. Elsoukkary SS, Mostyka M, Dillard A, Berman DR, Ma LX, Chadburn A, et al. Autopsy Findings in 32 Patients with COVID-19: A Single-Institution Experience. Pathobiology. 2021;88(1):56–68.

44. Hughes KT, Beasley MB. Pulmonary Manifestations of Acute Lung Injury: More Than Just Diffuse Alveolar Damage. Arch Pathol Lab Med. 2017;141(7):916–22.

45. Oikonomou A, Hansell DM. Organizing pneumonia: the many morphological faces. Eur Radiol. 2002;12(6):1486–96.

46. Roberton BJ, Hansell DM. Organizing pneumonia: a kaleidoscope of concepts and morphologies. Eur Radiol. 2011;21(11):2244–54.

47. Beasley MB, Franks TJ, Galvin JR, Gochuico B, Travis WD. Acute fibrinous and organizing pneumonia: a histological pattern of lung injury and possible variant of diffuse alveolar damage. Arch Pathol Lab Med. 2002;126(9):1064–70. Available from: http://www.ncbi.nlm.nih.gov/pubmed/12204055

48. Lee JH, Yum H-K, Jamous F, Santos C, Campisi A, Surani S, et al. Diagnostic procedures and clinico-radiological findings of acute fibrinous and organizing pneumonia: a systematic review and pooled analysis. Eur Radiol. 2021;31(10):7283–94.

49. Onishi Y, Kawamura T, Higashino T, Mimura R, Tsukamoto H, Sasaki S. Clinical features of acute fibrinous and organizing pneumonia: An early histologic pattern of various acute inflammatory lung diseases. PLoS One. 2021;16(4 April):1–16. Available from: http://dx.doi.org/10.1371/journal.pone.0249300

50. Gomes R, Padrão E, Dabó H, Soares Pires F, Mota P, Melo N, et al. Acute fibrinous and organizing pneumonia: A report of 13 cases in a tertiary university hospital. Medicine (Baltimore). 2016;95(27):e4073. Available from: https://journals.lww.com/00005792-201607050-00038

51. Lu J, Yin Q, Zha Y, Deng S, Huang J, Guo Z, et al. Acute fibrinous and organizing pneumonia: two case reports and literature review. BMC Pulm Med. 2019;19(1):141. Available from: https://bmcpulmmed.biomedcentral.com/articles/10.1186/s12890-019-0861-3

52. Copin M-C, Parmentier E, Duburcq T, Poissy J, Mathieu D, Lille COVID-19 ICU and Anatomopathology Group. Time to consider histologic pattern of lung injury to treat critically ill patients with COVID-19 infection. Intensive Care Med. 2020;46(6):1124–6.

53. Flikweert AW, Grootenboers MJJH, Yick DCY, du Mée AWF, van der Meer NJM, Rettig TCD, et al. Late histopathologic characteristics of critically ill COVID-19 patients: Different phenotypes without evidence of invasive aspergillosis, a case series. J Crit Care. 2020;59:149–55. Available from: https://doi.org/10.1016/j.jcrc.2020.07.002

54. Meduri GU, Belenchia JM, Estes RJ, Wunderink RG, El Torky M, Leeper K V. Fibroproliferative phase of ARDS; Clinical findings and effects of corticosteroids. Chest. 1991;100(4):943–52. Available from: http://dx.doi.org/10.1378/chest.100.4.943

55. Meduri GU, Chinn AJ, Leeper K V., Wunderink RG, Tolley E, Winer-Muram HT, et al. Corticosteroid rescue treatment of progressive fibroproliferation in late ARDS. Patterns of response and predictors of outcome. Chest. 1994;105(5):1516–27.

56. Zhang L, Wang Z, Xu F, Ren Y, Wang H, Han D, et al. The Role of Glucocorticoids in the Treatment of ARDS: A Multicenter Retrospective Study Based on the eICU Collaborative Research Database. Front Med. 2021;8(6):1–9. Available from: https://onlinelibrary.wiley.com/doi/10.1111/eci.13496

57. Junhai Z, Bangchuan H, Shijin G, Jing Y, Li L. Glucocorticoids for acute respiratory distress syndrome: A systematic review with meta-analysis and trial sequential analysis. Eur J Clin Invest. 2021;51(6):1–15.

