



Case 62: Headache

- **Chief complaint**
 - 35-year-old female presents with dizziness and headache.
 - Location is in Breckenridge, CO (9,600 ft elevation; if requested, can share that patient is on vacation)
- **Vital signs**
 - **HR: 108** BP: 134/92 RR: 22 Sat: 89% on RA T: 37.2°C Wt: 80 kg
- **Patient appearance**
 - Patient appears uncomfortable and sleepy. You watched her walk into the exam room and her gait was ataxic.
- **Primary survey**
 - Airway: speaking normally
 - Breathing: no respiratory distress, clear lungs
 - Circulation: dry and cool, normal capillary refill, 2+ distal pulses
 - Disability: Opens eyes spontaneously. Answers questions correctly. Moves all extremities spontaneously (GCS 15). Distal sensation intact.
 - Exposure: no obvious traumatic injuries, change the patient into a gown prior to full exam
- **Action**
 - Place patient on the monitor
 - Oxygen by NC (improves to 95%)
 - Place two large bore peripheral IV lines (draw rainbow top)
 - POC glucose (85, must ask)
 - Order Stat ECG ([Figure 62.1](#)- sinus tachycardia)
- **History**
 - Source: Patient
 - HPI: a 35 year-old female presents with a one day history of dizziness, loss of appetite, headache, weakness and nausea with NBNB emesis x2. She arrived in Denver 2 days ago and immediately drove up to a cabin at the base of Breckenridge where she is volunteering for an adaptive skiing program. Yesterday morning, she woke up feeling as if she was hungover but denies having any alcohol or drugs. This morning she was supposed to help with the ski program when her friend noticed she wasn't walking right

and brought her to the ED. No one else at the lodge has similar symptoms. No trauma. ROS otherwise negative.

- PMHx: negative
- PSHx: appendectomy (age 8)
- Allergies: none
- Meds: none
- Social: socially drinks alcohol, denies smoking, or drugs, lives on the Outer Banks of North Carolina. Arrived late two nights ago to the mountains by small plane, ascended 10,000 feet (must ask specifics).
- FHx: noncontributory
- Code Status: full code

- **Physical Exam**

- **General:** appears sleepy but arousable, uncomfortable
- HEENT: normal, unable to visualize for papilledema (if asked)
- Neck: normal
- Chest: nontender
- Heart: normal
- **Lungs:** minimally tachypneic, but otherwise normal
- Abdomen: normal
- Extremities: normal
- Back: normal
- **Neuro:** oriented to person. CN II-XII intact. No focal weakness. No sensory deficit. Ataxic on tandem gait. Normal Romberg's test. Normal reflexes.
- **Skin:** scattered abrasions to upper and lower extremities. Otherwise normal.

- **Instructor Prompt:** learners should discuss differential diagnosis

- **Action**

- Order Labs
 - CBC, BMP, LFT, PT/INR, PTT, urinalysis, urine pregnancy test
- Order Imaging
 - CT head without contrast
 - CXR
- Order Meds
 - IV/IM/PO Dexamethasone (8-10mg IV, IM, or PO x 1, then 4mg IV, IM, or PO q6 hours)
 - IV/PO antiemetic
 - Analgesic (such as ibuprofen, ketorolac, or acetaminophen for headache)
- Consult hospital at lower altitude such as Denver: express concern for HACE and need for transfer to a lower altitude to resolve patient symptoms

- **Response/Results**

- Patient reevaluation and repeat vitals:
 - Vitals after oxygen: HR: 98 BP: 130/86 Sat 95% on NC
 - Vitals if no O2: HR: 98 BP: 130/86 **Sat: 89% on RA** (Prompt: administer oxygen)
 - If no consult or attempt to transfer, patient will exhibit deterioration of neurologic condition and require intubation
- [Case 62 Lab Results](#) (normal)
- CXR ([Figure 62.2](#): normal)
- CT head ([Figure 62.3](#): cerebral edema)

- **Action**

- Hyperbaric chamber or bag (if available; pressurized to 1.2 atm)
- Immediate transfer/decant to lower altitude

- **Diagnosis**

- Primary Diagnosis: High Altitude Cerebral Edema (HACE)

- **Critical actions**

- Obtain history of rapid ascent
- Supplemental oxygen
- Point of care glucose
- Treatment with steroids
- Transfer for rapid descent

- **Instructor Guide**

- This is a case of acute mountain sickness (AMS) progressing to high altitude cerebral edema (HACE) without high altitude pulmonary edema (HAPE) in a patient presenting with ataxia, headache, dizziness, fatigue and nausea. This patient complained of feeling “hungover” on day 1 indicating the likely presence of AMS. Given the symptoms on day 2, this patient progressed to HACE. The patient does not have respiratory symptoms suggestive of HAPE despite a low O2 saturation that is typical at this altitude. Early in the case you may give indications that the patient is at high altitude, but wait for the learner to ask specific questions about how rapidly the patient ascended to altitude. Important early actions include supplemental oxygenation, treatment with steroids, and rapid descent. If time allows, consider a hyperbaric chamber/gamow bag but do not allow this to delay descent. If the important early actions do not occur, the patient will decompensate with increased lethargy and ultimately require intubation. The patient should be dispositioned to a lower altitude facility for continued care.

