Bronchiectasis and COVID-19 infection: a two-way street

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Abstract

Bronchiectasis (BE) has been linked to past viral infections such as influenza, measles, or adenovirus. Two years ago, a new pandemic viral infection severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) broke out and it still persists today, and a significant proportion of surviving patients have radiological and clinical sequelae, including BE. Our aim was to thoroughly review the information available in the literature on the bidirectional relationship between SARS-CoV-2 infection and the development of BE, as well as the impact of this infection on patients already suffering from BE. Available information indicates that only a small percentage of patients in the acute phase of coronavirus disease 2019 (COVID-19) pneumonia develop BE, although the latter is recognized as one of the radiological sequelae of COVID-19 pneumonia, especially when it is caused by traction. The severity of the initial pneumonia is the main risk factor for the development of future BE, but during the COVID-19 pandemic, exacerbations in BE patients were reduced by approximately 50%. Finally, the impact of BE on the prognosis of patients with COVID-19 pneumonia is not yet known.

Keywords: Bronchiectasis; Coronavirus; COVID-19; SARS-CoV-2

Introduction

It has been more than two years since the onset of the coronavirus disease 2019 (COVID-19), the illness caused by severe acute respiratory syndrome coronavirus 2 (SARS-CoV-2) and one of the most critical global health emergencies.[1] Each successive variant of the SARS-CoV-2 virus to date has been more transmissible than the previous one.[2] The new variants have led to multiple waves of COVID-19 around the world, each of them with different characteristics as regards the variant's virulence and power of transmission, the country's vaccination rate, the clinical severity, and the characteristics of affected individuals (with the first waves bringing higher mortality rates).[3-9]

At the level of the respiratory tract, COVID-19 infection presents a wide variety of complications ranging from self-limiting upper respiratory tract infection to acute respiratory failure due to diffuse bilateral pulmonary infiltrates.[10] While most people recover from COVID-19-associated pneumonia, in some individuals the lung damage persists even after the disease has receded inducing persistent symptoms and lung function abnormalities that may take months to improve.[11-15] This delayed recovery of symptoms has been termed "post-COVID-19 syndrome" or "long COVID."[16-18]

Thoracic imaging of pulmonary sequelae of COVID-19 has revealed fibrotic changes including parenchymal bands, irregular interfaces, and reticular opacities, with or without honeycomb-like changes.[19] However, several follow-up studies carried out during these two pandemic years have shown that the appearance of long-term post-infectious traction bronchiectasis (BE) in previously disease-free patients can also be a significant complication of SARS-CoV-2 infection.[20-22]

Many patients, however, already had BE before the start of the COVID-19 pandemic.[23] In such cases — as well as in those with previous other chronic respiratory diseases, especially chronic obstructive pulmonary disease (COPD) or asthma, the COVID-19 pandemic situation has presented further challenges and opportunities.[23-31] Severe COVID-19 seems to be more frequent in patients

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with BE than in those without it.[32] Conversely, it was also noted that social distancing measures during the first 12 months of the COVID-19 pandemic were associated with a marked reduction in BE exacerbations, although there were no changes in individual chronic respiratory symptoms.[33]

This bidirectional relationship between COVID-19 infection and BE suggested a need to review the existing literature on the reciprocal influences between these two pathological conditions and then describe them in narrative form.

We must duly emphasize that our analysis of the relationship between COVID-19 infection and the subsequent development of BE refers to the abnormal bronchial dilations observed in chest images that usually do not produce symptoms (a parameter that the latest international recommendations for the diagnosis of BE require for its correct syndromic diagnosis).[34] However, we still do not know whether these bronchial dilations will produce correlated symptoms in the future, as is the case in post-infectious BE secondary to viral infections (e.g., influenza, measles virus, or adenoviruses). Indeed, the time passed since the formation of BE is still too short to be able to confirm this hypothesis, but the impact of SARS-CoV-2 infection in individuals already suffering from BE is based on a solid clinical-radiological diagnosis supporting the presence of BE.[34]

BE and the Acute Phase of COVID-19 Pneumonia

The percentage of patients with BE observed in the early phase of pneumonia is highly variable, according to the studies undertaken (6.3–52.0%).[35-42] Bao *et al*[20] performed a systematic review with meta-analysis to provide a more accurate estimate of the detection of COVID-19 by chest computed tomography (CT) and reported the most common findings in imaged chest CT. The results showed that although ground-glass opacity was, as expected, the most common CT feature to be detected, in 83.3% of 2738 cases, BE was observed in only 5.4% of cases. However, the authors acknowledged that there were only two studies that had reported the presence of BE. Li *et al*[21] noted, in a systematic review of 84 articles covering 5340 patients with COVID-19, that information on BE was only offered in 12 of them. In these studies, BE was associated with ground-glass opacities in 32.4% of cases. Five studies focused on adult patients, and the prevalence of BE was 7.4% in these patients. No studies specifically examined the prevalence of BE in children with COVID-19.