58. Lin P, Zhao Y, Li X, Jiang F, Liang Z. Decreased mortality in acute respiratory distress syndrome patients treated with corticosteroids: an updated meta-analysis of randomized clinical trials with trial sequential analysis. Crit Care. 2021;25(1):122. Available from: https://doi.org/10.1186/s13054-021-03546-0

59. Lorente JA, Ballén-Barragán A, Herrero R, Esteban A. Acute respiratory distress syndrome: Does histology matter? Crit Care. 2015;19(1):4–5. Available from: http://dx.doi.org/10.1186/s13054-015-1022-6

60. Cornejo R, Llanos O, Fernández C, Carlos Díaz J, Cardemil G, Salguero J, et al. Organizing pneumonia in patients with severe respiratory failure due to novel A (H1N1) influenza. BMJ Case Rep. 2010;2010:1–5.

61. Epler GR. Bronchiolitis obliterans organizing pneumonia, 25 years: a variety of causes, but what are the treatment options? Expert Rev Respir Med. 2011;5(3):353–61. Available from: http://www.ncbi.nlm.nih.gov/pubmed/21702658

62. Poletti V, Casoni GL, Cancellieri A, Piciucchi S, Dubini A, Zompatori M. Diffuse alveolar damage. Pathologica. 2010;102(6):453–63. Available from: http://dx.doi.org/10.1378/chest.130.2.553

63. Parambil JG, Myers JL, Aubry M-C, Ryu JH. Causes and prognosis of diffuse alveolar damage diagnosed on surgical lung biopsy. Chest. 2007;132(1):50–7. Available from: http://www.ncbi.nlm.nih.gov/pubmed/17475632

64. Desai SR, Hansell DM. Lung imaging in the adult respiratory distress syndrome: current practice and new insights. Intensive Care Med. 1997;23(1):7–15.

65. Simon M, Braune S, Laqmani A, Metschke M, Berliner C, Kalsow M, et al. Value of Computed Tomography of the Chest in Subjects With ARDS: A Retrospective Observational Study. Respir Care. 2016;61(3):316–23.

66. Nöbauer-Huhmann IM, Eibenberger K, Schaefer-Prokop C, Steltzer H, Schlick W, Strasser K, et al. Changes in lung parenchyma after acute respiratory distress syndrome (ARDS): assessment with high-resolution computed tomography. Eur Radiol. 2001;11(12):2436–43.

67. Xiong Y, Sun D, Liu Y, Fan Y, Zhao L, Li X, et al. Clinical and High-Resolution CT Features of the COVID-19 Infection: Comparison of the Initial and Follow-up Changes. Invest Radiol. 2020;55(6):332–9.

68. Ding X, Xu J, Zhou J, Long Q. Chest CT findings of COVID-19 pneumonia by duration of symptoms. Eur J Radiol. 2020;127(5):109009.

69. Zhou M, Dong C, Li C, Wang Y, Liao H, Shi H, et al. Longitudinal changes in COVID-19 clinical measures and correlation with the extent of CT lung abnormalities. Int J Med Sci. 2021;18(5):1277–84. Available from: http://www.ncbi.nlm.nih.gov/pubmed/33526989

70. Liang T, Liu Z, Wu CC, Jin C, Zhao H, Wang Y, et al. Evolution of CT findings in patients with mild COVID-19 pneumonia. Eur Radiol. 2020;30(9):4865–73.

71. Wang Y, Dong C, Hu Y, Li C, Ren Q, Zhang X, et al. Temporal Changes of CT Findings in 90 Patients with COVID-19 Pneumonia: A Longitudinal Study. Radiology. 2020;296(2):E55–64.

72. Kobayashi H, Itoh T, Sasaki Y, Konishi J. Diagnostic imaging of idiopathic adult respiratory distress syndrome (ARDS)/diffuse alveolar damage (DAD) histopathological correlation with radiological imaging. Clin Imaging. 1996;20(1):1–7.

