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Obesity and Diabetes Mellitus

*Raman PG

M.G.M. Medical College, India

Introduction

History Taking on Obesity

• History of drugs which increase weight, eg. Steroids, tricyclic antidepressants and atypical antipsychotics

• History of alcohol and food preferences

• Whether emotionally triggered

• Depression, anxiety, mental illness, psychological trauma

• Drug abuse or smoking

Measurement of Obesity

A. **Body weight-** In epidemiological studies it is conventional to accept +2 Standard deviation from median weight for Height as a cut off point for obesity.

Other indicators:

1. Body mass index (Quetetlet's index)
   = Weight (in Kg) / Height^{2}mts.

2. Ponderal index
   = Height (in cm) / Cube root of body weight (in Kg)

3. Broca index
   = Normal Body Weight (kg) = [Height (cm) - 100]

Example: What is the ideal weight for a man weighing 175 centimeters?

Height = 185 cm
Normal weight = 185 - 100 = 85 kilograms
Ideal body weight = 85kg - 10% = 76.5 kilograms

4. Corpulence index
   = Actual weight / Desirable weight

   Should not exceed 1.2

B. **Skin fold thickness** – Measured with harpender skin calipers, at 4 sites–mid triceps, biceps, sub scapular and suprailiac regions. The sum should be less than 40 in boys and 50 in girls.

Body Mass Index or BMI

It is the measurement of choice for many physicians and researchers studying obesity. BMI uses a mathematical formula that takes into accounts both a person's height and weight. BMI equals a person's weight in kilograms divided by height in meters squared (BMI=kg/m^2)

***Corresponding author:** Raman PG, M.G.M. Medical College, India. E-mail: drpgraman@yahoo.com **Mob**: (91)9302104798

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Table 1: Obesity Classification

Classification	BMI
Underweight	<18.50
Normal	18.50 to 24.99
Overweight	>25
Pre-obese	25-29.99
Obese Class-I	30 to 34.99
Obese Class-II	35 to 39.99
Obese Class-III	> 40

Risk of associated disease according to BMI and waist size			
BMI		**Waist less than or equal to 40 in. (men) or 35 in. (women)**	**Waist greater than 40 in. (men) or 35 in. (women)**
18.5 or less	Underweight	-	No risk
18.5 – 24.9	Normal	-	No risk
25.0 – 29.9	Overweight	Increased	High
30.0 – 34.9	Obese	High	Very High
35.0 – 39.9	Obese	Very high	Very high
40 or greater	Extremely obese	Extremely high	Extremely high

Causes of Obesity

Genetic syndromes causing obesity

- Monogenetic disorders
    - Leptin mutation causing Leptin deficiency
    - Melanocortin four mutation
- Prader willi syndrome
- Bardet – Biedl syndrome

Causes

- Positive caloric balance
- Sedentary life-style
- Genetic predisposition
- Environmental causes

Anatomic manifestations

- Adipocyte hypertrophy
- Visceral, pericardial, perivascular and other periorgan
- Adipocity
- Growth of adipose tissue beyond its vascular supply
- Increased number of adipose tissue cells
- Ectopic fat deposition in other body organs

Pathophysiological manifestation

- Impaired adipogenesis
- Increased free fatty acid
- Increased leptin TNF-α and increased mineral corticoids
- Increased pro-inflammatory responses

Clinical manifestations

- Hyperglycemia
- High blood pressure
- Increased VLDL, triglycerides and apo B (small dense LDL)
- Low HDL-C

• Metabolic syndrome

• Atherosclerosis

• Fatty liver

• Hyperandrogenemia in women

•Hypoandrogenemia in men

•Cancer

Table 3: Clinical manifestations and conditions associated with PCOS

Associated conditions	Prevalence
Obesity	42% to 80%
Insulin resistance	71% To 77%
Impaired fasting glucose	31.1% to 35%
Type 2 diabetes mellitus	6.6% to 7.5%
Arterial hypertension	9% to 21%
Dyslipidemia	32% to 46.3%
Metabolic syndrome	43% to 47.3%

