

Pulmonary cavitation – an unexpected finding in late stage COVID-19 pneumonia (case report)

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ABSTRACT

The typical CT features of COVID-19 pneumonia include multifocal and bilateral ground-glass opacities with or without consolidation, found in both lungs, predominantly at peripheral, and posterior regions, bronchovascular thickening, crazy pavement appearance (ground-glass opacities with superimposed interlobular septal thickening). Atypical imagistic findings such as lung cavitation were rarely reported. In this report we describe the case of a 42 years old, healthy man with severe COVID-19 pneumonia who developed two pulmonary cavities during recovery. The pulmonary cavitations formed in the area of the lung where patchy air space opacification was seen in early stages. There were no signs of invasive fungal or bacterial infection and the complementary investigations have ruled out other possible etiology for lung cavitation. Although the pathophysiological mechanism involved in the origin of the pulmonary cavities is not fully known, it could be closely related to diffuse alveolar damage in severe COVID-19 pneumonia.

Keywords: COVID-19, severe pneumonia, lung cavities, serial recovery

INTRODUCTION

The new coronavirus SARS-CoV-2 has been spreading pandemically since the end of 2019 and became the most serious medical challenge of the century. The main route of infection is airborne, through respiratory secretions. Although the virus has also been isolated from feces, blood, eye secretions and semen, the other transmission routes are questionable. The clinical manifestations are variable, most being asymptomatic, mild or moderate forms. Severe forms require additional oxygen supply and can occur in 15% of cases, 5% of which can become critical forms, which require specific interventions of intensive care (1). The severity of the evolution is influenced by the viral genetic variant, by the co-morbidities and behavioral risk factors of

the host or by environmental factors, such as pollution or climate (1,2). The long-term consequences of pulmonary and systemic SARS-CoV-2 infection are still unknown, but systematic post-COVID clinical, imaging and biological monitoring will be able to clarify risk factors and the prevalence of complications and sequelae.

CASE PRESENTATION

A 42-years old man, living in rural area, presented on December 2020 with a 3 weeks history of high fever up to 39°C, dry cough, shortness of breath, extreme night sweats, fatigue. The patient denied smoking, traveling in the last two weeks, contacts with sick people and any history of medical problems.

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Physical examination on admission revealed an overweight patient, with a normal body temperature, mild dyspnea, with a respiratory rate of 24/minute and pulse oximetry of 90% on room air. He was hemodynamically stable, and the systemic examination of other system were within normal limits.

Given the current epidemiological context, RT-PCR SARS-CoV-2 of the nasopharyngeal swab was performed and the positive result confirmed the infection with the novel coronavirus.

Laboratory examination revealed normal blood leukocytes with a increase rate neutrophils/lymphocytes, inflammatory biologic syndrome, increased lactate dehydrogenase, elevated D-dimer and high ferritin level (Table 1).

Chest radiography showed atchy air space opacification projected in the lower 2/3 of both lung areas. The first diagnosis was COVID-19 pneumonia with respiratory failure and severe prognosis. The provided treatment included antivirals (Favipiravir and Remdesivir), low molecular weight heparin prophylactic dose, corticosteroids (Dexamethasone), antibiotic (Ceftriaxone), supplemental oxygen therapy and “prone position” for 16 to 18 hours daily.

After seven days, the patient was admitted to medical Intensive Care Unit due to progressively worsening clinical status with severe dyspnea, hypoxia and respiratory failure. In addition to his previous treatment, the patient received non-invasive ventilation with continuous positive airway pressure (CPAP) and Tocilizumab (800 mg) with slightly improvement in clinical and biochemical parameters.

The patient was readmitted in the infectious diseases department after two weeks. Physical examination revealed restlessness, fatigue, nasal bridge pressure ulceration, armpit temperature of 37.8°C, tachycardia (113/min), normal blood pressure

(108/81 mmHg), respiratory rate of 24-27/min and pulse oximetry of 94-95% with oxygen pressure of 6-8 l/min on a simple mask (Figure 1).

Repeated chest radiography describes patchy air space opacification in both mid and lower zones of the left lung with erasure of the heart contour, low intensity opacity in the right lower zone and residual patchy air space opacification in the right upper zone (Figure 2).

