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Original Article

Pulmonary cavitation in follow-up COVID 2019 cases: An etiological perspective

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ABSTRACT

Background: The current COVID-19 pandemic is an ongoing global healthcare challenge that has caused morbidity and mortality at unprecedented levels. Since the post-COVID pulmonary complications are evolving and challenging, a study was carried out to assess pulmonary cavitation in follow-up COVID cases from an etiological perspective. The aim of this study was to assess the incidence of pulmonary cavitation and describe its etiology and evolution in moderate and severe post-COVID pneumonia patients.

Methods: A prospective observational study of all patients admitted to our institution with moderate or severe COVID pneumonia was carried out. Some of these patients again became symptomatic after discharge and developed pulmonary cavitation on imaging.

Results: 6.2% (n = 37) out of 589 patients admitted to our institution with moderate or severe COVID pneumonia developed pulmonary cavitation on follow-up. We describe the imaging characteristics of post-COVID cavitation and present these patients' clinical, laboratory, and microbiological parameters.

Conclusion: Cavitary lung disease in patients with moderate to severe COVID-19 disease is not uncommon, and an etiological workup is necessary to institute timely and correct therapy.

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Introduction

Severe acute respiratory syndrome coronavirus 2 (SARS −COV 2) disease has inflicted a global challenge for the world community, causing colossal morbidity and mortality among the affected. Coronavirus disease 2019 (COVID-19) inflicts the lungs primarily. The lung injury is of varying severity, potentially causing severe acute respiratory distress syndrome (ARDS). After recovery from COVID, many patients also experience a decline in their health status and function. Dysregulation in immune regulation after hyperinflammatory acute response has been implicated as the mechanism behind post-COVID complications.

Post-COVID prolonged and persistent effects on lungs have been elucidated in various studies. In the United States, it was observed that a meagre 6.75% of patients achieved their previous health function after suffering from COVID. In the United Kingdom, around one-third of individuals among 47,780 patients who were followed up until an average of 140 days, were readmitted with one or the other symptom.[1] All these studies emphasize the need for long-term follow-up of COVID patients. The post-COVID complications are evolving and are being recognized more frequently as the research and literature on the subject is emerging. Post-COVID pulmonary fibrosis is the most frequently observed sequelae and it has been extensively studied. The other pulmonary complications like post-COVID cavitary disease and airway disease have also been reported, albeit in case series.[1] The common findings seen on computed tomography (CT) of the chest in patients with COVID-19 have been elucidated in detail,[2] in comprehensive reviews and meta-analyses[3] of as many as 55 studies. Of all the CT scans studied, peripheral ground-glass opacities were the most frequent abnormality detected followed by consolidation in 44% (95% CI 1−71%). Some chest CT findings are not frequently associated with COVID-19 viz. cavity and cysts.[4]

Pulmonary cavitation in follow-up cases of COVID, although infrequent, is not a rare entity. The etiology of these cavities is multifactorial and warrants comprehensive workup and timely treatment. Therefore, we did a study to ascertain the frequency, etiology, and outcome of cavitary lung lesions in critically ill patients with COVID-19 on post-discharge follow-up.

Material and methods

The study was carried out at a tertiary care chest center in northern India. A record of all patients admitted with COVID-19 from April 2021 to July 2021 was maintained. All moderate to severe COVID pneumonia cases (definition as per national guidelines) that were discharged and reported for follow-up in pulmonary OPD of post-COVID clinic were included in the study. Baseline demographic characteristics, comorbidities, symptoms, duration of hospitalization, and duration of systemic steroids were noted. We followed up with 589 patients affected with moderate or severe COVID-19 pneumonia out of which 37 patients had lung cavitation as seen on follow-up CT chest. Baseline chest radiographs of these patients at initial admission were reviewed, which revealed radiographic changes characteristic of COVID pneumonia, and no cavities were observed by the radiologist on those chest radiographs. A baseline CT scan on admission was not carried out for all patients in concurrence with the national guidelines as a CT scan was not considered mandatory for either COVID diagnosis or treatment and was not feasible in such an overwhelming public health care scenario. Baseline CT at admission was available for 15 of these 37 patients, which revealed characteristic COVID pneumonia findings, and no cavitation was seen. Subsequently, on follow-up, patients who were symptomatic were investigated based on symptomatology. These patients underwent contrast-enhanced computed tomography (CECT) of the chest and those with suspected embolism based on clinical profile and raised d dimer levels underwent CT pulmonary angiography. The CT scan was performed by a 256-slice CT scanner. All CT scans were reviewed by a radiologist and imaging characteristics including pulmonary cavitation, number, size, location of cavities, and features of other fungal, superadded infection or pulmonary artery thrombus or dilatation was noted. All patients with cavities on the CT chest were further investigated to ascertain the etiology. They were subjected to sputum microscopy and culture for pyogenic organisms; cartridge-based nucleic acid amplification test CBNAAT (gene Xpert for mycobacterium tuberculosis [MTB]) and fungal stain. Sputum-negative cases were subjected to Bronchoalveolar lavage (BAL), which was sent for microscopy, CBNAAT, fungal PCR, and galactomannan assay.

Data were expressed as mean ± SD when normally distributed. Proportions were used as descriptive statistics for categorical variables. Statistical analysis was performed using SPSS software version 20.0 (SPSS Inc, Chicago, IL, USA).

