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Acute and Chronic High Altitude Maladaptation Disorders: Pulmonary Hypertension

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Abstract

The most noticeable feature of a high altitude environment is alveolar hypoxia, which has well-known repercussions for the cardio-pulmonary system, including the development of pulmonary hypertension. High altitude pulmonary edema (HAPE), a life-threatening condition that occurs at high elevations in non-acclimatized healthy humans, is caused by pulmonary hypertension caused by excessive hypoxic pulmonary vasoconstriction. Despite a strong physiologic rationale for utilising vasodilators in the prevention and treatment of HAPE, there have been no rigorous trials of their efficacy to far. Based on considerable clinical experience with nifedipine in HAPE prevention in susceptible persons, calcium-channel blockers are now suggested for pharmacological prophylaxis in high-risk individuals with a clear history of recurrent HAPE. Hypoxia causes pulmonary vascular remodelling and the development of pulmonary hypertension, which increases the pressure inside the lungs. Chronic mountain sickness, another high-altitude maladaptation illness, may be complicated by pulmonary hypertension and severe erythrocytosis. Other factors, such as chronic heart and lung disorders, thrombotic or embolic diseases, may potentially underpin and/or contribute to pulmonary hypertension at high altitude. Extensive clinical experience with medicines in patients with pulmonary arterial hypertension shows that they could be used to treat pulmonary hypertension at high altitudes. Small studies have shown that they can lower pulmonary artery pressure in those who live at high altitudes. To far, no medicines for the treatment of chronic high altitude pulmonary hypertension have been approved. This paper presents a review of the literature on the involvement of pulmonary hypertension in the pathophysiology of acute and chronic high altitude maladaptation illnesses, as well as a summary of current knowledge regarding potential treatment options..

A considerable fraction of the human population has lived in high-altitude environments such as the Andes, Tibet, Ethiopian highlands, Pamir, and Tian-Shan mountains. Furthermore, the number of individuals travelling to high elevations for economic or recreational reasons has been steadily increasing over the previous few decades. High altitude is one of the most challenging extreme settings, with numerous obstacles. The most common is alveolar hypoxia, which has well-known repercussions for the cardio-pulmonary system, including the development of pulmonary hypertension. High altitude pulmonary edema (HAPE) is a life-threatening condition that occurs at high altitudes in non-acclimatized healthy individuals due to acute pulmonary hypertension caused by increased hypoxic pulmonary vasoconstriction (HPV). Chronic exposure to high altitude hypoxia causes pulmonary vascular remodelling and the development of long-term pulmonary hypertension, which increases the pressure load on the right ventricle and leads to right heart failure and death. This paper presents a review of the research on the involvement of pulmonary hypertension in the development of acute and chronic high altitude maladaptation syndromes, as well as a summary of current treatment options. The pulmonary circulation's response to alveolar hypoxia is known as HPV. Acute HPV is thought to be an adaptive response of the pulmonary circulation to regional alveolar hypoxia, in which blood flow is diverted from poorly ventilated to optimally ventilated lung segments, optimising ventilation-perfusion matching and gas exchange. Acute HPV is induced by local alveolar hypoxia and is restricted to the afflicted lung segments. Local HPV does not cause an increase in pulmonary artery pressure, which is important to note (PAP). HPV, on the other hand, is involved in the entire pulmonary circulation under global alveolar hypoxia, which occurs at high altitude, resulting in increased pulmonary vascular resistance and an increase in PAP. HPV's exact mechanisms are yet unknown. Nonetheless, the oxygen sensing and signal transduction machinery of the pulmonary precapillary vessels is well known. Reactive oxygen species produced by mitochondria are thought to be important mediators in HPV infection, according to mounting evidence.

Importance of research

Hypertension is the world's most common chronic disease, and a better understanding of its origin, prevention, and therapy, as well as its comorbidities, is critical. Animal models of hypertension have been and will continue to be very beneficial in offering insights into the pathophysiology of hypertension as well as novel therapeutic possibilities. Clearly, investigators must make well-informed decisions about which animal model to use for a given application, and experiments must be carefully planned, carried out, and interpreted. We summarise a few key points in this scientific statement that are especially relevant for those working in the field and may help propel it forward.

Biography

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Akylbek Sydykov has her expertise in evaluation and passion in improving the health and wellbeing. He developed his research in Department of Internal Medicine, Excellence Cluster Cardio-Pulmonary Institute (CPI), Justus Liebig University of Giessen, Aulweg 130, 35392 Giessen, Germany; Member of the German Center for Lung Research (DZL), Department of Internal Medicine, Excellence Cluster Cardio-Pulmonary Institute (CPI), Justus Liebig University of Giessen, Aulweg 130, 35392 Giessen, Germany;

Importance of institute and laboratory

Giessen is a town north of Frankfurt in central Germany. The Mathematikum, a math museum with hands-on experiments, is well-known. The Liebig Museum, next next door, features chemistry exhibits as well as the lab of 19th-century scientist Justus von Liebig. The Botanical Garden, which is part of the University of Giessen, was founded in 1609 and houses hundreds of plant varieties. Burg Gleiberg castle, to the northwest, offers a panoramic view of the town.

The University of Giessen is a significant public research university in Giessen, Hesse, Germany. Its official name is Justus Liebig University Giessen[3] (German: Justus-Liebig-Universität Gießen). Justus von Liebig, the pioneer of modern agricultural chemistry and inventor of artificial fertiliser, is its most famous faculty member. Arts/humanities, business, dentistry, economics, law, medicine, science, social sciences, and veterinary medicine are all included. Its university hospital, which has two The University of Giessen is one of the oldest higher education institutions in German-speaking countries. Because the all-Hessian Landesuniversität (the adjacent University of Marburg (Philipps-Universität Marburg) in Marburg, Hesse-Kassel (or Hesse-Cassel)) had become Reformed, it was created in 1607 as a Lutheran university in Giessen, Hesse-Darmstadt (that is, Calvinist). At October 1607, Louis V, Landgrave of Hesse-Darmstadt, who gave the university its original name "Ludoviciana," established his own institution of higher study in Giessen. locations in Giessen and Marburg (the latter of which is the University of Marburg's teaching hospital), is Germany's sole private university hospital.



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