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CASE REPORT

Point-of-Care Ultrasound Diagnosis of Acute High Altitude Illness: A Case Report

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With the advent of high-quality portable ultrasound machines, point-of-care ultrasound (POCUS) has gained interest as a promising diagnostic tool for patients with high altitude illness. Although POCUS is used successfully in hospital environments to detect interstitial pulmonary edema and increased intracranial pressure, the relationship between specific sonographic criteria and high altitude illness is still unclear. We report the case of a healthy 32-y-old male who developed acute respiratory distress and neurologic impairment at 4321 m while participating in a high altitude medical research expedition. We discuss the potential of POCUS to diagnose acute high altitude illness by lung ultrasound, optic nerve sheath diameter measurement, and echocardiography. Ultrasound in combination with clinical findings helped us to exclude relevant differential diagnoses, start on-site treatment, and organize an evacuation. We used serial clinical and ultrasound examinations to assess the patient over time. Although its role in high altitude medicine needs further investigation, we believe that POCUS can be a valuable tool to aid clinical decision-making in remote, high altitude environments.

Keywords: high altitude pulmonary edema, high altitude cerebral edema, acclimatization, ataxia

Introduction

Acute high altitude illness includes acute mountain sickness (AMS), high altitude pulmonary edema (HAPE), and high altitude cerebral edema (HACE). These illnesses are caused by hypobaric hypoxia. They can develop within a few days after ascent to altitudes above 2500 m. In rare cases, individuals can develop symptoms at elevations as low as 2000 m.[1] Although AMS and HACE usually develop relatively quickly after arrival at altitude, with the onset of symptoms within 4 to 12 h for AMS and within 24 h for HACE, HAPE usually develops within 1 to 5 d. The risk of developing AMS and HACE increases with altitude, rate of ascent, and degree of acclimatization. HACE rarely occurs at altitudes below 4000 m. The prevalence of HACE between 4200 and 5500 m is estimated to be 0.5 to 1%.[2] It can occur at lower altitudes, however, especially if concomitant with HAPE.[3] The risk of HAPE increases with rapid ascent, higher altitude, respiratory infection, history of HAPE, male sex, lower temperature, and physical exertion.[4]

Field diagnosis of high altitude illness is traditionally made with clinical signs and symptoms, field tests such as tandem gait, and the use of basic tools such as lung auscultation and pulse oximetry. With the development of high-quality portable ultrasound machines, point-of-care ultrasound (POCUS) has gained interest as a promising diagnostic tool for patients with high altitude illness. POCUS is ultrasonography used in combination with physical examination to search for specific pathologic findings and diagnose problems wherever a patient is being treated. It is easily repeatable if the patient's condition changes and is therefore useful for monitoring disease progression and treatment efficacy.[5]

In a hospital, point-of-care lung ultrasound (LUS) can be used to diagnose interstitial pulmonary edema by the presence of 3 or more B-lines in 2 or more bilateral

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intercostal spaces.[6] The relationship between the presence of B-lines and HAPE is not yet clearly understood. Although an increased number of B-lines is also a common finding in healthy individuals ascending to high altitude, patients clinically diagnosed with HAPE have a significantly greater number of B-lines than healthy controls.[7,8] The number of B-lines in healthy individuals has an inverse relationship with arterial oxygen saturation. The presence of B-lines in these individuals might be a manifestation of subclinical pulmonary edema.[8-11]

Serial optic nerve sheath diameter (ONSD) measurements have shown promise in sedated patients in intensive care units when invasive measurement of intracranial pressure (ICP) is not indicated or is unavailable. There is no universally accepted cutoff value above which ONSD can be considered abnormal. In intensive care medicine, measurements above 5 mm correspond to elevations in ICP above 20 mm Hg.[12] A meta-analysis showed a high degree of variation among studies, in which sensitivity and specificity ranged from 88 to 95% and 74 to 91%, respectively. The ONSD threshold values that optimized sensitivity and specificity ranged from 4.8 mm to 6.3 mm.[13] Although early reports indicated a positive correlation between AMS and increasing ONSD values,[14,15] later studies failed to reproduce these results. Instead, they showed an altitude-related increase in ONSD that seems to be independent of AMS symptoms and disappears after descent to lower altitudes.[16-19]

