CASE SERIES

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Maps of Death in Patients With Covid-19

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ABSTRACT: Introduction: Coronavirus disease (COVID-19) is a highly lethal infectious disease caused by the SARS-CoV-2 virus, a novel coronavirus that led to a pandemic. We know that COVID-19 affects the lungs, but it can also affect vital organs, damage blood vessels, and lead to ARDS. We hypothesized that this toxic plasma leads to mechanical destruction of the microvascular capillary endothelium, especially in lung tissues, and contributes to mortality caused by ARDS due to diffuse pulmonary capillary endotheliitis. We aimed to present the common specific radiologic findings of deceased patients from COVID-19. **Methods:** We retrospectively re-evaluated the radiological findings of 15,373 patients diagnosed with COVID-19 at VM / Medical Park Samsun Hospital, Samsun, Türkiye, between March 2020 and January 2023, and we aimed to present the specific radiologic findings of deceased patients from COVID-19. **Results:** The majority of patients died due to complications of hypoxemia caused by lung involvement, known as ARDS with or without tissue damage, resulting in barotrauma and other mechanical complications. **Conclusion:** COVID-19 starts as an infectious, contagious disease and leads to death in infected individuals because it may render blood plasma toxic. We hypothesized that this toxic plasma leads to mechanical destruction of the microvascular capillary endothelium, especially in lung tissues, and the mortality caused by ARDS due to diffuse pulmonary capillary endotheliitis.

KEYWORDS: Covid-19, ARDS, Death, Radiology, Toxic plasma

INTRODUCTION

In December 2019, cases of pneumonia of unknown cause were reported in Wuhan/China, and in January 2020, a newly identified coronavirus, which had not been detected in humans before, was identified. Initially referred to as 2019-nCoV, the disease was later named COVID-19. After emerging in China, it rapidly spread worldwide within about three months. As of March 2020, the COVID-19 outbreak, declared a pandemic by the World Health Organization, had evolved into a globally deadly epidemic and continues to be observed as an endemic. Many parameters have been studied to understand this disease and the relationship between severity and death worldwide, and efforts have been made to prevent mortality and elucidate the cause of death [1,2].

Death from COVID-19 is mainly caused by lung involvement, known as ARDS (Acute Respiratory Distress Syndrome), with severe hypoxemia and tissue damage resulting in barotrauma, namely pneumothorax, as mechanical complications. We believe that the underlying cause of this is the tissue damage caused by a fundamental pathology known as toxic plasma, which is characterized by an increase in circulating proinflammatory cytokines stemming from endotheliitis [3].

We hypothesized that this toxic plasma leads to mechanical destruction of the microvascular capillary endothelium, especially in lung tissues, and the mortality caused by ARDS due to diffuse pulmonary capillary

endotheliitis, and aimed to review and present the common specific radiologic features of deceased patients from COVID-19.

MATERIALS AND METHODS

We retrospectively examined the radiological findings of 15,373 patients diagnosed with COVID-19 at VM / Medical Park Samsun Hospital, Samsun, Türkiye, between March 2020 and January 2023, and we aimed to identify shared radiologic features among the deceased patients.

The study was performed in accordance with the principles of the Declaration of Helsinki. The written informed consents were obtained from patients.

Patient Selection

Patients who were suspected to be infected with COVID-19 were confirmed with clinical, laboratory, and/or radiologic results and included in the study. The radiological features were obtained and re-viewed from files of deceased patients with COVID 19.

COVID-19 testing

Respiratory samples were obtained by nasopharyngeal and nasal swabs and analyzed by RT-PCR assay. Confirmation of a COVID-19 tests were made according to the positive results of reverse-transcriptase-polymerase-chain-reaction (RT-PCR) assay and with the consistent high-resolution CT(HRCT) findings. Also, we included in the study those whose multiple nasopharyngeal swab samples were negative for COVID-19 by RT-PCR, but then serological IgM/IgG antibodies against SARS-CoV-2 were detected by a rapid antibody test.

CASES AND RESULTS

Between March 2020 and April 2021, a total of 15,373 COVID-19 patients who applied to the Chest Diseases Clinic of Samsun Medicalpark Hospital were retrospectively evaluated. The mean age was 60.1 ± 18.1 years.