Treatment of COVID patients was done as per national guidelines on COVID issued by ICMR which were evolving. Steroids were administered to patients with moderate to severe COVID. Tocilizumab was used for those in cytokine release storm based on clinical and laboratory parameters as per national guidelines. Empirical antibiotics and antifungals were not routinely administered unless suspicion of secondary infection was high. Thromboprophylaxis was given as per existing guidelines. Ventilation was done using a high-flow nasal cannula or non-invasive ventilation as per ARDS protocol.

Results

Among 589 patients, 6.2% (n = 37/589) were found to have cavities on imaging. Table 1 describes their baseline characteristics, clinical variables, and outcomes.

The mean age of patients was 52 years (ranging from 28 to 76 years), 57% (n = 21) were males, and 43% (n = 16) were females. A total of 43% (n = 16) patients had diabetes mellitus (DM). Other comorbidities observed were hypertension, rheumatic heart disease (RHD), and renal transplant recipient as depicted in Table 1. Symptoms of patients are shown in Fig. 1. The mean duration of hospitalization was 29 days, ranging from 15 to 60 days. Steroids were exhibited to all patients with a mean duration being 13 days (ranging from 7 to 21 days). On follow-up, in addition to findings of fibrosis of

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Table 1 – Baseline demographic, radiological, and etiological characteristics of all patients.

Patient	Age	Sex	Comorbidities	Hospitalisation	Steroids	Symptoms (Yes-1, No-2)					Sputum	D Dimer	Broncho alveolar lavage	Gene Xpert	Fungal PCR	Galacto	CT findings	Final diagnosis	Treatment	Outcome
				(no. of days)	(days)	Fever	Cough	Dyspnea	Hemoptysis	Weight loss	Gene Xpert		Pyogenic C/S							
1	42	M	RTR, DM, HTN	60	21	1	1	1	2	2	–	Normal	–	–	Positive	Raised	Cavity RML with soft tissue contents	Fungal	Antifungal	Resolution
2	53	M	RHD	30	21	1	1	1	2	2	–	Normal	–	–	Positive	Raised	Cavity RUL	Fungal	Antifungal	Resolution
3	76	F	DM	30	15	2	2	1	2	2	–	Elevated	–	–	–	–	Cavity LUL	PTE	Anticoag ulation	Resolution
4	54	M	–	15	10	1	1	2	2	2	MTB Det	Normal	–	–	–	–	Cavity RUL	TB	ATT	Resolution
5	45	F	–	30	21	2	1	1	1	2	–	Normal	–	–	Positive	Raised	Cavity RML with soft tissue contents	Fungal	Antifungal	Resolution
6	63	M	DM	19	10	1	1	2	2	2	–	Normal	Pseud onomas	–	–	–	Cavity RLL, Lung abscess	Pyogenic absecss	Antibiotics	Resolution
7	38	F	–	30	15	2	1	1	2	2	–	Elevated	–	–	–	–	Cavity RML with filling defect Rt MPA	PTE	Anticoag ulation	Resolution
8	73	M	HTN	60	15	1	1	2	2	2	–	Normal	–	MTB det	–	–	Cavity RUL	TB	ATT	Resolution
9	36	M	–	30	10	2	2	1	2	2	–	Elevated	–	–	–	–	Cavity LLL with filling defect lobar A	PTE	Anticoag ulation	Resolution
10	28	F	–	30	21	1	1	2	2	2	–	Normal	–	MTB det	–	–	Cavity RUL	TB	ATT	Resolution
11	52	M	DM	42	15	2	1	1	2	2	–	Normal	–	–	Positive	Raised	B/L Cavity	Fungal	Antifungal	Resolution
12	72	M	DM	30	15	2	2	1	2	2	–	Elevated	–	–	–	–	Cavity RUL with filling defect lobar A	PTE	Anticoag ulation	Resolution
13	54	F	DM	20	10	1	1	2	1	1	–	Normal	–	MTB det	–	–	Cavity RUL, Sup seg RLL	TB	ATT	Resolution
14	43	F	DM	21	10	1	1	1	1	2	–	Normal	–	MTB det	–	–	Cavity LUL	TB	ATT	Resolution
15	62	F	DM	20	7	1	1	2	2	2	MTB det	Normal	–	–	–	–	Cavity RUL	TB	ATT	Resolution
16	48	M	–	35	15	1	1	2	2	2	–	Normal	–	–	Positive	Raised	B/L Cavity	Fungal	Antifungal	Resolution
17	57	F	DM	42	15	2	1	1	2	2	–	Normal	–	MTB det	–	–	B/L Upper lobe Cavity	TB	ATT	Resolution
18	45	F	–	15	10	2	1	1	2	2	–	Elevated	–	–	–	–	Cavity RUL and filling defect MPA	PTE	Anticoag ulation	Deceased
19	48	F	Hypothyroidism	21	10	2	1	1	2	2	–	Normal	–	–	–	–	Cavity RLL	POST COVID	Symptomatic	Resolution
20	42	M	–	30	10	2	2	1	2	2	–	Normal	–	–	–	–	Cavity B/L LL	POST COVID	Symptomatic	Resolution
21	50	F	DM	40	21	1	1	2	2	2	–	Normal	–	–	Positive	Raised	B/L multiple cavity	Fungal	Antifungal	Deceased
22	66	M	HTN	17	7	1	1	2	2	2	–	Normal	–	MTB det	–	–	Cavity RUL	TB	ATT	Resolution
23	63	M	DM	30	14	1	1	2	2	2	–	Normal	Klebseilla	–	–	–	Cavity lingula	Pyogenic absecss	Antibiotics	Resolution
24	54	M	DM	30	15	2	1	1	2	2	–	Normal	–	–	Positive	Raised	Cavity RUL with halo sign	Fungal	Antifungal	Resolution
25	63	F	DM	30	15	1	1	2	2	2	–	Normal	–	MTB det	–	–	Cavity RUL	TB	ATT	Resolution
26	66	M	–	30	15	1	1	2	2	2	MTB det	Normal	–	–	–	–	B/L Upper lobe Cavity	TB	ATT	Resolution
27	60	F	HTN	25	10	2	1	1	2	1	–	Normal	–	MTB det	–	–	Cavity LUL	TB	ATT	Resolution
28	62	M	DM	45	15	1	1	1	2	1	–	Normal	–	–	Positive	Raised	cavity RUL	Fungal	Antifungal	Resolution

