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Arterial hypertension as a sequel to COVID-19. Clinical case report

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Abstract: The COVID-19 disease has caused one of the most important health crises in history. In these terms, this clinical case is reported, which is related to a health worker with no medical history or cardiovascular risk factors. This patient was infected, presenting sequelae of arterial hypertension that required pharmacological treatment. It was showed the need to continue the pathophysiological study of the consequences generated by this pathology, especially regarding arterial hypertension and the potential to establish a critical condition and cardiovascular dysfunction.

Key words: hypertension; coronavirus infections; complications

1 Introduction

December 2019 marks the beginning of one of the most significant health crises in history: the emergence of the disease caused by a new strain of coronavirus genetically identified in January 2020 [1]. In February, China reported around 70,000 cases and enacted lockdown and social distancing measures. Its virulence led to global spread, and a pandemic was declared [2]. By November, approximately 50 million people had been infected and 1,300,000 deaths had been reported worldwide [3].

Some groups are more vulnerable to COVID-19: older adults and people with pre-existing conditions. In Spain, the average age of patients was 69.4 years, and 21% of cases aged 70–79 died. In this context, 50% of those affected had high blood pressure (HBP) [4]. Furthermore, those infected who have pre-existing cardiovascular and metabolic diseases face a higher risk of developing complications [5].

This disease can also trigger sequelae and symptoms following recovery, primarily cardiovascular conditions, with an increase in resting heart rate to over 20 bpm and the presence of hypertension requiring pharmacological treatment [6].

There are reports of elevated blood pressure during the active and convalescent phases, with no apparent relationship to biological factors such as sex, age, and cardiovascular risk factors [7]. This situation may increase the severity of symptoms, raising the likelihood of a fatal outcome [8].

Acute respiratory conditions can trigger the activation of coagulation pathways, pro-inflammatory effects, pro-inflammatory effects, and endothelial cell dysfunction. A study of lung tissue demonstrated that 83% of cells expressing ACE2 were type II alveolar epithelial cells, suggesting they may serve as a reservoir for viral invasion. SARS-CoV-2 can cause myocardial damage and dysfunction, with elevated cardiac enzymes and electrocardiographic abnormalities [9,10].

The renin-angiotensin system plays a fundamental role in the regulation of hypertension and acute lung injury caused

by viruses such as SARS and H7N9, which induce changes in the activity of this cardiovascular regulatory system [11,12]. In this regard, there is a hypothesis regarding the harmful effects of angiotensin-converting enzyme inhibitors and angiotensin receptor blockers [10].

2 Development

We present the case of a patient who works as a healthcare professional at COVID-19 patient care sites. The patient presented at the triage station with a 3-day history of severe holocranial headache, retrocular pain, asthenia, arthralgia, myalgia, and fever.

The patient has no significant personal history or cardiovascular risk factors. On physical examination, the patient is conscious, alert, in pain, and sweating.

Height: 1.65 m

Weight: 60 kg

BMI: 22

Vital signs: blood pressure 149/99; heart rate: 119 bpm; temperature: 38.6°C; 24 breaths per minute; SpO2: 89–90%; and News score: 7.

Skin: warm.

Gait and facial expression not characteristic of pathological processes.

Head: normocephalic, conjunctival erythema, erythematous oropharynx.

Neck: symmetrical with no palpable lymphadenopathy.

Symmetrical chest: rhythmic heart sounds.

Lungs: preserved expandability, mild use of accessory muscles and intercostal retractions, decreased vesicular breath sounds in both fields, predominantly in the left field.

Abdomen, pelvic region, and extremities without abnormalities. No neurological focal signs.

2.1 Additional tests

These included:

- Computed tomography (CT) scan of the chest showing bilateral patchy cotton-wool-like infiltrates, as well as peripheral and subpleural infiltrates (Figure 1).
- Positive RT-PCR from nasal swab.
- Complete blood count, coagulation times, platelet count, urea, creatinine, and fasting glucose, all of which were within normal parameters.

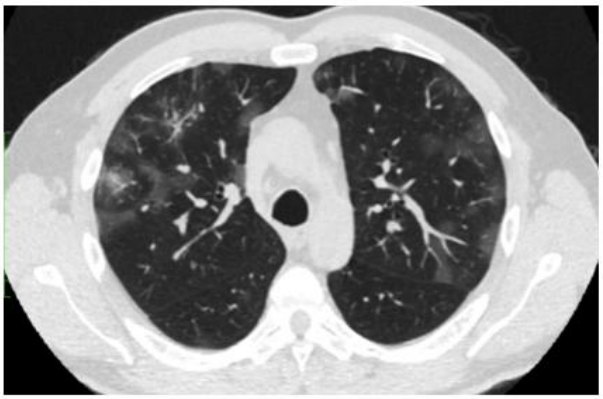

Figure 1. Chest CT

2.2 Therapeutic approach and clinical course

At 24 hours, the patient was monitored at home, and it was observed that he continued to have mild febrile and respiratory symptoms.