- **Case Teaching Points**

- The differential diagnosis for headache, weakness, nausea and vomiting in a patient at high altitude should include acute mountain sickness, HACE, traumatic injury, infection,

alcohol or drug intoxication, CO poisoning, and intracranial bleeding. For any patient with respiratory symptoms at high altitude, HAPE (high altitude pulmonary edema) should also be considered.

- **What is the difference between HACE and Acute Mountain Sickness (AMS)? How often does AMS progress to HACE?**

- AMS is characterized by flu-like symptoms including headache with a combination of nausea, vomiting, dizziness, anorexia, weakness, decreased urination, fatigue or sleep disturbance with recent ascent to greater than 2000m (6560 ft). Symptoms typically abate between 15 - 94 hours with symptomatic treatment. Appropriate treatment includes oxygen, NSAIDs, acetazolamide, +/- steroids and descent.
- HACE occurs in those who recently ascended to > 8200 ft (2500m) who have progressive neurologic deterioration. This typically starts with symptoms of AMS, then the patient begins to exhibit ataxia or altered mental status. Any neurological deficit should be concerning for HACE.
- AMS progresses to HACE in 1-5% of cases.
- The mainstays of treatment of HACE are rapid descent and steroids. Hyperbaric oxygen (Gamow bag) may be used as a temporizing measure. Acetazolamide may be given for suspected Acute Mountain Sickness but offers limited benefit in HACE.
- Death from HACE has been reported at as low as 8200 ft. HACE is most common at >12,000 ft.

- **Why is treatment of HACE so important?**

- Long-term neurologic sequelae of HACE including ataxia and cognitive impairment have been reported after recovery. Once coma occurs, mortality exceeds 60%.
- Oxygen decreases intracranial blood flow at high altitude and steroids are likely to reduce long-term deficits.

- **What is considered high altitude?**

- High altitude is a hypoxic environment with an elevation of >2440 m or >8000 ft
- Although partial pressure of oxygen remains the same at altitude, the air pressure decreases.
 - In Denver at 5280 ft, the air pressure is 17% less than at sea level.
 - In Aspen at 8600 ft, the air has 26% less oxygen.
 - This is a common altitude in Colorado and several mountainous areas of the western United States.

- **How can one acclimatize and prevent HAPE/HACE?**

- Natural acclimatization occurs when people hyperventilate causing respiratory alkalosis and bicarb diuresis.
- Prophylactic treatment with Acetazolamide (125 mg PO q12 hours, started 48 hr before ascent and continued for 48 hr after ascent) works by causing bicarb diuresis and metabolic acidosis, thus triggering hyperventilation and speeding acclimatization.

- Steroids may also be used for treatment (Decadron 8-10 mg IV, IM, or PO x 1, then 4 mg IV, IM, or PO q6 hours) to help reduce vasogenic cerebral edema.
- **What is the normal range of O2 saturation at high altitude? Does this change with acclimatization?**
 - At Sea level SaO2 96%
 - 5000 ft (1520 m) SaO2 95%
 - 7500 ft (2290 m) SaO2 92-93%
 - 15000 ft (4570 m) SaO2 86%
 - 20000 ft (7000 m) SaO2 76%
 - This changes minimally with acclimatization
- **What is the Pathophysiology of HACE?**
 - High-altitude exposure → hypoxemia → fluid retention and cerebral hypoxemia (mediator-induced permeability) → sympathetic discharge/peripheral vasoconstriction and increased pulmonary/cerebral blood volume → altered cerebral hemodynamics and increased cerebral blood flow and volume → capillary pressure increase → vasogenic edema → brain swelling → increased ICP = HACE
- **What is HAPE? How often are HAPE and HACE associated?**
 - High Altitude Pulmonary Edema (HAPE) is the most common lethal altitude illness. This may occur at altitude >3000m (9500ft).
 - Patients present with pneumonia-like symptoms and shortness of breath at rest, cough, hypoxia and fever. Clinical progression towards ARDS is likely to occur.
 - Treatment of HAPE should include oxygen, rapid descent, nifedipine/PDEIs (for pulmonary HTN), and hyperbaric oxygen.
 - HAPE is concomitant in approximately 50% of cases of HACE.
- **Attributions**
 - **Author:** Dr. Lindsay Davis
 - Editor(s): Dr. Therese Mead
 - Editor-in-Chief: Dr. Dana Loke, Dr. Kristen Grabow Moore
 - **References:**
 - Chris Davis, Kurt Power Elfling. Chapter 216: High-Altitude Disorders. In: Judith E. Tintinalli, O. John Ma, et al, editors. Tintinalli's Emergency Medicine: A Comprehensive Study Guide (9th ed). New York: McGraw-Hill; 2020.
 - Hackett PH and Roach RC. High altitude cerebral edema. High Altitude Medicine and Biology, 2004; 5(2):136-46N.
 - Stuart Harris. Chapter 136: High-Altitude Medicine. In: Ron Walls, Robert Hockberger, Marianne Gausche-Hill et al, editors. Rosen's Emergency Medicine: Concepts and Clinical Practice (9th ed). Philadelphia: Elsevier, Inc; 2018.
 - Bhandari Sanjeeb Sudarshan, Gehner Jessica R.A.. Altitude-Related Illness. In: Mattu A and Swadron S, ed. CorePendium. Burbank, CA: CorePendium, LLC.

