Hyperventilation syndrome following mild COVID-19 in a health care worker

Leila Laouar MD, Nadia Dammene Debbih MD, Sonia Nouioua MD

Abstract

Hyperventilation syndrome (HVS) is a frequently overlooked complication in the aftermath of SARS-CoV-2 infection. We describe the case of a 53-year-old female dental surgeon who, several weeks after recovering from COVID-19, developed severe exertional dyspnea (mMRC grade 4), palpitations, and marked anxiety (HADS score 14/9). Despite normal findings on pulmonary function tests, chest computed tomography, and echocardiography, clinical suspicion was raised for HVS based on a high Nijmegen score (49/64) and the presence of hypocapnia (26.9 mmHg) on post-exercise arterial blood gas following a six-minute walk test (6MWT), which also revealed reduced exercise capacity (67% of predicted distance). A multidisciplinary management plan including respiratory physiotherapy, psychotherapy, and pharmacologic anxiolysis was implemented. Follow-up demonstrated significant symptom improvement and functional recovery, enabling a return to professional activity. This case underscores the relevance of considering HVS in the differential diagnosis of post-COVID dyspnea and illustrates the utility of simple tools such as the 6MWT and Nijmegen questionnaire in both diagnosis and therapeutic monitoring.

Keywords : Hyperventilation syndrome, post-COVID-19, Nijmegen questionnaire, hypocapnia, exercise testing.

Introduction

Post-COVID-19 syndrome refers to the persistence of symptoms of varying severity following infection with SARS-CoV-2.[1] Among these, chronic post-COVID dyspnea is a common complaint that requires a thorough investigation to exclude other potential causes such as respiratory disease, cardiovascular conditions, thromboembolic events, deconditioning, respiratory muscle involvement, or hyperventilation syndrome (HVS).[2,3] This syndrome is defined as a set of somatic symptoms resulting from inappropriate excessive ventilation, which can be partially or fully reproduced through voluntary hyperventilation.[4] The underlying pathophysiological mechanisms remain controversial. Despite the wide range of somatic and psychological symptoms it may provoke due to physiologically inappropriate hyperventilation, the syndrome is often overlooked.[5,6]

Post-COVID-19 HVS appears to be a frequent occurrence, affecting approximately 15.6% of survivors according to Cherif et al., with suggestive evidence of a significant psychosomatic component.[7]

Diagnosing HVS can be challenging and requires recognizing often misleading symptoms, performing suggestive diagnostic tests, and ruling out other etiological factors. Although no gold standard for diagnosis exists, a combination of clinical history, the Nijmegen questionnaire.[8] and the hyperventilation provocation test constitutes a robust diagnostic approach. Despite its extremely low mortality, diagnosing HVS remains

Corresponding author: **Leila Laouar**
Contact Information: Laouar_Leila@yahoo.fr
DOI: 10.12746/swjm.v14i58.1545

crucial, as chronic hyperventilation significantly impairs social and occupational quality of life and is amenable to rehabilitative interventions.[9,10]

Case

Initial COVID-19 episode

The initial illness occurred several months prior and was characterized by anosmia, dry cough, moderate fever, and paroxysmal shortness of breath. Clinical examination revealed a respiratory rate of 20 breaths/min, heart rate of 100 beats/min, oxygen saturation (SpO_2) of 98% on room air, blood pressure of 97/60 mmHg, and normal pulmonary auscultation. The SARS-CoV-2 PCR test was positive. Chest computed tomography (CT) showed 10% ground-glass opacities, with no features suggestive of severity.

Biological investigations revealed no leukocytosis, moderate lymphopenia (1,000 cells/mm^3), normal renal and liver function tests, and D-dimer levels within the reference range.

The treatment regimen included antibiotics, analgesics, vitamins C and D, zinc, and magnesium supplementation.

The clinical course was marked by resolution of the fever and disappearance of the cough, but persistent resting dyspnea remained, despite a normal physical examination and follow-up chest CT showing complete resolution of the initial lesions without new abnormalities.

Post-COVID-19 assessment characteristics of the current episode

Two months after the acute COVID-19 episode, the patient continued to experience persistent dyspnea, described as progressive breathlessness occurring at rest and becoming markedly disabling. This was associated with uncomfortable palpitations and profound fatigue, which significantly impaired her quality of life.

She also reported two episodes of near syncope at work, which she attributed to enclosed environments and prolonged mask use suggestive of contextual hypersensitivity to stressful and confined settings.

A specialized psychiatric evaluation ruled out social phobia but revealed a persistent anxiety state, likely related to her post-infectious experience and pre-existing emotional vulnerability.