Medical conditions triggered by obesity

• Insulin resistance

• Type-2 diabetes

• Obstructive sleep apnoea

• Liver steatosis

• Polycystic ovary disease

• Depression

Differential diagnosis of obesity

• Cushing's disease

• Hypothyroidism

Hormonal changes in obesity

• Low SHBG and free testosterone

• Increased oestrogen

• Low LH, FSH

• Increased cortisol

• Vitamin D deficiency

Adipose tissue produces the following

• Leptin

• PAI-1

• NF-κβ

• Adiponectin

• TNF-α

• Resistin

• IL-6

Hazards of Obesity

1. The increased body fat, increased central fat deposits and increased weight gain are all associated with increased risk of death. Four large and many smaller epidemiologic studies have supported these findings. The studies include.

a. Build and BP study of 1979.

b. American Cancer Society Study.

c. Norwegian population study.

d. Nurses health trial.

A. Obesity and heart disease: Data from nurse's health trial, the risk of CAD in US females increase 3.3 fold with Body Mass Index > 29 Kg/m^2 when compared with females with Body Mass Index< 21Kg/m^2. Of more importance is the inverse relationship between HDL cholesterol and Body Mass Index. Low HDL increase risk of CAD. Obesity increases the cardiac work which will lead to cardiomyopathy and heart failure.

B.Obesity and Respiratory System: The major effects are increased residual volume associated with increased abdominal pressure on diaphragm. Sleep apnea is associated with obesity. The hypothesis is that the increase neck circumference and fat deposits in pharyngeal area may lead to sleep apnea.

C. Obesity and DM- Obesity is strongly associated with diabetes.

D. Gall bladder disease – Obesity leads to Cholelithiasis. For example:

If there is 10 Kg increase in body fat, the cholesterol increases to the tune of as much cholesterol in yolk of one egg. This goes in bile and leads to stone formation.

E. Certain cancers are significantly increased in obesity
In Males - Neoplasm's of colon, rectum and prostate.
In Females - Neoplasm's of reproductive tract and gall bladder.

This may be due to increased production of estrogen by adipose tissue.

F. Changes in Bones, joints, muscles, connective tissue and skin. Obesity leads to:-
- Osteoarthritis

- Acanthosis nigricans

- Hirsutism may reflect altered reproduction status.

G. Endocrinal - The most important are reproductive. Irregular menses and frequent anovulatory cycles are seen.

H. Psychosocial function – Obesity is a stigmatized condition. They are exposed to the consequences of public disapproval.

Leptin In Pathogenesis of Obesity and Type-2 DM

Leptin is a product of the ob'gene and is secreted by fat cells. It has role in appetite regulation, energy expenditure and possibly modulation of insulin sensitivity. The site of action seems to be hypothalamus. Leptin is closely related to body mass index and waist circumference and also to fasting and two hour insulin levels. Studies also suggest its role in type-2 DM and insulin resistance. The hypothalamic mediated resistance to leptin causes a rise in leptin and initiates hyperinsulinemia and insulin resistance in obesity.

Increased insulin levels leads to increased body fat, dyslipidemia (due to lipogenesis and selective insulin resistance in muscles). The increase adipose tissue produces more leptin but due to down regulation of leptin receptors in hypothalamus unregulated and continuous feedings occurs and vicious cycle of hyperinsulinemia, more fat and more leptin resistance is perpetuated. Other possible mechanism is that increased fat predominantly central accumulated due to insulin resistance may be responsible for increased leptin production. This leads to down regulations of leptin receptor and loss of appetite control and hence perpetuation of vicious cycle.

Obesity is frequently associated with low grade inflammation of adipose tissue and the increase in adipose tissue macrophages is linked to an increased risk of type-2 diabetes.

Fat People Have More Fat Cells than Skinny People

Obese subjects have on the average a 40% increase in cell size and an increase of 190% in cell number. Studies document that the number of adipocytes remain constant in an adult.