There is limited data available on the use of point-of-care echocardiography at high altitude. HAPE is a noncardiogenic type of pulmonary edema. Pulmonary artery pressure (PAP) increases significantly in healthy individuals ascending to high altitude. Although there is no correlation between PAP and the number of B-lines in healthy individuals, patients with HAPE have both elevated PAPs and high numbers of B-lines.[9] In healthy individuals at high altitude, left ventricular (LV) function remains normal and cardiac output increases with faster heart rates. With increased PAP and increased afterload, there is increased right ventricular (RV) strain and a decreased myocardial performance index. It is not known whether this is an adaptive mechanism to hypoxia or a pathologic response in susceptible individuals.[10]

We describe the case of a healthy 32-y-old male who developed acute respiratory distress and neurologic impairment while participating in a high altitude medical research expedition to the Khumbu valley of Nepal. We discuss the use of POCUS to diagnose acute high altitude illness using LUS, ONSD measurement, and echocardiography. We believe this case illustrates the possibilities and limitations of POCUS in the evaluation of acute high altitude illness. We hope to inspire further research.

Case Report

The patient was a member of a trekking group that had started in Lukla (2860 m) and was following a carefully planned ascent profile to Island Peak (Figure 1). Every evening, each participant completed a medical questionnaire and underwent clinical examination by a physician. During the examination, participants were evaluated for sonographic signs of high altitude illness using a handheld ultrasound machine (Sonosite iViz) to measure B-lines and ONSD. LUS was performed using a modified protocol with 4 chest areas per side instead of the usual 8, to limit examination time in a cold, high altitude environment.[6] LUS findings were recorded for the upper anterior and basal lateral chest areas of each side.

The patient's symptoms began on the evening of Day 6 at Phortse (3840 m) with a minor headache. His vital signs and the POCUS examination were normal. He had no B-lines on LUS and had an ONSD of 6 mm, comparable to his previous ONSD, starting at 2800 m. He was feeling better on Day 7. He continued trekking at a high pace and was also seen running up to different viewpoints along the trail to take pictures. That evening in Dingboche (4321 m), his blood pressure was 142/92 mm Hg, his heart rate was 72 beats·min^{-1}, his respiratory rate was 22 breaths·min^{-1}, and his SpO_2 was 89%. The normal range of SpO_2 at 4300 m is 85 to 95%.[20] On examination, he was alert, oriented, and showed no signs of ataxia with tandem gait. Lung auscultation was normal, with absence of inspiratory crackles. LUS, however, showed 1 to 2 B-lines in 4 of the 4 lung regions (Figure 2). The ONSD measurement remained unchanged at 6 mm (Figure 3).

The patient became increasingly dyspneic during the night and developed severe orthopnea in the early morning hours of Day 8. At breakfast, blood pressure was 144/104 mm Hg, heart rate was 90 beats·min^{-1}, respiratory rate was 34 breaths·min^{-1}, and SpO_2 was 78%. Lung auscultation was normal. He did not have a cough or a fever. He was mildly ataxic with tandem gait but had normal mental status without other neurologic findings. On LUS, the number of B-lines in all regions had increased compared to the previous evening. There were still no more than 2 B-lines in any region. There were no sonographic signs of pneumothorax or pneumonia. The ONSD had increased to 7 mm. This abrupt increase in ONSD coincided with the development of the patient's symptoms and was higher than the reported normal values. We interpreted it as a possible sign of early HACE (Figure 4).

To investigate pulmonary embolism (PE) as a potential cause of the clinical deterioration, we performed point-of-care echocardiography (parasternal long axis,

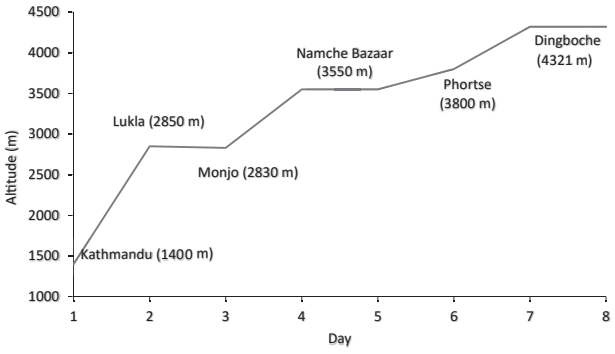

Figure 1. Ascent profile of the patient.