Notably, her dyspnea improved during morning walks outdoors, especially near the sea, suggesting a functional or psychogenic component to the clinical picture.

Professionally, she had suspended her practice twice due to the severity of her symptoms and their impact on her functional capacity. She had been on medical leave for 45 days at the time of evaluation, reflecting significant social and occupational dysfunction.

First-line investigations

a. Clinical examination at rest and during exertion

Pulmonary and cardiovascular examinations were strictly normal. Peak expiratory flow (PEF) was measured at 450 L/min (reference value: 464 L/min). Psychometric evaluation using the Hospital Anxiety and Depression Scale (HADS) revealed a score of 14 for anxiety and 9 for depression. The Nijmegen questionnaire, used to screen for hyperventilation syndrome, yielded a strongly positive score of 49/64.

b. Additional investigations

Complete blood count showed no anemia. Renal function, thyroid-stimulating hormone (TSH), and D-dimer levels were all within normal limits. Resting spirometry was normal, as were the electrocardiogram (ECG) and transthoracic echocardiography, with no structural or functional abnormalities. Resting arterial blood gases were also normal.

The six-minute walk test (6MWT) revealed a walking distance of 399 meters, corresponding to 67% of the predicted value, without oxygen desaturation. However, the test induced tachypnea (29 breaths/min) and tachycardia (141 bpm), both clinically well-tolerated without the need to interrupt the test.

Table 1. Laboratory and Functional Assessment

Examinations	Parameters	Unit	Reference Range	Results	Comments
Biology	WBC	$10^3/mm^3$	04–10	7,3	Normal
	RBC	$10^6/mm^3$	3,6–5,8	4,6	Normal
	Hb	g/dl	12–16	12,7	Normal
	Platelets	$10^3/mm^3$	150–400	290	Normal
	Urea	g/l	0,15–0,45	0,19	Normal
	Creatinie	mg/dl	07–14	9,84	Normal
	Blood Glucose	g/dl	1,03	0,70–1,10	Normal
	D-dimers	mg/ml	<500	370	Normal
	TSHus	uU/ml	0,25–4,67	2,17	Normal
Baseline Spirometry	FVC	Litres	4,38	4,11	94% of predicted
	FEV1	L/s	3,54	3,63	102% of predicted
	FEV1/FVC ratio (Tiffeneau Index)	%	81,41	88,36	Normal
Arterial Blood Gas – Rest	ph		7,35–7,45	7.42	Normal
	PaO_2	mmHg	>85	91,7	Normal
	$PaCO_2$	mmHg	35–45	38,3	Normal
	HCO_3^-	mmol/L	22–28	25,6	Normal
	SaO_2	%	95–100	98,1	Normal
Arterial Blood Gas – Post-exercise (6MWT)	ph		7,35–7,45	7,51	Elevated (alkalosis)
	PaO_2	mmHg	>85	101,4	Hyperoxia
	$PaCO_2$	mmHg	35–45	26,9	Hypocapnia
	HCO_3^-	mmol/L	22–28	21,3	Decreased
	SaO_2	%	95–100	98,5	Normal
6MWT		meters	597 (predicted)	399	67% predicted distance

WBC: White Blood Cells; RBC: Red Blood Cells; Hb: Hemoglobin; FVC: Forced Vital Capacity; FEV1: Forced Expiratory Volume in 1 Second; IT: Tiffeneau Index; PaO_2: Arterial Partial Pressure of Oxygen; $PaCO_2$: Arterial Partial Pressure of Carbon Dioxide; HCO_3^-: Bicarbonate Ion Concentration; SaO_2: Arterial Oxygen Saturation; TSHus: Ultrasensitive Thyroid-Stimulating Hormone; 6MWT: Six-Minute Walk Test.

A second arterial blood gas sample, drawn immediately after exercise, showed hyperoxia (PaO_2 101.4 mmHg) and hypocapnia ($PaCO_2$ 26.9 mmHg), with normal pH and bicarbonate levels (see Table 1). Post-exercise questioning revealed reproduction of typical hyperventilation symptoms: palpitations, shallow inspiratory dyspnea, and perioral paresthesias.

Management and follow-up

Given the persistent disabling dyspnea, the high Nijmegen score, and the absence of cardiorespiratory or metabolic abnormalities, a diagnosis of hyperventilation syndrome (HVS) was considered likely. The patient was informed of this diagnosis, which was explained as a physiological and functional dysregulation potentially triggered or maintained by emotional, post-infectious, and behavioral factors.

A multidisciplinary therapeutic plan was proposed and initiated, including:

– Respiratory physiotherapy focused on re-education of breathing patterns, with exercises targeting thoraco-abdominal coordination and slowing of the respiratory rate.