Thus caloric deprivation results in a decrease in cell size to normal, but the adipose hypercellularity persists. In man, it is estimated that adipose cells are still increasing in number upto early adolescence. Thus the cell numbers increase in infancy and adolescence. The obese patient has an excess number of adipocytes which are enlarged as well; it is the effect of early feeding experience and the interplay of such experience with genetic and early psychological events really where the answers to endemic obesity are to be found.

It was Jules Harsh who devised the method of determining both the number and size of human adipocytes. Could restriction and reformulation of early feeding aid in the prevention of obesity in adult life? Answers to such questions are still being sought.

Hormonal Changes in Obesity

Tissue	Abnormalities associated with obesity
Adrenal	Increases cortisol turn over Increase cortisol response to stress in female energy expenditure
Thyroid	Normal levels. Possible association between T3 and resting
Gonadal	Decrease Sex Hormone Binding Globulin.
Prolactin	Normal basal levels Decrease stimulated levels.
Growth Hormone	Decrease Growth Hormone secretion Insulin Like growth factor Decrease in IGF Decrease in IGF
Endocrine Pancreas	Increasing fating levels
Insulin	Peripheral tissues insulin resistance Altered β cell pulsatility
Adipose tissue	Increase in tumor Necrosis Factor alpha, Leptin levels correlation with fat mass.
Men	Increase aromatization of adrenal androgens into estrogens Decrease in Free testosterone. Pattern of hypogonadotrophic hypogonadism with severe disease.
Women	Increase aromatization of adrenal androgens into estrogens. Increase in free testosterone

Table 4: Factors that mediate the intersection of metabolism and immunity in obesity

Factors	Metabolic regulation	Effects
TNF-α	Increased	Promotes insulin resistance
IL-6	Increased	Promotes insulin resistance
Leptin	Increased	Central anti-obesity action; suppresses appetite; multiple effects on immune function
Adeponectin	Decreased	Anti-inflammatory; promotes insulin sensitivity; stimulates FA oxidation
Vistatin	Increased	Early B-cell growth factor; insulin mimetic
Resistin	Variable	Induce endotoximea and inflammation; promotes insulin resistance
IL-1	Increased by hyperglycemia	Pro-inflammatory; regulates insulin secretion; involved in central leptin action
IL- 1Rα	Increased	Anti-inflammatory, opposes leptin action

Factors	Metabolic regulation	Effects
IL-8	Increased	Pro-atherogenic
IL-10	Increased	Anti-inflammatory
IL-18	Increased	Pro-atherogenic
MCP-1	Increased	Pro-atherogenic
MIF	Increased	Produces IR; inhibit macrophage migration; stimulates adipose growth
M-CSF	Increased	Produces IR; inhibit macrophage migration; stimulates adipose growth
TGF-β	Increased	Inhibit adipose tissue development
c-reactive protein	Increased	Pro-inflammatory, atherogenic and risk factor for DM
Haptoglobin	Increased	Pro-inflammatory

*FA= fatty acid; MCP-1 = monocyte chemotactic protein-1; MIF= macrophage migration inhibitory factor.

Ref: Kathryn E, Wellen and Gokhan S. Hotamisligil, Inflammation, stress and diabetes, The Jr. of Clinical Investigation, Vol.115,No.5,pg 1111-1119

DM & Obesity

A central body fat distribution with a high waist hip ratio and an android and apple shaped habitués in association with increase insulin resistance, type-2, hyperlipidemia and premature mortality.

A peripheral gynaecoid or pear shaped distribution with low waist hip ratio is found in individuals who exercise and does not carry these diseases.

The insulin resistance has been demonstrated in obese patients using hyperglycemic and euglycemic insulin clamp technique. The insulin mediated glucose metabolism is decreased by 50% in obesity. Fatty acid metabolism is enhanced in obesity and type-2 DM and this may interfere with glucose utilization. Overweight in a super imposed factor which perhaps by demanding excessive insulin secretion to overcome insulin resistance contributes to β cell exhaustion and a final decline to type-2 DM. This is supported by the demonstration that insulin secretion in impaired in all patients with type-2.