(*continued on next page*)

Table 1 – (continued)

Patient	Age	Sex	Comorbidities	Hospitalisation (no. of days)	Steroids (days)	Symptoms (Yes-1, No-2) Fever	Cough	Dyspnea	Hemoptysis	Weight loss	Sputum Gene Xpert	D Dimer	Broncho alveolar lavage Pyogenic C/S	Gene Xpert	Fungal PCR	Galacto	CT findings	Final diagnosis	Treatment	Outcome
29	42	M	–	21	7	2	1	1	2	2	–	Normal	–	–	–	–	Cavity RML	Post COVID	Symptomatic	Resolution
30	45	M	–	25	7	2	1	2	1	1	MTB det	Normal	–	–	–	–	Cavity RUL	TB	ATT	Resolution
31	36	M	–	35	15	2	1	1	2	2	–	Normal	–	–	Positive	Raised	B/L multiple Cavity	Fungal	Antifungal	Deceased

Patient	Age	Sex	Comorbidities	Hospitalisation (no. of days)	Steroids (days)	Symptoms (Yes-1, No-2) Fever	Cough	Dyspnea	Hemoptysis	Weight loss	Sputum Gene Xpert	D Dimer	Broncho alveolar lavage Pyogenic C/S	Gene Xpert	Fungal PCR	Galacto	CT findings	Final diagnosis	Treatment	Outcome
32	47	M	RA	35	10	2	1	1	2	2	–	Normal	–	–	–	–	Cavity RML	Post COVID	Symptomatic	Resolution
33	56	M	DM	25	10	2	1	2	2	2	–	Normal	–	–	–	–	Cavity RUL	Post COVID	Symptomatic	Resolution
34	35	M	–	20	7	1	1	2	1	1	–	Normal	–	MTB det	–	–	Cavity LUL	TB	ATT	Resolution
35	41	F	–	15	7	1	1	2	2	2	–	Normal	Pseud omonas	–	–	–	Cavity RLL	Pyogenic absecss	Antibiotics	Resolution
36	50	F	DM	20	7	1	1	2	1	2	MTB Det	Normal	–	–	–	–	Cavity RUL	TB	ATT	Resolution
37	57	F	HTN	21	10	2	1	1	2	2	–	Normal	–	–	–	–	Cavity LLL	Post COVID	Symptomatic	Resolution

(M – male, F – female, DM – diabetes mellitus, HTN – hypertension, RHD – rheumatic heart disease, RTR – renal transplant recipient, MTB – *Mycobacterium tuberculosis*, det – detected, micro – microscopy, C/S – culture, PCR –polymerase chain reaction, galacto – galactomannan assay, RUL – right upper lobe, RML – right middle lobe, RLL – right lower lobe, LUL – left upper lobe, LLL – left lower lobe, B/L – bilateral, PTE – pulmonary/thromboembolism, TB – tuberculosis, ATT – antitubercular therapy).

post-COVID sequelae, 37 patients had cavities on the CT chest. 81% (n = 30) patients had single unilateral cavity and 19% (n = 7) had bilateral cavities. 38% (n = 14) patients had tuberculosis (TB), which was detected on sputum microscopy (n = 5) or BAL (n = 9). 24% (n = 9) patients had cavity secondary to fungal pneumonia diagnosed on the basis of BAL fungal PCR and galactomannan assay. 8% (n = 03) patients had a cavity secondary to pyogenic infection, and 14% (n = 05) had an underlying pulmonary embolism (PE). Findings of BAL are depicted in Fig. 2. In 16% (n = 06) patients, no cause could be ascertained and the cavity was attributed to COVID infection per se. The etiological profile of cavities in post-COVID patients is depicted in Fig. 3. After confirmations of etiology, patients were administered antitubercular therapy for TB, antifungal therapy for fungal pneumonia, and anticoagulation for PE. Patients in whom no etiological agent was identified were managed symptomatically. All patients were followed up monthly until 6 months or recovery. Repeat imaging (CT scan) was performed after 3 months and 6 months. Two patients who were suffering from fungal pneumonia were readmitted and subsequently passed away. While one patient developed pneumomediastinum and pneumothorax, the other had disseminated mucormycosis and sepsis. Thirty-five patients showed clinical and radiological resolution. Representative images are depicted in Figs. (4–7).