Laboratory analysis demonstrated high white blood cell with neutrophilia, inflammatory increased markers, elevated procalcitonin, D-dimer, and liver enzymes. Clinic and biologic data suggested the possible development of bacterial sepsis with nosocomial origin and the medical decision was the antibiotic empiric treatment with Vancomicine, Meropenem and Fluconazole. The urine and blood microbial culture were negative, but coagulase negative staphylococcus (CoNS) was isolated from the nasal bridge wound and the pharyngeal samples was positive for staphylococcus aureus, without influence on antibiotic treatment. The patient’s physical condition and laboratory tests slowly improved with a decline in oxygen needs. The RT-PCR SARS-CoV-2 test became negative in the fourth week from the onset, concomitant with fever relapse and health care associated diarrhea with *Clostridium difficile*, recovered after standard treatment.

Chest radiography repeated for a third time showed opacity in both apical and subclavicular right zone with 2 lung cavitations with maximum walls of 4.5-5 mm thick which were new from previous imaging (Figure 3).

The cavity image required investigations that ruled out other differential diagnosis, as primary and metastatic lung cancer, bacterial, fungal or tuberculous lung abscess, pulmonary emboli, granulomatous autoimmune diseases, traumatic pneumatocele or congenital malformations of aerial tree, pulmonary sequestration or bronchogenic cysts.

TABLE 1. Laboratory data on COVID-19 severe pneumonia

| | December 2020 | | ICU | January 2021 | | | |
|----------------|---------------|-----------|-----|--------------|-------------|----------|------------|
| | 25.12 | 30.12 | | 15.01 | 19.01 | 25.01 | 31.01 |
| WBC | 7,200 | 8,400 | | 27,000 | 9,600 | 14,300 | 11,800 |
| N/Ly | 8 | 12 | | 23 | 14.7 | 6 | 6.2 |
| ESR | 69 | 72 | | 62 | 50 | 32 | 20 |
| RCP | 51.7 | 221.5 | | 98.9 | | 67.98 | |
| Fybrinogen | 514 | 578 | | 593 | 485 | 485 | 439 |
| Procalcitonine | neg | neg | | 10 | neg | | neg |
| Lactate acid | 32.3 | | | 16.2 | | | 39 |
| ALT | 47.5 | 53.3 | | 166.2 | 224 | 109.6 | 130 |
| D-dimers | 669 | 2,476 | | 4,803 | 911 | | 810 |
| Feritine | 1,061 | >1,200 | | 1,093 | 1,141 | 994 | |
| Creatinine | 1.08 | 1.0 | | 1.15 | 0.77 | 0.74 | 0.74 |
| Glycemia | 133 | 84.5 | | 60,4 | 129 | 63 | |

