CLINICAL VIGNETTE

From Diving to Skiing to Your ED: A Case of HAPE

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Case

A 26-year-old male is brought to the ED by a friend after landing at Los Angeles International Airport 20 minutes away due to shortness of breath. He arrived on a direct 2-hour flight from Aspen, CO where he had spent the prior 3 days skiing. He felt well at the start of his skiing vacation. On the second day, he noted some shortness of breath with a dry cough. He was ambivalent about boarding the flight to LAX as he had to stop a few times to catch his breath. He denied chest pain, leg pain or swelling, PND or orthopnea. He had no history of cancer or hypercoagulable state. He was not a smoker and did not use drugs. Prior to going to Aspen, he had spent 4 days in Florida on a scuba diving trip, before flying directly to Aspen. He also reported that his symptoms were improving during the flight from Aspen to LAX and continued to improve since arriving at LAX.

In the ED, triage vitals Temp 37.4, HR 105, BP 142/84, RR 16, O2 sat 96% RA.

On exam, he was speaking full sentences with no respiratory distress. There was no JVD. Crackles were heard in the right lower lung field. Heart sounds were unremarkable and there was no edema. EKG showed sinus tachycardia without right heart strain. Laboratory studies including normal d-dimer, BNP and troponin. Chest radiograph showed a small right lower lobe opacity. In the ED he was placed on supplemental oxygen at 2L NC with oxygen saturation improving to 100%. After several hours, he was asymptomatic and was discharged home with the diagnosis of suspected High Altitude Pulmonary Edema, HAPE. He was given information regarding the diagnosis and at follow up with his PCP one week later, he was feeling well with no symptoms.

Discussion

High altitude illnesses (HAI) are neurological and pulmonary syndromes that develop after ascent to altitude in a non-acclimatized individual.[1,2] The neurological disorders include acute mountain syndrome (AMS) and high altitude cerebral edema (HACE.)[1,2] AMS is a constellation of non-specific symptoms that usually develop between 6-10 hours after arrival to high altitude but may develop within the first hour.[2] AMS is defined by the Lake Louise Scoring System with headache and one more symptom which may include insomnia, fatigue, dizziness, or GI symptoms.[1-3] These will worsen over 1-2 days and then self-resolve. HACE is the life threatening neurologic manifestation of HAI with ataxia and altered mental status. HACE usually presents as a global encephalopathy but may include focal neurological deficits.[1-3] Death results from brain herniation.

High altitude pulmonary edema (HAPE) is the pulmonary manifestation of HAI. It is the potentially life-threatening development of non-cardiogenic pulmonary edema after ascent to 2000 – 3000 meters.[1-3] HAPE has been reported as early as the late 18th century but was identified as noncardiogenic in the 1960s.[3] HAPE develops within 2-4 days after arrival to altitude. HAPE rarely develops after 4 days, due to cellular and biochemical adaptation to altitude. Of individuals with HAPE, 50% have AMS and about 15% have HACE.[2]

HAPE manifests as shortness of breath, decreased exercise tolerance, chest congestion, and nonproductive cough which may progress to pink frothy sputum to frank blood.[1-4] Chest pain, syncope and orthopnea are rare.[4] Vital signs may include low grade fever, tachycardia, increased respiratory rate, hypertension and varying degrees of hypoxia.[1-4] Exam may show rales. These tend to be right sided but may progress to bilateral.[1-4] Labs are rarely abnormal. EKG can show tachycardia and right heart strain.[2] Chest radiographs show patchy alveolar infiltrates in the right middle and lower lobes in mild cases which can progress to bilaterally lung involvement with worsening disease.[1-3]

Risk factors include rapid ascent, higher altitude, colder ambient temperatures, increased exertion, existing respiratory infection particularly in children, prior history of HAPE and certain medical conditions.[1-3] Men are more susceptible.[1,2] Physical fitness is not protective.[2] Certain medical illnesses including hypertension, coronary artery disease, COPD, diabetes, and pregnancy do not seem to affect susceptibility.[2] Interestingly, long term high altitude habitants who spend time at lower altitude may develop reentry HAPE, and children are especially susceptible.[1]

The best prevention is gradual ascent. Recommendations are to ascend 300-350 meters/day above 2500 meters and an extra day for every 600-1200 meters over 2500 meters.[1] Patients with history of HAPE or thought to be high risk for development of HAPE may benefit from prophylaxis. Nifedipine, a nonselective calcium channel blocker reduces pulmonary vascular resistance and PA pressure as well as systemic blood pressure.

It is not effective against AMS.[1-3] The phosphodiesterase-5 (PDE-5) inhibitors tadalafil and sildenafil have been effective as prophylaxis by augmenting pulmonary vasodilatory effects of nitrogen oxide, but may worsen AMS.[1] Dexamethasone has also decreased incidence of HAPE but has been used more for the treatment of AMS/HACE given the side effect profile.[1] If an individual develops recurrent HAPE <2500 meters they should be evaluated for intracardiac, intrapulmonary shunts, pulmonary hypertension, and valvular stenosis.[2]

If HAPE develops the most effective treatment is descent of at least 500-1000 meters.[1-3] With mild to moderate symptoms the patient can be kept at altitude with rest and supplemental oxygen for 48-72 hours.[1-3] If symptoms are severe, HACE can develop and oxygen saturation can remain <90% even with supplementation and descent is imperative.[1-3] Passive descent is ideal, keeping the individual warm without exertion.[1] Intubation and mechanical ventilation are rarely necessary.[2] Nifedipine may be used as adjunctive therapy if descent is not feasible. There is no data to recommend the use of PDE-5 inhibitors for treatment.[4] Hyperbaric bags can also be used as a temporizing measure.[1-3]

REFERENCES

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