

# High Altitude Pulmonary Edema: About a Case

Edema pulmonar de las alturas. A propósito de un caso

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Pulmonary edema  
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## Palabras clave (DeCS)

Mal de altura  
Edema pulmonar  
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## Summary

High altitude pulmonary edema is a clinical condition that develops in individuals who have been in regions at sea level and who subsequently have a rapid ascent to altitudes above 2500 meters. We present the case of a 26-year-old male with clinical presentation and radiological findings typical of this entity, in whom coronavirus infection was initially suspected given the signs and symptoms in context of the current pandemic.

## Resumen

El edema pulmonar de las alturas es una condición clínica que se desarrolla en individuos que han estado en regiones a nivel del mar y que posteriormente ascienden rápidamente a altitudes por encima de los 2500 metros. Se describe el caso de un paciente de 26 años con cuadro clínico y hallazgos radiológicos típicos de esta patología, en quién inicialmente se sospechó infección por coronavirus dada la sintomatología y el contexto de pandemia actual.

## Introducción

High altitude pulmonary edema is a potentially fatal clinical condition that develops in individuals who have previously been in sea level regions, but rapidly ascend to altitudes above 2500 m.a.s.l. It usually occurs in healthy young adults with some individual susceptibility. It usually occurs in healthy young adults with some individual susceptibility.

This article presents the case of a 26-year-old man, with no relevant history, who consulted for a clinical picture of 24 hours of evolution consisting of productive cough, dyspnea and fever, after having direct contact with foreigners in La Guajira. The initial suspicion was COVID-19 infection given the context of the current pandemic, but the final diagnosis was high altitude pulmonary edema.

A chest X-ray was taken with the finding of non-confluent alveolar opacities that compromised the patient, and a chest CT scan was performed, which showed multiple alveolar opacities of predominantly peri-bronchovascular occupation, of “patchy” distribution located in both lung fields, some surrounded by “ground-glass” halo, with a tendency to consolidation at the bases (Figure 2). The results of other paraclinical tests performed, HIV test, serial smear microscopy and respiratory panel (FilmArray) were negative. Two days after admission, the patient’s symptoms improved significantly; the control chest X-ray showed almost complete resolution of the findings of the initial study (figure 3), so the clinical picture was considered compatible with high-altitude pulmonary edema.

## Case presentation

A 26-year-old male patient from La Guajira, with no relevant medical history, reported staying in a hostel and direct contact with foreigners. He was admitted to a local hospital in Bogota for clinical symptoms of 24 hours of evolution consisting of productive cough associated with asthenia, adynamia, respiratory distress and fever. Physical examination revealed rales in both lung bases with no other relevant findings. Paraclinical findings: leukocytosis and preserved renal function. Given the symptomatology and the epidemiological link, the patient was initially considered to have respiratory symptoms of viral etiology and a probable case for coronavirus infection (COVID-19).

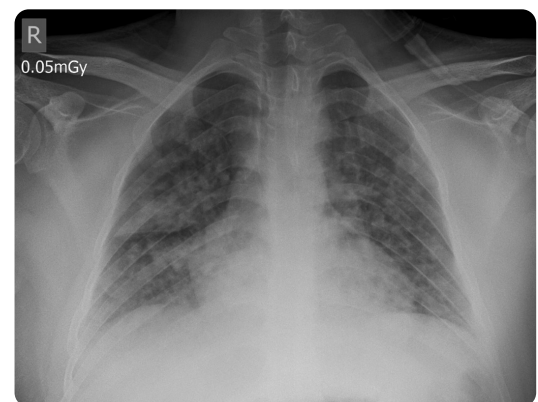


Figure 1. Initial chest X-ray. Alveolar occupation opacities, non-confluent, involving both lung fields.