Two examples of patients with BE that developed in the acute phase of the disease have been reported. In the first, the enlargement of the bronchi and evolution into BE occurred after 5 days of hospitalization,[43] whereas the second shows a rapid progression from pneumonitis to cystic BE.[44] The frequency of BE in the acute phase of SARS-CoV-2 pneumonia depends on several factors, such as the severity and distribution of the pneumonic process, the need for admission to an intensive care unit, and the days elapsed between the onset of symptoms and the CT scan.[36,37,42]

The probability of BE occurrence becomes greater as the number of days increases. Ding *et al*[36] examined 112 patients with COVID-19 and observed that the prevalence of BE increased as the time between the onset of symptoms and the CT scan increased, from a prevalence of 6.3% when the CT scan was performed 0 to 4 days after the onset of symptoms to 45.2% when the period between the onset of initial symptoms and the CT scan was >28 days. However, when CT examination was conducted in the early stages of COVID-19 (range: 0–7 days), the BE rate was statistically significantly higher in the most severe patients (85.7% *vs.* 47.1%; $P = 0.007$).[42] Furthermore, a study involving 85 patients with COVID-19 pneumonia demonstrated not only that lesions adjacent to the pleura were associated with a high rate of respiratory failure, but also that the occurrence of traction BE was much more frequent in the presence of lesions where the subpleural areas were not spared than in those that did spare these areas (57% *vs.* 8%, $P < 0.001$).[37]

There is also documentation of the presence of BEs that should not be considered traction-related. In a study by Devie *et al*[35] involving 158 patients, it was observed that 25.3% had BE unrelated to the pneumonic condensation process, whereas in 9.5% BE were related to traction processes. Both forms were significantly more frequent in more severe cases than in non-severe cases (41.4% *vs.* 16.0%, $P < 0.001$ for non-traction BE; and 17.2% *vs.* 5.0%, $P = 0.011$ for traction BE, respectively). These data are consistent with those of a small Chinese study which included 53 patients with confirmed COVID-19 pneumonia, of whom 12 (22.6%) showed the presence of BE with lower lobe distribution as a prominent feature, but BE was considered traction related in only 3 cases.[41] These findings suggest that most patients with COVID-19 pneumonia had BE at the time of presentation, but neither study specified any exclusion of patients with BE before infection. Consequently, the pre-existence of BE cannot be ruled out in some of these cases.

Only one study specifically focused on the relationship between acute COVID-19 infection and the presence of BE.[39] This was a retrospective study of 115 patients convalescing after COVID-19 pneumonia that excluded subjects with COPD, asthma and BE before infection. The prevalence of BE was 28.7% (82 patients had no BE and 33 had BE), mainly in the lung bases and located in the inflamed parenchymal areas. Risk factors for its occurrence were: female gender ($P = 0.008$), more advanced age ($P < 0.005$), and a greater degree of inflammatory lesions in the CT scan ($P = 0.006$).

The prevalence of BE in the early phase of pneumonia is highly variable and depends on several factors, such as the severity and distribution of the pneumonic process, age and gender as well as the number of days elapsed between the onset of symptoms and the CT scan. It is not known whether some of these patients had BE previously.

BE as a Pulmonary Sequela of COVID-19 Pneumonia

A substantial number of previously hospitalized survivors of SARS-CoV-2 infection will have abnormalities in a CT scan over time, especially those with more severe acute infection with parenchymal or subpleural bands, reticular abnormalities ("honeycombing"), pleural thickening, and BE being the most common.[45]

As regards BE specifically, several studies have provided information on its appearance at one month and more than a year after the acute phase of infection.[45-51] BE is usually associated with an established residual fibrotic pattern and therefore caused by traction in most cases, increases over time, and its incidence is higher in patients with more severe initial pneumonia.[34,43,45,46]

Of special interest is the study carried out by Ding *et al*[36] in 68 patients with serial CT (up to six scans) from the acute to the follow-up phases, in which the prevalence of BE increased from 6.3% in the CT performed in the first 4 days after the onset of symptoms to 45.2% in the CT performed more than one month after the acute process.