Adipose Tissue as an "Endocrine Organ"

I. Leptin – It is a 16-K dalton protein produced exclusively in the adipocytes.

II. Tumor Necrosis factor alpha – Secreted by not only the classic inflammatory cells but also by adipocyte. It has been postulated to be responsible for insulin resistance by alternation in the phosphorylation status of insulin receptors substrate one (IRS-1) and to after insulin receptor

The relationship between body mass index and risk of diabetes was shown by the Health Professionals study. It was found that when body mass index was less than 24 Kg/m2. The risk was lowest. At a body mass index of 35 Kg/m2. The relative risk increased 40 fold of 4000%.

Management of Obesity

Obesity Treatment

Approaches can be conservative like diet therapy, pharmacotherapy, behavior modification.

Invasive therapy

Bariatric surgery, gastrointestinal electrical stimulation, invasive endoscopic techniques like intragastric balloons, bezoars.

Diet

Low carbohydrate, high protein diet, vegetarian diet, diet based on plant and fibrous foods rich in complex carbohydrates with low glycemic index when used, increase satiety and lowers BMI. Fiber based dietary supplement also helps. But diet regimen has very little sustainability. 5% weight reduction for more than a year is considered a success.

Reasons for inability to lose weight

- If patient is on steroids, tricyclic antidepressants and atypical antipsychotics
- Dietary shortfalls
- Emotionally triggered disordered eating behavior
- Bringe eating

• History of depression, anxiety, mental illness and psychological trauma or abuse, smoking, alcohol and substance abuse

Table 5: A) Effect of weight reduction on glucose tolerance

Trial –	Body Mass Index	Body Mass Index
1. Health Professionals Study	24kg/m2. Low risk of DM	35 kg/m2. Risk increase 40 fold or 4000%
2. Nurses Health Trial	22kg/m2. Low risk of DM	>35 kg/m2. Risk increase by 60 fold or > 6000%

A) Effect of weight reduction on glucose tolerance

Using the body mass index at age 15 years, Colditz and Coworkers showed that a 20 kg weight gain increased risk for diabetes by 15 fold where as a weight reduction of 20 kg decreased risk to almost zero.

In a Swedish obesity study; Sizostrone and colleagues observed that diabetes was present in 13% to 16% of their obese subjects. Of those who underwent gastric by pass and subsequently lost weight 60% of subjects with diabetes were cured and diabetes developed in only 0.5% of those who did not have diabetes at baseline. In contrast in the obese control group who lost no weight there was a small (16%) cure rate and a 7.8% incidence of new cases of diabetes.

Thus we see that weight reduction decrease the incidence of diabetes.

B) Role of Diet and Exercise

Diet – The standard weight reduction diet is of 1000 cal/day. This diet helps patients to loose 1 to 2 pound/wk. Under very careful medical supervision in hospitalized patient 200 cal. Semi starvation diets can be used for 1 week followed either by diet containing 1200 cal or very low protein sparing diet 600 to 800 cal/day.

Very low calories (protein sparing) modified diet (600 to 80 cal/day) includes

1. 1.5 gm protein/kg or 75 gm protein 50gms carbohydrates.

2. Potassium 30 mEq/day.

3. Multivitamins and minerals.

4. Sodium Chloride 5gms.

5. Calcium Carbonate 4 tab/day.
6. 1.5L fluids.

Patients on very low caloric diet should monitor urine for ketosis biweekly electrolyte estimation. Should undergo Electrocardiography after every 3 months, particularly after losing 50 1bs (22.5 kg) to check cardiac irregularities.

Exercise

From their Meta analysis Epstein and Wing reported that exercise training was modestly effective at reducing body weight (0.09Kg/wk). Exercise induced weight loss was dependent on weekly exercise energy expenditure, initial body mass and frequency of exercise.