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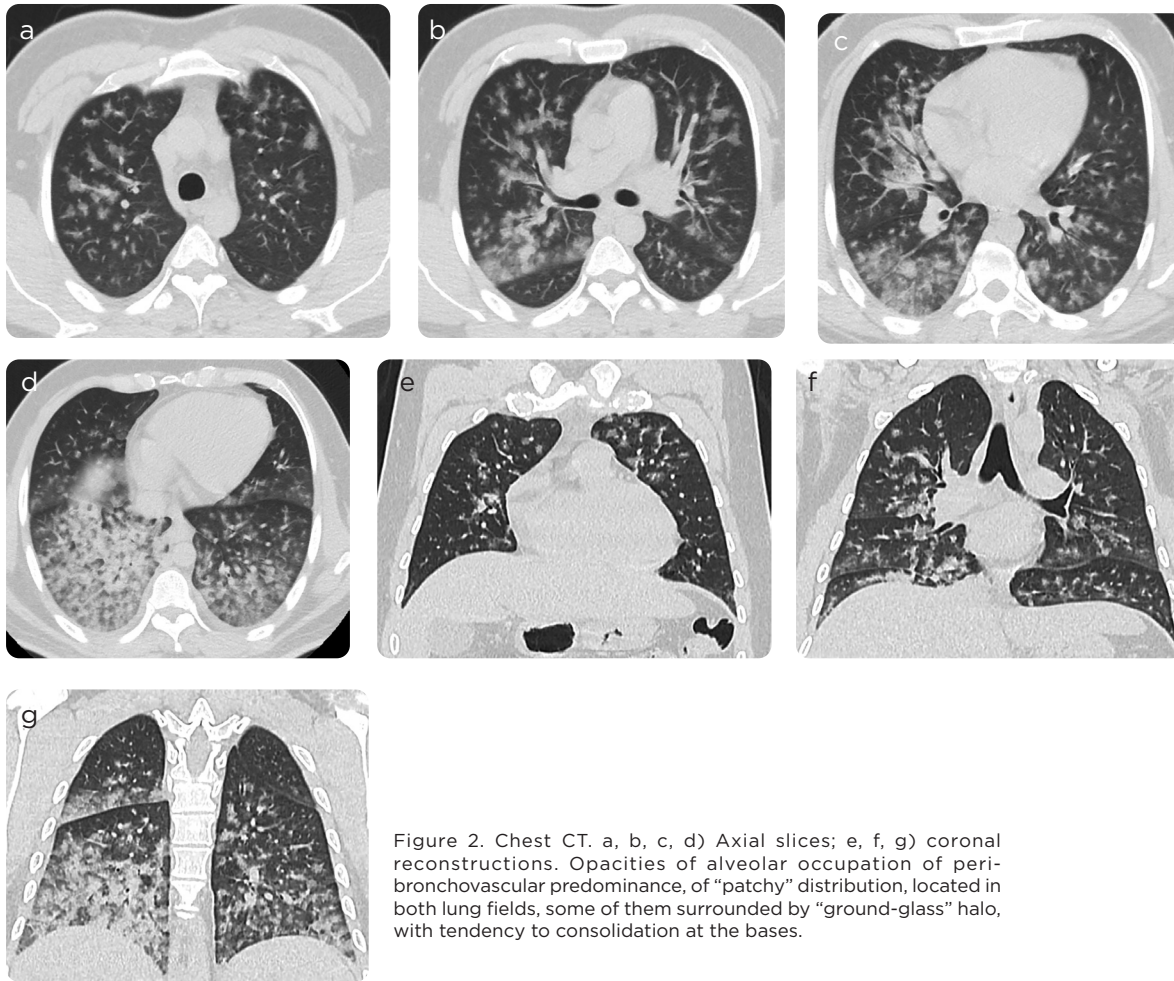


Figure 2. Chest CT. a, b, c, d) Axial slices; e, f, g) coronal reconstructions. Opacities of alveolar occupation of peribronchovascular predominance, of “patchy” distribution, located in both lung fields, some of them surrounded by “ground-glass” halo, with tendency to consolidation at the bases.