Other studies have analyzed the prevalence of BE 3–6 months after the acute process.[45,46,48-51] The reported percentages fluctuate, in most studies, between 15% and 30%, remaining fairly stable from month 3 onward. Such stability over time suggests that this BE might be an irreversible marker of fibrosis, but the irreversibility of BE has been questioned by some authors. Hu *et al*[52] observed in a retrospective study the presence of BE traction in 21 of 41 COVID-19 survivors with acute respiratory distress syndrome. BE did not completely disappear in only seven of these patients after more than four months of follow-up, but even then it was significantly alleviated. Also, in a study by Caruso *et al*[53] that evaluated lung damage in patients with COVID-19 pneumonia at a 6-month follow-up CT examination compared with CT chest examination at baseline, the prevalence of BE decreased in the former, confirming the regression of traction BE, which, accordingly, should not be understood as a trustworthy irreversible marker of fibrosis in a setting subsequent to lung injury.

This BE should be referred to as pseudobronchiectasis, which is the term used to describe bronchial dilatations that frequently develop after an infection because of the intense inflammatory process involved but resolve after the acute infection has healed.[54] In this regard, it is worth pointing out that when the radiological findings in the acute infection phase of patients with COVID-19 pneumonia were compared with those of patients with influenza A (H1N1) pneumonia, BE was more frequently observed in H1N1 infections (30.0% *vs.* 3.3%, $P = 0.012$).[55] In any case, the patients in a study by Hu *et al*[52] with incomplete disappearance of traction BE were more likely to exhibit reticulation on the last CT images than those with complete disappearance of traction BE.

In a systematic review of 45 studies including 4410 patients with COVID-19 pneumonia subjected to CT studies, BE was observed in 18% of patients.[56] Unfortunately, it is not specified whether these CT scans were performed in the acute phase of the disease or in the follow-up, but it is reported that 14.5% of patients presented with bronchial wall thickening, an indirect marker of active airway inflammation.

In almost all studies, the most severe forms of COVID-19 were those with a higher prevalence of BE, which reached even 50% of cases.[34,46,51] Conversely, in the study by Vijayakumar *et al*,[48] a review of chest CT abnormalities present after up to one year of follow-up in 80 COVID-19 survivors showed a stable prevalence of traction BE from 3 months to 1 year of follow-up in only 6.8% of cases. In this study, the severity of patients is not specified, so the low prevalence of BE could be a consequence of non-severe COVID-19.

Furthermore, the presence of traction BE at discharge (odds ratio [OR] = 13.6, $P = 0.012$) was an independent risk factor for the development of pulmonary fibrosis after seven months, even more important than other risk factors such as age (OR = 1.078, $P = 0.049$), steroid therapy (OR = 12.9, $P = 0.01$), and discharge opacity score (OR = 1.565, $P = 0.034$).[51] In an interesting study, Sugino *et al*[57] correlated CT images of post-COVID-19 pulmonary sequelae with histopathological characteristics of specimens from two patients undergoing video-assisted thoracoscopic surgery. BE can be seen in both CT images and biopsy.

Between 15% and 30% of patients will have BE after few months of the acute infection [Table 1]. The vast majority of these BE are associated with a residual fibrotic pattern. However, it is not uncommon for the presence of pseudobronchiectasis which disappears after the acute infection.

Risk Factors for BE after COVID-19 Pneumonia

Several studies have analyzed the risk factors associated with the occurrence of BE during the follow-up in recovered COVID-19 patients. Among these risk factors, the initial severity of pneumonia is undoubtedly the most crucial.[34,45,46,50,51] A meta-analysis focusing on CT characteristics of patients with COVID-19 pneumonia analyzed 15 articles that collectively reported data from 1453 patients with mild pneumonia and 697 with severe pneumonia.[58] Traction BE appeared in 31% (12–55%) of patients with non-severe pneumonia and 52% (30–73%) of those with severe pneumonia. Therefore, based on the CT images, patients with non-severe pneumonia exhibited traction BE less frequently (OR = 0.40; $P = 0.002$) when compared with severe patients. On the other hand, the prevalence of bronchial wall thickening was 13% (4–26%) in non-severe patients and 47% (19–77%) in those with severe pneumonia, but this feature did not exhibit any significant association with the severity of disease (OR = 0.15; $P = 0.064$). This finding could be of great importance, since bronchial wall thickening is recognized as a factor of active inflammation of the bronchial wall,[59] which in turn could be related to a future formation of clinically active BE, or to its evolution due to the disruption of local defense mechanisms.[60]

Table 1: Main studies with information on BE as a sequela from COVID-19 pneumonia.

