

Atypical Post-COVID Sequel: Bronchiectasis

Atipik Post-COVID Sekeli: Bronşektazi

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Abstract

Although there have been many studies determining the occurrence of post-COVID pulmonary fibrosis and thromboembolism, there are a limited number of studies and case reports in literature on the development of bronchiectasis. The present study presents a case of bronchiectasis sequel in the post-COVID 11th month. A 49-year-old male, non-smoker with diabetes mellitus and hypertension was admitted with exertional dyspnea. The patient had been followed up in the hospital 11 months earlier for 1.5 months with severe COVID-19 pneumonia and respiratory failure for which he was treated with Favipiravir, pulse methylprednisolone and broad-spectrum antibiotics. There was no need for invasive mechanical ventilation, and no secondary bacterial infection was detected. Compared to the previous CT, a chest CT revealed that bronchiectasis had persisted despite the disappearance of fibrotic changes. In the coming years, one of the first questions raised regarding the etiology of bronchiectasis may be the patient's COVID-19 history.

Key words: COVID- 19, bronchiectasis, post-COVID sequel.

Öz

Post- COVID pulmoner fibrozis ve tromboemboli gelişimine dair çok sayıda yaygın olmasına rağmen, bronşektazi gelişimi ile ilgili literatürde sınırlı sayıda yayın ve olgu sunumları bulunmaktadır. Biz de bu çalışmamızda post-COVID 11. ayda bronşektazi sekeli saptanan olgumuzu sunmayı amaçladık. Kırk dokuz yaşında, erkek, diabetes mellitus ve hipertansiyon tanılı, non-smoker hasta, efor dispnesi ile başvurdu. On bir ay önce ağır COVID-19 pnömonisi ve solunum yetmezliği ile hastanede 1,5 ay süreyle yatırılarak izlenmişti. Favipiravir, pulse metilprednizolon ve geniş spektrumlu antibiyotik verilen hastanın invazif mekanik ventilatör ihtiyacı olmamıştı ve sekonder bakteriyel enfeksiyon saptanmamıştı. Toraks BT'sinde eski BT'leri ile kıyaslandığında fibrotik değişiklikler kaybolmasına rağmen bronşektazisinin sebat ettiği görüldü. Gelecek yıllarda bronşektazi etyolojisi ilk sorgulayacağımız nedenlerden biri COVID-19 geçirme öyküsü olabilir.

Anahtar Sözcükler: COVID- 19, bronşektazi, post-COVID sekeli.

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Bronchiectasis is indicated by the inner diameter of the bronchus being larger than the accompanying vessel on computed tomography (CT) (bronchus/arterial ratio greater than 0.7), by the disappearance of bronchial narrowing or by the appearance of bronchi within 1 cm of the pleural surface (1), or by the permanent and abnormal dilatation of the bronchi resulting from the destruction of elastic tissues and muscles (2,3).

While immunodeficiency syndromes, and genetic and metabolic defects rank in the first place in the etiology in developed countries, bacterial, viral and fungal infections are at the forefront in developing countries (4). Respiratory infections in childhood are usually severe. Viral infections lead to mucociliary clearance damage, thereby allowing the infection of the respiratory tract. Continued infection leads to the prolongation of the inflammatory process and bacterial colonization, leading to a repetitive cycle that triggers progressive lung damage. With the release of elastase, metalloproteinases and reactive oxygen species by neutrophils, it damages elastin and basement membrane collagen, and proteoglycans are involved in the weakening of the bronchial wall and bronchial enlargement (5). Elastase causes epithelial cell damage, goblet cell hyperplasia and mucosal hypersecretion (6).

Although viral agents are mentioned in many studies, the effect of Coronavirus Disease 2019 (COVID-19) on the development of bronchiectasis remains unclear. Post-COVID chronic cough, fibrotic lung disease, pulmonary vascular diseases and bronchiectasis have all been defined as potential respiratory problems (7). Although many publications have determined the occurrence of post-COVID pulmonary fibrosis and thromboembolism, there have been a limited number of studies and case reports to date assessing the development of bronchiectasis (8). The present study presents a case of bronchiectasis sequel in the post-COVID 11th month.

CASE

A 49-year-old male patient was admitted with a complaint of exertional dyspnea. A physical examination revealed no pathological findings except for crepitation sounds in the auscultation of the thoracic baselines. Partial oxygen saturation in room air was 94%. The patient, who had been diagnosed previously with diabetes mellitus and hypertension, had no smoking history, and had been hospitalized for approximately 1.5 months due to severe COVID-19 pneumonia (SARS-CoV2 PCR test obtained with nasopharyngeal sampling was positive) and

respiratory failure 11 months earlier. A chest X-ray revealed diffuse ground-glass infiltration (Figure 1a). Diffuse ground-glass consolidations, including an air bronchogram, were observed on chest CT taken in the post-COVID first month (Figure 1b, c and d). The patient's file revealed that the patient had been treated with Favipiravir, low molecular weight heparin, piperacillin-tazobactam and 1gr/day methylprednisolone (withdrawn and reduced) therapy for three days along with high-flow nasal oxygen therapy, and an oxygen concentrator was prescribed for home use at the time. No secondary bacterial infection agents grew in the sputum culture. The patient had no history of being treated for pneumonia or tuberculosis other than COVID-19, including in childhood; and there was no chronic cough or sputum complaint. A chest CT obtained at the post-COVID 11th month revealed bilateral bronchiectasis, peripheral air cyst in the left lung and peripheral atelectasis bands. Significant regression in fibrotic appearance (Figure 2) was noted when compared to the chest CT performed in the post-COVID third month. The patient was informed about the necessity of flu, pneumococcal and COVID-19 vaccines and was followed up.

