

Medical Complications of Severe Eating Disorders

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Goals

- Know basic diagnosis and epidemiology of Anorexia and Bulimia Nervosa
- Recognize “typical” physiologic abnormalities associated with severe starvation
- Identify potentially deadly complications of refeeding and know how to manage them
- Understand common clinical complaints in these patients
- Identify ACUTE center at Denver Health as an option for referring patients

Case

- A 26 year old woman with restrictive anorexia nervosa weighs 85 pounds at 5'5" tall.
- Due to a hypoglycemic seizure she is admitted to a step-down unit in her home state of Texas where her vital signs are: Temp 33 C, HR 35, BP 75/50, glucose 23.
- Exam is notable for extreme emaciation and lanugo.
- For three weeks she receives D10 NS at 100 cc/hr through a PICC line, is encouraged to eat whatever might appeal without a formal dietary plan.
- At the end of those three weeks she weighs 100 pounds, has deep pitting edema and a sacral decub, and her father calls to ask that she be transferred to Denver Health's ACUTE unit.

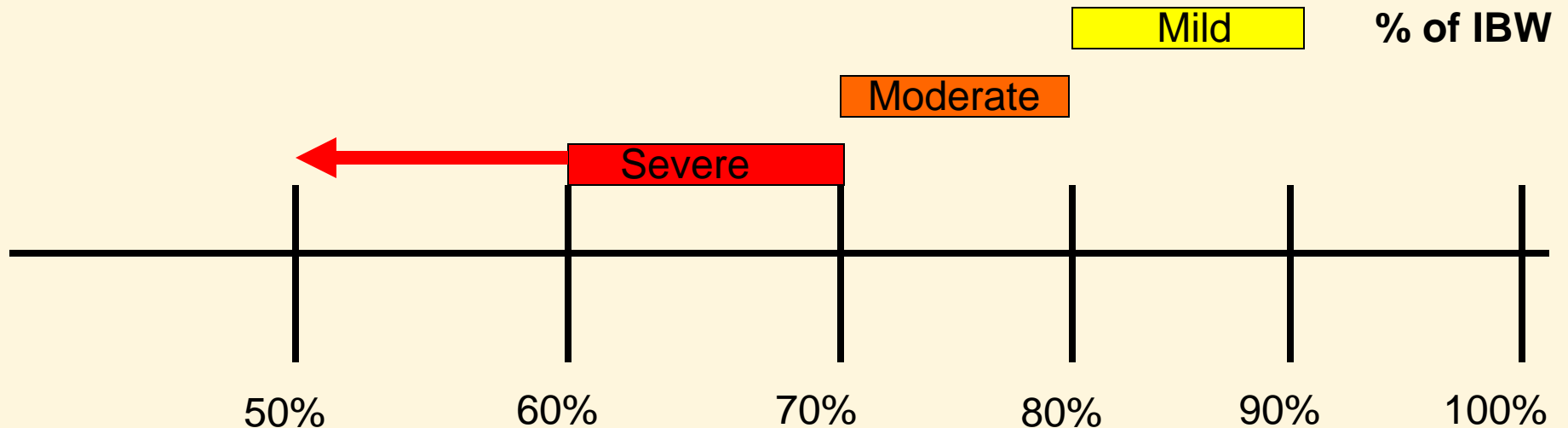
What is Anorexia Nervosa?

- Defined by four criteria from DSM-IV
 - Refusal to maintain body weight at or above 85% of that expected for height
 - Intense fear of gaining weight or becoming fat, even though underweight
 - Disturbance in the perception of one's body weight or shape
 - In postmenarchal women, the absence of three consecutive menstrual periods
- Subtypes: Restrictive or binge/purge type

Ideal Body Weight

Ideal Body Weight (IBW):

- 100 lbs for first five feet of height
- Then 4-5 lbs for each additional inch



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What is Bulimia Nervosa?

- Defined by DSM-IV:
 - Recurrent episodes of binge eating
 - Recurrent compensatory behavior to prevent weight gain
 - Self-evaluation overly influenced by body weight and shape
 - Symptoms occur at least twice a week and persist at least three months
 - Behaviors not occurring exclusively during episodes of Anorexia Nervosa

Epidemiology

- Anorexia Nervosa (AN)
 - In Western industrialized countries, prevalence 0.5-4% of the female population
 - Woman make up 90% of anorexics
 - Mortality is the **highest of any psychiatric diagnosis**: .56% per year, or 5.6% per decade, 12 times higher than the annual death rate of women 15-24 years old

Epidemiology

- Bulimia Nervosa (BN)
 - In Western industrialized countries, 1-5% of female population, <1% of men

What is the A.C.U.T.E. unit?