Table 6 Other Studies

Meta analysis Epstein & Wingh	Mode Walk / run	Frequency 2.5 times / wk	Duration 6-20 wks	Weight loss 0.09 kg / wk
Bailor & Keesay	Walk/run Cycle	3-4 times/wk	Ave. 6.8 wks	0.1 kg/wk
Garrow & Summerbell	Walk/run cycle	3-4 times/wk	8-52 wks	0.1 kg/wk
Hadijolova et al	Sports	Daily	45 days	0.6kg /wk
Lee et al	Military Training	5 days /wk	5 mth	1.8 kg/wk

Recently it has been found that high intensity exercise but not prolonged endurance type training increase skeletal muscle beta hydroxyacyl CoA dehydrogenase activity, a key enzyme in beta oxidation pathway of fatty acid metabolism. This can promote the oxidation of fatty acids not only during exercise but during non exercising periods, thereby promoting further fat loss.

Exercise training can reduce adiposity. Obese patients should have minimum of 150 minutes of moderate exercise per week. Bariatric surgery is becoming more useful treatment for obesity. It helps to reach BMA target, improve metabolic parameters, reduce inflammatory markers and improve insulin sensitivity.

Metformin

Metformin is a dimethylbiguanide. It belongs to the class of biguanides.

Mechanism of action – Metformin therapy improves insulin sensitivity as shown by a reduction in fasting plasma glucose and insulin concentration. It is not effective in absence of insulin. In patients of type-2 glucose lowering is attributed mainly to low hepatic glucose output and increased peripheral glucose uptake. Several other actions may contribute such as increased intestinal use of glucose and decreased fatty acid oxidation. Metformin also increase translocation of GLUT-1 and GLUT-4 isoarm of glucose transporters in different types of cells, and it prevents the development of insulin resistance in cultured hepatocytes and adipocytes for long periods to high insulin concentrations.

Comparison of Sulphonylurea and Metformin Therapy

Metformin and Sulphonylurea cause similar decrease in fating plasma glucose concentration in patients with Type-2 DM.

Both sulphonylurea and insulin can cause weight gain but this does not occur with metformin. Sulphonylurea can induce hypoglycemia, whereas this is rare with metformin therapy. Therefore metformin has antihypoglycemia action where as sulphonylurea and insulin has hypoglycemic action. Sulphonylurea increase fasting plasma insulin concentration where as metformin may decrease it. In theory, the reduced plasma concentration of insulin or plasminogen activator inhibitor type I could decrease the risk of macrovascular disease.

Thus we see that metformin is complimentary to sulphonylurea and dietary therapy and represents a useful additional drug for management of type-2.

Appetite reducing agents in obesity

There are several groups of drugs used as appetite suppressants –

Bulk e.g. – psyllium promotes gastric filing

Psychogenic e.g. – impramine and amitryptylline

Anorexia (amphetamines) – most effective anorexics e.g.–

Dextroampheteramine and methamphetamin.

Anticonvulsant – Phenytoin

Anti-histamines - Chlorpheniramine

Grape Fruit Pill

Carbohydrate wafers

Hormones

A) Anterior pituitary hormones. Human Chorionic gonadotropin

B) Vasopressin

C) Starch Blockers (alpha amylase inhibitors)

D) Thyroid hormones.

Hypolipidemic agents – Gemfibrozil – 600mg twice daily

Antidepressive drugs like fluoxitine hydrochloride may be useful in some and help in weight reduction. Oral sustained release fenfluramine 20 mg was used to suppress appetite. Now the drug is banned due to its side effect pulmonary hypertension.

Pioglitazone and Ramipril improve adipose tissue function by increasing adiponectin secretion and may contribute to reduction in vascular disease. But pioglitazone causes weight gain.