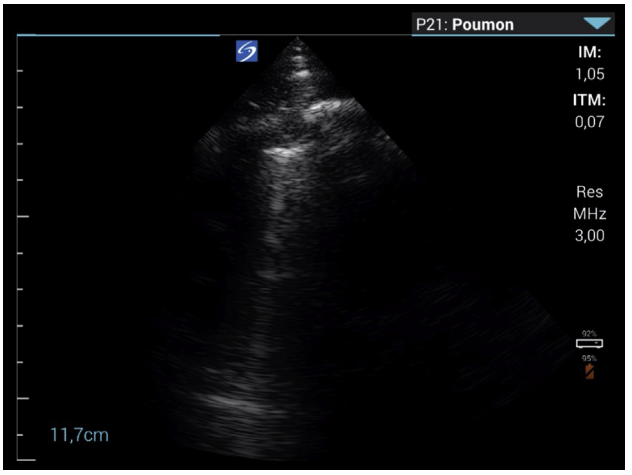

Figure 2. Lung ultrasound on the evening of Day 7.

parasternal short axis, and apical 4-chamber view). These views showed a slightly enlarged RV with an RV/LV ratio greater than 0.6, which allowed us to exclude a large, central PE. Although we could not exclude peripheral PE, the patient did not have typical symptoms such chest pain or a cough, which made us question peripheral PE as a differential diagnosis. We interpreted the RV enlargement as an indirect sign of pulmonary hypertension that was most consistent with HAPE (Figure 5).

The patient's rapid clinical deterioration together with the dynamic changes on POCUS prompted us to evacuate him by helicopter to a hospital in Kathmandu (1400 m). We gave the patient 20 mg of nifedipine by mouth. The patient remained in a seated position. His condition did not deteriorate while he was waiting for the helicopter to arrive. He did not receive oxygen because it was not available at our lodge or any of the neighboring lodges. Because the evacuation took place within 3 h of diagnosis, we did not contact the Himalayan Rescue Association aid post in Pheriche for treatment with a portable hyperbaric chamber. A dose of oral dexamethasone was discussed by the expedition leaders but was not administered.

The availability of real-time ultrasound images helped the patient, a physician himself, to understand and accept the decision to evacuate. Although shared decision-making is not always possible or appropriate in emergency situations, showing the dynamic changes on ultrasound was helpful in convincing our patient that descent was the appropriate course of action. Shortly after admission to the hospital in Kathmandu, a chest x-ray and transthoracic echocardiography were performed. The chest x-ray showed normal lungs with no signs of consolidation, interstitial pulmonary edema, pneumothorax, or pleural effusion (Figure 6). According to the hospital report, the transthoracic echocardiography showed no signs of pulmonary artery hypertension. The patient was discharged on the evening of the same Day. Three days after the evacuation, the patient developed a cough, muscle aches, and fever, most likely due to a respiratory infection. These symptoms subsided within 5 d with symptomatic treatment and without antibiotics. One month later, back home in Switzerland, the patient was mountaineering at altitudes up to 3000 m without any issues.

Discussion

The patient developed symptoms of HAPE and HACE during the first night after ascending to a new higher altitude and after performing a high level of physical exertion. Considering the severity of his respiratory symptoms, the number of B-lines seen was lower than expected and not enough to diagnose HAPE using the established sonographic criterion for interstitial pulmonary edema of 3 or more B-lines per intercostal space.[6] The same observation was reported in another study, in which B-lines were seen in healthy individuals ascending to high altitude. In 1 clinically diagnosed case of HAPE, the LUS findings did not meet the sonographic criteria for interstitial pulmonary edema.[11] As in our patient, the researchers noted an increase in B-lines that coincided with the onset of the patient's symptoms.

The relationship between the presence of B-lines and HAPE is not yet clear. When performing POCUS, it is recommended to perform repeated ultrasound assessments.[5,21] In this way, the patient's own images can serve as a control and help physicians to correlate dynamic changes on ultrasound with the development of new clinical symptoms.[22] For example, a sudden increase in B-lines in a patient with exertional dyspnea could prompt