DISCUSSION

Bronchiectasis can occur rapidly and cause sequel in cases of COVID-19 infection, and comorbidities and secondary infections may be predisposing factors for bronchiectasis (5). The presented case had diabetes mellitus and hypertension, and so blood sugar regulation may have been impaired during the period of steroid therapy. Despite the predisposition to secondary infections after high-dose steroids, no growth was detected in the patient's sputum culture.

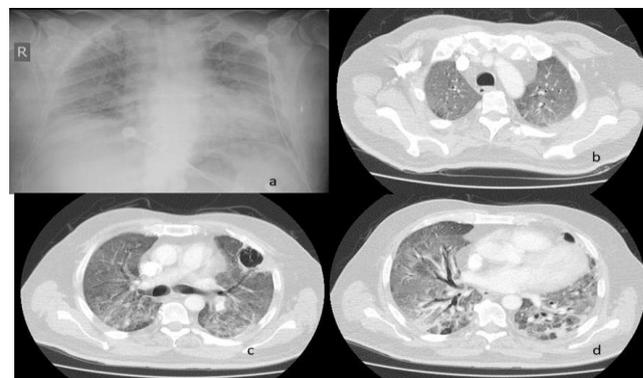


Figure 1: Chest X-ray at the time of hospitalization revealing bilateral ground-glass infiltration (a), in the 1st month post-COVID, revealing a peripheral air cyst in the left upper lobe, diffuse ground-glass and consolidated areas, and bronchiectasis in the axial sections of a chest CT (b,c,d)

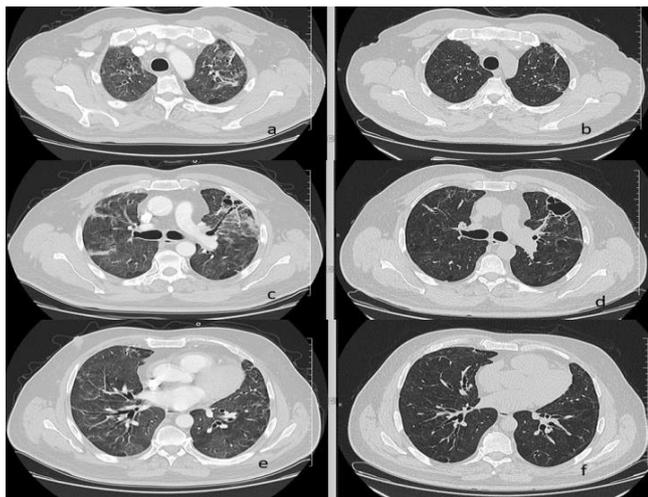


Figure 2: Chest CT axial sections showing persistent bronchiectatic changes despite persistent regression in fibrotic findings at the third month post-COVID (a,c,e) and 11th month post-COVID (b,d,f)

High-resolution CT findings 3 months after discharge in China have revealed interstitial thickening (27.27%), pure ground glass opacity (7.27%) and crazy paving (5.45%) findings (9), and traction bronchiectasis secondary to post-COVID fibrosis is also common. Traction bronchiectasis is a subtype of bronchiectasis in which the bronchi become dilated secondary to mechanical traction due to fibrosis of the adjacent lung parenchyma, and lung injury resulting from invasive mechanical ventilation may also contribute to the process. It is not known how much of the bronchiectasis persists following the resolution of interstitial pneumonia. Enlarged or convoluted bronchi lose their ability to clear mucus effectively and may predispose the patient to recurrent infections (10). Although fibrotic changes were observed in our patient's post-COVID 3rd-month chest CT, most had regressed by the 11th month, although the bronchiectasis image persisted.

Bronchiectasis associated with COVID-19 is an atypical finding. In a retrospective study, bronchiectasis changes were described in one of 121 COVID-19 patients (11). Secondary bacterial infections, prolongation of weaning from mechanical ventilation, and length of hospital stay may lead to the formation of bronchiectasis (12). Our patient did not need invasive mechanical ventilation during his prolonged hospital stay of 1.5 months.

Increased interleukin-6 has been associated with the incidence of severe bronchiectasis in tuberculosis patients (13). Interleukin-6 is an acute phase reactant that forms in the early phase of inflammation, and high Interleukin-6 levels have also been associated with poor prognosis in COVID-19 (14). Based on this relationship, we suggest that bronchiectasis may be seen more frequently in

COVID-19 cases with severe pneumonia and respiratory failure.

CONCLUSION

Apart from post-COVID fibrosis and pulmonary thromboembolism, the present study draws attention also to isolated bronchiectasis. One of the first questions posed regarding the etiology of bronchiectasis in the years to come may be the patient's COVID-19 history.

CONFLICTS OF INTEREST

None declared.

AUTHOR CONTRIBUTIONS

Concept - E.A.; Planning and Design - E.A.; Supervision - E.A.; Funding - E.A.; Materials - E.A.; Data Collection and/or Processing - E.A.; Analysis and/or Interpretation - E.A.; Literature Review - E.A.; Writing - E.A.; Critical Review - E.A.

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