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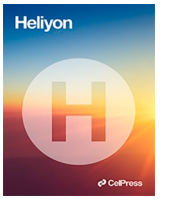

Case report

Adult-onset asthma induced by COVID-19: A case report

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ABSTRACT

COVID-19 commonly presents respiratory symptoms that can linger after the acute phase. Asthma onset or exacerbations have been documented after viral infections although rarely in adults.

We report a case of adult-onset asthma triggered by mildly symptomatic COVID-19 in a 24-year-old female without previous respiratory issues.

Fatigue, exercise dyspnea, and intermittent cough persisting a month after the infection, led to spirometry testing, revealing reduced lung function, with normal CT imaging. Budesonide/formoterol therapy improved symptom control and repeated spirometry testing showed improving but reduced lung function after five months. Methacholine testing was thus conducted and confirmed bronchial hyperreactivity and adult-onset asthma.

Clinicians should be attentive to persistent respiratory symptoms and suggest appropriate testing. Further research should focus on underlying mechanisms of this phenomenon.

1. Introduction

COVID-19, the disease caused by the coronavirus SARS-CoV-2, emerged as a pandemic in early 2020 and has since posed substantial disruption to public health and healthcare systems functioning worldwide. Symptomatic cases of COVID-19 can present with diverse symptoms, the most common are respiratory, including rhinitis, cough, dyspnea with fever and fatigue. Severe cases progress to pneumonia, respiratory distress, and multiple organ failure, requiring hospitalization and intensive care [1–4].

Some individuals experience persistent symptoms long after the initial infection has resolved, that is known as Long COVID or Post-Acute Sequelae of SARS-CoV-2 infection (PASC) [5]. These lingering symptoms, which can endure for months or even years, affect up to 10 %–30 % of individuals who have been infected with COVID-19. Common manifestations of Long COVID include persistent fatigue, dyspnea on exertion, chest discomfort, neurological and psychological alterations, ageusia, and anosmia [7–10].

While most patients experience a decreasing trend in symptom severity over time, up to 10 % of cases may develop chronic conditions following COVID-19 infection. Cohort studies and reports indicate an increased risk of cardiovascular problems [11], autoimmune diseases [12], and other complications. Respiratory sequelae remain the most frequently reported [13,14], including the most worrisome lung fibrosis [15] and interstitial lung disease [16], especially after severe infections. Nevertheless, the interaction between COVID-19 infections and the development of new onset or pre-existing asthma is still not clear, particularly in the adult population.

Asthma, an airway chronic inflammatory disorder, presents with dyspnea, cough, wheezing associated with reversible airflow obstruction, and bronchial hyperresponsiveness [17]. Its etiology, although not fully clear, likely origins from genetic, environmental,

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and immunological factors [18–23]. While most cases of asthma present and are diagnosed in childhood or adolescence, it can develop and manifest at any age [24,25]. Some evidence has linked asthma onset to triggering viral infections including from coronaviruses, especially in younger patients, more rarely in adults [26–32].

Understanding how COVID-19 and adult-onset asthma could be linked can provide insights for physicians and patients that face post-infection symptoms. Furthermore, investigating its underlying pathophysiological mechanisms could help explain lingering respiratory symptoms, such as long COVID syndrome. In this report, we present the case of a previously healthy adult patient who experienced likely asthma onset following a mildly symptomatic COVID-19 infection.

2. Case presentation

A 24-year-old female without personal or family history of respiratory symptoms, chronic illnesses, allergies, or medication use, experienced a mildly symptomatic SARS-CoV-2 infection in September 2021. At the time the patient had received two doses of the Pfizer-BioNTech COVID-19 vaccine and at blood antigen testing had evidence of satisfactory IgG response to the Spike protein. The infection was confirmed after 2 days from symptom onset through a positive nasopharyngeal swab and reverse transcription-polymerase chain reaction (RT-PCR) test. The patient presented with mild symptoms, specifically fever, myalgia, rhinorrhea, and anosmia, which resolved within five days of onset. A summary of the patients' clinical history can be found in Table 1.

At medical evaluation two days after symptom onset, the patient's blood oxygen saturation and heart rate were within the normal range, and no abnormal lung sounds were detected. Blood work revealed mildly elevated C-reactive protein (CRP), normal lactate dehydrogenase (LDH). Complete blood count was within normal range from blood differential test emerged lymphocytopenia and monocytosis, while maintaining the total WBC at 4.5×10^{9}. Blood laboratory values returned to the normal range after three days.

Symptoms improved with the use of only paracetamol and nonsteroidal anti-inflammatory drugs (NSAIDs). A follow-up swab test conducted 14 days after symptom onset, as per previous guidelines, returned negative for SARS-CoV-2. However, following the resolution of the infection, the patient reported persistent fatigue, dyspnea on exertion, and sporadic cough. Long-COVID syndrome was suspected and a spirometry test performed 30 days after the onset of COVID-19 symptoms revealed globally reduced lung function, affecting forced vital capacity (FVC), forced expiratory volume (FEV1), and forced mid-expiratory flow (FEF25-75 %) [33,34].