- Acute Comprehensive Urgent Treatment of Eating Disorders
 - Dr. Mehler's expertise
 - Unique model of care
 - Goals for patients
 - When and why do patients need ACUTE level care?
 - Goal: Become a Center of Excellence.

A.C.U.T.E. Medical Center



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The A.C.U.T.E. Medical Center at Denver Health is a unique medical unit for individuals who are suffering from serious medical complications of an eating disorder.

As an inpatient medical intensive care service for patients whose eating disorders have become life threatening, the A.C.U.T.E. Center at Denver Health Medical Center is one of the only hospitals in the United States able to care for the most complex medical complications of both anorexia nervosa and bulimia, regardless of the percentage below ideal body weight.

Physicians in the Center work closely with referring physicians and psychiatrists about their patients' medical progress, and help facilitate a safe transfer to other levels of care after they are medically stabilized.

During hospitalization, medical care is administered by nationally recognized eating disorder specialist Philip S. Mehler, M.D., Chief Medical Officer at Denver Health, and Glassman Professor of Medicine at the University of Colorado School of Medicine, with his team of physicians, nurses and dietitians. Psychiatric services during hospitalization are provided by Kenneth L. Weiner, M.D., and Emmett "Rick" Bishop, M.D., from the Eating Recovery Center, who are also nationally recognized eating disorder experts.

Medical treatment provided by the A.C.U.T.E. Center may be covered under the patient's health insurance benefit.

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Follow-On From ACUTE: local and national options



The Hospitalized Patient with BN

- Often not underweight, admitted instead for volume depletion and severe electrolyte abnormalities
- Efforts to replete potassium often unsuccessful
 - a) Due to underestimation of actual K depletion
and
 - b) If coexisting volume depletion-induced metabolic alkalosis isn't treated first

The Hospitalized Patient with BN

- **Pseudo-Bartter's syndrome**

- Patients who are chronically hypovolemic (due to starvation or purge-behaviors) develop a highly sodium-avid state in which kidneys upregulate adrenal aldosterone secretion, increasing sodium and water reabsorption in the renal tubules → EDEMA!
- Characterized by hypokalemia and metabolic alkalosis
- Efficacy of K repletion poor unless volume is restored
- Go slow with IVF! NS 50-75 cc/hr...NOT bolus dosing
- Edema usually goes away in a few weeks
- Spironolactone can be used to help with peripheral edema

The Hospitalized Patient with AN

- Goal: Refeed the patient with AN to achieve around 75% IBW, at which point starvation-induced cognitive deficits subside and meaningful psychiatric work can begin/resume
- Patients weighing less than 75% IBW should generally be hospitalized for careful monitoring of the refeeding process

The Hospitalized Patient with AN

- Two main categories to think about with refeeding:
- **Biochemical complications**
 - Refeeding syndrome
 - Electrolyte dysfunction
 - Organ dysfunction
- **Common clinical complaints and how to manage them**

Biochemical Complications

- Case:
 - Two mornings after oral intake is initiated for a young woman with severe anorexia nervosa, her chemistry panel shows:

132	100	32	Glu 55
3.3	32	1.2	Ca 8.0 Phos 1.5 Mg 1.5

***What should you be most concerned about and attentive to this morning?

Biochemical complications

- Refeeding syndrome
 - A problem of **hypophosphatemia** (but also of hypoK and hypoMg)

Refeeding Syndrome

- As starving person is fed, body shifts from catabolic to an **anabolic state**, thus from fat to carbohydrate metabolism
- Glucose load in the food stimulates **insulin release**, which shifts phosphate and potassium into cells, causing serum lows
- **Newly synthesized tissues** incorporate Phos, K, and Mg which further deplete serum levels

Hypophosphatemia

- Causes **depletion** of ATP (for energy) and 2,3-DPG (helps transfer oxygen into cells)
- **Low ATP and 2,3-DPG** cause diaphragmatic muscle fatigue with respiratory failure, RBC and WBC dysfunction, rhabdo, seizures, CHF, and general tissue hypoxia
- (Also, hypoK can cause severe cardiac and neurologic sequelae)

Refeeding Syndrome

- Who's at risk?
 - Severely underweight, haven't eating for a week or more and then suddenly get high-dextrose containing foods