- Cognitive-behavioral psychotherapy sessions aimed at addressing the patient's anxiety and helping her understand and manage the somatic manifestations of stress.
- Pharmacological treatment, which included low-dose anxiolytics (hydroxyzine) and magnesium supplementation, particularly in light of her history of irritable bowel syndrome and emotional fragility.

The patient adhered strictly to the program, with sessions scheduled twice weekly for three weeks, followed by a reassessment.

At follow-up, the patient reported a marked improvement in symptoms, including less frequent palpitations and dyspnea, improved tolerance to daily activities, and a return to part-time professional activity. A repeat six-minute walk test showed a slight improvement in distance (420 meters) and normalization of respiratory and cardiac parameters post-exercise, with no recurrence of hypocapnia. The Nijmegen score decreased to 29/64 (Table 2).

The patient's quality of life improved significantly, as reflected in a reduction of the HADS anxiety score from 14 to 9, and she resumed her professional duties in full, albeit with adjustments to workload and stress management.

Discussion

Post-COVID-19 sequelae both organic and functional are increasingly reported in the literature, particularly in terms of respiratory impairment. However, certain persistent symptoms, such as chronic dyspnea despite normal thoracic imaging, remain unexplained and necessitate the exploration of alternative diagnoses, including cardiac sequelae and hyperventilation syndrome (HVS).[1,2] Interestingly, the risk factors for post-COVID HVS appear to contrast with those of severe acute COVID-19. While severe forms are more prevalent among older males with comorbidities such as obesity and cardiovascular disease, HVS after COVID-19 typically affects younger, active women with no history of cardiopulmonary disease, often following mild infection and in the presence of an anxious or emotionally vulnerable background.[11–13]

In its classic presentation, HVS primarily affects young, professionally active individuals—especially women. A triggering event, often acute or chronic psychological stress, initiates inappropriate hyperventilation, leading to dyspnea due to respiratory muscle overuse. This symptom becomes a source of anxiety, perpetuating a vicious cycle of hyperventilation and anxiety. Psychiatric comorbidities such as anxiety and depression are frequently associated with HVS, further complicating its recognition and management.[13]

Clinically, HVS presents with cardiorespiratory symptoms (dyspnea, sighing, yawning, chest pain, palpitations) as well as systemic and neurovegetative signs (fatigue, dizziness, cognitive impairment, paresthesia, muscle cramps), many of which overlap with symptoms seen in both acute and long COVID syndromes.[14–16] ECG changes, visual disturbances, tinnitus, tremors, migraines, or even muscle spasms may also occur.

Table 2. Evolution of Clinical and Biological Parameters Under Treatment

Paramtes	Initial	Day 7	Day 14	Day 21	Day 30
mMRC scale parameters (Dyspnea)	3	3	3	1	0
HADs (Anxiety/Depression)	13/9	13/8	10/8	10/9	10/7
Nijmegen score	49/64	49/64	34/64	28/64	18/64
6MWT Distance (meters)	399 (67%)	367 (61%)	410 (69%)	490 (82%)	497 (83%)
	Arterial blood gas at Day 30				
	ph	PaO_2	$PaCO_2$	HCO_3^-	SaO_2
	7,39	97 mmHg	39 mmHg	24 mmol/l	98

HADs: Hospital Anxiety and Depression Scale; mMRC: modified Medical Research Council Dyspnea; 6MWT: Six-Minute Walk Test.

Our case is consistent with this profile: a middle-aged female with a long-standing anxiety disorder following a traumatic personal loss, who experienced mild COVID-19 and subsequently developed persistent, unexplained symptoms. Her clinical picture was dominated by cardiorespiratory manifestations and profound asthenia, with no structural abnormalities, prompting a diagnostic orientation toward post-COVID HVS.

The classical approach to assessing dysfunctional breathing relies on clinical assessment and validated screening tools such as the Nijmegen Questionnaire (NQ).[17] Originally developed for detecting HVS, the NQ comprises 16 items, each rated from 0 (never) to 4 (very often), assessing symptoms such as dyspnea, dizziness, paresthesia, and palpitations, for a maximum score of 64. A score of ≥23/64 is strongly suggestive of HVS, with a reported sensitivity of 91% and specificity of 95% in populations without known respiratory disease[18] It is important to note that the NQ does not assess the severity or progression of HVS, but only the likelihood of its presence. In our case, the patient's score was 49/64, significantly above the diagnostic threshold. Furthermore, the questionnaire revealed symptoms the patient did not spontaneously report, considering them unimportant or unrelated. Thus, the NQ helped uncover functional symptoms and supported the diagnostic hypothesis of post-COVID HVS.