Discussion

Post-COVID complications like secondary infections, pulmonary function test disorders, pulmonary thromboembolism, pulmonary hypertension, and lung fibrosis have all been studied in various studies, although mostly in retrospective analysis. It has been observed that 10–35% of patients who do not require hospitalization and regardless of comorbidities develop post-COVID symptoms, and in hospitalized patients, it has been reported as high as in 80%.[1] Garg et al in their pictorial review of CT findings in sequelae of COVID-19 and its complications elucidated the wide spectrum of abnormalities seen secondary to fibrosis, bacterial infection, fungal infection, mycobacterial infections, and PE.[5]

The causes of cavitation in COVID-19 patients are multifactorial, with varying aspects like infection i.e. bacterial and fungal infection, pulmonary TB possibly secondary to the immunosuppressive effects of glucocorticoids, and tocilizumab (interleukin-6 inhibitor). Many other factors like SARS-CoV-2 specific inflammatory pathways, and COVID-19-related predisposition to venous thromboembolism subsequently leading to infarct and micro-infarcts have been implicated in COVID-19 pulmonary cavitation.[4]

It is uncommon for viral pneumonia (including SARS-CoV and MERS-CoV) to cause pulmonary cavitation even in advanced and severe viral infections.[2] However, cavitary lesions have been published in case series and case reports. Zoumot et al[2] found cavities in 1.7% of a cohort of 689 COVID-19 cases. In their retrospective review of cavities seen in 12 cases, all patients were on invasive ventilation, and DM was the most common comorbidity among them, six patients had died, and eight were treated for fungal infection.[2] Among our cohort of 37 patients, only 1 patient was on the ventilator and

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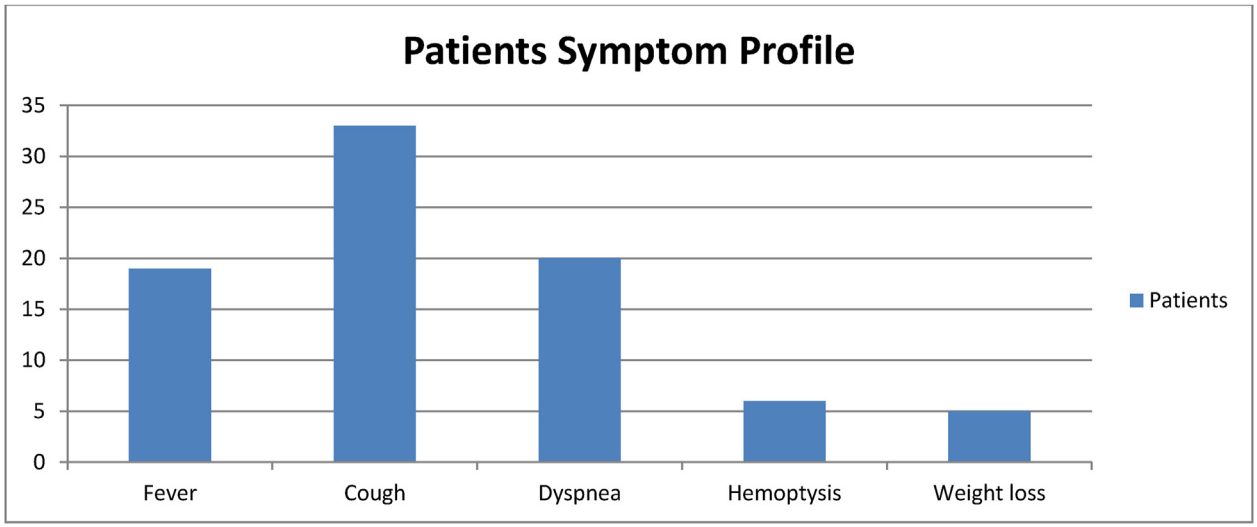

Fig. 1 – Symptoms of patients.

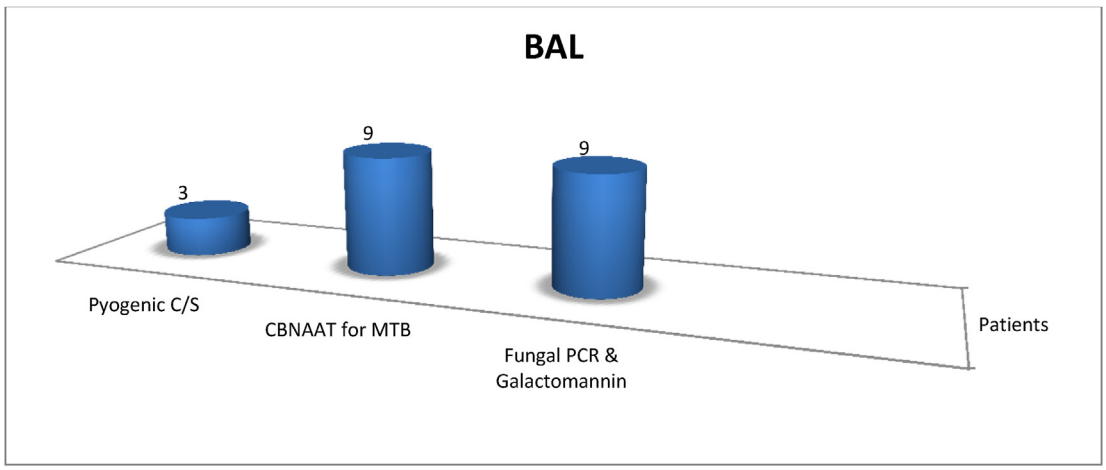

Fig. 2 – Bronchoalveolar lavage Lresults.