We lost the majority of the patients due to complications related to hypoxemia caused by lung involvement known as ARDS, with or without tissue damage resulting in pneumothorax and mechanical complications. We believe that the underlying cause of this is the tissue damage caused by a fundamental pathology known as toxic plasma, which is characterized by an increase in circulating proinflammatory cytokines stemming from endotheliitis. We present radiological images that support this hypothesis, especially in our deceased patients (Figures 1-8).

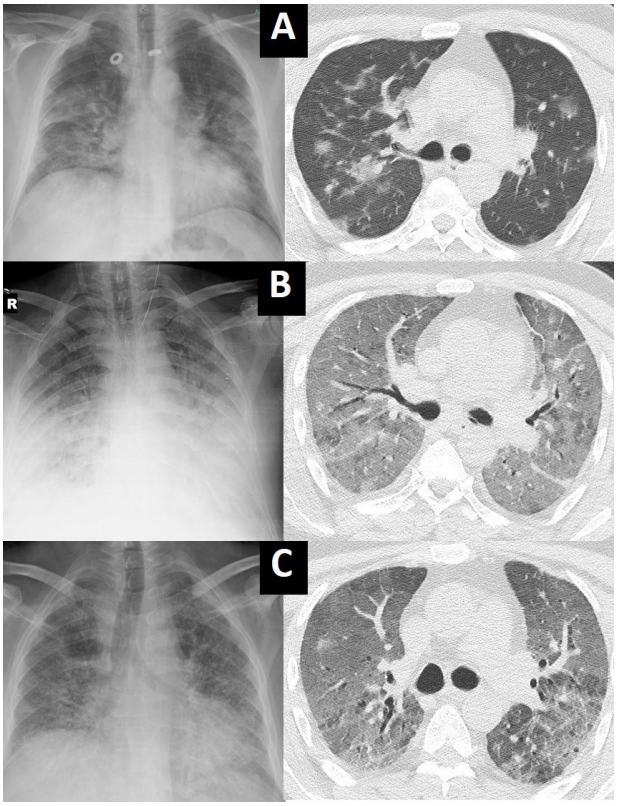

FIGURE 1. 53 years-old male patient with COVID-19 PCR positive. Early phase lung involvement is shown in Figure 1A as small patchy GGO's and consolidations just around the pulmonary capillary. The progressive (1B) and severe phases (1C) are showing the bilaterally diffuse GGOs and fibrotic changes consistent with "white lungs" due to chemically "baked or boiled" lung parenchyma as a result of toxic plasma.

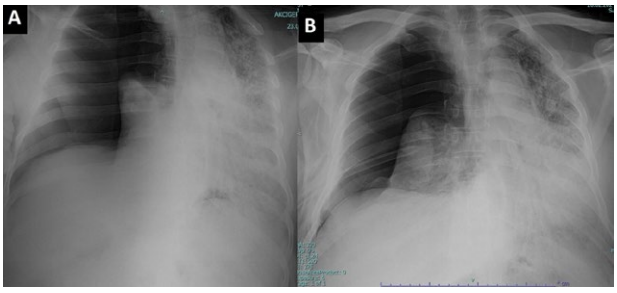

FIGURE 2. Pneumothorax and totally collapsed right lung (2A) is shown in 1st patient with COVID-19. Also, there is no expansion despite the chest tube insertion due to the loss of elastic recoil of the pulmonary parenchyma is shown in Figure 2B.