<https://www.emrap.org/corependium/chapter/recucVS255aTs01tW/Altitude-Related-Illness>. Updated October 24, 2022. Accessed October 24, 2022.

■ Image References

- ECG from NYU ECG Database
<https://education.med.nyu.edu/ecg-database/app/search/results/18609>
- XR Chest from Radiopedia
<https://radiopaedia.org/cases/normal-chest-x-ray-1?lang=us>
- CT Head from Radiopedia
<https://radiopaedia.org/cases/cerebral-edema-evolution-in-time-1?lang=us>

Case 62 Lab Results

Basic Metabolic Panel:

Na	138 mEq/L
K	4.8 mEq/L
Cl	106 mEq/L
CO ₂	22 mEq/L
BUN	15 mg/dL
Cr	0.9 mg/dL
Gluc	86 mg/dL

Liver Function Panel:

AST	32 U/L
ALT	14 U/L
Alk Phos	90 U/L
T bili	1.1 mg/dL
D bili	0.3 mg/dL
Lipase	40 U/L
Albumin	4.5 g/dL

Complete Blood Count:

WBC	$9.1 \times 10^3/\mu\text{L}$
Hb	14.1 g/dL
Hct	42.5%
Plt	$250 \times 10^3/\mu\text{L}$

Urinalysis:

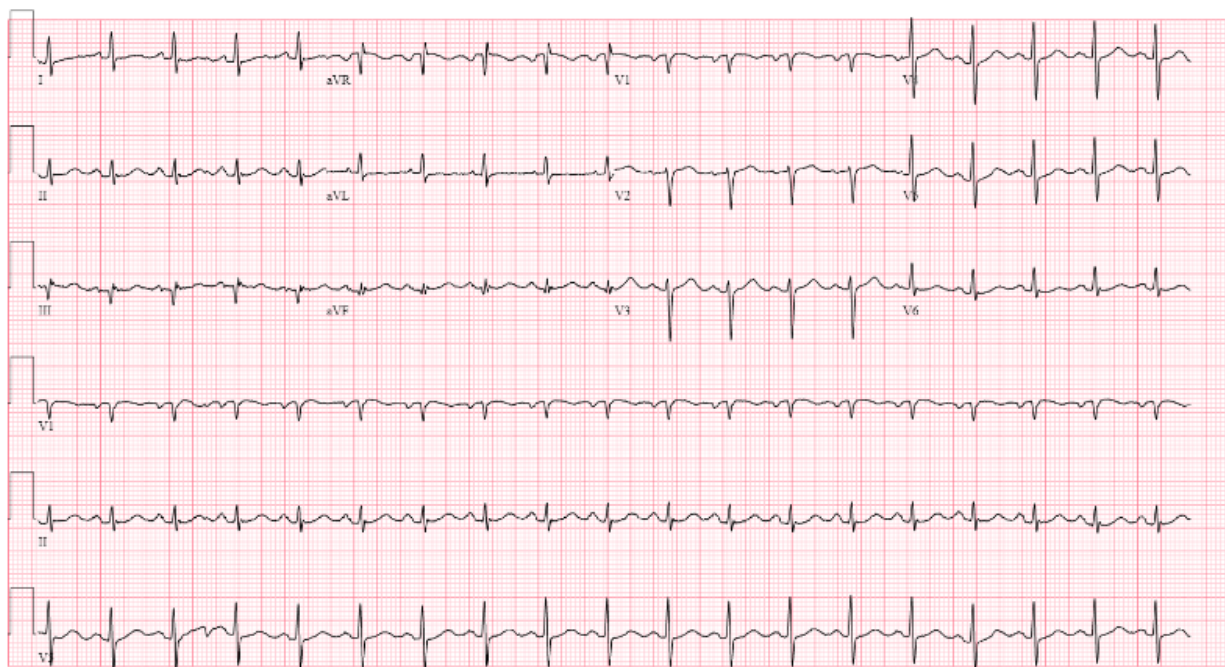
SG	1.018
pH	6.8
Prot	Neg
Gluc	Neg
Ketones	Neg
Bili	Neg
Blood	Neg
LE	Neg
Nitrite	Neg
Color	Yellow

