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CASE REPORT

High Altitude De-Acclimatization Syndrome (HADAS): A case report

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ABSTRACT

High Altitude De-acclimatization Syndrome (HADAS) is a clinical entity that arises when individuals return abruptly from high-altitude environments to lower altitudes without undergoing the necessary acclimatization processes. While altitude-related illnesses have been extensively studied, HADAS remains relatively underreported and may pose diagnostic challenges due to its varied and nonspecific clinical presentation. This case report aims to shed light on the importance of recognizing HADAS as a potential consequence of inadequate acclimatization, emphasizing the need for a thorough understanding of altitude-related disorders in individuals with a history of rapid altitude changes.

Keywords: *Acetazolamide, de-acclimatization, high altitude, hypoxemia.*

INTRODUCTION

High Altitude De-acclimatization Syndrome (HADAS) occurs following abrupt return of individuals from high-altitude to lower altitudes without undergoing necessary acclimatization processes.[1]

CASE REPORT

A 38-year-old male presented with a chief complaint of headache and insomnia persisting for the previous three days. He is a resident of Ahmedabad city in western part of India, which is 53 meters or 174 feet above sea level. A week before presentation he undertook a pilgrimage tour to different levels of higher altitudes with rapid ascent and descent by air-travel (flights and helicopter service) during major part of the journey to Badrinath (3300 m; 10827 ft) and Kedarnath (3583 m; 11755 ft) in Northern India, and Muktinath (3710 m; 12172 ft) in Nepal. After the whirlwind tour, in Ahmedabad, he started having throbbing occipital headache and sleep disturbance. His self-monitoring revealed fluctuating oxygen saturation levels (SpO_2) between 86% and 96%. After 3 days of suffering on and off headache and disturbed sleep he consulted physician. Despite being hemodynamically stable with a clear chest and cardiovascular examination, the patient's symptoms prompted an extensive diagnostic workup.

Investigations, including echocardiography, pulmonary function tests, and diffusion capacity (DLCO) assessment, yielded normal results, ruling out common pulmonary or cardio-vascular causes of hypoxemia. Arterial blood gas analysis demonstrated a pH of 7.42, PaO_2 of 90 mm Hg, and $PaCO_2$ of 32 mm Hg on room air, indicative of normoxemia. A detailed travel history brought to light that the patient went up by helicopter, did not take acetazolamide, stayed at different high altitudes for total 3 days and came down by helicopter, leading to insufficient de-acclimatization to high altitude, raising suspicion for HADAS. During stay at different higher altitude locations the person didn't experience any significant problem and hence SpO_2 was not monitored. Upon returning to Ahmedabad the problems started and he self-monitored his SpO_2 which was fluctuating between 86 to 96%.

The diagnostic journey involved a systematic approach to exclude potential causes of hypoxemia. The absence of pathological hypoxemia on arterial blood gas analysis, coupled with the patient's travel history, led to the consideration of HADAS as the primary aetiology. This diagnosis was further supported by the patient's symptoms and the rapid ascent and descent without adequate acclimatization.

The patient received a therapeutic intervention consisting of acetazolamide 250 mg twice daily for two days. It is a diuretic with carbonic anhydrase inhibitory properties known to aid in acclimatization. Concurrently, hydration and electrolyte supplementation were initiated to address potential imbalances. The patient's symptoms improved rapidly following the administration of acetazolamide, and his SpO_2 levels stabilized at 95%.

DISCUSSION

HADAS represents a unique challenge in the realm of altitude-related disorders. The mechanism underlying High Altitude De-acclimatization Syndrome is rooted in the intricate interplay between environmental changes and the body's physiological response to high altitudes. Unlike more widely recognized conditions such as acute mountain sickness (AMS) or high-altitude pulmonary oedema (HAPE), HADAS may not present with overt symptoms immediately upon descent.[2] Instead, its manifestations, as illustrated in this case, may include headache, insomnia, and fluctuating oxygen saturation levels.[3] The diagnostic journey underscores the importance of a detailed travel history in patients presenting with such symptoms, especially in those with recent high-altitude exposure.

One key aspect involves the process of acclimatization, wherein the body gradually adjusts to lower oxygen levels at higher altitudes. This adaptation includes increased ventilation, changes in blood flow distribution, and alterations in red blood cell production. Prolonged exposure to high altitudes allows for these adjustments to occur, optimizing oxygen delivery to tissues.[4]

However, when individuals abruptly return to lower altitudes without affording the body sufficient time to undergo this acclimatization process, a state of de-acclimatization ensues. The abrupt descent hampers the body's ability to cope with the sudden shift in oxygen levels, leading to a mismatch between the physiological adaptations acquired at high altitudes and the oxygen-rich environment at lower altitudes.[5]

Furthermore, the role of hypoxia-inducible factor-1 (HIF-1) in HADAS cannot be understated. HIF-1 is a transcription factor that orchestrates cellular responses to low oxygen levels. In high-altitude environments, sustained hypoxia triggers HIF-1 activation, promoting adaptive changes. However, when individuals rapidly descend to lower altitudes, HIF-1 continues to be upregulated, contributing to an array of symptoms associated with HADAS.[6]

The pathophysiological cascade also involves alterations in cerebral blood flow and vasoreactivity. Rapid descent disrupts the delicate balance achieved at high altitudes, potentially leading to cerebral vasodilation and increased permeability of the blood-brain barrier. This, in turn, may contribute to the development of symptoms such as headache and insomnia.

The prompt resolution of symptoms observed in our patient following acetazolamide administration aligns with the medication's mechanism of action. Acetazolamide, a carbonic anhydrase (CA) inhibitor, induces a metabolic acidosis that mimics the acid-base changes associated with acclimatization.[7]

This pharmacological intervention aids in the restoration of acid-base balance and facilitates the correction of respiratory alkalosis induced by hyperventilation at high altitudes.

High Altitude De-acclimitaziation Syndrome (HADAS) is a diagnosis of exclusion. There was a strong suspicion from his travel history to different higher altitudes and rapid descent. His clinical examination, blood reports and imaging studies (chest Xray, CT head and chest, Lung perfusion scan) was unremarkable except for hypoxemia in pulse oximeter reading.

He responded quickly with rest, hydration and acetazolamide therapy. Recognition of HADAS is essential for timely intervention and prevention of potential complications. Acetazolamide, by promoting renal excretion of bicarbonate, facilitates metabolic acidosis, thereby aiding in acclimatization and symptom relief. This case emphasizes the need for healthcare providers to remain vigilant to altitude-related disorders, even in the absence of severe hypoxemia or overt clinical signs, particularly in individuals with suboptimal acclimatization practices. Further research is warranted to enhance our understanding of HADAS and refine its management strategies.

To conclude, the presented case illuminates the significance of considering High Altitude De-acclimatization Syndrome (HADAS) in individuals returning from high-altitude environments without adequate acclimatization. This clinical entity, though less recognized, can manifest with subtle yet impactful symptoms, such as headache, insomnia, and fluctuating oxygen saturation levels. The successful management of our patient underscores the importance of a comprehensive diagnostic approach, encompassing a detailed travel history and exclusion of other potential causes of hypoxemia. Recognition of HADAS allowed for timely intervention with acetazolamide, resulting in a rapid resolution of symptoms and the restoration of oxygen saturation levels to normalcy.

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