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Case Report

Susceptibility - weighted imaging: A valuable diagnostic tool for early detection of high-altitude cerebral edema: A case report ☆

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ABSTRACT

High altitude cerebral edema (HACE) is a clinical spectrum of high-altitude illness. The working diagnosis of HACE should be based on the history of rapid ascent with signs of encephalopathy. Magnetic resonance imaging (MRI) can be crucial in the timely diagnosis of the condition. A 38-year-old female was airlifted from Everest base camp due to sudden onset of vertigo and dizziness. She had no significant medical or surgical history, and routine laboratory tests showed normal results. MRI was performed, which showed no abnormalities except for the detection of subcortical white matter and corpus callosum hemorrhages on susceptibility-weighted imaging (SWI). The patient was hospitalized for 2 days and treated with dexamethasone and oxygen, and had a smooth recovery during follow-up. HACE is a serious and potentially life-threatening condition that can occur in individuals who rapidly ascend to high altitudes. MRI is a valuable diagnostic tool in the evaluation of early HACE, and can detect various abnormalities in the brain that may indicate the presence of HACE, including micro-hemorrhages. Micro-hemorrhages are tiny areas of bleeding in the brain that may not be visible on other MRI sequences but can be detected on SWI. Clinicians especially radiologists, should be aware of the importance of SWI in the diagnosis of HACE, and ensure that it is included in the standard MRI protocol for evaluating individuals with high altitude-related illnesses for early diagnosis and appropriate treatment to prevent further neurological damage and improve patient outcomes.

Abbreviations: AMS, Acute Mountain Sickness; CT, Computed Tomography; MRI, Magnetic Resonance Imaging; HACE, High Altitude Cerebral Edema; SWI, Susceptibility Weighted Imaging.

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Introduction

High-altitude illness (HAI) encompasses acute mountain sickness, high-altitude pulmonary edema (HAPE), and high-altitude cerebral edema (HACE) that occur in an individual living at low altitude following a rapid ascent to higher altitude without acclimatization [1]. Clinically acute mountain sickness and HACE represent a continuum of the cerebral form of HAI, with HACE being the end stage of the spectrum [2]. Common symptoms of acute mountain sickness are headache, nausea, vomiting, loss of appetite, fatigue or malaise (particularly at rest), sleep disturbance, and dizziness/lightheadedness [3]. The onset of general neurological signs such as encephalopathy and ataxia signifies the transition from AMS to HACE. HAPE and HACE are a life-threatening emergencies requiring immediate treatment, with a descent to lower altitude (or higher pressure artificial environment) as quickly as can be safely arranged and executed [3]. The working diagnosis of HACE is made with a history of recent ascent and signs of encephalopathy [4]. Cerebral edema is demonstrated in MRI and usually shows intense T2 and fluid-attenuated inversion recovery (FLAIR) signals in the white mater, especially the splenium of the corpus callosum, however there may be exceptions. In this case report, we present a case of HACE and discuss the relevance of susceptibility weighted imaging (SWI) sequence in the timely diagnosis of the condition.

Case presentation

The patient in our case is a 38-year-old female of Chinese nationality who presented with symptoms of vertigo and dizziness for 1 day. She had been rescued by helicopter from Everest base camp, where she had spent 5 days ascending to an altitude of 5364 meters. The patient reported a history of on-and-off dizziness, headache, and vomiting lasting for 3 hours but denied experiencing any shortness of breath, cough, palpitations, or chest pain. Her medication history includes the use of ibuprofen for the treatment of headache, but it was not successful. The patient denied consuming tobacco and alcohol. Her medical and surgical histories were unremarkable.

Upon examination, the patient was found to be tachypneic and tachycardic, with a saturation of 92% while receiving oxygenation via nasal prongs. The physical exam did not reveal any notable abnormalities. The results of her complete blood count (CBC), basic metabolic panel (BMP), lipid panel, liver enzyme panel, and thyroid-stimulating hormone (TSH) test were all within normal limits.

On further evaluation with MRI brain shows normal in T2 weighted, FLAIR- weighted, T1 weighted and diffusion-weighted imaging sequences. (Figs. 1-3). The only positive finding was in SWI sequence (Figs. 4-6) which showed micro-hemorrhages in subcortical white matter and the corpus callosum.

Following the imaging findings, diagnosis of HACE was confirmed and the patient was admitted to the hospital and treated with dexamethasone 8mg intravenously every 6 hours for 2 days and supplemental oxygen (2L/min) via nasal prongs. The patient was clinically improved after the second day of hospitalization and discharged on oral dexamethasone every 6 hours for 7 days. At the follow-up appointment 1 week later, the patient did not experience any complications.