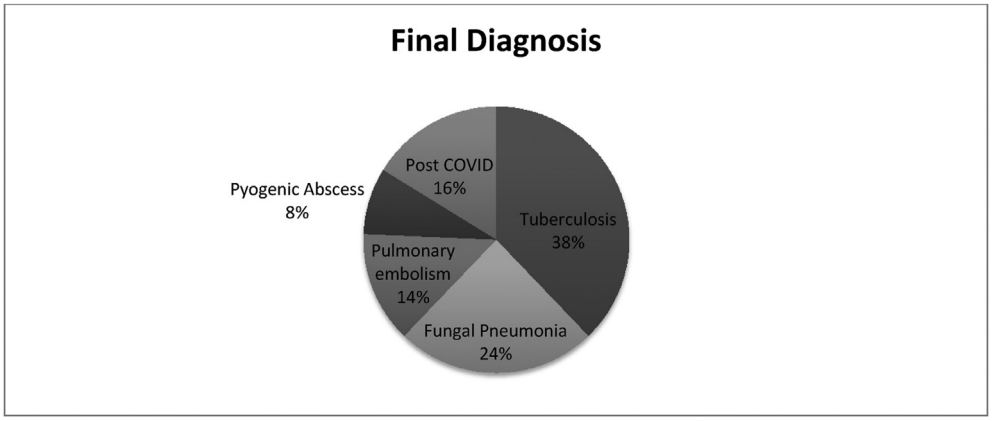

Fig. 3 – Final etiological diagnosis.

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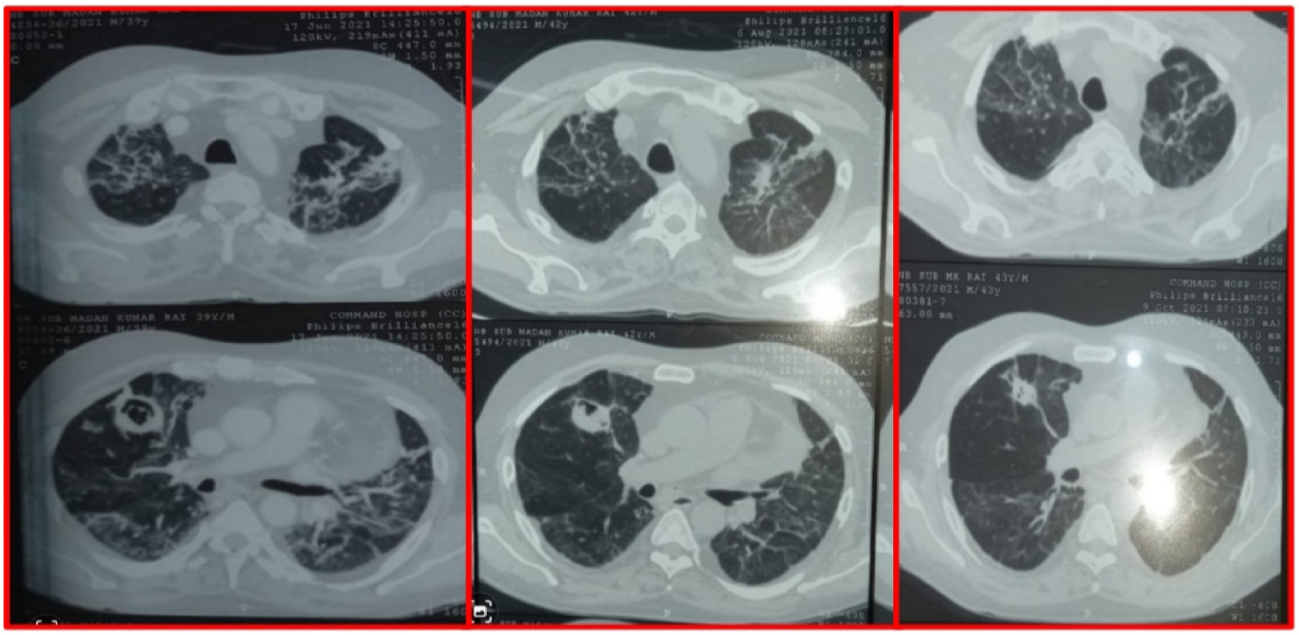

Fig. 4 – Patient no 1, a case of renal transplant recipient with post-COVID fungal pneumonia. (a) CT chest showing cavity RML. (b) Follow-up CT after 6 weeks. (c) CT after 6 months showing significant resolution of disease.

