High-Altitude Cerebral Edema manifesting as T2/FLAIR Hyperintensity and Microbleeds in the White Matter on MRI Brain

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Abstract

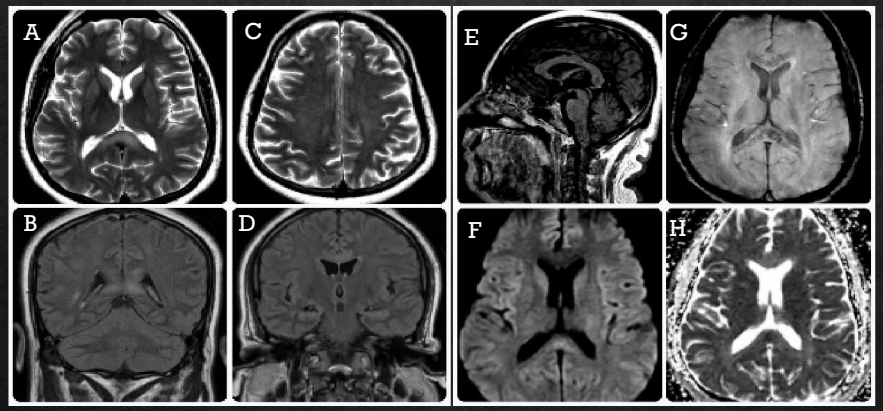
High-altitude cerebral edema (HACE) is a rare type of acute mountain illness characterized by consciousness disruption and truncal ataxia. Here we discuss a 40-year-old non-diabetic, non-smoker male who went on a tour to Nanga Parbat. On returning home, the patient developed symptoms of headache, nausea, and vomiting. Subsequently, he developed lower limb weakness and shortness of breath. CT chest was interpreted as Covid pneumonia despite being Covid PCR negative his MRI brain revealed T2/FLAIR hyperintense and T1 hypointense signals in the bilateral semi-oval centrum, posterior periventricular white matter, and corpus callosum genu, body, and splenium. These abnormal signals were discovered to be more evident in the corpus callosum's splenium. Moreover, susceptibility-weighted imaging revealed micro hemorrhages in the corpus callosum (SWI). This verified the diagnosis that the patient is suffering from HACE. Within five days, his symptoms resolved and he was discharged with full recovery.

Introduction

Living at sea level while traveling to high altitudes could have dire consequences if adequate measures are not taken on time. At higher altitudes, our body is exposed to low atmospheric pressure that results in a low partial pressure of oxygen which may impede adaptation to high-altitude regions(1). Although acclimatization is a natural physiological adaptation to certain environmental situations, the body requires a certain time to adapt to a new environment. High-altitude cerebral edema (HACE) is a rare type of acute mountain illness characterized by consciousness disruption and truncal ataxia(2). These symptoms are caused by vasogenic edema, and microbleeds in the white matter and corpus callosum are frequent MRI findings(3). Sometimes, verbal and visual memory deficits can be identified in a patient with corpus callosum splenium lesions(4).

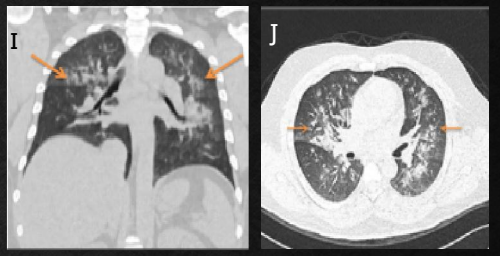
Case Presentation

A 40-year-old nondiabetic, nonsmoker adult male went on a tour to Nanga Parbat (3,850 m to 8,126 m in height) situated in the higher altitudes of northern Pakistan. On returning home the patient suddenly experienced fever, vomiting, nausea, and headache. He visited local hospital where his routine workup was performed, during his hospital stay he developed lower limb weakness and shortness of breath. CT chest was performed which revealed bilateral perihilar and smooth interlobular septal thickening which was diagnosed as Covid pneumonia despite negative PCR. When his health further deteriorated he came to our hospital where his routine workup was performed including mri brain and whole spine. MRI brain revealed T2/FLAIR hyperintense and T1 hypointense signals in the bilateral semi-oval centrum, posterior periventricular white matter, and corpus callosum genu, body, and splenium. , susceptibility-weighted imaging revealed micro hemorrhages in the corpus callosum (SWI). This verified the diagnosis that the patient is suffering from HACE.



A,B,C,D: T2/FLAIR Hyperintense signals in the periventricular deep white matter and the genu, splenium and splenium of the corpus callosum

E: T1 hypointense signals in the genu, body and splenium of the corpus callosum. **F:** Diffuse micro hemorrhages in the corpus callosum on SWI. **G,H:** Diffusion restriction in the splenium of corpus callosum.



I,J: Bilateral perihilar airspace opacification with smooth interlobular septal thickening representing interstitial pulmonary edema.

Discussion

HACE is described as an end-stage acute high-altitude illness. At high altitudes, the "partial pressure of the oxygen" is 13.3kPa (68%) at 3000 m height while at sea level it is 19.6kPa.(5,6) This difference in partial pressure disrupts the autoregulatory vascular system in non-acclimatized individuals. Additionally, the reduced partial pressure of oxygen at extreme heights induces vasodilation followed by transient failure of the autoregulatory mechanism making an increase in the capillary hydrostatic pressure causing cerebral edema with further injury to the blood-brain barrier(7). Studies have proposed two theories regarding the pathogenesis of HACE. These theories include vasogenic and cytotoxic edema. Vasogenic edema occurs due to the release of multiple neurohormones responsible for hemodynamic instability including nitric oxide, free radicals, growth factors, and cytokines. All these neurohormones collectively disrupt the cerebral blood-brain barrier and cause cerebral edema.Cytotoxic edema occurs due to disruption of the Na+/K+ ATPase pump caused by inadequate oxygen supply to the tissues(8). Reduced oxygen supply causes tissue injury by free radicals formation. This breach in the blood-brain barrier results in extensive microhemorrhages which have a predilection of corpus callosum(8).

Conclusion

MRI brain findings alone are insufficient for the diagnosis of HACE, adequate clinical history must be obtained in reaching the accurate diagnosis.

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