Discussion

Acute mountain sickness (AMS) is a self-limiting illness that can progress to HACE, which is a potentially fatal neurologic syndrome. The diagnosis of AMS is made clinically, as no diagnostic modalities or physical findings can reliably confirm it. The Lake Louise Consensus Committee definition of AMS

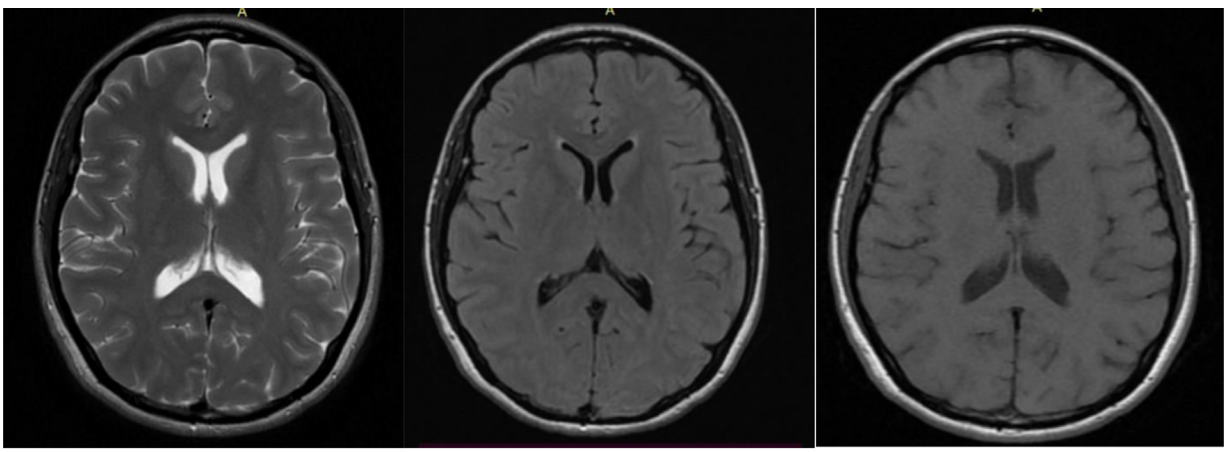

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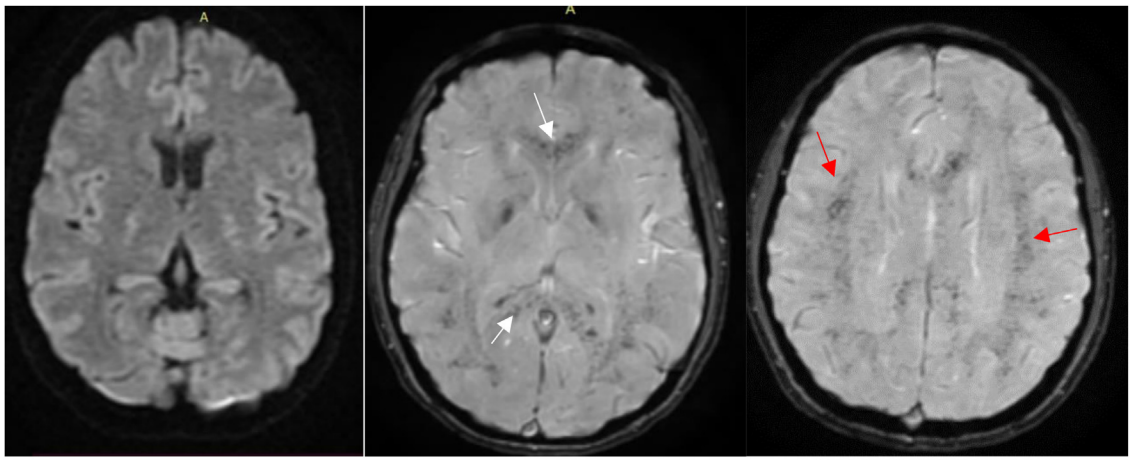
Fig. 1–3 – Axial MRI T2 (1), FLAIR (2) and T1 WI show normal signal intensity. No focal lesions were seen.



4 5 6

Fig. 4-6 – (4) Axial DWI sequence appears normal. No areas of restriction were seen. (5, 6) Axial SWI sequences show punctate blooming foci in the corpus callosum (white arrows) and subcortical white matter (red arrows).

includes headache and one or more of the following: anorexia, nausea, or vomiting; fatigue or weakness; dizziness or lightheadedness; or difficulty sleeping [5].