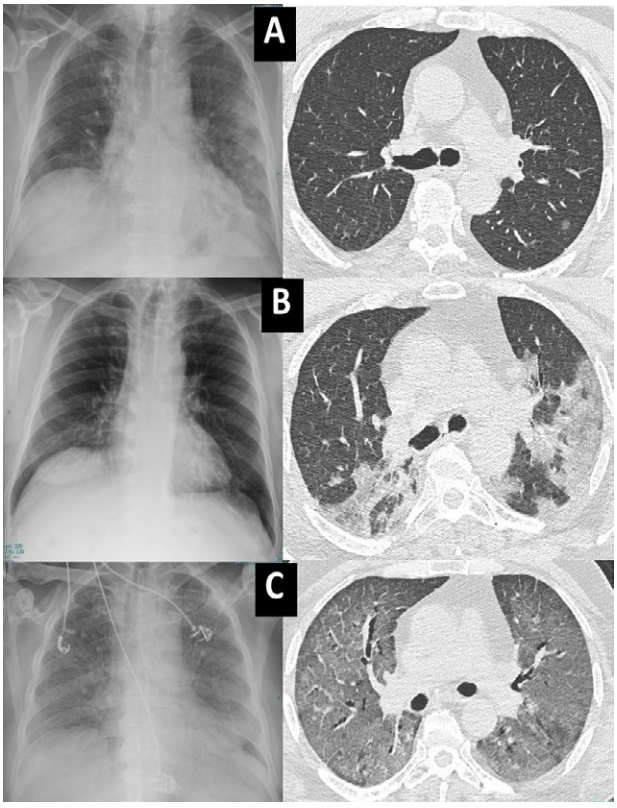

FIGURE 3. The small nodular GGO (3A) is shown as a first involvement in a 69-year-old male patient with PCR-positive COVID-19. And, bilaterally alveolar consolidations were noted after 6 days (3B), and "white or cooked lung" developed (3C) 2 weeks later. He died from Hypoxemic respiratory failure.

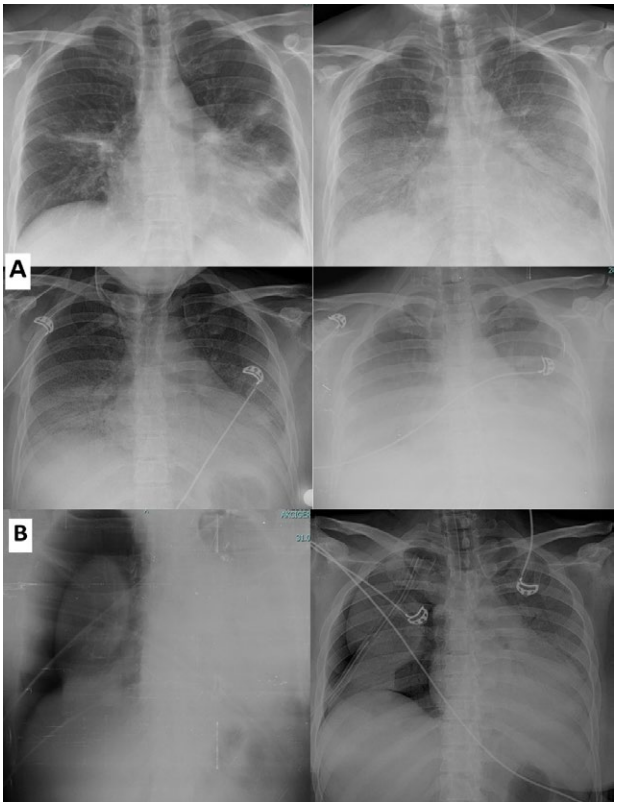

FIGURE 4. Chest X-ray images are showing the early, progressive and severe phases of COVID 19 in a 33 years-old female patient within two weeks (4A). Pneumothorax was noted and there is no expansion despite the chest tube insertion due to loss of elastic recoil of pulmonary parenchyma is shown in Figure 4B. And, she died from pneumothorax and respiratory failure.

One of the most common causes of death in COVID-19 patients is pneumothorax. When a pneumothorax occurs in a COVID-19 patient, partial or complete expansion does not occur despite the insertion of a chest tube. This condition results from the loss of elastic recoil as a result of chemical cooking of the lung parenchyma by toxic plasma.

DISCUSSION

As of May 2023, a total of 687 million COVID-19 cases have been reported worldwide, and the number of deaths has reached 6.8 million. Although the pandemic situation has ended, COVID-19 continues to be observed in society as a disease that still causes death[4]. This disease has four stages [3]:

Stage 1 – In the transmission and infection stage, COVID-19 generally spreads through respiratory transmission, typically when an infected person coughs, sneezes, talks, or breathes. An infected person can also be asymptomatic or show symptoms such as fever, sore throat, etc.

