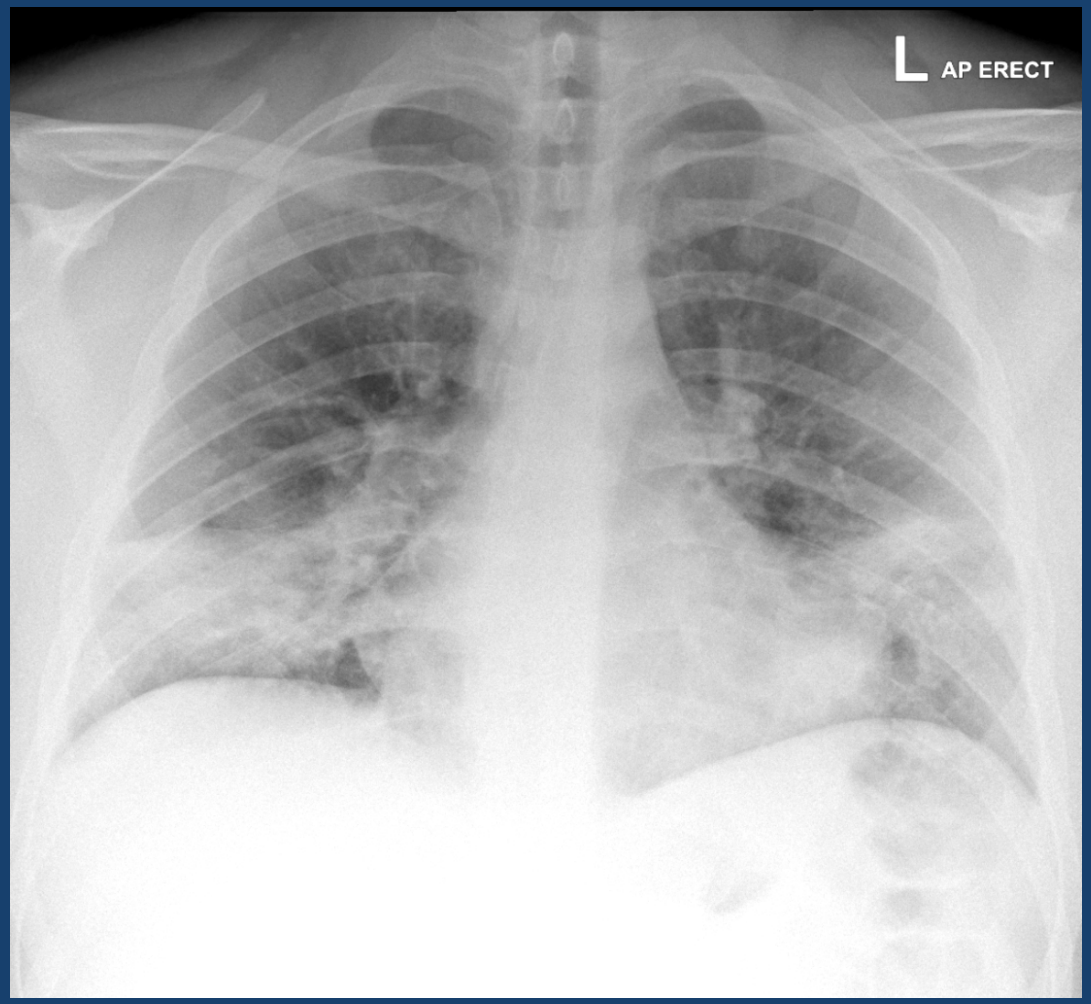
Can COVID-19 Pneumonia Cavitate?

Divya Nelson, Danyal Jajbhay, James May, Siva Mahendran, Anita I Rhodes

Kingston Hospital NHS Foundation Trust

Background



- 20 year old University student
- Presents to A & E with a one week history of fever, cough, worsening breathlessness and chest pain.
- PMH mild Asthma.
- Positive COVID-19 PCR throat swab.

Figure 1: Admission chest radiograph shows bilateral, dense lower zone consolidation.

