433 Teams

MEDICINE

11 | Obesity

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Objectives:

1. Definition and classification of obesity
2. Pathogenesis of obesity
3. Approach to an obese patient
4. Management of obesity: lifestyle, medical and surgical

Obesity:

- Is Abnormal or excessive fat accumulation in adipose tissue, to the extent that health is impaired.
- Presence of an abnormal absolute amount or relative proportion of body fat.

Surrogate measures of adiposity:

1. Ideal body weight
2. Weight
3. Anthropometric measures
4. BMI, **(Relatively reliable except in):**
    1. Extremes of age or height.
    2. Very fit individuals with muscular build.

WHO recommended definition of obesity:

5.26 Quantifying obesity with body mass index (weight/height²)

BMI (kg/m²)	Classification*	Risk of obesity comorbidity
18.5–24.9	Normal range	Negligible
25.0–29.9	Overweight	Mildly increased
> 30.0	**Obese**	
30.0–34.9	Class I	Moderate
35.0–39.9	Class II	Severe
> 40.0	Class III	Very severe

Classification "according to body fat distribution":

1. Central Obesity (visceral):

- Central obesity is associated with more metabolic disease:
  1. DM2
  2. HTN
  3. Dyslipidemia
- More common in men.
- **The intra-abdominal fat draining into the portal vein and directly to liver. thus many factor that are released from adipose tissue (including free fatty acids such as tumor necrosis factor -alpha, adiponectin, resistin and steroid hormones) may increase in the liver and induce insulin resistance and promote DM2.**
- Diagnosed by:
  - MRI
  - Dual X-ray absorptiometry (DEXA).
  - Single CT slice L4/L5
  - Waist: hip ratio
  - Waist circumference

Central (Visceral) obesity is more dangerous and associated withcomplications (DM and HTN)

The easiest and cheapest method to measure central obesity is:

- **Waist : Hip ratio**
- **Waist circumference**

2. Gynoid, (pear-shaped):

a. **Fat deposited around the hips or gluteal region.**
b. **More common in female.**

Mechanism of (hunger):

1. **Signals from peripheries are carried out by neurotransmitters and hormones to CNS in presence or absence of food.**
2. **Signal from fat by hormone leptin to hypothalamusto reduce food intake and increase sympathetic activity and energy expenditure (obese people are leptin resistant). In other word they have more leptin but it doesn't work).**
3. **Gastric distension and contraction send signal for satiety and hunger.**
4. **Fall in blood sugar send signals to CNS for hunger.**
5. **Sympathetic activity from food thermogenesis leads to reduce food intake.**
6. **Ghrelin (hunger"orexigenic"hormone)Secreted in the stomach and acts onhypothalamus to stimulate appetite (Peak before meal and decrease after).**

Etiology & Pathogenesis:

- Imbalance between energy intake and energy expenditure.
- Body weight is ultimately determined by the interaction of:
  1. Genetic
  2. Environmental and
  3. Psychosocial factors
  4. energy expenditure
  5. food intake

Classification "according to etiology":

Neuroendocrine disease	• **Ventromedial hypothalamus damage:** - Tumors - Inflammatory lesions • **Other hypothalamic disease** • **cushing disease**
Drug-induced	• **Hyperinsulinism** - Insulin - Sulfonylureas • **Antidepressants** • **Antiepileptics** • **Neuroleptics**
Dietary	• High carbohydrate diet • Hi fat diet
Reduced energy expenditure	• **Resting metabolism:** - 800 to 900 kcal/m2/24hr, Females < Males - Declines with age. • **Physical exercise:** - ~ 1/3 of daily energy expenditure, most easily manipulated. • **Dietary thermogenesis (thermic effect of food):** - In the obese, the thermic effects of food are reduced (especially in patients with diabetes). • **Adaptive thermogenesis**
Genetic factors	• **Dysmorphic or syndromic obesity:** - Bardet-Biel syndrome - Alström syndrome - Carpenter syndrome - Cohen syndrome - Prader-Willi syndrome • **Single-gene cause of obesity:** - Leptin and leptin gene deficiency - POMC deficiency • **Genetic defects with nonsyndromic obesity :** - Melanocortin receptor system abnormalities

Complications of Obesity:

A. HTN, heart disease, hyperlipidemia, DM2, stroke, osteoarthritis, liver disease, cancer, obstructive sleep apnea and depression.
B. Obesity is associated with a significant increase in mortality.