The patient was advised to begin treatment with budesonide/formoterol in low doses (160/4.5 mcg twice per day) and repeat spirometric testing after 15 days. High-resolution computed tomography (CT) imaging conducted at 40 days post-infection showed no abnormalities in the lung parenchyma or airways, excluding long lasting lung modifications or fibrosis. A follow-up spirometry test performed at 45 days post-infection showed a slight improvement in lung function, however, dyspnea symptoms persisted, and the patient reported exacerbation of symptoms when the corticosteroid and long-acting beta-agonist (LABA) therapy was temporarily suspended.

Five months after the initial infection, dyspnea symptoms were mainly controlled by inhalation therapy with satisfactory patient

Table 1
Patients' clinical history.

Demographics	
Ethnicity	Caucasian
Age, y	24
Sex	Female
Body mass index, kg/m2	26.57
Sars-Cov-2 infection history	
Sars-Cov-2 IgG antibody titer, AU/mL[a]	1.270,40
Date of COVID-19 RT-PCR diagnosis	13 September 2021
Date of asthma diagnosis	15 February 2022
Comorbidities and risk factors	
COPD #	No
Tuberculosis/chronic lung infections	No
Previous allergic diseases	No
Smoking history	Nonsmoker
Family history of asthma	No
Occupational risk of exposure	No
Respiratory symptoms	
Dyspnea	Yes
Cough	Yes
Sputum	No
Wheezing	No
Treatment	
Inhalatory	Low-dose ICS/LABA §
Systemic Steroids	None

Chronic obstructive pulmonary disease.
§ Inhaled Corticosteroids/Long-Acting Beta2-Agonists.
[a] SARS-CoV-2 IgG assay, including neutralizing antibodies directed against RBD of the S 1 subunit of the Spike protein, Chemiluminescence method, cutoff for positive result: >50.

Table 2
Spirometry values at subsequent follow up testing. Values were calculated trough a specialized software complying with ATS/ERS 2019 guidelines.

Parameter	Theoretical value	Measured (% theoretical value)			
FVC, L	**3.77**	2.47 (66 %)	2.71 (72 %)	2.68 (71 %)	2.92 (77.5 %)
FEV1, L	**3.29**	1.87 (57 %)	2.3 (70 %)	2.45 (74.5 %)	2.66 (81 %)
FEV1/FVC, %	**84.4**	75.7 (90 %)	84.9 (100 %)	91 (107 %)	91 (107 %)
PEF, L/s	**7.21**	2.67 (37 %)	4.78 (66 %)	5.18 (72 %)	6.71 (93 %)
FEF 25–75, L/s	**4.13**	1.74 (42 %)	–	3.19 (77 %)	3.92 (95 %)
Date of spirometry (months from Sars-cov-2 infection)		12/10/21 (1)	27/10/21 (1.5)	15/02/2022 (5)	22/03/23 (18)

Values were calculated trough the winspiropro software, complying with ATS/ERS 2019 guidelines.
FVC: forced vital capacity; FEV1: Forced expiratory volume in 1 s; FEV1/FVC: percentage of FVC expired in 1 s; PEF: Peak expiratory flow, maximal flow during maximally forced expiration; FEF 25–75: Forced expiratory flow from when 25 percent of the FVC has been exhaled to when 75 percent of the FVC has been exhaled.

adherence. However, when the therapy was suspended, symptoms exacerbated again. Given symptoms presentations, and the response to empirical therapy, asthma was suspected and methacholine challenge test, diffusion capacity (DLCO) testing, and plethysmography were conducted. Control spirometry showed slight improvement of measured values and DLCO testing was normal. The methacholine challenge test was positive (20 % fall in the forced expiratory volume in 1 second [FEV1]) for bronchoconstriction at a 1100 mcg dose, confirming the diagnosis of asthma. A summary of clinical testing can be found in Table 2, values were calculated trough a specialized software complying with ATS/ERS 2019 guidelines [34].

Over the subsequent year, the patient experienced two additional SARS-CoV-2 infections documented through nasal swab testing at 6 months and 14 months after the initial infection. Both infections presented as mildly symptomatic, with fever, cold-like symptoms, and myalgia, and resolved uneventfully with the use of NSAIDs and a slightly increased need for a rescue dose of inhaled budesonide/formoterol.

Prick allergy testing was advised per usual asthma diagnosis workup, and a mild reaction to Dermatophagoides farinae and pteronis allergens was detected; the patients reports dust as a mild trigger for asthma symptoms. Follow-up spirometry at 1 year from the asthma diagnosis (18 months from SARS-CoV-2 infection) showed a still reduced lung function compared to expected values, although it had improved from the first spirometry (see Table 2). According to severity classification guidelines, our patient's symptoms are categorized as mild persistent asthma [35].

The indication for chronic treatment with budesonide/formoterol at the dose of 2 or 3 daily inhalations of 160/4.5 mcg was confirmed. The treatment provided good symptom control, and specific blood work conducted during the follow-up did not indicate any significant alterations, alpha 1 antitrypsin and protein electrophoresis was within range.