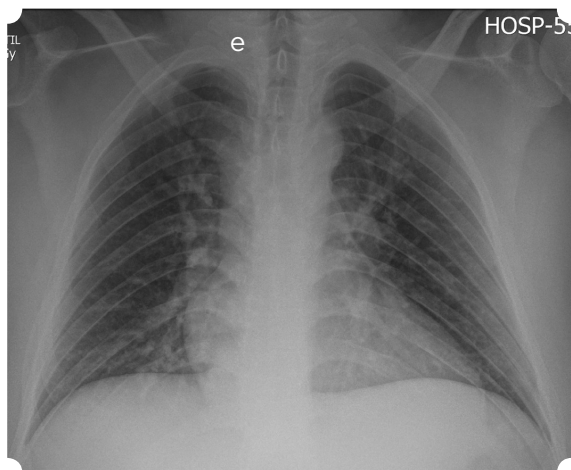


Figure 3. Chest X-ray control at 48 hours. Almost complete resolution of the alveolar opacities visualized in the initial study.

## Discussion

High altitude pulmonary edema is a potentially fatal entity that is triggered approximately 24–48 hours after rapid ascent to altitudes above 3000 m.a.s.l., and less frequently at moderate altitudes between 2500 and 3000 m. It usually develops in young male adults, and the risk of developing it is closely related to the speed of ascent, the altitude itself, and individual susceptibility (1). It usually develops in young male adults, and the risk of developing it is closely related to the rate of ascent, the altitude itself, and individual susceptibility (1). The risk of developing this disease is from 6% to 15% when the ascent is made in one or two days, respectively, and can even reach up to 60% in individuals with a history of pulmonary edema at high altitudes. The diagnostic approach is fundamental in its clinical course, because this entity can have a mortality rate close to 50% in untreated subjects (2, 3).

Pulmonary edema at high altitudes is part of the non-cardiogenic spectrum of this pathology; although its pathophysiology is controversial, a dysfunctional response to hypoxia at high altitudes is accepted as a theory. The triggering factor is considered to be an increase in mean pulmonary artery pressure above 35–40 mm Hg, which causes an alteration of the alveolocapillary membrane. This, coupled with intense vasoconstriction in response to prolonged exposure to a lower oxygen partial pressure environment, causes protein-rich and mildly hemorrhagic edema, but without diffuse alveolar damage. This process is rapidly reversible with descent or with supplemental oxygen administration (4, 5).

Pulmonary edema at high altitude corresponds to the extreme form of acute mountain sickness; although it may initially manifest with dry cough and dyspnea with exercise, it can rapidly evolve to dyspnea at rest, productive cough with pink and frothy sputum, cyanosis and even neurological involvement due to associated cerebral edema. Physical examination may identify tachycardia, tachypnea, low-grade fever and desaturation (1, 5).

In imaging studies it is common to find findings that simulate an underlying infectious process; however, a clinical-radiological dissociation is essential to establish the definitive diagnosis. Chest radiographic findings vary according to the degree of hypoxemia; initially bilateral patchy alveolar opacities of central predominance are described, which with the evolution of the disease become confluent with diffuse involvement, as observed in the case presented. In tomography, the appearance of ground-glass and areas of multilobar consolidation with diffuse involvement of both lung fields is described with the progression of the disease (5).

The main differential diagnosis is pneumonia, especially in older adults with comorbidities in whom the diagnosis of this entity should be by exclusion. However, pulmonary edema due to other etiology, both cardiogenic and non-cardiogenic, alveolar proteinosis and diffuse alveolar hemorrhage, are part of the wide spectrum of differential diagnoses; however, the diagnosis of pulmonary edema at high altitudes is considered in healthy patients with a characteristic clinical history (5).

In a patient with high altitude pulmonary edema, saturation levels are rapidly corrected with oxygen administration. The imaging findings described above associated with a clinical history of rapid ascent above 2500 m.a.s.l. allow the diagnosis of this pathology to be established with certainty (pathognomonic); its timely recognition guarantees a timely intervention and favors a good outcome (5).

## Conclusion

The initial approach to the diagnosis of high-altitude pulmonary edema is clinical, but images in a specific clinical context establish its diagnosis. Recognizing this entity is essential to improve the outcome of patients, because if treatment is not established in half of the cases it can be death, as opposed to a timely diagnosis and treatment that ensures a rapid and complete recovery in most cases.

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