Respiratory provocation tests aim to reproduce at least two symptoms listed in the NQ, thereby increasing diagnostic confidence.[19] Various techniques can be used to assess ventilatory response:

Arterial blood gas ($PaCO_2$), Transcutaneous capnography (e.g., earlobe), End-tidal CO_2 ($PETCO_2$) via nasal capnograph[20]

In HVS, hypocapnia (low $PaCO_2$) is a typical finding, resulting from excessive ventilation and thought to be responsible for many functional symptoms. While resting ABG may be normal in the absence of a current hyperventilation episode, exercise-induced ABG abnormalities can be revealing.

Contraindications to provocation testing include ischemic heart disease, cerebrovascular disease, hypercapnic respiratory failure, and sickle cell disease (due to thrombosis risk). In this case, provocation tests could not be performed due to equipment unavailability.

Voluntary Hyperventilation Test (HVPT) involves guided over breathing for several minutes and evaluating whether typical HVS symptoms are provoked. Though its sensitivity and specificity remain imperfectly established, many authors consider it a useful diagnostic tool[21]

Cardiopulmonary Exercise Testing (CPET) may help diagnose HVS in patients who show preserved capacity but evidence of relative deconditioning.[22] Common ventilatory findings include Effort-induced hypocapnia: Rapid shallow breathing, Mouth breathing, Lack of end-expiratory pause.[22,23]

A comparative study showed that HVPT is superior to CPET in reproducing HVS symptoms, suggesting it is more effective in provoking the characteristic clinical picture.[24]

Six-Minute Walk Test (6MWT), although not validated for diagnosing HVS, provides a reliable and reproducible assessment of submaximal exercise capacity, integrating cardiovascular, respiratory, and muscular responses[25] It is widely used in chronic respiratory disease evaluation and rehabilitation follow-up.

In our case, due to lack of capnography and rehabilitation facilities, the 6MWT was used to:

- Rule out significant physical deconditioning
- Evaluate ventilatory response during exertion
- Perform post-exercise arterial blood gas

This revealed exercise-induced hypocapnia, confirming hyperventilation. In addition, the patient reported recurrence of multiple Nijmegen-related symptoms post-exercise, further supporting the diagnosis of exercise-induced HVS in the post-COVID context.

Conclusion

Hyperventilation syndrome (HVS) is a frequent manifestation in the post-COVID-19 period and may present as disabling chronic dyspnea. In the presence of suggestive clinical symptoms, and after ruling out pulmonary, vascular, or cardiac pathology, the diagnosis primarily relies on the Nijmegen Questionnaire and the voluntary hyperventilation test. However, HVS is often diagnosed late due to the complex interplay between post-COVID syndrome and functional breathing disorders, as well as the limited availability of highly specialized diagnostic centers. This diagnostic delay frequently leads to significant patient anxiety and results in numerous costly, sometimes invasive investigations with limited diagnostic yield.

While capnography remains the gold standard for detecting abnormal ventilatory patterns and hypocapnia, its availability is restricted in many healthcare facilities. In this context, the six-minute walk test (6MWT), commonly employed in pulmonary and cardiopulmonary evaluation, followed by arterial blood gas analysis, may offer a pragmatic and promising alternative for identifying post-exercise hypocapnia and supporting the diagnosis of post-COVID HVS.

Article citation: Laouar L, Debbih ND, Nouioua S. Hyperventilation syndrome following mild COVID-19 in a health care worker. The Southwest Journal of Medicine. 2026;14(58):30–36
From: Youcef El Khatib University of Health Sciences, Algiers, Algeria (LL, NDD, SN)
Conflicts of interest: none