Refeeding Syndrome

- How to **prevent**?
 - Start low, go slow (25-30 kcal/kg)
 - We start patients at or below their Basal Energy Expenditure (BEE) as calculated by the Harris-Benedict equation which our RDs help us with
 - Caloric increases are done slowly over a few days, checking a chem 10 daily and repleting phos aggressively. Later on, QOD chemistry appropriate

Refeeding Syndrome

- What are the **clinical features** to watch out for?
 - In addition to the above list, patients should be on telemetry at all times to watch for HR (typically in the 40-50 range for AN) jumping to 80-90, watch for any peripheral edema or decreased O2 saturations, and check lytes

Hyponatremia

- Predominant mechanism: **hypovolemic hyponatremia**
- The AN version of “beer drinker’s potomania” where too little solute is ingested and thus less free water can be excreted daily
- Usually resolves in first few weeks of treatment as patient increases caloric intake and drinks an appropriate (but not excessive) amount of fluid

Organ Dysfunction

- Case:
 - A patient with AN who has been steadily increasing her caloric intake over the last week complains of dyspnea. On exam, her pulse is 75, she has bilateral rales, and she has 1+ pitting edema in her legs.

***What should you be concerned about?

Organ Dysfunction: CHF

- Rare
- The heart has a reduced mass and decreased CO, which may struggle with increased blood volume in refeeding
- Low ATP and hypoK may impair contractility
- Tele, pulse ox, pulse rate, and presence of edema are key...watch for changes from baseline
- Usually risk gone by 2-3 weeks into refeeding

Organ Dysfunction: Pancreatitis

- Can occur in both starvation and refeeding states, mechanisms not totally clear
- **Refeeding-induced**
 - Gallstone pancreatitis can occur as stones develop with weight loss
- **Starvation-induced**
 - Many models posited

Organ Dysfunction

- Case:
 - A young woman with severe AN arrives in transfer from an acute hospital out of state. She is 4'9" and weighs 46 pounds. Her LFTs are in the 600s, and her admission glucose is 16 without significant symptoms. Records from the outside hospital show negative viral studies; U/S shows mild steatosis but no gallbladder disease. She is on no hepatotoxic medications.

***What is likely going on?

Organ Dysfunction: Hepatitis

- Acute mild hepatic steatosis is common in starvation and refeeding.
- Elevations in AST and ALT in starvation is a marker of severe malnutrition
 - Etiology not well understood

Organ Dysfunction: Hepatitis

- With refeeding, AST and ALT can rise, followed by Alk Phos and bilirubin, thought due to excessive dextrose calories causing fat accumulation in the liver cells: hepatic steatosis.
- Typically resolves after further refeeding, which sometimes has to continue at a slower rate.

Hypoglycemia

- Found both in starving state and in refeeding state.
- **In starvation**, caused by depleted hepatic glycogen reserves and gluconeogenesis substrates. Can be severe/life-threatening
- **In refeeding**, the increased glucose load causes insulin release which may overwhelm the depleted hepatic stores of glycogen and cause further hypoglycemia.
 - Don't discontinue PEG or TPN abruptly
 - Practically, check FS q4h for the first 24 hours of admission at least, then space as appropriate
 - Replete any fingerstick glucose <50 mg/dL

Organ Dysfunction

- Case:
 - A young woman with progressively worse AN develops new onset severe abdominal pain and vomiting shortly after eating either solids or liquids. LFTs, lipase, and a plain film are unremarkable.
- ***What else might cause these symptoms?

Organ Dysfunction

- **Superior Mesenteric Artery (SMA) Syndrome**
 - Characterized by compression of the third portion of the duodenum by the abdominal aorta and the overlying superior mesenteric artery due to loss of retroperitoneal fat.
 - Rare
 - Diagnosed by CT or small bowel follow-through
 - Often a jejunostomy tube must be placed to feed the patient beyond the obstruction until weight restoration “cures” the SMA syndrome

Clinical Issues

- **Early satiety/bloating**
 - “Every time I eat something my stomach feels bloated and painful.”
- **Constipation**
 - “I only move my bowels a few times a week, and I need to stay on my senna.”
- **Edema**
 - “The last time I was in the emergency room, they gave me some IV fluids and I swelled up like a balloon.”