AMS typically presents within 24 hours after ascent to a new altitude and usually resolves within 2-3 days at a consistent altitude [2]. HACE generally occurs after 2 days above 4000 m but can occur at lower elevations (2500 m) and with a faster onset. Some individuals may suffer from symptoms of AMS before transitioning to HACE, and some may also have concomitant HAPE. The absence of concomitant HAPE or symptoms of AMS prior to deterioration does not rule-out the presence of HACE, which can occur in isolation, but is rare [4].

Risk factors for developing HACE include a prior history of high-altitude illness, a lack of acclimatization, heavy physical exertion and an abrupt ascent from lower altitudes [4]. The working diagnosis of HACE is made with a history of recent ascent and signs of encephalopathy [4]. HACE is characterized by truncal ataxia, decreased consciousness, and usually a mild fever. Headaches that are poorly responsive to nonsteroidal anti-inflammatory drugs (NSAIDs) and vomiting indicate probable progression of AMS to HACE [6].

It is important to exclude other disorders that may have signs and symptoms overlapping with HACE, such as dehydration, hypoglycemia, hypothermia, or hyponatremia. Early symptoms may be misinterpreted as exhaustion. Though rarely available, laboratory testing may show an elevated white blood cell count in the setting of HACE [4].

Without appropriate treatment of HACE, coma may evolve rapidly, followed by death from brain herniation within 24 hours [6].

CT may show cerebral edema, but MRI is a better study to evaluate for more subtle early signs of edema [4]. Magnetic resonance imaging in patients with HACE shows vasogenic edema and microhemorrhages that are located predominantly in the corpus callosum. Vasogenic edema is characteristically evident as an intense T2 and fluid-attenuated inversion recovery (FLAIR) signal in the white mater, especially the splenium of the corpus callosum, with no gray mater lesion [7,8]. In our particular case, the MRI results showed normal findings in T1, T2, FLAIR, and DWI sequences. The cause of these findings could be that the initial vasogenic and cytotoxic edema which is seen typically in the early period of HACE has resolved and progressed to the phase of microvascular disruption and microbleed [9].

The SWI sequence revealed evidence of microhemorrhage in the subcortical white matter of the frontal, parietal, and occipital lobes, as well as within the corpus callosum.

Small foci of blood or hemosiderin deposits frequently indicate hemorrhages, particularly those due to microvascular pathology or traumatic brain injury. These tiny lesions can be challenging to identify using traditional MRI sequences due to their low signal intensity and proximity to surrounding tissues. SWI is specifically designed to enhance the visibility of these small hemorrhagic lesions. By exploiting the susceptibility effects caused by paramagnetic substances such as deoxyhemoglobin and hemosiderin, SWI increases the sensitivity to blood products, making them appear as hypointense signals against the background tissue. This improved contrast resolution helps differentiate even tiny hemorrhages from the surrounding normal brain tissue. Susceptibility-Weighted Imaging (SWI) is a powerful 3D MRI sequence that plays a crucial role in the detection of small hemorrhages and calcifications, overcoming the limitations of conventional sequences. SWI takes advantage of the interaction between paramagnetic, diamagnetic, and ferromagnetic compounds with the local magnetic field, resulting in a phase alteration and signal change in the surrounding tissue. This unique capability enables SWI to enhance the visibility of subtle hemorrhagic lesions that may be missed by other techniques [10].

Microhemorrhage in subcortical white matter can be seen in conditions like diffuse axonal injury (DAI), trauma-related fat embolism, and cerebral amyloid angiopathy (CAA) in elderly patients [11,12]. Accurate diagnosis requires ruling out trauma, considering clinical context, and investigating the

patient's medical history and physical examination for insights into the underlying cause [13].

HACE is a critical medical condition that requires prompt medical intervention. The treatment for HACE involves administering dexamethasone and descending from high altitudes. If the body's oxygen level drops below 90%, immediate oxygen therapy should be initiated. The use of a portable hyperbaric chamber can offer temporary symptom relief and facilitate the transfer of patients to a medical facility. To prevent HACE, acetazolamide should be administered prior to ascending to high altitudes, and a gradual ascent should be maintained [14].

Conclusion

Microhemorrhages visible on MRI through the SWI sequence can serve as an early indicator of HACE. Identifying these micro-hemorrhages should be a cause for concern for clinicians, as they may require prompt and aggressive management of the underlying cause. Radiologists, in particular, should be diligent in evaluating the SWI sequence when assessing patients suspected of having HACE, as this can help ensure timely diagnosis and appropriate treatment.

Patient consent

Written informed consent was obtained from the patient for publication of this case report and accompanying images. A copy of the written consent is available for review by the Editor-in-Chief of this journal on request.

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