During this hospital admission…

- Admitted to the High Dependency Unit for 24 hours of CPAP (Continuous Positive Airway Pressure) therapy due to type 1 respiratory failure.
- A CTPA was performed due to persistent hypoxia (paO2 of **8.54** KPa (normal range 11-14.5 KPa)) and raised d-dimer (695 ng/ml (raised above 300ng/ml)).
- ECHO showed septal flattening, suggestive of right heart strain and a small pericardial effusion.
- Anticoagulated with apixaban, treated for possible super-added bacterial pneumonia with intravenous Piperacillin-Tazobactam for 5 days and discharged home after 9 days.

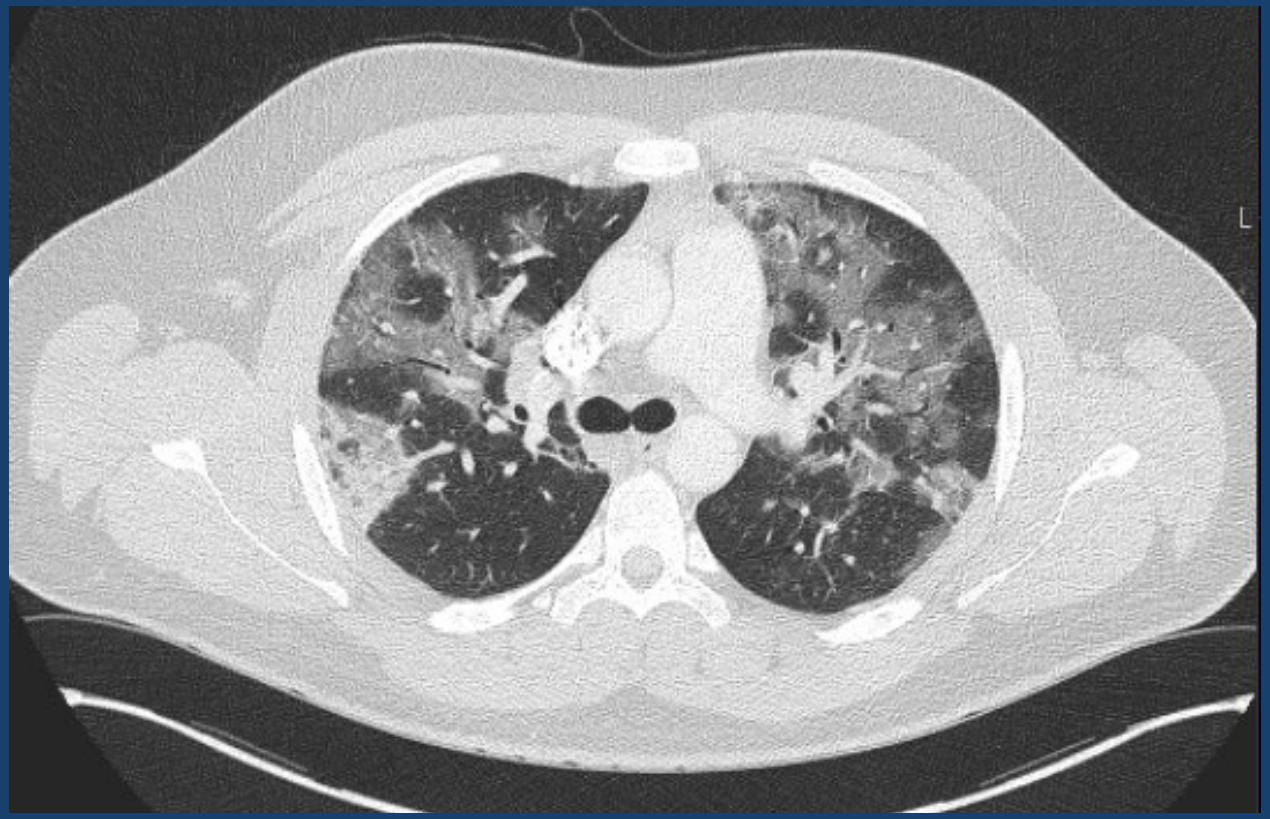
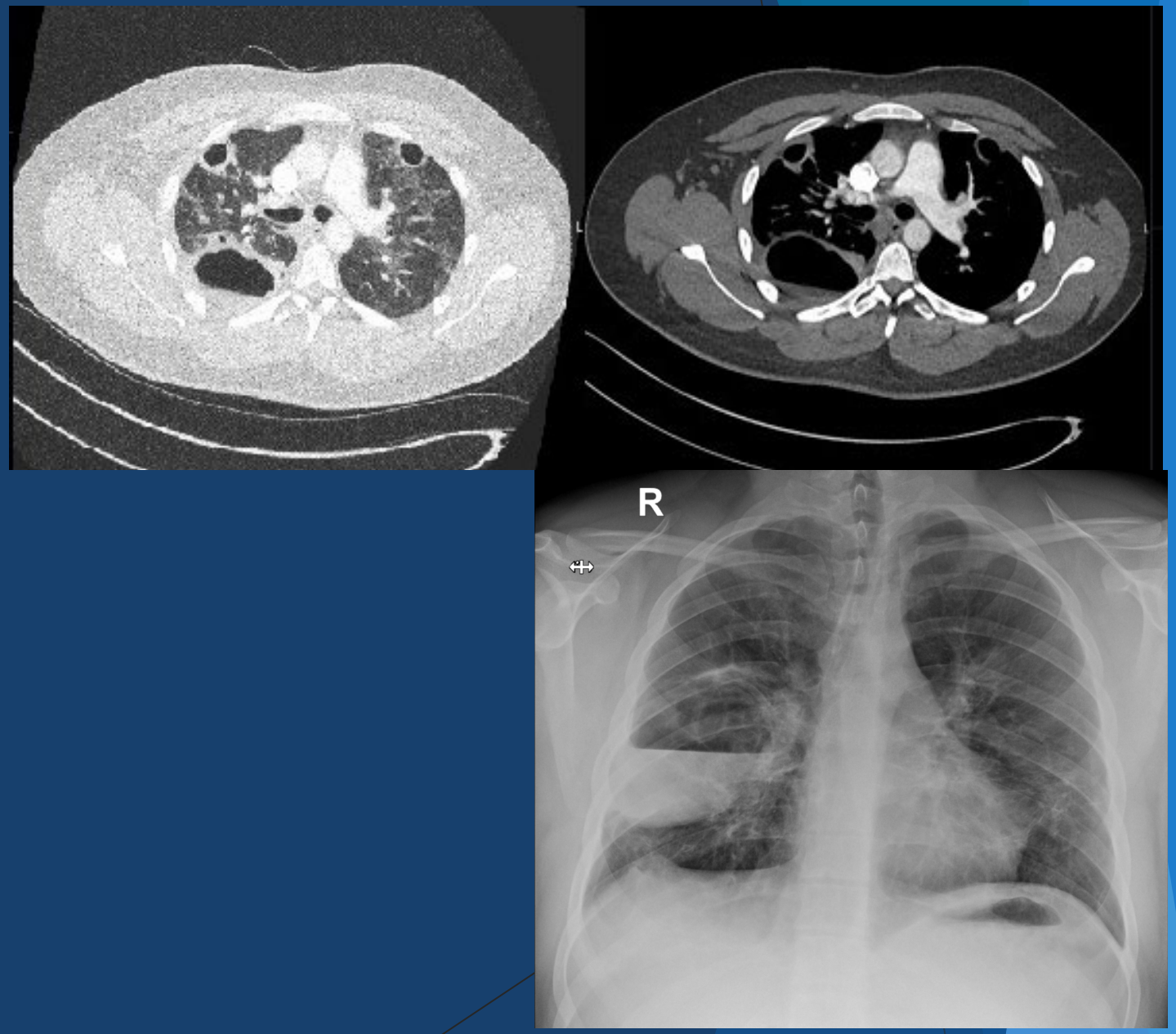


Figure 2: The first CTPA shows diffuse ground glass change and dense consolidation in the right middle lobe and lingula consistent with a severe, classical COVID-19 pneumonia pattern [1].
There were filling defects in the lower lobe subsegmental vessels, in keeping with small bilateral pulmonary emboli.
No peripheral vessel dilatation or enlargement was seen within or travelling towards these areas of ground glass shadowing, which have been described as possible signs of peripheral vessel thrombosis [2]. There was also a small left pleural effusion.