Vital signs: blood pressure 160/100; heart rate: 105 bpm; temperature: 37.6 °C; respiratory rate: 24 breaths per minute; SpO2: 87–90%.

2.3 Therapy

As non-pharmacological treatment, a low-salt diet, oral rehydration, respiratory physiotherapy, strict monitoring of warning signs, and daily blood pressure monitoring are indicated (Table 1).

Table 1. Blood pressure values during daily monitoring

Day	Blood Pressure Reading
1	155/95
2	165/98
3	160/95
4	150/89
5	155/87
6	145/90
7	142/90
8	145/85
9	155/95
10	145/95
11	140/90
12	138/89
13	135/89
14	145/90
15	142/90
16	145/85
17	148/89

Pharmacological treatment included acetaminophen and metamizole to control persistent fever. Given the persistence of elevated blood pressure readings (right arm: 160/110, left arm: 155/95), 10 mg of enalapril was prescribed in the morning.

The patient completed 28 days of isolation and rest. After discharge, a weight loss of 6 kilograms was noted, with persistent headache, fatigue, and tachycardia, and blood pressure readings ranging between 160/95 and 155/90. Therefore, it was decided to increase the enalapril treatment with an additional 10 mg dose at night, successfully controlling the symptoms.

2.4 Follow-up

The treatment prescribed at discharge was followed for 5 months, with self-monitoring showing normal levels, and medication administration was discontinued. For the following two weeks, he was asymptomatic and his blood pressure readings remained within normal limits; however, he subsequently developed a holocranial headache, prompting him to seek medical evaluation. His blood pressure was recorded at 147/104, necessitating the resumption of treatment with 10 mg of enalapril daily in the morning.

In addition, an electrocardiogram and blood count were performed, both of which were within normal parameters.

3 Discussion

SARS-CoV-2, a virus that emerged in a province of China in December 2019 [13], has now affected millions of people of all ages and health conditions, with morbidity and mortality rates continuing to rise as more effective therapeutic and immunological treatments are developed [14].

A high prevalence of hypertension was observed in patients with COVID-19 in a study of 138 patients, where approximately 60% of those with severe symptoms had high blood pressure [15].

The mechanisms underlying the possible relationship between hypertension and COVID-19 are not fully understood; but the most widely accepted hypothesis relates to a dysregulation of the renin-angiotensin-aldosterone system and angiotensin-converting enzyme 2, present in the lungs, heart, and systemic vasculature (to a lesser extent in the latter two). This enzyme is involved in the metabolism of bradykinins [16,17,18,19].

Angiotensin II is a potent vasoconstrictor that modifies blood pressure through two mechanisms (inhibiting arterioles, which increases peripheral vascular resistance, and direct retention of salt and water by the kidneys or secretion of aldosterone by the adrenal glands), leading to vascular inflammation and long-term cardiac remodeling [16,17,18,19].

The rehabilitation phase for patients following infection must be continuous and range from to chronic. The main sequelae are related to immobilization, respiratory, cardiovascular, neurological, and psychological syndromes [20], with reports of respiratory distress, cough, fatigue, muscle weakness, post-traumatic stress, limitations in daily activities, pulmonary fibrosis, and cardiovascular and renal disorders [21].

The study of the hypertensive sequelae of the disease caused by the novel coronavirus is of interest to the health sciences. In this regard, sequential monitoring of blood pressure is recommended, considering systolic blood pressure ≥140 mm Hg and/or diastolic blood pressure ≥90 mm Hg as elevated [22,23].

4 Conclusion

The presented clinical case of COVID-19 serves as evidence of the need to continue the pathophysiological study of the sequelae it generates, particularly those related to hypertension and the potential for critical conditions and cardiovascular dysfunction. The patient had no history of cardiovascular disease or risk factors; however, following SARS-CoV-2 infection, he developed sustained hypertension that required pharmacological treatment.

Conflicts of Interest

The author declares no conflicts of interest regarding the publication of this paper.

References

[1] Wang C, Horby PW, Hayden FG, Gao GF. A novel coronavirus outbreak of global health concern. The Lancet [Internet]. 2020 [citado 2 Nov 2020]; 395: 470-473. Disponible en: https://www.thelancet.com/action/show Pdf?pii=S0140-6736%2820%2930185-9. https://doi.org/10.1016/ S0140-6736(20)30185-9.