Author	*n*	Design	Serial CT	Number (%) of BE	Pneumonia severity
Han *et al*[45]	114	Prospective	At 17 ± 11 days	8/114 (7.0%)	Severe
			At 175 ± 20 days	27/114 (23.7%)	
Liu *et al*[46]	52	Prospective	At admission	0	32 (61.5%) moderate
			At discharge	16 (31%); moderate 19%, severe 50%	20 (38.5%) severe
			At 1 month	16 (31%); moderate 19%, severe 50%	
			At 3 months	14 (27%); moderate 13%, severe 50%	
			At 6 months	13 (25%); moderate 13%; severe 45%	
Mumoli *et al*[47]	88	Retrospective	At 3 months	13 (15%)	Hospitalized patients
Vijayakumar *et al*[48]	80	Prospective	At 3 months	5/41 (12.2%)	Hospitalized patients
			At 12 months	5/41 (12.2%)	
Zhou *et al*[49]	120	Prospective	At 314.5 (296–338) days	14.4% (moderate 15.7%; severe 7.1%)	16 (13.3%) severe
Ding *et al*[36]	112	Prospective	At 0–4 days	3/47 (6.4%)	Hospitalized patients
			At 5–9 days	7/54 (13.0%)	
			At 10–14 days	13/67 (19.4%)	
			At 15–21 days	23/68 (33.8%)	
			At 22–28 days	19/59 (32.2%)	
			>28 days	24/53 (45.3%)	
Dai *et al*[50]	50	Prospective	Discharge	5/48 (10.4%)	Hospitalized patients
			At 6 months	3/45 (6.7%)	
Liu *et al*[51]	41	Prospective	At discharge	12 (29%), 7% NFG and 83% FG	Hospitalized patients
			At 3 months	14 (34%), 7% NFG and 100% FG	
			At 7 months	12 (29%), 0 NFG and 100% FG	

BE: Bronchiectasis; COVID-19: Coronavirus disease 2019; CT: Computed tomography; FG: Residual fibrosis group; NFG: Non-residual fibrosis group.

There are also other possible risk factors or variables associated with the occurrence of BE. Han *et al*[45] observed in a prospective study of 114 patients with severe COVID-19 pneumonia that subjects who developed fibrotic changes in the months following the event were those with a significantly higher prevalence of BE, as compared to those patients who did not develop these changes (23.0% *vs.* 4.1%; $P = 0.004$). Persistence of cough 3 months after the acute episode is another clinical risk factor. Mumoli *et al*[47] reported a very strong correlation ($r = 0.57$; $P = 0.016$) between the persistence of cough after 3 months and persisting consolidation in a follow-up chest CT scan in 88 patients with COVID-19 pneumonia.

The main factor for the development of BE after the COVID-19 pneumonia is the severity of the infection process and the extension of the ground-glass and fibrotic CT lesions. However, a percentage of patients present BE with bronchial wall thickening as a marker of persistent bronchial inflammation. This finding is of great importance in the development of future clinically active BE in post-COVID patients.

Impact of BE on the Severity of COVID-19 Infection

Data on the possible relationships between various chronic respiratory diseases of the airways, especially COPD and asthma, and their impact on the severity of SARS-CoV-2 infection are extremely contradictory,[23,61-63] with some authors reporting that pre-existing COPD and asthma are risk factors for greater disease severity,[64,65] whereas others have found no relationship between these disorders and the severity of COVID-19 infection.[66,67] Some studies have even found that COPD or asthma protects these patients from infection, perhaps because of a reduced respiratory exposure, or because of the effect of their anti-inflammatory medication, particularly inhaled corticosteroids.[68-70]

As regards BE, the results are similarly contradictory. On the one hand, there is evidence of a possibility that COVID-19 is more frequent in patients with BE than in those without it. A Korean study examining 8070 patients with a confirmed diagnosis of COVID-19 and 121,050 controls not infected with COVID-19 observed that the risk of presenting with underlying BE was significantly higher in the COVID-19 cohort than in the controls (OR = 1.22; 95% CI = 1.01–1.45), and that the proportion of patients with severe COVID-19 was significantly higher in patients with BE than in those without BE (30.3% *vs.* 13.1%, $P < 0.001$).[32] Moreover, these patients required significantly more oxygen and mechanical ventilation, and they presented significantly higher mortality.