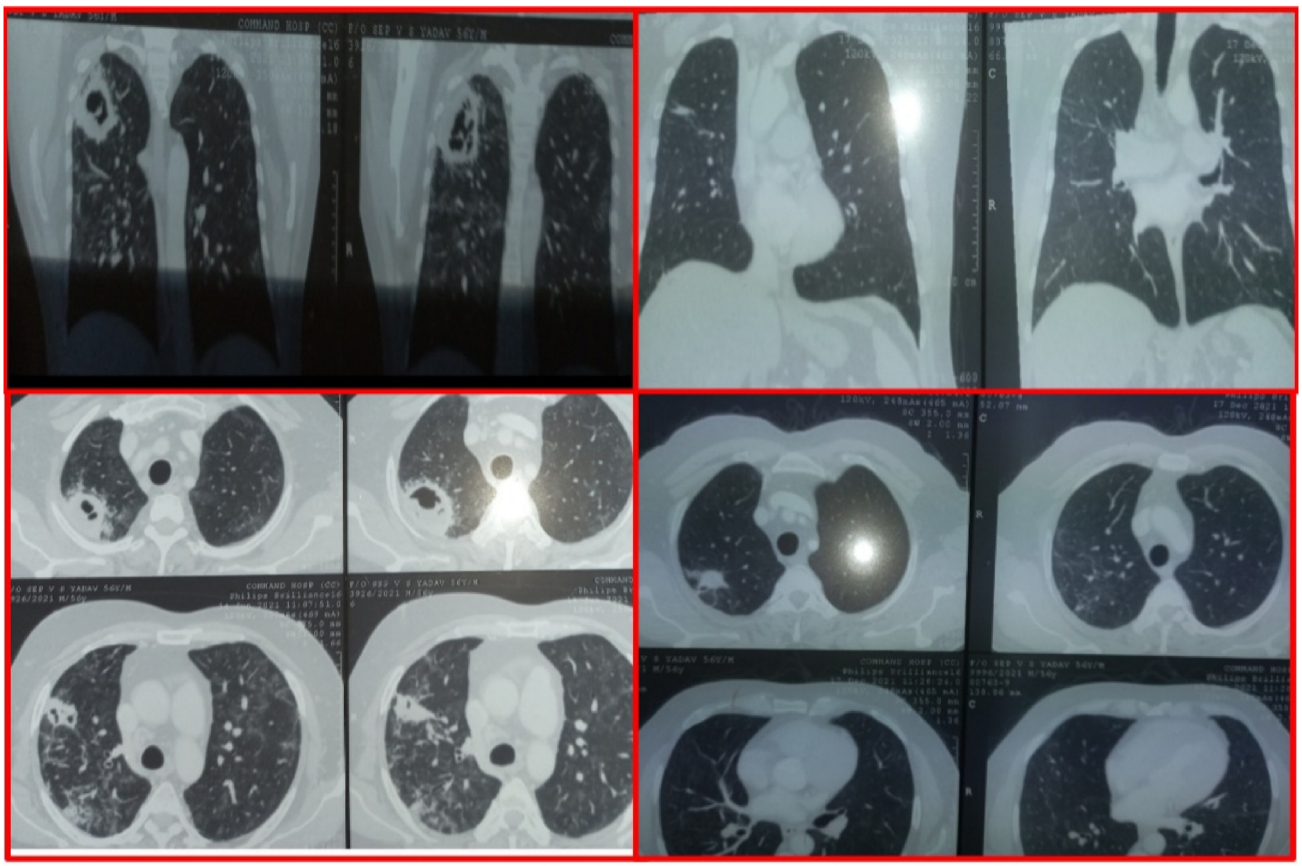

Fig. 5 – Patient no 28, known case of diabetes with post-COVID fungal pneumonia. (a) CT chest showing cavity RUL. (b) Follow-up CT after 3 months showing significant resolution of disease.

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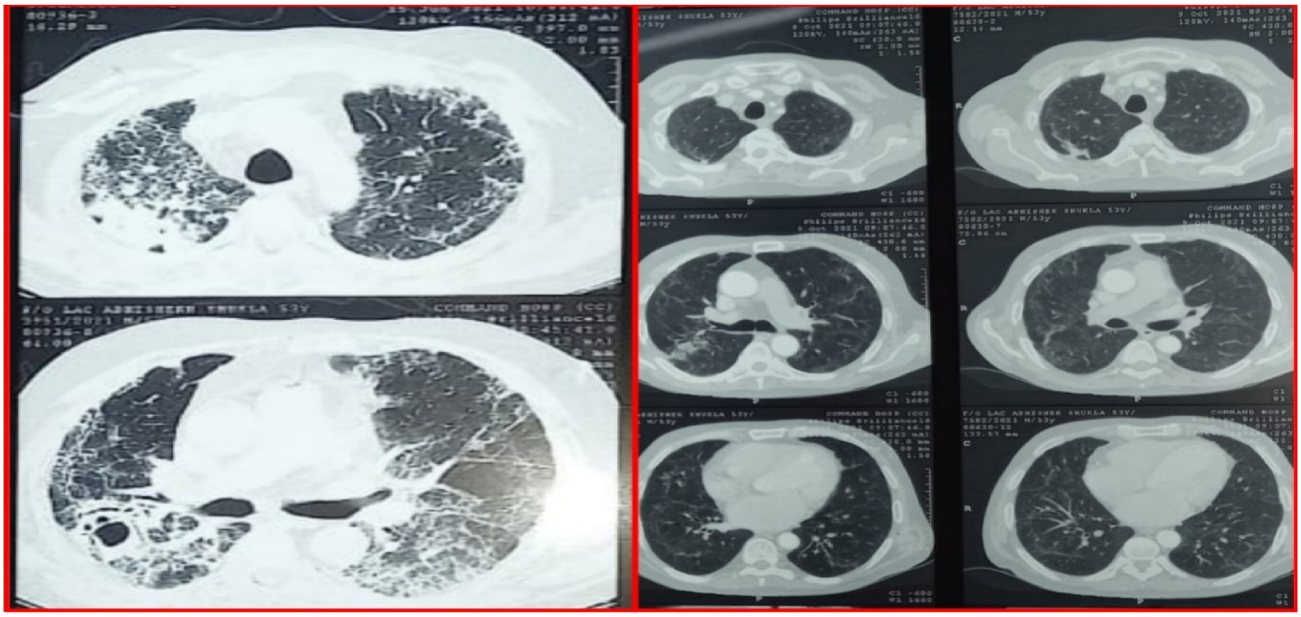