[2] The Novel Coronavirus Pneumonia Emergency Response Epidemiology Team. Vital Surveillances: The Epidemiological Characteristics of an Outbreak of 2019 Novel Coronavirus Diseases (COVID-19) — China, 2020. CCDC weekly [Internet]. 2020 [citado 4 Nov 2020]; 2(8): 113-122. Disponible en: http://weekly.chin acdc.cn/en/article/id/e53946e2-c6c4-41e9-9a9b-fea8db1a8f51.

[3] World Health Organization. Coronavirus disease (COVID-19) [Internet]. Ginebra: WHO; [actualizado 11 Oct 2020; citado 11 Nov 2020]. Disponible en: https://www.who.int/docs/default-source/coronaviruse/situation - reports/20201012-weekly-epi-update-9.pdf.

[4] Casas-Rojo JM, Antón-Santos JM, Millán-Núñez-Cortés J, Lumbreras-Bermejo C, Ramos-Rincón JM, Roy-Vallejo E, et al. Clinical characteristics of patients hospitalized with COVID-19 in Spain: Results from the SEMICOVID-19 Registry. Rev Clin Esp [Internet]. 2020 [citado 21 Oct 2020]; 220(8): 480 – 494. Disponible en: https://reader.elsevier.com/reader/sd/pii/S001425652030206X?token=36F2D15E28727572FE2B4871C7593EB13FBA89A58659A67D260FF8B6035E42C3F11CAC371E62E6352DDFC0932738F494.

[5] Li B, Yang J, Zhao F, Zhi L, Wang X, Liu L, et al. Prevalence and impact of cardiovascular metabolic diseases on

COVID-19 in China. Clinical Research in Cardiology [Internet]. 2020 [citado 7 Nov 2020]; 109: 531–538. Disponible en: https://link.springer.com/article/10.1007%2Fs00392-020-01626-9. https://doi.org/10.1007/s00392-020-01626-9.

[6] Xiong Q, Xu M, Li J, Liu Y, Zhang J, Xu Y, et al. Clinical sequelae of COVID-19 survivors in Wuhan, China: a single-centre longitudinal study. Clin Microbiol Infect. [Internet]. 2021 [citado 1 Ene 2021]; 27(1): 89-95. Disponible en: https://www.clinicalmicrobiologyandinfection.com/article/S1198-743X(20)30575-9/fulltext.

[7] Silva P, Taborda A, Ahumada L, Fosco MJ, Fundación Favaloro. Asociación entre aislamiento social preventivo obligatorio e hipertensión arterial severa en el servicio de emergencias [Internet]. Buenos Aires: Comunicaciones SAC; 2020 [citado 21 Nov 2020]. Disponible en: https://www.sac.org.ar/trabajos-cientificos/asociacion-entre-aislamiento-social-preventivo-obligatorio-e-hipertension-arterial-severa-en-el-servicio-de-emergencias/.

[8] Unger T, Borghi C, Charchar F, et al. 2020 International Society of Hypertension Global Hypertension Practice Guidelines. Hypertension [Internet]. 2020 [citado 7 Nov 2020]; 75(6): 1334-1357. Disponible en: https://www.ahajournals.org/doi/10.1161/HYPERTENSIONAHA.120.15026.

[9] Salazar M, Barochiner J, Espeche W, Ennis I. COVID-19, hipertensión y enfermedad cardiovascular. Hipertensión y Riesgo Vascular [Internet]. 2020 [citado 10 Nov 2020]; 37(4): 176 – 180. Disponible en: https://www.sciencedirect.com/science/article/pii/S1889183720300659?via%3Dihub.https://doi.org/10.1016/j.hipert.2020.06.003.

[10] Mandeep RM, Sapan SD, SreyRam K, et al. Cardiovascular Disease, Drug erapy, and Mortality in Covid-19. N Engl J Med [Internet]. 2020 [citado 12 Nov 2020]; 382(25): e102. Disponible en: https://www.nejm.org/doi/pdf/10.1056/NEJMoa2007621?articleTools=true.

[11] Martínez-Pizarro S. Inhibidores del sistema renina-angiotensina en pacientes con COVID-19. Rev. cuba. cardiol. cir. cardiovasc. [Internet]. 2020 Abr [citado 25 Nov 2020]; 26(2): 1-2. Disponible en: http://revcardiologia.sld.cu/index.php/revcardiologia/article/view/973/pdf.