On the other hand, a Chinese nationwide retrospective cohort study of 39,420 laboratory-confirmed patients concluded that greater severity of COVID-19 infection defined with a composite endpoint (need for invasive ventilation, admission to intensive care unit or death within 30 days after hospitalization) was associated with previous COPD (OR = 1.7; 95% CI = 1.44–2.03) or asthma (OR = 1.45; 95% CI = 1.05–1.98), but not with BE (OR = 0.91; 95% CI = 0.67–1.23).[23] BE frequently overlapped with COPD (50.7%) and asthma (15.9%), but apparently the presence of an overlap with both COPD (OR = 0.87; 95% CI = 0.37–2.01; $P = 0.738$) and asthma (OR = 0.93; 95% CI = 0.20–4.38, $P = 0.923$) did not seem to induce a greater risk of reaching the composite endpoint. Paradoxically, when mortality within 30 days after hospitalization was analyzed separately, the presence of BE seemed to be a protective factor (OR = 0.38; 95% CI = 0.21–0.70), an effect that was lacking when BE

overlapped with COPD or asthma. As the authors themselves acknowledge, it is possible that the non-inclusion of some important variables in the study, such as pulmonary function, previous hospitalizations, and prescribed medication, may have influenced these results.

The information about the relationship between pre-existent BE and the severity of COVID-19 infection is contradictory. There is a need for future well-designed studies to better understand this point.

Impact of COVID-19 Pandemic on Patients with BE

An analysis of how COVID-19 infection has affected patients with chronic inflammatory airway disease in general,[71-75] and with BE in particular, could certainly provide information of great clinical interest. A prospective observational Scottish study involving 147 patients with CT-confirmed BE, 57.1% women, aged 70 years on average, showed that during the pandemic (from 2020 to March 2021), there was an overall reduction in exacerbations of up to 50%, from 2.08 exacerbations per patient per year in 2018–2019 to 1.12 in 2020–2021, and in hospitalizations, which decreased from about 15.0% to 8.8%, but there was no change in individual chronic respiratory symptoms compared to those observed in the two years before the pandemic (2018–2019). These findings are remarkably similar to those found in other diseases such as COPD.[72] It is likely that this clear reduction in exacerbations might have several causes, such as the social distancing measures, increase in respiratory protection measures and decrease in environmental pollution observed during the pandemic period.

Probably due to the respiratory protection, the decrease in environmental pollution and social distancing measures, the number of BE exacerbations significantly decreased during the pandemic as that occurred in other airways diseases such as COPD. Future studies should be developed to analyze the impact of these measures in patients with chronic airways diseases even in non-pandemic periods.

Conclusions

It is an undeniable fact that a high percentage of individuals who have suffered pneumonia due to COVID-19, especially in its severe form, will present clinical, radiological and functional alterations as a sequela, but their long-term impact is not yet known. Although most studies agree that the majority of radiological images compatible with BE are associated with underlying fibrotic processes, in some cases, these bronchial dilations are associated with an increase in bronchial wall thickening, which indicates that an inflammatory component can continue to exist months after the acute SARS-CoV-2 infection.[59] However, to date, it is not known whether this persistent inflammation is due to the past SARS-CoV-2 infection or due to new bacterial infections since no serial microbiological studies have been done in post-COVID patients. It is also known that other viruses that have produced pneumonic episodes in some patients have been associated with BE that eventually produces BE-related symptoms. In contrast, some forms of BE observed in the acute process of the disease in images with a high inflammatory load could disappear over time as the pulmonary inflammatory state improves.[52]

It is still too early to know whether any of these instances of BE will produce symptoms, exacerbations, or bacterial bronchial infections in the individual suffering from them, or whether they will persist as traction BE. It is possible that both forms may coexist and that there are certain risk factors associated with respiratory symptoms, or an increase in them related to BE.

Special attention must be paid to those patients who already present an underlying pathology, including those who already suffered from pre-existing BE, to assess whether SARS-CoV-2 infection is going to produce a change in their natural history. It will also be important to check whether the different current or future variants of the virus (especially the current omicron variant, whose future impact is still really very little known) will influence the evolution of BE in different ways.

In the meantime, it seems vital to monitor those patients with radiological sequelae (including BE) to assess the evolution of these sequelae. In fact, an international consensus on the research priorities on the long-term sequelae of COVID-19 established the need for an identification of the predictors of a new diagnosis of BE after COVID-19 in CT scans at 6 months after hospital discharge (e.g., disease severity, length of stay, and clinical history of sputum production and sputum microbiology at ≥3 months).[34]

Conflicts of interest

None.

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