Fig. 6 – Patient no 2, known case of rheumatic heart disease with post-COVID fungal pneumonia. (a) CT chest showing cavity RUL. (b) Follow-up CT after 3 months showing significant resolution of disease.

similarly, DM was also the most common co-morbidity observed. The commonest etiology in their cases was fungal, and they found that four patients had a bacterial infection. However, none had TB.[2] In contrast, our study showed MTB as the most common etiology, probably also related to the endemicity of TB in this region. In a retrospective case series, MTB was found as a common cause of lung cavitation in COVID cases.[6]

Although no standard diagnosis or treatment algorithm has been described in the management of cavities due to COVID-19, the authors have emphasized the need for detailed evaluation and investigations (including laboratory and microbiological examination) with a focus on fungal infections, especially aspergillosis and TB.[1]

Aggarwal et al. elucidated a pictorial review of COVID-19 pneumonia with cavitation and cystic lung changes on CT

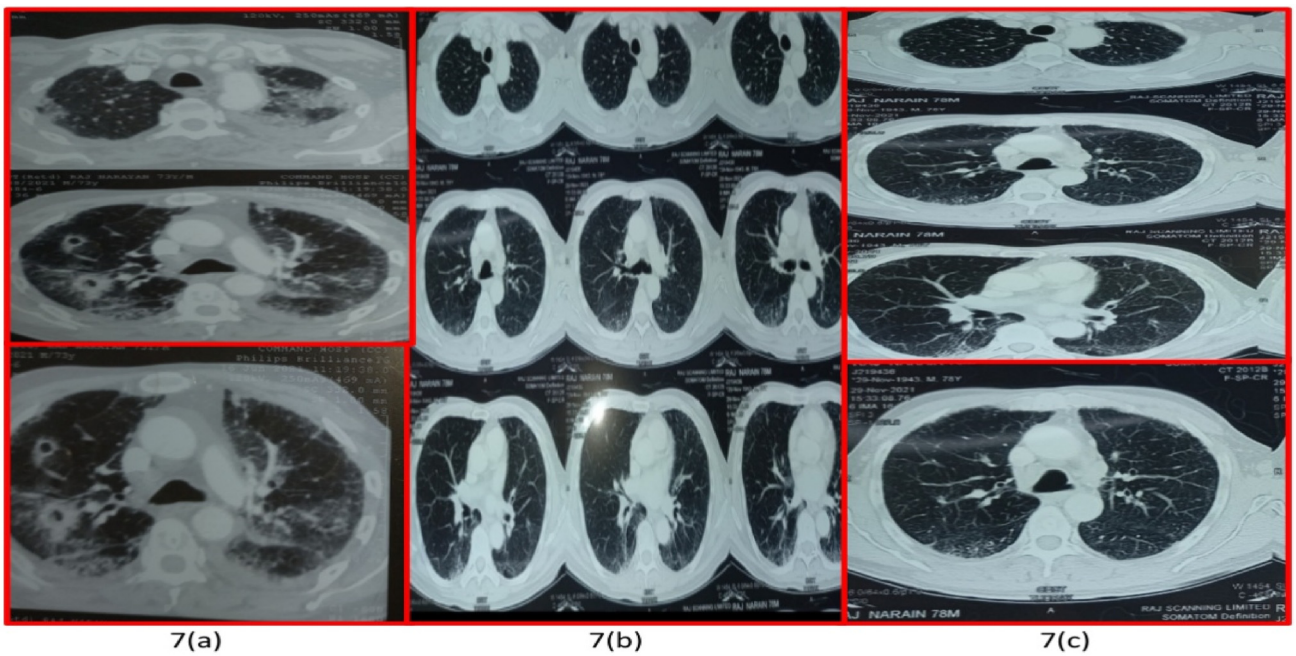

Fig. 7 – Patient no 15, known case of diabetes with post-COVID tuberculosis. (a) CT chest showing cavity RUL. (b) Follow-up CT after 3 months and (c) CT after 6 months showing significant resolution of disease.

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encompassing a gamut of etiologies. They observed that cavitation could be due to COVID infection per se or manifestation of other ominous etiologies like coinfection with bacteria, fungus, mycobacteria, or incidental malignancy/metastasis.[7]

Cavitation primarily due to COVID-19 has been seen in the absorption stage of disease, typically after 2 weeks. Although the precise mechanism is unknown, a histopathological finding seen in autopsy reports has been of diffuse alveolar damage, intra alveolar hemorrhage, and necrosis of parenchymal cells.[8] Cavities due to COVID-19 infection are seen in the areas of previously existing ground-glass opacities or consolidation. Authors also highlighted that other etiologies of cavities like superadded bacterial, fungal, or mycobacterial infection and cavitatory infarct should be excluded by detailed workup before labelling the COVID-19 infection per se as the etiology of the cavity.[7]

Pulmonary TB is one of the most common infective lung diseases in developing countries.[9] The concurrence of COVID-19 and pulmonary TB leads to more severe consequences.[10] This syndemic of COVID-19 and TB is a challenge for developing nations as it may lead to severe mortality and morbidity. Furthermore, it has been observed that patients with pre-existing lung disease secondary to pulmonary TB show a poor outcome when inflicted with COVID-19 pneumonia.[11] Such patients with the concurrence of COVID-19 and pulmonary TB can pose a diagnostic challenge for the treating physician.[12] Correct and apt diagnosis of these two co-existing diseases is of paramount importance to reduce the affliction of residual fibrosis in patients.[9] Sputum/BAL smear microscopy for acid-fast bacilli is the easiest and one of the most essential investigations for the detection of pulmonary TB.[13] Further molecular tests like CBNAATs may be used to aid diagnosis and identification. Cavitation secondary to pulmonary TB in endemic countries like India warrants prompt testing.