73. Ichikado K, Suga M, Gushima Y, Johkoh T, Iyonaga K, Yokoyama T, et al. Hyperoxia-induced diffuse alveolar damage in pigs: correlation between thin-section CT and histopathologic findings. Radiology. 2000;216(2):531–8. Available from: http://www.ncbi.nlm.nih.gov/pubmed/34159661

74. Henkel M, Weikert T, Marston K, Schwab N, Sommer G, Haslbauer J, et al. Lethal COVID-19: Radiologic-Pathologic Correlation of the Lungs. Radiol Cardiothorac Imaging. 2020;2(6):e200406. Available from: http://pubs.rsna.org/doi/10.1148/ryct.2020200406

75. Bradley BT, Maioli H, Johnston R, Chaudhry I, Fink SL, Xu H, et al. Histopathology and ultrastructural findings of fatal COVID-19 infections in Washington State: a case series. Lancet (London, England). 2020;396(10247):320–32. Available from: http://www.ncbi.nlm.nih.gov/pubmed/32682491

76. Duarte-Neto AN, Monteiro RAA, Silva LFF, Malheiros DMAC, Oliveira EP, Theodoro-Filho J, et al. Pulmonary and systemic involvement in COVID-19 patients assessed with ultrasound-guided minimally invasive autopsy. Histopathology. 2020;77(2):186–97. Available from: https://onlinelibrary.wiley.com/doi/10.1111/his.14160

77. Schaller T, Hirschbühl K, Burkhardt K, Braun G, Trepel M, Märkl B, et al. Postmortem Examination of Patients With COVID-19. JAMA. 2020;323(24):2518. Available from: https://jamanetwork.com/journals/jama/fullarticle/2766557

78. Kianzad A, Meijboom LJ, Nossent EJ, Roos E, Schurink B, Bonta PI, et al. COVID-19: Histopathological correlates of imaging patterns on chest computed tomography. Respirology. 2021;26(9):869–77.

79. Zare Mehrjardi M, Kahkouee S, Pourabdollah M. Radio-Pathological Correlation of Organising Pneumonia (OP): A Pictorial Review. Br J Radiol. 2017;20160723. Available from: http://www.birpublications.org/doi/10.1259/bjr.20160723

80. Torrealba JR, Fisher S, Kanne JP, Butt YM, Glazer C, Kershaw C, et al. Pathology-radiology correlation of common and uncommon computed tomographic patterns of organizing pneumonia. Hum Pathol. 2018;71:30–40.

81. Garrana SH, Som A, Ndakwah GS, Yeung T, Febbo J, Heeger AP, et al. Comparison of Chest CT Findings of COVID-19, Influenza, and Organizing Pneumonia: A Multireader Study. AJR Am J Roentgenol. 2021;

82. Kim YS, Kang UR, Kim YH. The Spectrum of CT Findings of COVID-19 Pneumonia: Acute Alveolar Insult and Organizing Pneumonia as Different Phases of Lung Injury and Repair. J Korean Soc Radiol. 2021;82(2):359.

83. Bernheim A, Mei X, Huang M, Yang Y, Fayad ZA, Zhang N, et al. Chest CT Findings in Coronavirus Disease-19 (COVID-19): Relationship to Duration of Infection. Radiology. 2020;295(3):200463.

84. Zhou S, Zhu T, Wang Y, Xia L. Imaging features and evolution on CT in 100 COVID-19 pneumonia patients in Wuhan, China. Eur Radiol. 2020;30(10):5446–54. Available from: http://www.ncbi.nlm.nih.gov/pubmed/32367418

85. Chung MP, Nam BD, Lee KS, Han J, Park JS, Hwang JH, et al. Serial chest CT in cryptogenic organizing pneumonia: Evolutional changes and prognostic determinants. Respirology. 2018;23(3):325–30.

86. Lee JW, Lee KS, Lee HY, Chung MP, Yi CA, Kim TS, et al. Cryptogenic organizing pneumonia: serial high-resolution CT findings in 22 patients. AJR Am J Roentgenol. 2010;195(4):916–22.

87. Jeong YJ, Nam B Da, Yoo JY, Kim K Il, Kang H, Hwang JH, et al. Prognostic Implications of CT Feature Analysis in Patients with COVID-19: a Nationwide Cohort Study. J Korean Med Sci. 2021;36(8):e51.

88. Cereser L, Da Re J, Zuiani C, Girometti R. Chest high-resolution computed tomography is associated to short-time progression to severe disease in patients with COVID-19 pneumonia. Clin Imaging. 2021;70(June 2020):61–6. Available from: http://www.ncbi.nlm.nih.gov/pubmed/33125986

89. Carvalho A, Cunha R, Lima BA, Pereira JM, Madureira AJ. Chest CT imaging features of COVID-19 pneumonia: First radiological insights from Porto, Portugal. Eur J Radiol Open. 2020;7(October):100294.

90. Wang Y, Jin C, Wu CC, Zhao H, Liang T, Liu Z, et al. Organizing pneumonia of COVID-19: Time-dependent evolution and outcome in CT findings. PLoS One. 2020;15(11):e0240347.

