

Comments on “High Altitude Pulmonary Edema in an Experienced Mountaineer. Possible Genetic Predisposition”

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Whitlow K, Davis B. High Altitude Pulmonary Edema in an Experienced Mountaineer. Possible Genetic Predisposition. *West J Emerg Med.* 2014;15(7):849–851.

To the Editor:

I read with interest the case report by Whitlow and Davis in the November 2014 issue of the Western Journal of Emergency Medicine regarding management of high altitude pulmonary edema (HAPE) in an experienced mountaineer.¹ The authors have appropriately highlighted the need of descent and supplemental oxygen for treating HAPE, a potentially fatal disease if left untreated. The patient in this study, a 25-year-old sea-level resident, was diagnosed as a case of HAPE on the basis of history of acute ascent to 3200m and onset of symptoms and signs suggestive of HAPE within 72 hours of high altitude exposure. He was treated with 100% oxygen, albuterol and ipratropium nebulizers, inhaled and intravenous dexamethasone, intravenous hydralazine, and intravenous furosemide. Subsequently, with improvement of symptoms, he was continued on intravenous dexamethasone. However, as per the Wilderness Medical Society (WMS) evidence-based guidelines of 2009 for clinicians for prevention and treatment of acute altitude illness, diuretics have no role to play in the treatment of HAPE, as these patients are likely to have co-existing intra-vascular volume depletion.² Moreover, dexamethasone is not recommended for treatment of all cases of HAPE and it has only a preventive role in HAPE-susceptible individuals.² Analysis of the history and examination of this patient reveals that a differential diagnosis of asthma was considered along with HAPE. As a reader, I was inquisitive to know if an X-ray facility was available at the community academic emergency department, and if so, an X-ray chest of the patient on arrival would have helped in confirming the diagnosis of HAPE.

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In reply:

We appreciate the letter to the editor and are pleased to respond regarding our recent case study regarding high altitude pulmonary edema in an experienced mountaineer. The letter raises some valid questions regarding our treatment decisions. With this, as with most emergency department (ED) patients, it must be understood that the initial treatment reflected the breadth of our differential diagnosis. The patient was receiving nebulizers as he was wheeled into the department for evaluation for a possible asthmatic condition. An initial chest x-ray in the ED revealed “multiple nodular opacities” and our bedside read could not exclude bilateral pulmonary edema of unknown etiology. Although retrospectively the patient’s history is consistent with High-altitude pulmonary edema (HAPE), for a patient with continued low oxygen saturation despite supplemental oxygen, diffusely coarse breath sounds, and a broad differential it seemed appropriate for a trial of Furosemide for hypervolemic causes of his apparent pulmonary overload as he was not hypotensive. Regarding his continued treatment outside of our department, we are unable to comment specifically given that we were his emergency providers, but again, continuing Dexamethasone for the possibility that he may have been experiencing some degree of reactive airway or an asthmatic response seems relatively low risk with significant potential gains. Given the patient’s rapid response to treatment during his very brief stay his differential was readily narrowed and he was discharged home in excellent condition after a very short stay. This case report was focused on the possible familial or genetic predisposition to HAPE and not intended as a complete review of the prevention and treatment of altitude related illnesses. Perhaps this was a limitation of our manuscript. We are pleased that our case report was read with such interest and welcome further discussion at any time.

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