Stage 2 – The stage of systemic inflammatory response; It depends on the viral load in the infected individual, the pathogenicity of the virus, and the immune response of the infected person. Various chemokines and cytokines, primarily including ferritin, are produced. These cytokines transform the plasma of the infected person into a toxic and destructive chemical fluid.

Stage 3 – The endothelial damage(endotelitis) stage; The plasma of the infected person transforms into a toxic and destructive chemical fluid, starting from the innermost layer of the vascular system, known as the endothelial layer, and vascular wall damage occurs. The thin walls of microvascular capillaries break down, the toxic and destructive plasma exits the

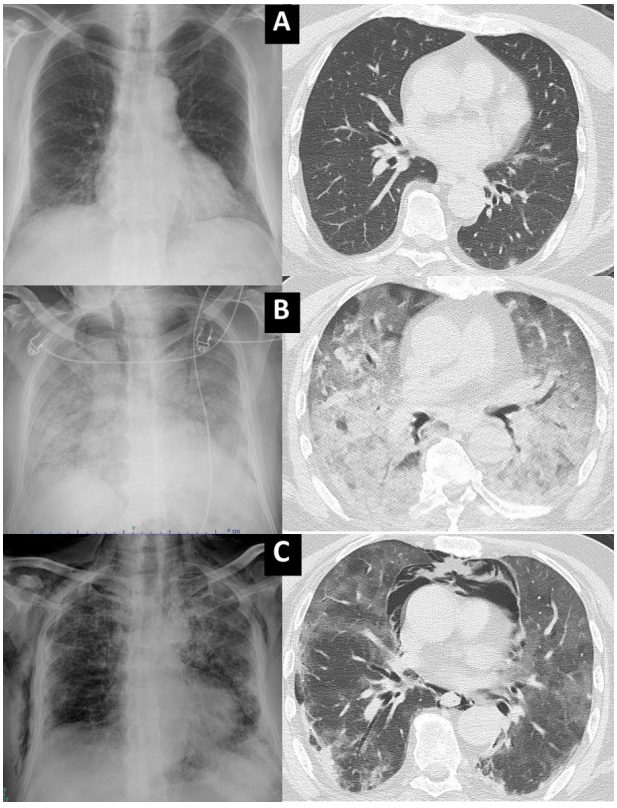

FIGURE 5. A 67-year-old male patient with COVID-19 is PCR positive. Early phase lung involvement is shown in Figure 5A as small patchy GGO's and consolidation. The severe phase (5B) is showing the bilaterally diffuse GGOs and consolidations consistent with "white lungs" due to chemically cooked lung parenchyma as a result of toxic plasma. Also, pneumomediastinum is seen in Figure 5C, and he died from respiratory failure and pneumomediastinum within 3 weeks of the early phase.