91. Jin C, Tian C, Wang Y, Wu CC, Zhao H, Liang T, et al. A Pattern Categorization of CT Findings to Predict Outcome of COVID-19 Pneumonia. Front Public Heal. 2020;8(September). Available from: https://www.frontiersin.org/article/10.3389/fpubh.2020.567672/full

92. Borczuk AC, Salvatore SP, Seshan S V., Patel SS, Bussel JB, Mostyka M, et al. COVID-19 pulmonary pathology: a multi-institutional autopsy cohort from Italy and New York City. Mod Pathol. 2020;33(11):2156–68.

93. Romagnoli M, Colby T V., Berthet J-P, Gamez AS, Mallet J-P, Serre I, et al. Poor Concordance between Sequential Transbronchial Lung Cryobiopsy and Surgical Lung Biopsy in the Diagnosis of Diffuse Interstitial Lung Diseases. Am J Respir Crit Care Med. 2019;199(10):1249–56.

94. Cordier J-F. Cryptogenic organising pneumonia. Eur Respir J. 2006;28(2):422–46. Available from: http://www.ncbi.nlm.nih.gov/pubmed/34217780

95. Rocha AS, Meireles M, Vilaça H, Guimarães TC, Martins MD, Santos LR, et al. Outcomes of Covid-19 organizing pneumonia in critically ill patients. J Infect. 2021;83(4):496–522. Available from: https://linkinghub.elsevier.com/retrieve/pii/S0163445321003224

96. Gómez-Gómez A, Martínez-Martínez R, Gotway MB. Organizing pneumonia associated with swine-origin influenza A H1N1 2009 viral infection. AJR Am J Roentgenol. 2011;196(1):W103-4.

97. Nuñez-Conde A, Marquez-Algaba E, Falcó V, Almirante B, Burgos J. Organizing pneumonia secondary to influenza infection: Two case reports and a literature review. Enfermedades Infecc y Microbiol Clin (English ed). 2020;38(3):123–6.

98. Schlesinger C, Koss MN. The organizing pneumonias : a critical review of current concepts and treatment. Treat Respir Med. 2006;5(3):193–206.

99. Cottin V, Cordier J-F. Cryptogenic Organizing Pneumonia. Semin Respir Crit Care Med. 2012;33(05):462–75.

100. Bradley B, Branley HM, Egan JJ, Greaves MS, Hansell DM, Harrison NK, et al. Interstitial lung disease guideline: the British Thoracic Society in collaboration with the Thoracic Society of Australia and New Zealand and the Irish Thoracic Society. Thorax. 2008;63 Suppl 5(Supplement 5):v1-58.

101. Flora AS, Simo H, Assaly R. Increasing Role of Macrolide Anti-inflammatory Therapy in Secondary Organizing Pneumonia. Am J Ther. 2017;24(4):e495. Available from: http://www.ncbi.nlm.nih.gov/pubmed/28166162

102. Pathak V, Kuhn JM, Durham C, Funkhouser WK, Henke DC. Macrolide use leads to clinical and radiological improvement in patients with cryptogenic organizing pneumonia. Ann Am Thorac Soc. 2014;11(1):87–91.

103. Radzikowska E, Wiatr E, Gawryluk D, Langfort R, Bestry I, Rudzinski P, et al. Cryptogenic organizing pneumonia – Results of treatment with clarithromycin versus corticosteroids. In: 15 Diffuse Parenchymal Lung Disease. European Respiratory Society; 2016. p. PA3907.

104. Watanabe K, Miyake A, Kaneko T. Secondary organizing pneumonia due to Mycobacterium abscessus lung disease: Case report and review of the literature. Int J Mycobacteriology. 2019;8(4):397.

105. Trungu J, Pirard L, Vandenhove T, Stanciu Pop C, Dahlqvist C, Sohy C. Secondary organizing pneumonia associated with sertraline: A case report. Respir Med Case Reports. 2020;31:101141.

106. Vieira AL, Vale A, Melo N, Caetano Mota P, Jesus JM, Cunha R, et al. Organizing pneumonia revisited: insights and uncertainties from a series of 67 patients. Sarcoidosis, Vasc Diffus lung Dis Off J WASOG. 2018;35(2):129–38. Available from: http://www.ncbi.nlm.nih.gov/pubmed/32476892

107. Cazzato S, Zompatori M, Baruzzi G, Schiattone ML, Burzi M, Rossi A, et al. Bronchiolitis obliterans-organizing pneumonia: an Italian experience. Respir Med. 2000;94(7):702–8.