Early satiety

- Once weight loss of 10-20 pounds occurs with pure food restriction, development of **gastroparesis** is almost universal.
- High fiber diets and prolonged time at <80% IBW worsen gastroparesis
 - Use of liquid food supplements as ½ the daily calories for the first week or two of refeeding can help as liquid emptying is generally normal.
 - Dividing intake into several snacks/smaller meals may help
 - Reglan 2.5-10mg AC may help
 - Generally resolves when weight is restored to 80-90% IBW
 - Rarely, a nuclear med gastric emptying study is indicated

Constipation

- Among restrictive anorexic patients, weight loss alone may slow colonic transit
- Extremely low caloric intake causes **reflex hypofunctioning** of the colon
- Hypokalemia and hypomagnesemia can contribute
- The cathartic colon syndrome is caused by long term use of stimulant laxatives (senna, bisacodyl) which can damage colonic nerve cells

Constipation

- Resolution occurs with weight restoration
- Remind patients of the broad range of normal bowel function
- Water drunk quickly can sometimes trigger the gastro-colic reflex. Low fiber (10 gm/day) diets can help
- Miralax or lactulose are acceptable. Mechanism is osmotic laxative.

Peripheral Edema

- During early refeeding, increased insulin secretion induces sodium retention by increasing renal tubular sodium resorption.
- Poor cardiac function may contribute
- Remember pseudo-Bartter's syndrome
 - RARELY do we use IV fluids in these patients: usually only when the bicarb is in the 30s or BUN is rising and patient can't/won't take in enough PO fluids
 - Severe edema can accumulate...if present and patient is stable, consider spironolactone diuresis, NOT lasix

Practical Points:

Standardize Admit Orders

- We're developing a computerized admit order set
- All patients on admission get:
 - Blinded height and weight
 - Fingerticks q4h
 - Minimal activity
 - Eating disorder diet managed daily by the RD's.
 - Calcium/Vit D and prn Miralax are standard
 - Labs including Prealbumin and Vitamin D levels
 - EKG, telemetry 24-7, and DEXA scan if none in past 2 years
 - OT/PT/wound care/chaplain consults
 - No sc heparin needed in this population, unless concurrent medical probs

Practical Points: VS

- Typical vital signs for a patient with severe AN include **hypothermia**, **bradycardia** (sometimes severe), and **hypotension**.
- We recalibrate “call for” orders in this population:
 - Temp <35, >37.5
 - HR <40, >100 or any acute change >10
 - SBP <80, >130 or any acute change >10

Practical Points: Lab abnormalities

- **CBC:**

- Often with chronic disease anemia, or iron deficiency anemia. We don't transfuse unless $\text{hct} < 20$ (unless bleeding). Minimize lab draws to reduce iatrogenic anemia. We use pedi lab tubes.
- Often with thrombocytopenia, reflecting a replacement of cell-producing bone marrow with a non-cell producing matrix due to starvation, improves with weight restoration
- Often with low WBC. We don't consider "neutropenic" until $\text{ANC} < 500$ usually.

Practical Points: Lab abnormalities

- Elevated **LFTS & electrolyte** abnormalities: As discussed before
- Low **T4**, high/normal **TSH** reflecting a euthyroid sick syndrome. We recheck in a few weeks and do not automatically replete with thyroid hormone
- High **BUN and CO2**: Often reflect a volume depleted state
- High **INR**: Nutritional deficiency and liver dysfunction

Practical Points: Lab Abnormalities

- The **albumin** paradox
 - Starvation per se does NOT cause low albumin
 - Many patients with pure restricting AN have normal albumin
 - If albumin is low, likely reflects concurrent medical problems (hepatic dysfunction, infection, etc.)

Practical Points: Lab Abnormalities

- Check daily chem 10 for a week, then move to QOD or even further spaced to “save blood”
- QOD LFTs while refeeding, then space further
- Check weekly CBC and INR unless there are acute abnormalities that need to be followed
- Vitamin K PO repletion is appropriate, 5-10 mg/day

Back to our vignette . . .

- The patient is admitted to ACUTE and seen by an internist, a registered dietician, the psychology team from ERC, and Dr. Mehler
- She's given spironolactone 50 mg daily, told to elevate her legs frequently, and over the course of a week and a half diureses 20 pounds of edema
- Glucoses are monitored every 4 hours, and juice is given when <50
- Wound consult is obtained for sacral decub
- Her diet is started at her BEE of around 1100 kcal/day and increased every few days to 1600 kcal per day with a goal of weight gain of about 2 lbs/week
- She was discharged to ERC at an approximate 70% IBW, after a 9 day hospitalization

In Sum . . .

- **Hospitalize** patients with severe AN, preferably in a place where a multidisciplinary team has experience managing these patients
- **Recognize** typical physiology/biochemistry in the severely starved state
- Know what to watch for to diagnose and treat **complications of refeeding**
- Contribute to **prevention** of eating disorders