Table 7: Newer drugs in obesity management

S.No.	Name of the drug	Side effects	Remark
1	Qunexa (Topiramate + Phentermine)	Constipation, tingling, dry mouth, altered taste and insomnia	Topiramate is anti epileptic and modulator of gamma amino butyric acid
2	Contrave (Bupropion and Naltrexon)	Nausia, constipation, vomiting, dry mouth	Bupropion is an anti depressive which releases MSH and produce ano-rectic effect
3	Empatic (Bupropion and Zonisamide)	Nausia, Headache and Insomnia	Zonisamide is used in Partial seizures, modulate Sodium channel and increase dopamine and Serotonin
4	Pramlintide and Metreleptin	-	Amylin is a short term Satiety signal and Lepin is a long term Satiety signal
5	GLP-1 analogue (Liraglutide - long acting OD) Extenatide (GLP-1 analogue, short acting)	Nausea, vomitting	Affects at hypothalamic level and produce Satiety
6	Orlistat and Cetilistat	Fatty diarrhoea flatulence	Inhibit, pancreatic lipase. Cetilistat has less of side effects
7	Velneperit, given orally 800 mg.	Nasopharyngitis, Otitits, URI, Sinusitis, Headache, increases HCT, Hb and RBC	Hypothalamic, NPY receptor antagonist
8	Thyroid hormone receptor agonist	Cardio vascular side effects are less	There are two receptors - TR and TR-β. TR-β is seen in liver and adipose tissue and has beneficial effect on metabolism
10	Bio active food ingredients	-	Promote thermogenesis and decrease weight by 2-5%
11	Flavonoid	-	Decrease IR, visceral mass. Act on AMPK pathway
12	Tantalus	Accelerate gastric emptying	Gastric electrical stimulation

Insulin required in type-2 only when

1 Oral hypoglycemic Agent failure

2 Intercurrent illnesses
- MI
- Surgical disease
- Occult infection

Problem of insulin therapy in obese DM

As seen earlier due to insulin resistance they require a higher dose of insulin. With hyperinsulinemia patient gets weight gain.

The dyslipidemia of obesity is best managed with total fat reduction, TG levels are reduced with weight loss and if necessary, fibrate therapy. RNYGB, LAGB and LSG are most commonly performed procedures, each of which has different anatomic and physiological properties. Most useful procedure is RNYGB which is superior for achieving weight loss as well as in improving metabolic parameters.

V-Block Therapy in diabetes and obesity

Vagal Nerve Modulation device is thought to be effective for the treatment of obesity. By using very high frequency but low energy electrical pulses, effects of

vagotomy are obtained. The maestro rechargeable system consists of electrical pulse generator, wire and electrodes implanted surgically in the abdomen. Stomach emptying and fullness signaling goes by vagus nerve to brain to satiety and hunger centre. Adverse effects of this device are nausea, pain at the site of implantation, heart burn, problems in swallowing, belching and chest pain.

Pharmacological therapy

• Adrenergic or serotonin agonist produce appetite suppression. Plain Phentermine, Phentermine Plus, Topiramite are used. Side effects include rise of BP and heart-rate.

• Drugs deactivating pancreatic lipase – Orlisitat.

Invasive antiobesity therapy

• Laparoscopic adjustable gastric banding, vertical-horizontal gastroplasty, gastric bypass (more effective)

• Cost, invasiveness, reversibility, adjustability, complications rate, recovery period, abrupt change in lifestyle and eating habits are important facts influencing selection of type of bariatric surgery.

Gastric lumen reduction

Transoral, endoluminal, gastroplasty. Food malaborption (duodeno, jejuna bypass) is an endoscopically implantable, impermeable polyethlene sleeve mimicking bariatric surgery. Short term weight loss and metabolic benefits occur. Complications are device migration and anchor dislocation. Continuous abdominal pain and nausea occurs.