3. Discussion

The case report describes adult asthma onset secondary to a mildly symptomatic SARS-CoV-2 infection without significant previous history or risk factors. Given the patient's persistent respiratory symptoms, inhalation therapy with corticosteroids and Long-Acting Beta-Agonist was initiated before a specific diagnosis and four months later spirometry and methacholine testing confirmed the diagnosis of asthma, while high-resolution CT imaging revealed no abnormalities, distinguishing this presentation to other described Long-COVID syndromes.

While asthma is a well-studied chronic respiratory condition, the potential role of COVID-19 in asthma development remains an area of ongoing research [29,31,36]. COVID-19 infections seem to be able to cause asthma exacerbations, although in population-wide studies their incidence was reduced during COVID-19 waves, as lockdowns, reduced social contact, increased mask use, and better hand hygiene, could have reduced overall rates of viral infections or reduced exposure to pollution [30,37–39].

Adult-onset asthma in individuals without prior respiratory issues or other risk factors is relatively uncommon. Viral infections such as those from Respiratory Syncytial Virus (RSV) or coronaviruses, have been linked to asthma onset, as they can induce airway inflammation, hypersecretion, cough, and bronchospasm, leading to airway architectural changes and altered immune function, influencing airway responses to insults [28,32].

In a large Korean cohort study by Kim et al. analyzing non-asthmatic subjects, COVID-19 increased asthma incidence of 2.1-fold compared to people never infected by Sars-Cov-2 [40]. As already reported in other cohort studies that described increased health care utilization for asthma after COVID-19 infection [41]. Among risk factors for asthma onset there was severity of COVID-19 infection, as described in children that had increased risk of developing asthma symptoms after a COVID-19 related hospitalization [42]. Older age, diabetes, hypertension and allergic rhinitis also increased asthma onset risk. Vaccination against Sars-Cov-2, was also found to be a protective factor [40].

It is hypothesized that interactions between viral factors, host immune responses, and individual susceptibility may contribute to asthma development following a COVID-19 infection, although further investigation on these mechanisms is still needed. It has been described that hyper eosinophilia (in subjects not treated with corticosteroids) can increase Sars-Cov-2 severity trough not fully cleared mechanisms [43]. The patient in our case displayed normal fractional exhaled nitric oxide, a favorable response to inhaled corticosteroids, and low environmental allergen sensitization, indicative of a probable T2-high nonallergic asthma phenotype. Further studies are warranted to ascertain if eosinophil-mediated inflammation [44], typical of T2-high nonallergic asthma, could contribute to asthma onset following COVID-19 infections, potentially opening new treatment or prevention strategies.

Available evidence is not yet conclusive but is plausible that asthma could be considered as a COVID-19 infection sequela, as a specific entity among the respiratory post-COVID syndromes. The subject of our case report was fully vaccinated, did not have any previous risk factors or a severe COVID-19 infection and would be considered a low-risk subject. A comprehensive evaluation of persisting respiratory symptoms especially if they worsen over time is needed for all patients and considering asthma testing can be indicated even in healthier and lower risk patients.

As this report comprises a single case, it can't draw definitive conclusions on the link between these diseases, additional investigations are needed to comprehensively understand the underlying mechanisms and potential impact of COVID-19 on adult asthma development. Raising awareness among healthcare providers regarding the potential link between COVID-19 and asthma development is vital to ensure prompt and suitable management for affected patients. Clinicians should consider asthma as part of the differential diagnosis for subacute or chronic respiratory symptoms following COVID-19 infection.

4. Conclusion

This case report presents an instance of adult-onset asthma following a mildly symptomatic SARS-CoV-2 infection in a previously healthy 24-year-old female.

Clinicians should be vigilant in monitoring patients recovering from COVID-19, especially those with persistent respiratory symptoms, for the development of asthma. Early recognition and management of asthma symptoms following COVID-19 recovery are essential for improved patient outcomes and tailored care. Further research is necessary to unravel the complex relationship between COVID-19 and adult-onset asthma, leading to enhanced preventive and treatment strategies.

Additional research is needed to understand the possible relationship between COVID-19 and adult-onset asthma, paving the way for better preventive strategies and treatment approaches.

Patient perspective

COVID has become a common illness, with lots of information available world-wide. Nonetheless it's not clear yet what's the extent of long term consequences of the disease. Arriving at a final diagnosis was a long process, as I was struggling with finding the right specialist and the right tests, most were stumped or had a myriad of hypothesis. Thankfully in the end asthma is controllable and my quality of life wasn't much affected, unlike other patients with COVID sequalae.

Ethics statement

The authors declare that appropriate written informed consent was obtained for the publication of this manuscript and accompanying data.

Availability of data and materials

All data collected for the present study are included in this published article.

CRediT authorship contribution statement

Federica Cucè: Writing – review & editing, Writing – original draft, Validation, Methodology, Investigation, Data curation. **Marco Visicaro:** Writing – review & editing, Writing – original draft, Data curation, Conceptualization.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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