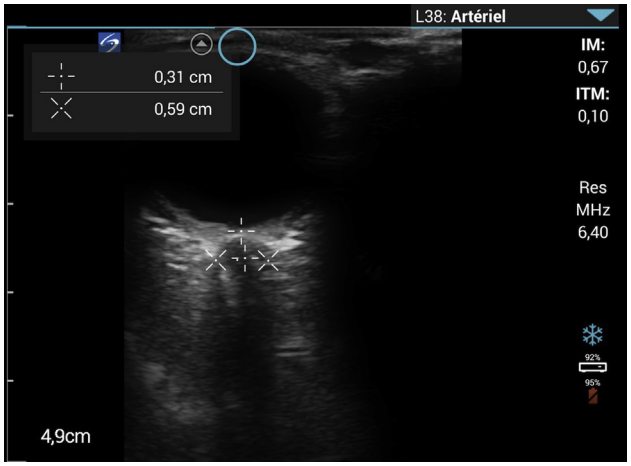

Figure 3. Optic nerve sheath diameter measurement on the evening of Day 7.

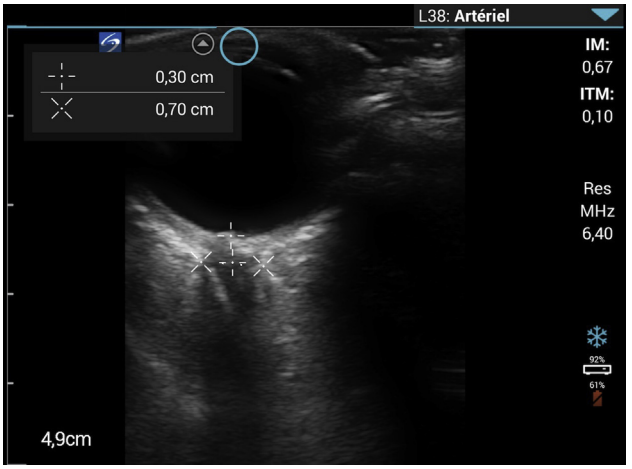

Figure 4. Optic nerve sheath diameter measurement on the morning of Day 8.

medical providers to recommend an extra layover day, followed by a slower ascent profile. If the patient develops clinical HAPE, ultrasound might be helpful in monitoring the effectiveness of treatment,[21] especially if the patient cannot be evacuated. A decreasing number of B-lines after treatment with nifedipine can provide additional information to show that the patient's condition is improving. Ultrasound monitoring of the course of HAPE has not been studied. It should be used to complement but not replace other clinical parameters, such as S_pO_2, to support clinical decision-making.

Because the exact ONSD cutoff value for predicting increased ICP and for the diagnosis of AMS and HACE is currently unknown, we believe that ONSD measurements should also be compared over time and not interpreted based on single absolute values. The sonographic images should always be correlated with clinical findings.[22] Because our patient developed mild ataxia in conjunction with an abrupt increase in ONSD, we interpreted these changes as a possible sonographic sign of early HACE.

We did not administer a dose of oral dexamethasone because we were able to rapidly evacuate the patient by helicopter. However, helicopter rescue services can be unreliable, and it is potentially dangerous to delay emergency treatment even if evacuation is imminent. Especially in remote, high altitude areas, helicopters can be subject to mechanical issues and delay due to changing weather conditions. In our case, care for the patient should have been improved by starting emergency treatment for HACE in the field. It would also have been prudent to obtain a portable hyperbaric chamber or oxygen while waiting for the helicopter. Patients with early signs and symptoms of HACE can deteriorate rapidly and develop a life-threatening illness.

A limitation of our examination protocol is that we only examined 4 of the usual 8 chest areas on each side.[6] It is therefore possible that we missed intercostal spaces with 3 or more B-lines, which could have facilitated the diagnosis of HAPE. Another limitation is that echocardiography was not part of the standard patient evaluation protocol. We only performed this examination on the morning before the evacuation. It therefore represents a single point value that we could not compare to previous measurements. We cannot say whether RV enlargement was a new development. It might have developed earlier when the patient initially arrived at high altitude.

A general limitation of ultrasound is that special precautions need to be taken to ensure machines work in cold, high altitude environments. Batteries can discharge quickly under these circumstances and can be difficult to recharge, even in highly developed trekking areas such as the Khumbu valley. Batteries should be protected from cold temperatures, and the need for alternative charging methods should be anticipated. We found that power was available in most guesthouses up to Namche Bazaar. At higher altitudes, we successfully charged our equipment with a combination of solar panels and external batteries.