[12] Rico-Mesa JS, White A, Anderson AS. Outcomes in Patients with COVID-19 Infection Taking ACEI/ARB. Current Cardiology Reports. Springer [Internet] 2020 [citado 6 Nov 2020]; 22(5): 31. Disponible en: https://pubmed.ncbi.nlm.nih.gov/32291526/.

[13] Citro R, Pontone G, Bellino M, et al. Role of multimodality imaging in evaluation of cardiovascular involvement in COVID-19. Trends in Cardiovasc Med [Internet]. 2021 [citado 1 Ene 2021]; 31(1): 17-19. Disponible en: https://www.sciencedirect.com/science/article/abs/pii/S1050173820301249?via%3Dihub.

[14] Hidalgo P, Villaquirán C, Celis C, García OM, Bermúdez M. Primer informe Grupo Covid Evidencia 110520. Universitas Médica [Internet]. 2020 [citado 16 Nov 2020]; 61(3): 1-6. Disponible en: https://revistas.javeriana.edu.co/index.php/vnimedica/article/view/29781/24214.

[15] Giralt-Herrera A, Rojas-Velázquez JM, Leiva-Enríquez J. Relación entre COVID-19 e Hipertensión Arterial. Rev haban cienc méd [Internet]. 2020 [citado 6 Nov 2020]; 19(2): e_3246. Disponible en: http://www.revhabanera.sld.cu/index.php/rhab/article/view/3246/2493.

[16] López-Ponce de León JD, Cárdenas-Marín PA, Giraldo-González GC, Herrera-Escandón A. Coronavirus – COVID 19: Más allá de la enfermedad pulmonar, qué es y qué sabemos del vínculo con el sistema cardiovascular. Rev Colomb de Cardiol [Internet]. 2020 [citado 13 Nov 2020]; 27(3): 142-152. Disponible en: https://www.sciencedirect.com/science/article/pii/S0120563320300784?via%3Dihub.

[17] Guzik TJ, Mohiddin SA, Dimarco A, et al. COVID-19 and the cardiovascular system: implications for risk assessment, diagnosis, and treatment options. Cardiovasc Res [Internet]. 2020 [citado 8 Nov 2020]; 116(10): 1666-1687.

Disponible en: https://pubmed.ncbi.nlm.nih.gov/32352535/.

[18] Pallarés V, Górriz-Zambrano C, Morillas C, et al. COVID-19 y enfermedad cardiovascular y renal: ¿Dónde estamos? ¿Hacia dónde vamos? SEMERGEN [Internet]. 2020 [citado 11 Nov 2020]; 46(1): 78-87. Disponible en: https://www.sciencedirect.com/science/article/abs/pii/S1138359320301441?via%3Dihub.

[19] Choi M, Aiello EA, Ennis IL, Villa-Abrille MC. El SRAA y el SARS-CoV-2: el acertijo a resolver. Hipertensión y Riesgo Vascular [Internet]. 2020 [citado 21 Nov 2020]; 37(4): 169-175. Disponible en: https://www.sciencedirect.com/science/article/abs/pii/S1889183720300568?via%3Dihub.

[20] Piedra JS, Rodríguez EI, Cuellar CT, García AL. Protocolo de rehabilitación integral para pacientes posinfección al virus SARS-CoV-2 COVID-19. Rev Cubana de Med Fís y Rehabilit [Internet]. 2020 [citado 17 Nov 2020]; 12(3): 1-22. Disponible en: http://www.revrehabilitacion.sld.cu/index.php/reh/article/view/545/596.

[21] González MA. Bloqueadores del sistema renina-angiotensina: enemigos o amigos en pacientes con COVID-19. Rev Venez de Endocrinol y Metabol [Internet]. 2020 [citado 15 Nov 2020]; 18(1). Disponible en: https://www.redalyc.org/journal/3755/375563116002/html/index.html.

[22] Díaz-Barreiro LA, Cossio-Aranda J, Verdejo-Paris J, Odín-De los Ríos M, Galván-Oseguera H, Álvarez-López H, Alcocer-Gamba MA. COVID-19 y el sistema renina, angiotensina, aldosterona. Una relación compleja. Cardiovasc Metab Sci [Internet]. 2020 [citado 14 Nov 2020]; 31(S3). Disponible en: https://www.medigraphic.com/cgi-bin/new/resumen.cgi?IDARTICULO=93945.

[23] Puntmann VO, Carerj L, Wieters I, et al. Outcomes of Cardiovascular Magnetic Resonance Imaging in Patients Recently Recovered from Coronavirus Disease 2019 (COVID-19). JAMA Cardiol [Internet]. 2020 [citado 10 Nov 2020]; 5(11): 1265-1273. Disponible en: https://jamanetwork.com/journals/jamacardiology/fullarticle/2768916.