108. Myall KJ, Mukherjee B, Castanheira AM, Lam JL, Benedetti G, Mak SM, et al. Persistent Post-COVID-19 Interstitial Lung Disease. An Observational Study of Corticosteroid Treatment. Ann Am Thorac Soc. 2021;18(5):799–806.

109. RECOVERY Collaborative Group, Horby P, Lim WS, Emberson JR, Mafham M, Bell JL, et al. Dexamethasone in Hospitalized Patients with Covid-19. N Engl J Med. 2021;384(8):693–704.

110. Bahl A, Johnson S, Chen N-W. Timing of corticosteroids impacts mortality in hospitalized COVID-19 patients. Intern Emerg Med. 2021;16(6):1593–603. Available from: http://www.ncbi.nlm.nih.gov/pubmed/33547620

111. Tomazini BM, Maia IS, Cavalcanti AB, Berwanger O, Rosa RG, Veiga VC, et al. Effect of Dexamethasone on Days Alive and Ventilator-Free in Patients With Moderate or Severe Acute Respiratory Distress Syndrome and COVID-19: The CoDEX Randomized Clinical Trial. JAMA. 2020;324(13):1307–16.

112. Monedero P, Gea A, Castro P, Candela-Toha AM, Hernández-Sanz ML, Arruti E, et al. Early corticosteroids are associated with lower mortality in critically ill patients with COVID-19: a cohort study. Crit Care. 2021;25(1):2. Available from: https://doi.org/10.1186/s13054-020-03422-3

113. Tortajada C, Colomer E, Andreu-Ballester JC, Esparcia A, Oltra C, Flores J. Corticosteroids for COVID-19 patients requiring oxygen support? Yes, but not for everyone: Effect of corticosteroids on mortality and intensive care unit admission in patients with COVID-19 according to patients' oxygen requirements. J Med Virol. 2021;93(3):1817–23. Available from: http://www.ncbi.nlm.nih.gov/pubmed/33107607

114. Go RC, Shah R, Nyirenda T, Oe Y, Sarfraz K, Panthappattu JJ, et al. Methylprednisolone and 60 Days in Hospital Survival in Coronavirus Disease 2019 Pneumonia. Crit Care Explor. 2021;3(7):e0493. Available from: https://journals.lww.com/10.1097/CCE.0000000000000493

115. Tang X, Feng Y-M, Ni J-X, Zhang J-Y, Liu L-M, Hu K, et al. Early Use of Corticosteroid May Prolong SARS-CoV-2 Shedding in Non-Intensive Care Unit Patients with COVID-19 Pneumonia: A Multicenter, Single-Blind, Randomized Control Trial. Respiration. 2021;100(2):116–26.

116. Jeronimo CMP, Farias MEL, Val FFA, Sampaio VS, Alexandre MAA, Melo GC, et al. Methylprednisolone as Adjunctive Therapy for Patients Hospitalized With Coronavirus Disease 2019 (COVID-19; Metcovid): A Randomized, Double-blind, Phase IIb, Placebo-controlled Trial. Clin Infect Dis. 2021;72(9):e373–81. Available from: http://www.ncbi.nlm.nih.gov/pubmed/33534047

117. Corral-Gudino L, Bahamonde A, Arnaiz-Revillas F, Gómez-Barquero J, Abadía-Otero J, García-Ibarbia C, et al. Methylprednisolone in adults hospitalized with COVID-19 pneumonia : An open-label randomized trial (GLUCOCOVID). Wien Klin Wochenschr. 2021;133(7–8):303–11. Available from: http://www.ncbi.nlm.nih.gov/pubmed/33534047

118. Bartoletti M, Marconi L, Scudeller L, Pancaldi L, Tedeschi S, Giannella M, et al. Efficacy of corticosteroid treatment for hospitalized patients with severe COVID-19: a multicentre study. Clin Microbiol Infect. 2021;27(1):105–11. Available from: http://www.ncbi.nlm.nih.gov/pubmed/32971254

119. Segala FV, Sgalla G, Salvati F, Berardini L, Negri M, Nardella E, et al. Adjunctive corticosteroid treatment for organizing pneumonia in COVID-19 patients with persistent respiratory failure. Respir Med. 2021;187:106571.