One month later, back in A & E with…

- Right sided pleuritic chest pain, a non-productive cough and exertional breathlessness.
- Afebrile but tachypnoeic and tachycardic with a mildly raised C-reactive protein (CRP) and a normal white cell count. Arterial blood gas revealed again a type 1 respiratory failure with a PaO2 of 7.98 kPa on room air.
- Two consecutive throat swabs for COVID-19 were negative.

- ***Figure 3:*** The second CTPA showed resolution of the pulmonary emboli and left pleural effusion.
  New multiple, bilateral and peripheral, cavitating lung lesions with predominantly smooth thickened walls of varying sizes were present. The largest cavity in the right lower lobe measured 11cm and contained an air fluid level (as seen on the radiograph).
- Moderately enlarged mediastinal lymph nodes are suggestive of severe COVID-19 pneumonia [3] or superadded infection.



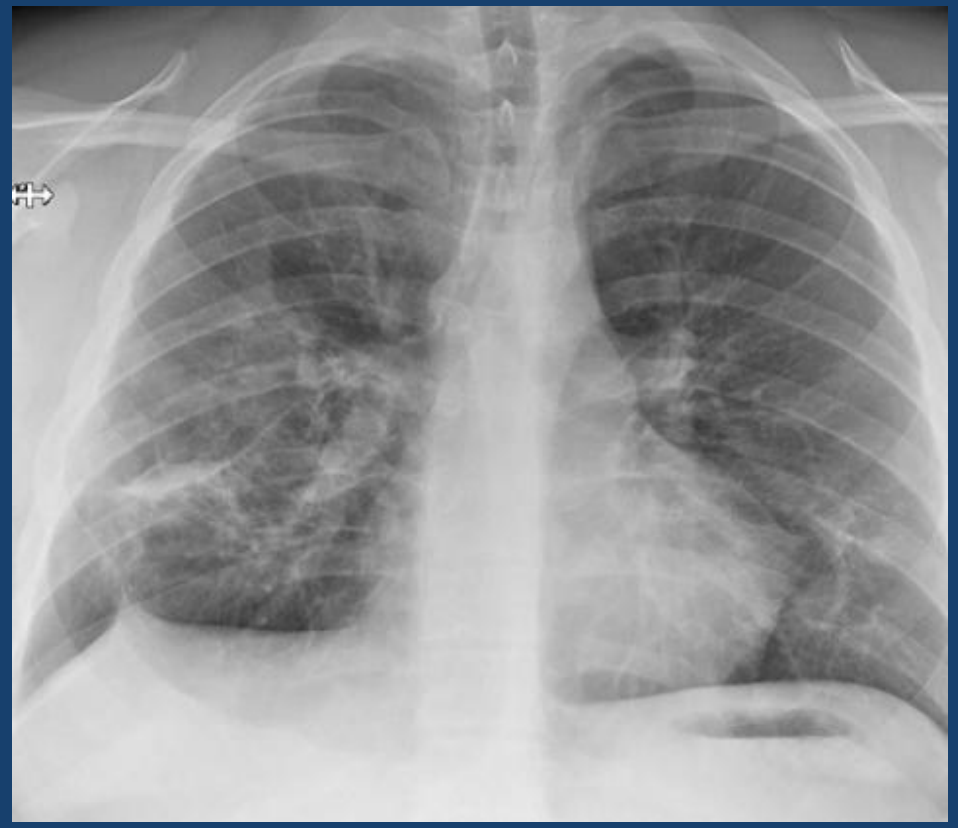
What happened next?