Coagulation Panel:

PT	13.1 sec
INR	1.0
PTT	26 sec

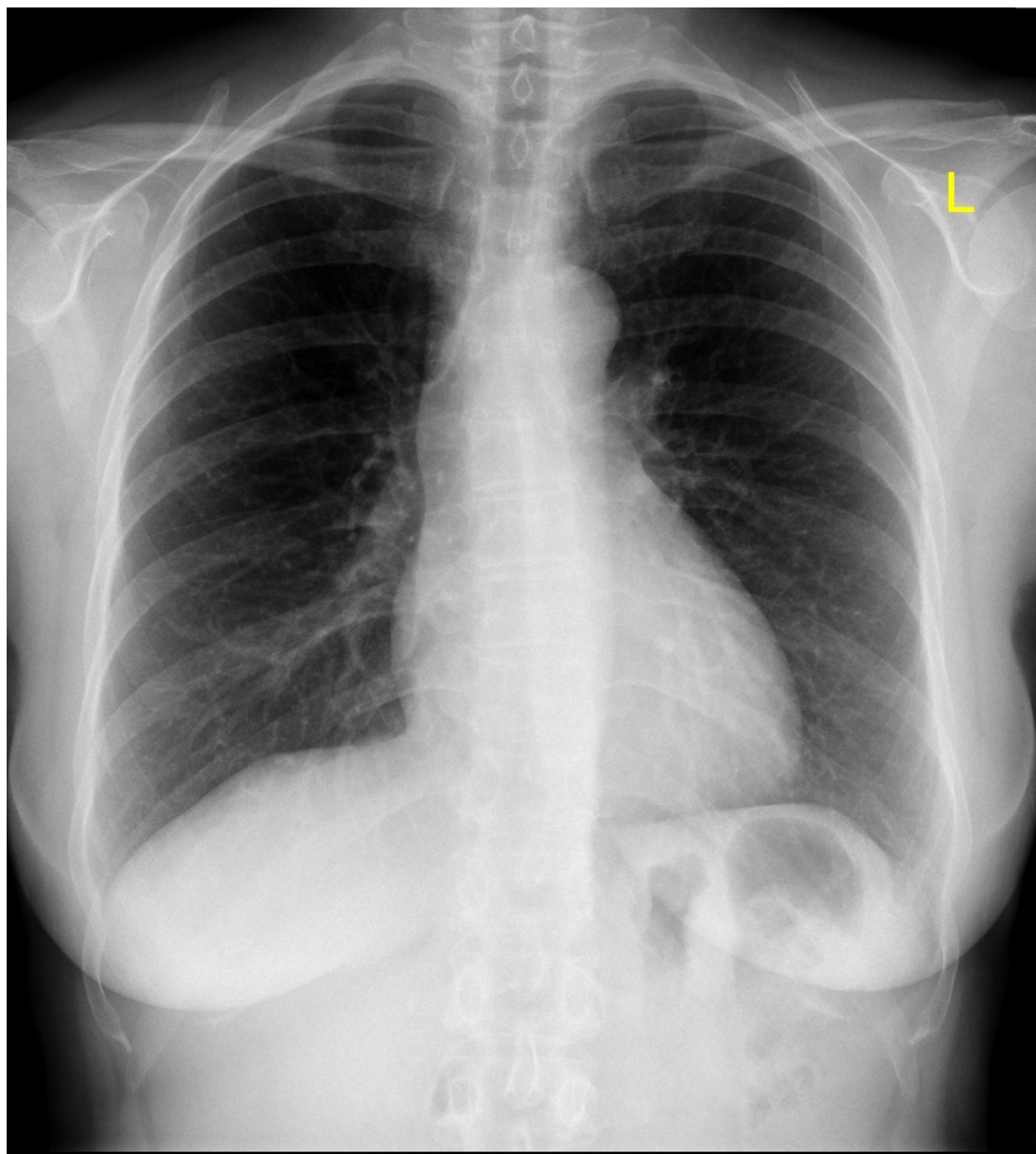
[Back to case](#)

Figure 62.1- ECG



[Back to case](#)

Figure 62.2- CXR



[Back to case](#)

Figure 62.3- CT Head w/o contrast



[Back to case](#)