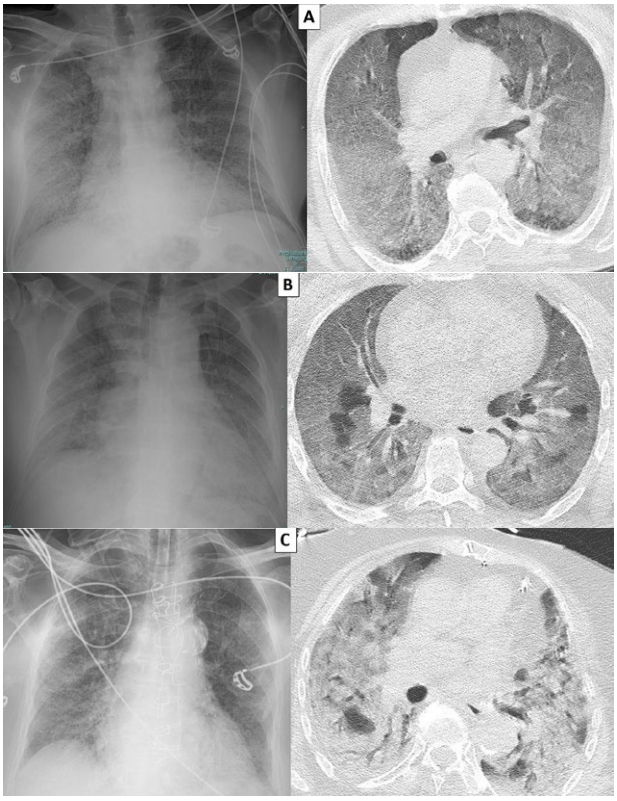

FIGURE 7. The "white or cooked lungs" were seen in 58-year-old male (7A), 58-year-old male (7B), and 84-year-old female (7C) patients with COVID-19. Diffuse bilaterally GGOs and alveolar consolidations are called "white or cooked lungs". It is caused by toxic plasma, and this intravascular toxic plasma leads to diffuse endotheliitis, vascular and tissue damage in patients with COVID-19.

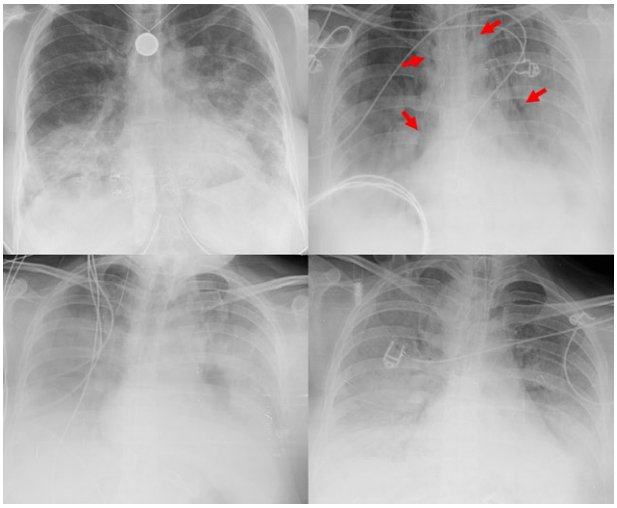

FIGURE 6. A 33-year-old female patient was admitted to the ICU with COVID-19 and respiratory failure. Chest X-ray images show the early, progressive, and severe phases within two weeks. Also, pneumomediastinum was developed (arrows). And, she died from respiratory failure.

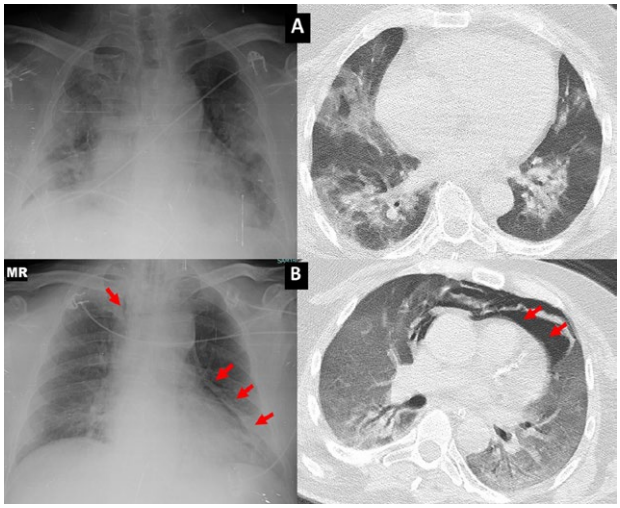

FIGURE 8. Bilateral alveolar consolidations and diffuse interstitial and GGO's were seen in Figure 8A and B. He died from pneumomediastinum (arrows) and hypoxemic respiratory failure.

blood vessels and causes tissue damage. In this context, the vascular system with the most concentrated microvascular capillaries and the thinnest wall structure is the pulmonary capillaries, primarily in the initial lungs, as a result of the endothelium (endotelitis), radiologically detectable ground-glass opacities are identified (Figures 9-14).

Stage 4 – Tissue damage and severe illness; The toxic and destructive plasma leads to tissue damage depending on the infected individual's response and the host's immune reaction. As a result of this, primarily respiratory failure, along with organ failure and death due to inadequate tissue perfusion related to endothelial dysfunction, arises.