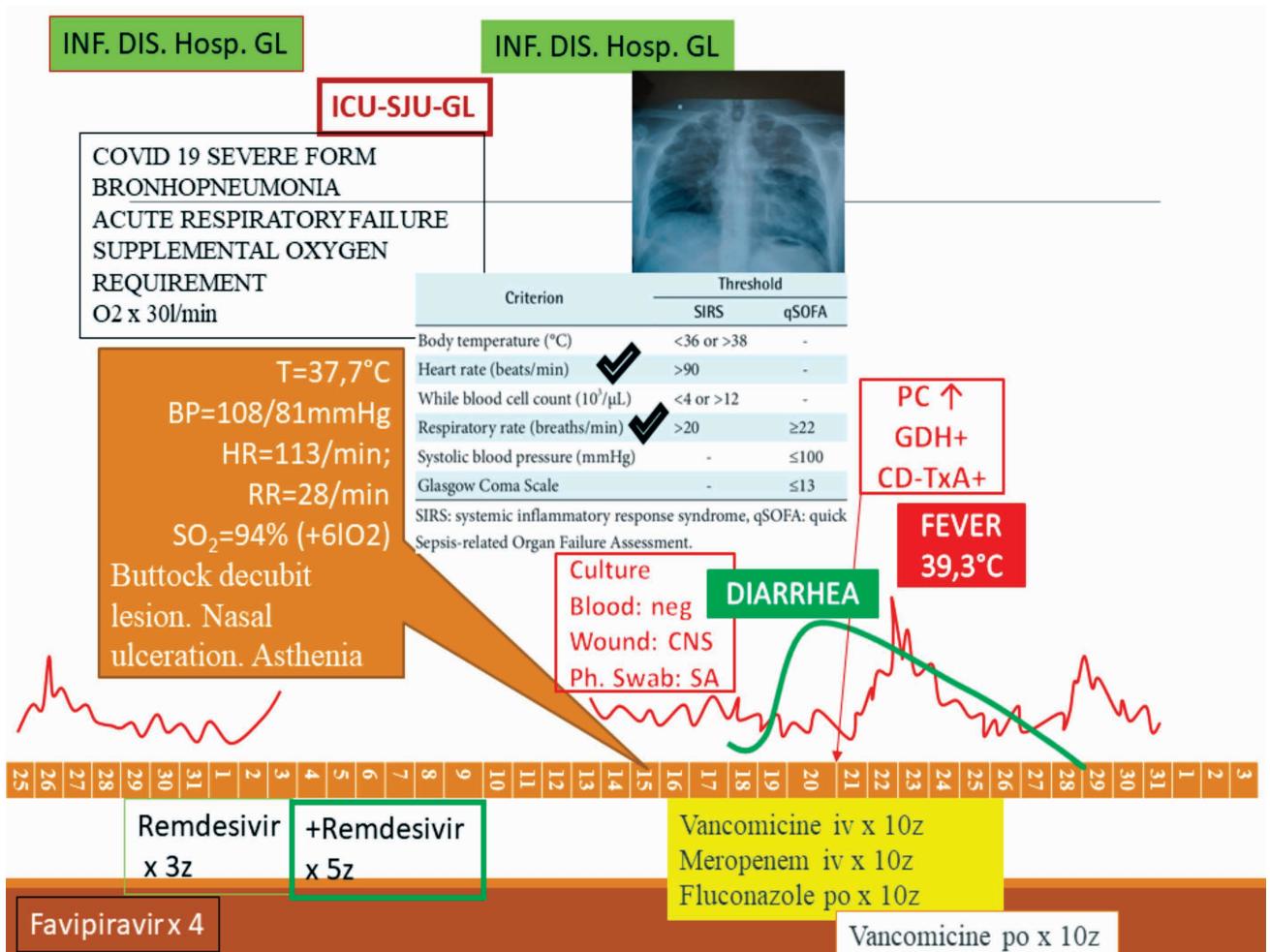


FIGURE 1. Synopsis of the clinical and treatment of COVID-19 severe pneumonia. CNS – coagulazo-negative Staphylococcus; Ph – pharyngeal; SA – Staphylococcus aureus; PC – procalcitonine; GDH – glutamat dehidrogenasis; CD – Clostridium difficile



FIGURE 2. Chest X-RAY showing patchy air space opacification in both mid and lower zones of the left lung, low intensity opacity in the right lower zone and residual patchy air space opacification in the right upper zone

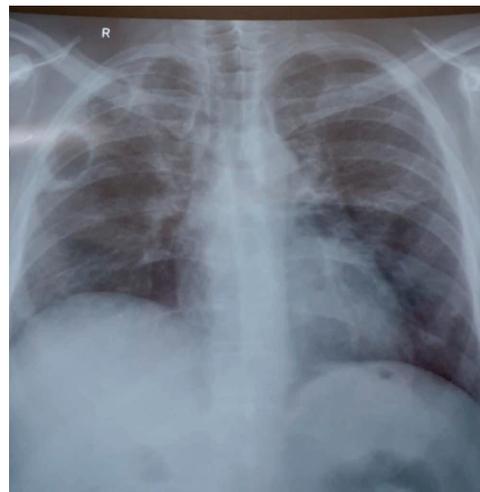


FIGURE 3. Control Chest X-RAY performed one month after admission show a reduction in size and intensity of left lung opacity, opacity in the right lower zone with unchanged appearance, small/medium apical and right subclavicular opacity with two new cavity images with maximum walls of 4.5-5 mm.

Computer tomography of the thorax revealed two pulmonary cavitation of 33/30 mm and 32/27 mm, in contact with each other, situated in the upper right zone, near thoracic pleura, in posterior contact with the right oblique cleft, and bilateral ground-glass opacities with superimposed air space consolidations in upper left zone (Figure 4a, 4b). The thoracic surgery and pneumology evaluation concluded that the cavitary images are bullous dystrophy, probably consecutive to COVID-19, and has no criteria for the surgery cure. The patient was discharged stable, regarding cardio-vascular, respiratory and digestive functions, but with important asthenia and low tolerance for the efforts and require medical rehabilitation.