COVID-19 patients are predisposed to coinfection with invasive pulmonary aspergillosis.[14] Severe COVID-19 alters both T-helper cell 2 (Th2) and Th1 responses leading to immune dysregulation,[15] although a direct immunomodulatory effect of COVID-19 on the antifungal host defence has not been proven conclusively. Unlike influenza infection, which has been identified as an independent risk factor for developing invasive pulmonary aspergillosis,[16] invasive pulmonary aspergillosis infection in COVID-19 patients is associated more with concomitant corticosteroid therapy. Fungal coinfection with COVID-19 has been observed in 20–35% of severely ill patients, possibly secondary to immune dysregulation and increasing use of steroids.[7]

Clinical and radiological signs of invasive pulmonary aspergillosis in non-neutropenic patients are generally nonspecific. Therefore, bronchoscopy is indicated in critically ill COVID-19 patients who are suspected to have a secondary infection, including fungal infection. Galactomannan testing of BAL is important for the diagnosis of invasive pulmonary aspergillosis. High galactomannan levels (galactomannan index >2.5) were seen in patients with suspected COVID-19-associated invasive pulmonary aspergillosis.[14]

Type 2 diabetes, immunosuppression, and corticosteroids are major risk factors for COVID-19 associated mucormycosis (CAM).[17] Molecular tests (PCR) for the detection of circulating Mucorales DNA are available for clinical use. But standardization of the mucor PCR method is ongoing.[18] This direct detection method with a short turnaround time can help in early diagnosis and timely treatment to arrest the rapid growth of mucor and progression of the infection. The key to improving outcomes in mucormycosis is surgical treatment with radical removal of infected tissues. Besides surgery, the timely institution of antifungal therapy is essential.

The incidence of PE in hospitalized patients with COVID-19 has been reported to be ranging from 1.9% to 8.9%.[19,20] The real incidence of PE may has been underestimated due to the retrospective nature of the studies and a relatively short period of follow-up. In one study, when the follow-up was increased from 1 to 2 weeks, the incidence of PE increased to 33.3%.[21]

The cause for the high frequency of PE in hospitalized COVID-19 patients is multifactorial. Firstly, the pathologic consequences of COVID-19, such as severe hypoxia, associated organ dysfunction, and pre-existing comorbidities, can predispose to hemostatic abnormalities. Hypoxia predisposes to thrombosis by increasing blood viscosity and through a signaling pathway dependent on a hypoxia-inducible transcription factor.[22,23] Individual patient-related risk factors and ICU-specific risk factors may also increase this risk, such as sedation, immobilization, use of central venous catheters, and vasopressor administration. Secondly, pro-inflammatory and procoagulant effects may be induced through complement activation and cytokine release due to endothelial dysfunction.[24,25] The procoagulant state of COVID-19 may continue for some weeks following the discharge of patients. Clinicians should have a high degree of suspicion of PE in COVID-19 patients readmitted to the hospital after discharge.[26]

Necrotizing pneumonia (NP) is a rare and severe complication of community-acquired pneumonia (CAP).[27,28] As NP progresses across its spectrum, the pulmonary vascular and bronchial supply is compromised, leading to devitalization of lung parenchyma.[29] Due to the copious administration of steroids in hospitalized COVID-19 patients, their immune status becomes further compromised. Bacterial coinfection with COVID has been reported in 7% of hospitalized patients, and 14% of patients admitted to intensive care units.[7]

Staphylococcus aureus and *Streptococcus pneumonia* have been described as the most common etiologic agents in NP[28]; however, *Pseudomonas aeruginosa* and *Klebsiella pneumonia* are more commonly associated if pulmonary gangrene is present.[30]

In our cohort, two cases were associated with *P. aeruginosa*, and one case showed growth of Klebsiella pneumonia. NP is a common, probably overlooked, complication in COVID-19 patients, which may be directly linked to increased mortality in the late phases of the disease. However, early diagnosis and prompt treatment with appropriate culture-sensitive antibiotics for an adequate duration can certainly improve patient outcomes.

Limitations of the study: Baseline CT images were not available for all patients as the decision to obtain baseline CT was clinically guided and not routinely performed. Chest radiographs were used to determine the radiographic changes at admission.

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Conclusion

Pulmonary cavitation in follow-up COVID patients is not a rare entity and has multifactorial etiology. These cavities could be a result of immune dysregulation as well as immunosuppression used for treatment in COVID patients. These cavities could be early indicators of other ominous etiologies or reflect the end stage of the disease itself. In our study also, variable etiologies for cavities like TB and fungal were seen in follow-up patients. Awareness of these etiologies can aid in timely evaluation and institution of correct therapy leading to improved outcomes.

Ethics statement

This is to certify that inform consent was taken to collect data. No new trial or interventions was carried out. The present manuscript was in-vitro examinations from records of patients coming for regular follow up, hence IEC certification was not accorded.

Disclosure of competing interest

The authors have none to declare.

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