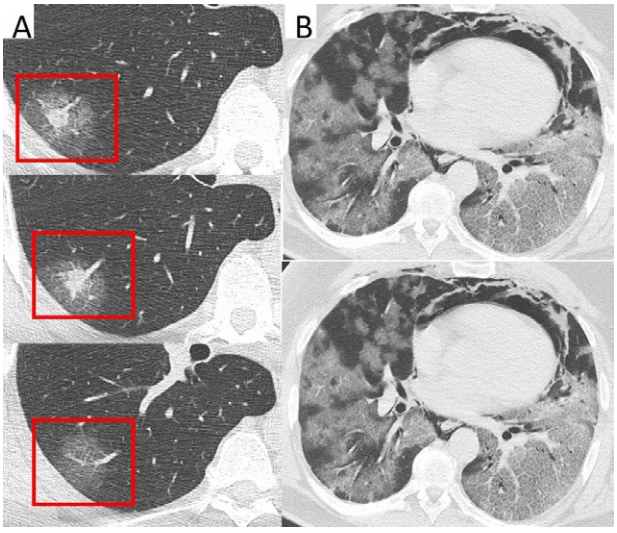

FIGURE 9. 56 years-old male presented with PCR-positive COVID-19. The first and early lesions start from pericapillary GGO and consolidation (9A). This pericapillar GGO is the best evidence of endotheliitis due to chemically destroyed to endothelium in pulmonary capillaries consisting only of the endothelial layer. And, it leads to chemically cooked lung parenchyma as a result of toxic plasma.

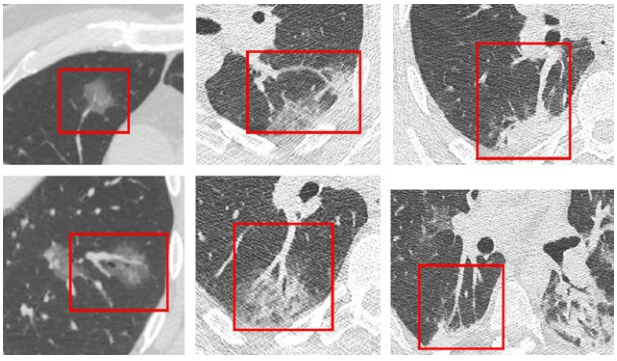

FIGURE 10. The pericapillary GGOs (early lesions) and consolidations (10 days later), like "lightning strikes," are shown in a 53-year-old male with PCR-positive COVID-19.

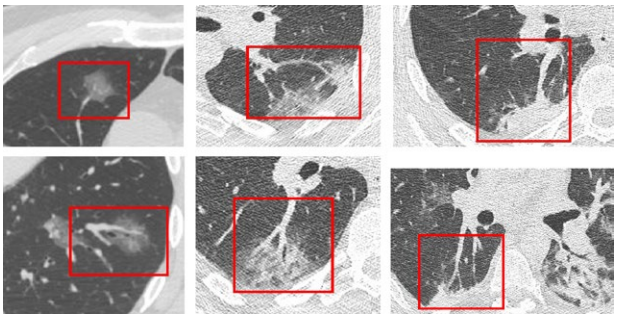

FIGURE 11. The GGOs are showing around the capillary in a 52-year-old female with COVID-19 in the early phase.

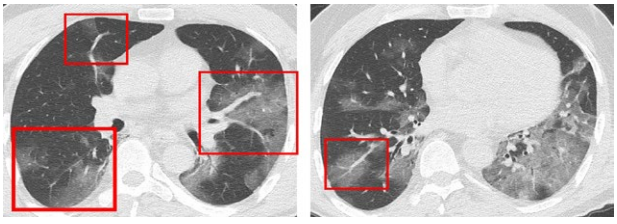

FIGURE 12. Bilaterally diffuse GGOs are showing, especially around the capillary in a 50-year-old male with COVID-19 in the progressive phase.

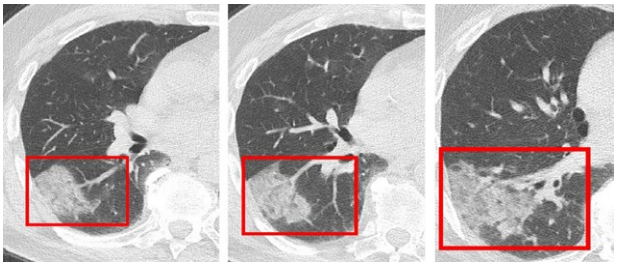

FIGURE 13. Localized GGOs with widening of the vessels are showing in a 74-year-old male with COVID-19.