Space occupying devices

They are endoluminal, non-surgical, reversible and repeatable treatment of obesity devices which reduce gastric volume and decrease food intake. Other effects are change in gastric motility and hormonal profile. There is 10% weight loss, patient adopts new eating habits. Balloons are inflated with 500-700 cc of saline or methylene blue solution. Other balloons are totally implantable. Intragastric prostheses – endogut, semistationary antral balloon, slimmed gastric balloon and butterfly.

Gastrointestinal electrical stimulation of stomach delay gastric emptying, affect vagal efferent impulses, antral muscle contraction and cause 40% weight loss. Improves Gherelin secretion in appetite disorders.

Bezoars

They are collection of foreign bodies, can be located in oesophagus, stomach, intestine, colon and rectum. Pharmoco Bezoars are made up of Aluminium Hydroxy Antacid, bulk forming laxatives, cellulose based tablets and vitamin. Complications are epigastric discomfort, nausea and vomiting. Complications like bleeding, gastric outlet obstruction, pressure necrosis and gastric ulcer can occur. Small bowel bezoars cause Rapunzel syndrome. There is mechanical obstruction at hepato pancreatic ampulla, iron deficiency, anaemia and frontal alopecia. Digestive resistant polyethylene bezoars are used recently.

A Sugar Tax – A Bitter Pill to reduce Obesity

WHO recently urged the Governments to levy taxes on sugar sweetened beverages to end childhood obesity. Other interventions suggested are increasing physical activity and improving access to healthy food.

Hungary, France, Finland and Mexico are among the many countries that have taken to such measures. 34 US states and Districts of Columbia have food taxes that affect sugar sweetened drinks. Mexico which has highest prevalence rates for obesity in the world (33% between ages of 2 to 8) has demonstrated significant reduction in consumption of sugar after taxation. It was found there is 6% drop in average quantity of purchase of unhealthy drinks. The risk of childhood obesity is greater in low socio-economic group. In 2014, 41 million children were either overweight or obese.

India may be one of the biggest contributors to the global pool of childhood obesity. India also has underweight children – 14% in Sikkim and 44% in Bihar. This is a big concern. Children who are at low birth weight are at a greater risk of becoming overweight and obese when they consume energy rich diets and have a sedentary lifestyle.

How diabetes improves with bariatric surgery?

• Caloric or energy restriction

• Increased GLP-1 secretion

What are the complications of bariatric surgery?

• Gastric pouch dilatation due to disordered eating traits

Indications	Contraindications
BMI equal or more than 35 kg/m^2 with comorbidities or over 40 kg/m^2 without any comorbities T2DM less than 10 years with BMI over 30kg/ m^2	Drug or alcohol abuse Psychiatric illness Lack of commitment During Child bearing age, malabsorptive surgery is contraindicated

• Late dumping syndrome causing post prandial reactive hypoglycemia due to dense carbohydrate intake

How will you treat late dumping syndrome?
• Treated by dietary modifications, acarbose, calcium channel antagonist and somatostatin analogue
What histopathological changes are seen in pancreatic islets after bariatric surgery?
• Extensive hypertrophy of islets and development of nesidioblastosis (insulinoma like symptoms with excessive islet cells hyperplacia of pancreas)

What are the psychosocial problems after bariatric surgery?
• Depression, suicide, self harm and divorce

What is a liver shrinkage diet?
• It is a low caloric diet given to shrink liver glycogen