References

1. Hellemons ME, Huijts S, Bek LM, et al. Persistent health problems beyond pulmonary recovery up to 6 months after hospitalization for COVID-19: a longitudinal study of respiratory, physical, and psychological outcomes. Ann Am Thorac Soc 2022 Apr;19(4):551–61. doi:10.1513/AnnalsATS.202103-340OC.
2. Fernández-Plata R, Higuera-Iglesias AL, Torres-Espíndola LM, et al. Risk of pulmonary fibrosis and persistent symptoms post-COVID-19 in a cohort of outpatient health workers. Viruses 2022 Aug 23;14(9):1843.
3. Taverne J, Salvator H, Leboulch C, et al. High incidence of hyperventilation syndrome after COVID-19. J Thorac Dis 2021 Jun;13(6):3918–22.
4. Lum LC. Hyperventilation: The tip and the iceberg. J Psychosom Res 1975;19(5–6):375–83.
5. Boulding R, Stacey R, Niven R, et al. Dysfunctional breathing: a review of the literature and proposal for classification. Eur Respir Rev 2016 Sep;25(141):287–94.
6. Tavel ME. Hyperventilation syndrome: why is it regularly overlooked? Am J Med 2021 Jan;134(1):13–15.
7. Cherif H, Mokaddem S, Debiche S, et al. Incidence and predictive factors of hyperventilation syndrome in patients after COVID-19 pneumonia: a prospective cohort study. F1000Re. 2024;13:1497.
8. van Dixhoorn J, Folgering H. The Nijmegen Questionnaire and dysfunctional breathing. ERJ Open Res 2015 May 15;1(1):00001-2015.
9. Nunes D, Bautin N, Perez T, et al. Impact of hyperventilation syndrome on work productivity. Rev Mal Respir 2018 Jan;35(Suppl 1):A77.
10. Gene Cand L, Maillard S, Szikora JP, et al. Dysfunctional breathing symptoms, functional impact and quality of life in patients with long COVID-19: a prospective case series. BMJ Open Respir Res 2023;10(1):e001767.
11. D'Alba I, Carloni I, Ferrante AL, et al. Hyperventilation syndrome in adolescents with and without asthma. Pediatr Pulmonol 2015 Dec;50(12):1184–90.
12. Motiejunaite J, Balagny P, Arnoult F, et al. Hyperventilation: a possible explanation for long-lasting exercise intolerance in mild COVID-19 survivors? Front Physiol 2021 Jan 18;11:614590.
13. Dafauce L, Plaza V, Callarisa L, et al. Psycho-demographic profile in severe asthma and effect of emotional mood disorders and hyperventilation syndrome on quality of life. BMC Psychol 2021 Jan 5;9(1):5.
14. Lewis RA, Howell JB. Definition of the hyperventilation syndrome. Bull Eur Physiopathol Respir 1986;22(2):201–5.
15. Gavriatopoulou M, Korompoki E, Fotiou D, et al. Organ-specific manifestations of COVID-19 infection. Clin Exp Med 2020 Nov;20(4):493–506.
16. Wang C, Pan R, Wan X, et al. A longitudinal study on the mental health of general population during the COVID-19 epidemic in China. Brain Behav Immun 2020 Jul; 87:40–8.
17. Courtney R, van Dixhoorn J, Greenwood KM, et al. Medically unexplained dyspnea: partly moderated by dysfunctional (thoracic dominant) breathing pattern. J Asthma 2011 Apr; 48(3):259–65.
18. Van Dixhoorn J, Duivenvoorden HJ. Efficacy of Nijmegen questionnaire in recognition of the hyperventilation syndrome. J Psychosom Res 1985;29(2):199–206.

19. Hornsveld H, Garssen B. The low specificity of the hyperventilation provocation test. J Psychosom Res 1996;41(5): 435–9.

20. Pauwen NY, Faoro V, Boucharessas F, et al. Validation Criteria for PETCO2 Kinetics during the hyperventilation provocation test in the diagnosis of idiopathic hyperventilation syndrome. J Clin Med 2022 Oct 31;11(21):6482.

21. Kinnula VL, Sovijarvi AR. Elevated ventilatory equivalents during exercise in patients with hyperventilation syndrome. Respiration 1993;60(5):273–8.

22. Brat K, Stastna N, Merta Z, et al. Cardiopulmonary exercise testing for identification of patients with hyperventilation syndrome. PLoS One 2019 Apr 23;14(4):e0215997.

23. ATS Committee on Proficiency Standards for Clinical Pulmonary Function Laboratories. ATS Statement: Guidelines for the six-minute walk test. Am J Respir Crit Care Med 2002 Jul 1;166(1):111–17.

24. Tiotiu A, Ioan I, Poussel M, et al. Comparative analysis between available challenge tests in the hyperventilation syndrome. Respir Med 2021 Apr; 179:106329.

25. Enright PL, Sherrill DL. Reference equations for the six-minute walk in healthy adults. Am J Respir Crit Care Med 1998 Mar;158(5 Pt 1):1384-7.

26. Resta E, Quarato CMI, Scioscia G, et al. Low-intensity rehabilitation in persistent post COVID-19 dyspnoea: the value of Spa health resort as appropriate setting. Ann Ig. 2024 Sep–Oct;36(5):597–613.

27. Herer B, Ghergan A. Mixed results of pulmonary rehabilitation in hyperventilation syndrome: A case series of six patients. Respir Med Res 2023 Jun;83:100935.