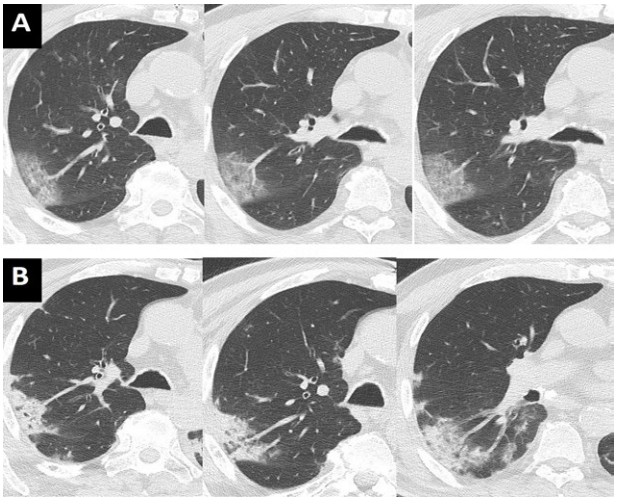

FIGURE 14. A 85 years old-male admitted to the hospital with PCR-positive COVID-19. Pericapillar GGOs were noted (14A), and fibrotic changes and consolidation were seen around the capillary, like "lightning strikes".

The lung involvements associated with COVID-19 can vary according to the radiological stages of the disease; they can primarily be observed as ground-glass opacities (GGOs), in addition to consolidations, linear interstitial infiltrations, reverse halo sign, and fibrotic areas[3,5].

We can interpret COVID-19 disease findings on CT imaging in 4 phases (early, progressive, severe, and dissipative). In the early phase, numerous small patchy shadows and interstitial changes appear and exhibit a distribution starting near the pleura or bronchi rather than the pulmonary parenchyma. When the lesions increase and enlarge, developing into multiple GGOs and infiltrating consolidation in both lungs, it corresponds to the progressive phase. In the severe phase, massive pulmonary consolidations and "white lungs" are recognized, but pleural effusion is uncommon. As the lesions began to change into fibrosis, the GGOs and pulmonary consolidations were completely absorbed, which implies the dissipative phase[3].

Our patients with diffuse interstitial involvement had higher mortality rates. We named this radiological appearance "baked or boiled lungs". Patients with this radiological appearance often experienced exitus due to hypoxemic respiratory failure. The most common complication leading to mortality was pneumothorax. In cases of pneumothorax, despite the presence of a chest tube, in many cases, lung expansion did not occur due to the loss of lung elasticity caused by the "baked or boiled lungs" effect. Pneumomediastinum is also a frequently observed complication, and despite its high frequency, it has better survival rates compared to patients with pneumothorax in terms of mortality. According to reports and articles, pneumothorax and pneumomediastinum are the most common causes of mortality in patients with COVID-19 due to mechanical complications [3,6-8].

We know that COVID-19 continues to persist as a fatal disease among us, and lung involvement, along with hypoxemic respiratory failure, continues to be the most frequent cause of death. Stage 2 of the disease plays a key role in patients with COVID-19. The stage of systemic inflammatory response depends on the viral load in the infected individual, the pathogenicity of the virus, and the immune response of the infected person. Various chemokines and cytokines, primarily including ferritin, are produced [3,9-16]. It has been shown that Interleukins (such as IL-1, IL-4, IL-6, IL-7, IL-10, IL-12, IL-17, and IL-18), IFN-γ, TNF-α, TGF-β, and NF-κB play major roles in the body's inflammatory response to SARS-CoV-2 infection [15]. These cytokines transform the plasma of the infected person into a toxic and destructive chemical fluid. This stage leads to the endotheliitis stage (stage 3) in depends on the patient's immune response.

One typical radiological finding in patients with COVID-19 is vascular enlargement within areas of ground-glass opacity. In our patients, the earliest pulmonary lesions appeared as GGOs surrounding the pulmonary capillaries (Figure 15). These findings may support the presence of endotheliitis caused by chemical damage to the endothelium of pulmonary capillaries, whose walls consist primarily of an endothelial layer. This process may contribute to chemically "cooked" lung parenchyma as a result of toxic plasma.