DISCUSSIONS

The present case revealed a severe COVID-19 pneumonia with respiratory failure supported by noninvasive ventilation and complicated with cavitation image, considered bullous dystrophy. Excepting the overweight, the patient had no significant risk factors for severe illness, including smoking, age over 60 and co-morbidities with diabetes mellitus, hypertension, cardiovascular disease or cerebrovascular diseases (3). However, he developed a severe COVID-19 pneumonia that required admission in the intensive care unit for two weeks. The therapy was complex, consisting intravenous antiviral, anticoagulant, corticosteroids, broad-spectrum antibiotic, anti-cytokine biologic medication, and non-invasive ventilation. Although the respiratory failure was improved, the patient recorded health care associated infections of the decubitus soft tissue lesions and *Clostridium difficile* diarrhea. Moreover, unusual course of the COVID-19 pneumonia with development of acute pulmonary cavi-

tation, in both apical and subclavicular right zone, was documented after virological cure and corresponds to the image of the lung patchy air space opacification, notified in early stages.

Although the pathophysiological mechanism involved in the genesis of the pulmonary cavities is not fully known, it could be closely related to diffuse alveolar involvement, intra-alveolar hemorrhages and parenchymal necrosis from severe forms of COVID-19 (4).

Computer tomography typical features of COVID-19 pneumonia include multifocal and bilateral ground-glass opacities with or without consolidation, found in both lungs, predominantly in peripheral, and posterior regions, bronchovascular thickening, crazy pavement appearance (ground-glass opacities with superimposed interlobular septal thickening). Atypical CT findings are rarely reported, such as pleural effusion, target sign, cavitation, spontaneous pneumothorax, hilar lymphadenopathy, halo sign, pulmonary nodules, tree-in-bud (5).

The pulmonary cavitation was reported in 1.7% of cases of COVID-19 pneumonia and was considered multifactorial involvement of bacterial and fungal co-infection, immunosuppressive effects of the glucocorticoids and tocilizumab, in addition of SARS-CoV-2 specific inflammatory pathways or predisposition to venous thromboembolism and potential to cause lung infarct and micro-infarcts (6,7). Considering the experience of lung sequelae following the severe cases of influenza, it is supposed the influence of noninvasive ventilation on appearance of pulmonary cavitation (8). Concordant to other previous medical reports (9,10), the evidence of pulmonary cavitation was discovered after 30 days from the onset, as an early sequela, requiring a long time follow-up.

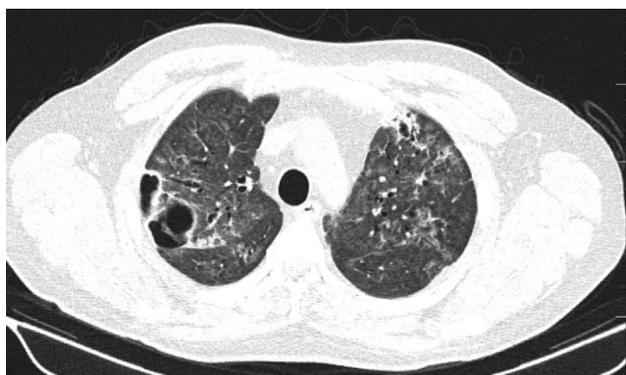


FIGURE 4a, 4b. Computer tomography of the chest – axial lung window – performed at discharge revealed 2 pulmonary cavitations in the upper right zone in contact with each other, near thoracic pleura and in posterior contact with the right oblique cleft (33/30 mm, 32/27 mm) with 3 mm walls, bilateral ground-glass opacities with superimposed air space consolidations in upper left zone

CONCLUSION

The pulmonary cavitation is an unexpected finding in the late stage COVID-19 pneumonia. The pathophysiological mechanism of the COVID-19 re-

lated lung cavities is incompletely understood, and further clinical, biological and imagistic studies are necessary in order to clarify the long term impact of the severe forms of infection.

Conflict of interest: none declared

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