Acute Mountain Sickness (AMS), High Altitude Pulmonary Edema (HAPE), & High Altitude Cerebral Edema (HACE)

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Assessment for altitude sickness should include:

- Elevation and rate of climb
- Home elevation
- Mental status changes
- Abnormal vital signs
- Skin color, temperature and condition
- Difficulty breathing
- Chest pain

Acute mountain sickness: headache in an unacclimatized person ascending to an altitude above 2,500m (8,000ft) with at least one of the following symptoms: nausea, vomiting, anorexia, lightheadedness, dizziness, weakness, fatigue, lassitude, dyspnea, or insomnia but a clear sensorium.

High altitude cerebral edema: a severe form of acute mountain sickness. A severe headache, vomiting and lethargy will progress to unsteadiness, confusion, drowsiness and ultimately coma. HACE can kill in only a few hours. A person with HACE will find it difficult to walk heel-to-toe in a straight line. Similar symptoms as AMS along with confusion, in-coordination, and obtundation.

High altitude pulmonary edema: a dangerous build-up of fluid in the lungs that prevents the air spaces from opening up and filling with fresh air with each breath. When this happens, the sufferer becomes progressively more short of oxygen, which in turn worsens the build-up of fluid in the lungs. In this way, HAPE can be fatal within hours. Presents with SOB at rest and with exertion, cough, crackles, etc.

Acute Mountain Sickness

Onset: typically delayed for 6-12 hrs, especially after the first night, can occur as rapidly as 1-2 hrs or as late as 24 hrs

Treatment:

- Descent to lower elevation (at least 500-1,000 m)
- Acetazolamide: 125-250 mg PO or Decadron 4 mg PO

Physical evaluation, vital signs, and pulse oximetry typically within normal limits

Differential diagnosis: if onset of symptoms is more than two days following ascent, absence of headache, absence of dyspnea, and failure to improve rapidly with supplemental oxygen; check for: carbon monoxide poisoning, migraine, dehydration, exhaustion, hyponatremia, viral syndrome, alcohol hangover, bacterial infection, subarachnoid hemorrhage, stroke, and intracranial mass

Epidemiology: 2,000-3,500 m is 10-40%, 3,800-4,000m is 25-25%, >4,000m is 50%

Special Notes

- High altitude is a hypoxic environment and most people have a hypoxic ventilator response
- Hypobaric hypoxia is the culprit in these syndromes, but other cytotoxic and vasogenic factors are involved with HACE and HAPE

HACE

Epidemiology: 0.1-2% >3,000-4,000 m

Patho: Increased blood-brain barrier permeability leads to reversible vasogenic brain edema with T2 signal increase in the corpus callosum and subcortical white matter

Diagnosis: ataxic gait, severe lassitude, progressive decline of mental function and consciousness (irritability, confusion, impaired mentation, drowsiness, stupor, and intracranial mass), most importantly encephalopathy from MRI testing lasting days to weeks

Differential diagnosis if: hemiparesis, slurred speech, or discrete visual deficit; look for possible ischemic stroke, intracranial hemorrhage, or hypoglycemia

Onset: 12-36 hours after onset of AMS, more quickly if co-occurring with HAPE

Treatment:

- descent to lower elevation or hyperbaric therapy
- Decadron 8-10 mg IV
- RSI if unable to protect airway
- SpO2 >90%
- ETCO2: 25-30 with hyperventilation
- avoid hypotension in the comatose patient using IV hydration with isotonic crystalloid and catheterization to assess fluid status

Special Considerations for Peds with AMS/HACE/HAPE

Children can be more susceptible to hypoxia due to: more compliant rib cage, reduced surfactant in pre-term infants, increased airway reactivity, lung volume at end expiration similar to closing volume in early infancy, reduced upper and lower internal diameters of the airways, fewer alveoli in early infancy, being 0-2 months of age

Risk factors: concurrent URI, congenital cardiopulmonary disease, down syndrome, systemic diseases affecting respiratory fx, <12 mo with hx of O2 requirements or pulmonary hypertension

Signs: decreased playfulness, fussiness, poor sleep, vomiting, and decreased appetite

Treatment:

- descent to lower elevation or hyperbaric therapy
- O2 >90%
- AMS: Acetazolamide 2.5 mg/kg q 12hrs
- HACE: Decadron 0.15 mg/kg q 6hrs
- HAPE: Nifedipine 0.5 mg/kg q 8hrs
- warmth and rest

References

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HAPE

Epidemiology: 0.2-6% >4,500 m

Patho: High mean PA pressure (>35-40 mmHg) leading to breakdown in pulmonary-gas barrier, triggered by hypobaric hypoxia leading to accumulation of water in the alveolar spaces and then alveolar hemorrhage.

Diagnosis: subtle, nonproductive cough, then dyspnea at rest, cough can become productive with pink, frothy sputum/frank blood, then severe hypoxemia with drowsiness, tachycardia, tachypnea, low-grade fever, inspiratory crackles, decreased SpO2 (40-50%)

Differential diagnosis if: appears as sick as assessment shows and slow response to O2/decent; check for pneumonia, ADHF, acute coronary syndrome, bronchitis, reactive airway disease; can coexist with infection

Onset: 2-4 days following ascent

Treatment:

- descent to lower elevation or hyperbaric therapy
- consider CPAP
- consider nifedipine to decrease PA (ex: 30-60 mg slow release q 12hrs)
- SpO2 >90%
- rest and warmth