5.23 Complications of obesity

Risk factors	Outcomes
'Metabolic syndrome' Type 2 diabetes Hypertension Hyperlipidaemia	Coronary heart disease Stroke Diabetes complications
Liver fat accumulation	Non-alcoholic steatohepatitis Cirrhosis
Restricted ventilation	Exertional dyspnoea Sleep apnoea Respiratory failure (Pickwickian syndrome)
Mechanical effects of weight	Urinary incontinence Osteoarthritis Varicose veins
Increased peripheral steroid interconversion in adipose tissue	Hormone-dependent cancers (breast, uterus) Polycystic ovary syndrome (infertility, hirsutism; p. 760)
Others	Psychological morbidity (low self-esteem, depression) Socioeconomic disadvantage (lower income, less likely to be promoted) Gallstones Colorectal cancer Skin infections (groin and submammary candidiasis; hidradenitis)

Management:

1. Diet, exercise, lifestyle modification are mainstays of treatment.
2. Drug therapy—for patients who have not succeeded in losing weight with diet and exercise, **(Orlistat**is a first -line agent).

Orlistat

A. It is a lipase inhibitor, reduces the absorption of dietary fat
B. Lowers Cholesterol (4-11%) & LDL (5-10%).
C. Major Contraindication: Chronic malabsorption Syndrome, Cholestasis, Pregnancy and breast feeding.
D. Max. period of treatment is 2 year.
E. Eating fat with Orlistat will cause: Diarrhea, Fecal incontinence (social problem)

3. Reassess for who Don't Lose Weight:

- Understanding and compliance with diet, physical activity, and drug regimen
- Accuracy of weight recordings
- Possible Fluid retention (salt intake, etc.)
- Changes in medical condition
- Motivation for change
- Social and personal stress.

Management: (Firstly, History and Investigations):

- To confirm that he is not taking any medication which cause obesity.
- To exclude endocrine problems
- To rule out complication

NOW START MANAGEMENT!!

4. Bariatric surgery:

A. Bariatric surgery is effective in reducing comorbidities associated with obesity, including hypertension, diabetes, obstructive sleep apnea, and hyperlipidemia. This translates into a 29% reduction in mortality. **Only indicated in patients who have earnestly tried other means of losing weight and have beenunsuccessful**.
B. Best evidence is for patients with BMI over40.
C. Bariatric surgery is based on 2 mechanisms: restriction of intake (via a small stomach reservoir) and malabsorption (via decreasing small bowel length). Restrictive techniques are technically easier, have lower complication rates, but result in less weight loss than malabsorptive techniques.
D. Most common procedure is the laparoscopic Roux-en-Y gastric bypass. The laparoscopic adjustable gastric banding (LapBand) has fewer complications, is reversible, but is not as effective in achieving weight loss as compared with the gastric bypass.

Problems of surgery:

- Most of them will end up with depression
- Dietary complications such as vit B12 deficiency and Iron deficiency.

Questions

1. Body mass Index (BMI) gives a measure of relative weight adjusted for height. The healthy range of BMI is between?
   A. 15-18.4
   B. 18.5-25.9
   C. 25-29.9
   D. 30-34.9

2. Which of the following is not a secondary cause of obesity?
   A. Hypothyroidism
   B. Cushing's syndrome
   C. Insulinoma
   D. diabetes mellitus

3. Rapid weight loss is a cause of:
   A. Electrolyte imbalance
   B. Diabetes mellitus
   C. Dehydration
   D. Osteoporosis

4. Orlistat is used when:
   A. BMI > 40.
   B. When trial of weight and exercise fail
   C. BMI > 30with co-morbidities
   D. Well-informed and motivated patients

5. Which one is not a type of Bariatric restrictive surgery ?
   A. Vertical banded-gastroplasy
   B. Gastric banding
   C. Roux-en-Y gastric bypass

Answers: B, D, A, B, C.

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