If the viral load and systemic inflammatory response are high, endotheliitis may affect all pulmonary capillaries and cause diffuse lung lesions. We identified diffuse interstitial involvement, which we refer to as "baked or boiled lungs," as the most lethal form of lung involvement, and pneumothorax as the most fatal complication.

We showed to endotheliitis in the biopsy section of the lung parenchyma. Haematoxylin&Eosin-stained sections from representative areas of lung parenchyma were seen with the mixed-type inflammatory-cell infiltration of lung tissue and exudative capillaritis with thickened microvascular walls, as reported in articles[15-17].

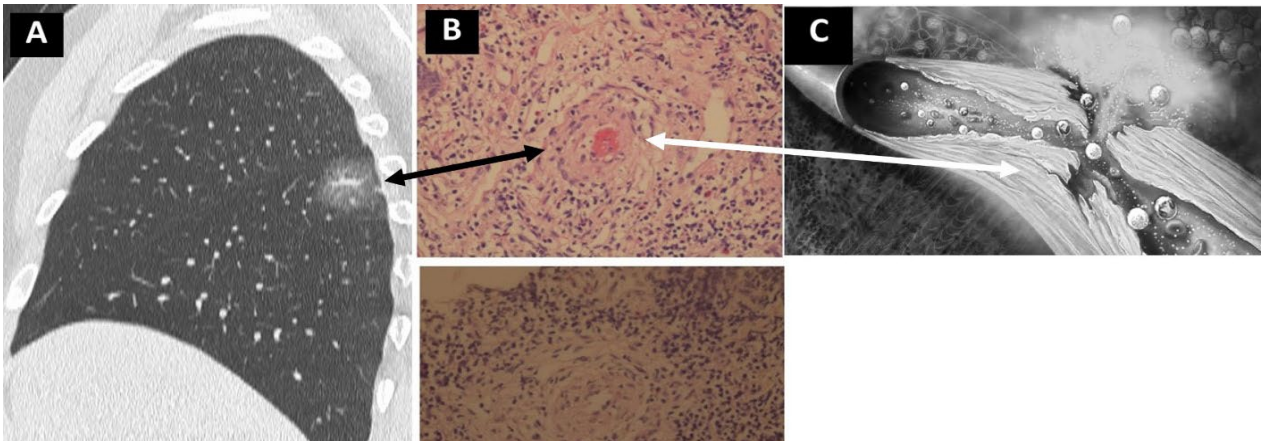

FIGURE 15. A perivascular ground-glass opacity with widening of the pulmonary vessels is seen in Figure 15A. The pulmonary histopathologic features and illustration of endotheliitis are shown in patients with COVID-19 (Arrows in Figure 15B and C*).
*Permission of Kenneth Warrington, MD. Mayo Clinic Minute: How vasculitis affects the body. June 21, 2023.

As a result, COVID-19 starts as an infectious contagious disease and leads to death in infected individuals because it renders the blood plasma toxic. We hypothesized that this toxic plasma leads to mechanical destruction of the microvascular capillary endothelium, especially in lung tissues, and the mortality caused by ARDS due to diffuse pulmonary capillary endotheliitis.

COMPETING INTERESTS: *No potential conflict of interest was reported by the authors.* **AUTHORS CONTRIBUTIONS:** *AD, SO, and TC contributed to the design and implementation of the research, to the analysis of the results, and to the writing of the manuscript. SO and AD contributed to the patient's care. SO and ZK contributed to the hypothesis of pathophysiological features. CB and DO contributed to the collection of data and figures from the files of cases. All authors have read and agreed to the published version of the manuscript.* **FUNDING STATEMENT:** *This research received no specific grant from any funding agency in the public, commercial, or not-for-profit sectors.* **FINANCIAL SUPPORT:** *The authors declare that no financial support was provided for the conduct of this study.*
DATA AVAILABILITY STATEMENT: *All data generated or analysed during this study are included in this article. Further enquiries can be directed to the corresponding author.*

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