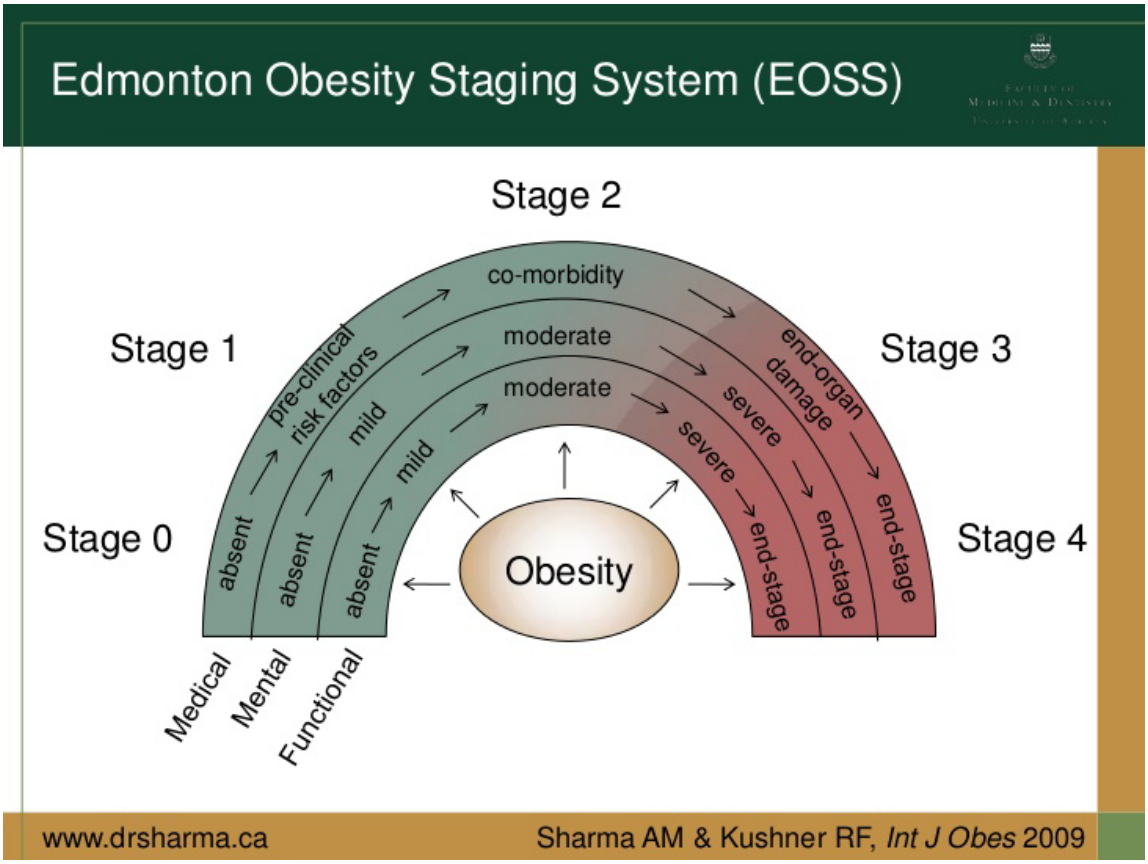
Table 9 Comparison of the characteristics of the two most common surgical interventions

Characteristics	Laparascopic adjustable gastric banding- LAGB	Laparascopic Roux en Y gastric bypass – RYGB
Principal technique	Reversible and restrictive	Irreversible, restrictive and malabsorptive
Weight loss	About 50% EWL at 1 year	About 70% EWL at 1 year
Type 2 diabetes	Higher than with diet but less than RYGB	Improvement or remission shortly after surgery (40-80%)
After care	Repeated follow up for band adjustments	Lifelong mineral and vitamin supplementation
Common complications	Band erosion, ulceration, band slippage, pouch dilatation and wound infection	Anastomatotic leaks, internal hernias, pulmonary embolus, sepsis and wound infection
Side effects	Nausea, vomiting and dyspepsia	Nausea, vomiting, dumping syndrome, vitamin deficiencies, malnutrition
Surgical mortality	0.05%	0.5% with higher risk of intra operative complication
Revision	Required in 10-25%, this is typically to RYGB	Less common and technically difficult

What is Edmonton Obesity staging system?
• This is a clinical tool to compliment BMI.

Conclusion

Pathogenesis and hazards of obesity are discussed. Type 2 DM patient may be obese. If they lose weight diabetes status may be improved. When diet fails metformin is the drug of choice. Obese type 2 Diabetics may need insulin occasionally due to sepsis or during surgery. When insulin is used it is required in higher dose due to peripheral insulin resistance.



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