Despite the limitations, we found that our ultrasound findings helped us to rule out several relevant differential diagnoses in the field. The absence of sonographic signs of pneumothorax and pneumonia, for example, made them unlikely causes of the patient's respiratory symptoms. In case of central PE, we would have expected a significantly higher RV/LV ratio (>1:1). Even if the rapid

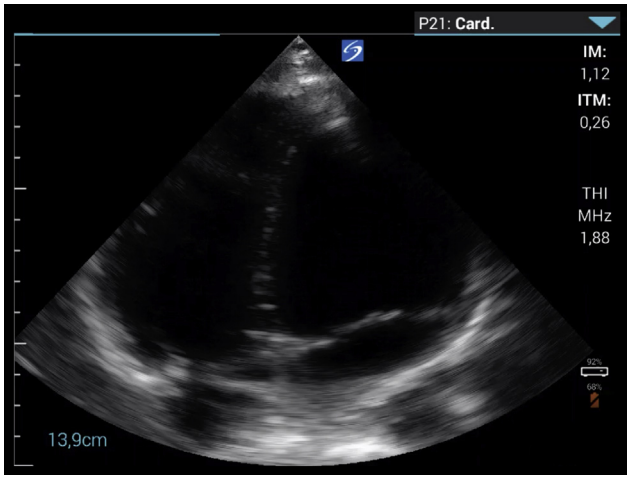

Figure 5. Apical 4-chamber view on the morning of Day 8.

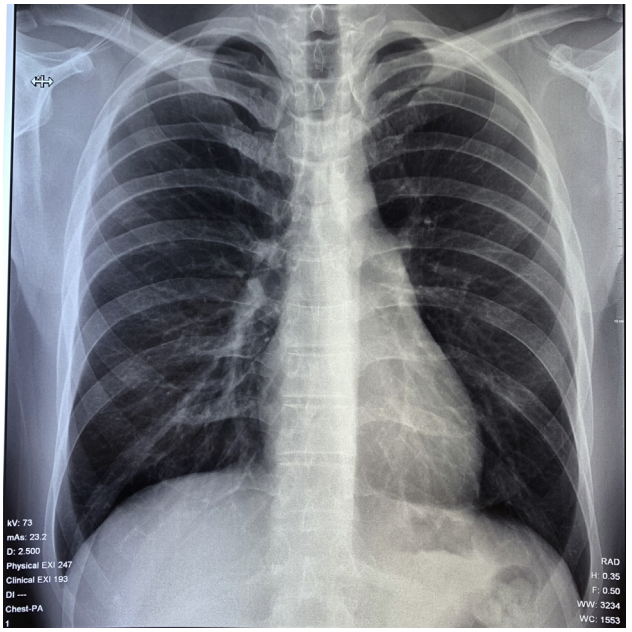

Figure 6. Chest x-ray after evacuation on Day 8.

resolution of symptoms and normalization of clinical findings at lower altitudes is most consistent with HAPE, we cannot rule out peripheral PE based on our findings and the further investigations done by the hospital in Kathmandu. With the reduction of pulmonary hypertension by nifedipine and the increase in partial pressure of alveolar oxygen by evacuation to a lower altitude, the symptoms caused by peripheral PE could also have improved.

The normal chest x-ray in Kathmandu could be explained by chest radiographic abnormalities that can lag behind the clinical signs of HAPE. Through timely treatment with nifedipine and descent to a lower altitude, it is also possible that sufficient interstitial fluid was reabsorbed by the time the patient arrived in Kathmandu and had the x-ray taken. Because the patient developed symptoms of an upper respiratory infection shortly after evacuation, it is possible that the respiratory infection had begun at high altitude. Nasal congestion may worsen hypoxemia, especially at night when excessive oxygen desaturation is common during episodes of periodic breathing.[23]

Conclusion

The development of high-quality, portable ultrasound machines has increased interest in the use of POCUS in high altitude medicine. The association between B-lines on LUS and the presence of HAPE is still unclear and needs further investigation. The correlation of increased ONSD with AMS and HACE is also still uncertain. Considering these limitations, there remains a need for clinical decision-making based on history and physical examination using basic tools such as auscultation and pulse oximetry. POCUS can, however, provide additional information to exclude relevant differential diagnoses and complement clinical findings with dynamic changes on ultrasound. When using POCUS, it is recommended to perform repeated ultrasound assessments. This allows medical providers to interpret sonographic changes over time and integrate these changes with clinical findings. We believe that POCUS can be a valuable tool for clinical decision-making in remote, high altitude environments.

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