- Atypical features of COVID-19 raised concern for other infection or additional diagnoses. Fungal (Beta-D Glucan and Aspergillus serology), atypical bacterial and vasculitic screens were negative.

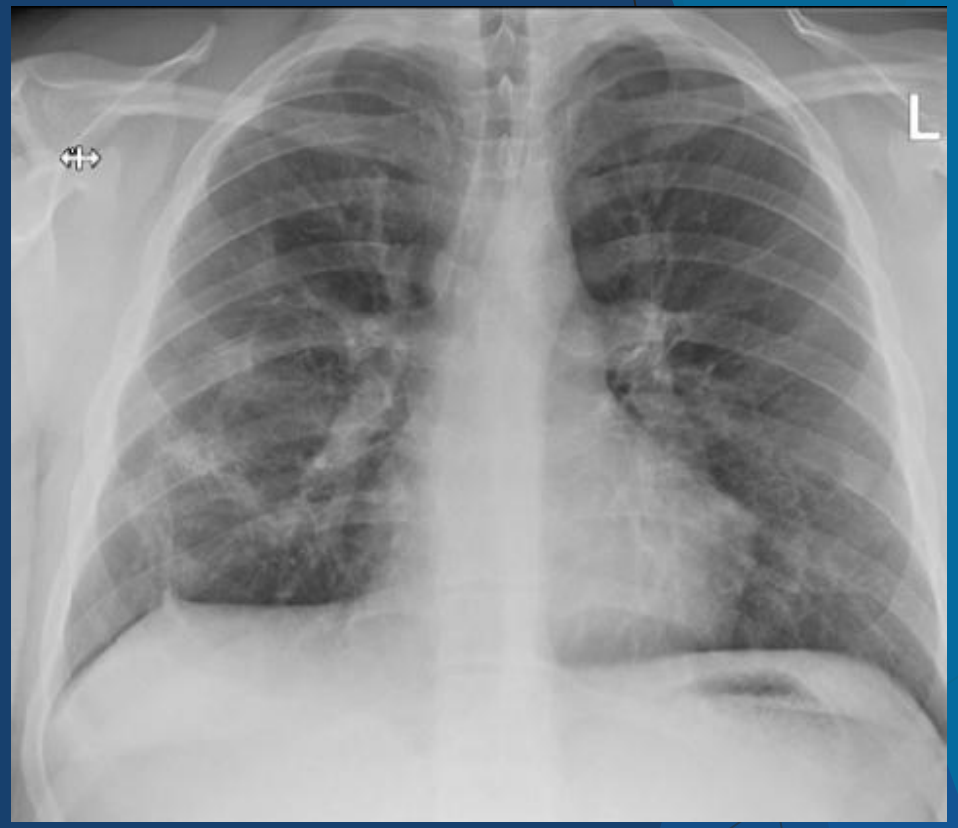
- Percutaneous drainage of the largest, fluid-filled cavity was considered, but not performed on MDT (Multi-Disciplinary Team) consensus, as the patient's modest symptoms did not warrant the risk of introducing pleural infection and creating a bronchopleural fistula.

- The patient was treated conservatively and monitored with serial CXRs for six weeks. These showed reduction in size of the cavities with resolution of the air-fluid level.

Follow-up Chest radiographs



- ***Figure 4:*** This three week follow up radiograph shows reduction in the size of the cavities, particularly the right mid zone cavity, with minimal fluid present in the dependent portion of the cavity.
- There is significant improvement in the consolidative shadowing in the periphery of both lungs.



- ***Figure 5:*** At six weeks, there is further reduction in the size of the cavities with atelectatic scarring in the right mid zone.

Differentials

Very large post COVID-19 cavities have not been previously reported such as in our case.

- **Post COVID-19 pneumatoceles**

Pneumatoceles are thin-walled parenchymal cysts that can complicate severe pneumonia. Histology of pneumatoceles shows inflammation and necrosis of airway walls with formation of direct communication between the bronchovascular interstitium and the pulmonary parenchyma [4].

Interval imaging can differentiate pneumatoceles from lung abscess, due to their characteristics of rapid change in appearance, size and location [5]. Pneumatoceles resolve spontaneously.

- **Secondary infection leading to lung abscess formation**

Differentials continued

- **Cavitating pulmonary infarcts due to COVID-19 related thrombo-embolic disease or micro-thrombotic process**

High incidence of pulmonary emboli (PE), up to 30%, has been reported in patients with COVID-19 [6]. A COVID-19 PE phenotype is suggested, as clots commonly occur in peripheral vessels, possibly due to thromboembolic disease and in situ immunothrombosis [7]. Cavitation, due to aseptic liquefaction or secondary infection [8], can complicate pulmonary infarction in 2.7-7% of cases [9] and was reported during the 2003 SARS pandemic [10].

- **Bronchopleural fistula**

The largest cavity in our patient, contained an air-fluid level which raised the suspicion of a broncho-pleural fistula: a communication between the pleural space and the bronchial tree. A definite airway-cavity communication was not identified after careful review of CT reconstructions in all planes, however this does not necessarily rule out a broncho-pleural fistula.

Teaching points

- Delayed cavitation is a rare, symptomatic feature of Covid-19 pneumonia.
- Exclusion of additional pathologies and close clinic-radiological cooperation should guide treatment.
- In well individuals, post Covid-19 cavitation may be successfully managed conservatively.
- Lymphadenopathy may be a predictor of worse outcome in severe COVID-19 pneumonia

Written informed patient consent for publication has been obtained.

References

- [1] British Society of Thoracic Imaging: Imaging in COVID 19 Infection Guidance for the Reporting Radiologist British Society of Thoracic Imaging (Version 2,updated 16th March 2020)
- [2]Lang M, Som A, Mendoza DP, et al (2020) Hypoxaemia related to COVID-19: vascular and perfusion abnormalities on dual-energy CT. Lancet Infect Dis. 2020;S1473-3099(20)30367-4 (PMID: 32359410)
- [3] Valette X, du Cheyron D, Goursaud S (2020) Mediastinal lymphadenopathy in patients with severe COVID-19. Lancet Infect Dis. 2020;S1473-3099(20)30310-8 (PMID: 32330440)
- [4] Quigley MJ, Fraser RS (1988) Pulmonary pneumatocele: pathology and pathogenesis. AJR Am J Roentgenol. 1988;150(6):1275-1277 (PMID: 3259364)
- [5] Kim SH, Chung YT, Lee KD, et al. (2005) Infected pneumatocele following anaerobic pneumonia in adult. Korean J Intern Med. 2005;20(4):343-345 (PMID: 16491835)
- [6] Leonard-Lorant I, Delabranche X, Severac F, et al. (2020) Acute Pulmonary Embolism in COVID-19 Patients on CT Angiography and Relationship to D-Dimer Levels. Radiology. 2020;201561 (PMID: 32324102)
- [7] van Dam LF, Kroft LJM, van der Wal LI, et al. (2020) Clinical and computed tomography characteristics of COVID-19 associated acute pulmonary embolism: A different phenotype of thrombotic disease? Thromb Res. 2020;193:86-89 (PMID: 32531548)
- [8] Morgenthaler TI, Ryu JH, Utz JP (1995) Cavitary pulmonary infarct in immunocompromised hosts. Mayo Clin Proc. 1995;70(1):66-68 (PMID: 7808055)
- [9] Libby LS, King TE, LaForce FM, Schwarz MI (1985) Pulmonary cavitation following pulmonary infarction. Medicine (Baltimore). 1985;64(5):342-348 (PMID: 4033411)
- [10] Oudkerk M, Büller HR, Kuijpers D, et al. (2020) Diagnosis, Prevention, and Treatment of Thromboembolic Complications in COVID-19: Report of the National Institute for Public Health of the Netherlands. Radiology. 2020;201629 (PMID: 32324101