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QUESTIONS :

1. What do you understand by primary obesity
2. How does drug therapy and congenital syndrome affect secondary obesity
3. Discuss the aetiology of cancer and its molecular basis

ANSWERS

1. Obesity is a medical condition in which excess body fat has accumulated to an extent that it may have a negative effect on health. People are generally considered obese when their body mass index (BMI), a measurement obtained by dividing a person's weight by the square of the person's height, is over 30 kg/m2; the range 25–30 kg/m2 is defined as overweight. Primary obesity is due to excessive energy intake and too little consumption. It's also known as diet-induced obesity and has the largest proportion in all types of obesity( about 95%).Primary obesity is characterized by a normal or increased growth rate with an acceleration of bone age maturation. When longitudinal growth slows down in the presence of obesity, a hormonal disturbance should be sought. Despite normal growth, simple obesity is characterized by a reduced growth hormone secretion evaluated by standard provocative tests, the administration of growth releasing hormone or spontaneous 24-hour secretion. It is also associated with high insulin and insulin-like growth factor I levels which may interfere in the complex endocrine interactions. It is not associated with clinical condition.
2. HOW DRUG THERAPY AFFECTS OBESITY.

• Medication treatment of obesity should be used only in patients who have health risks related to obesity. Medications should be used in patients with a BMI greater than 30 or in those with a BMI of greater than 27 who have other medical conditions (such as high blood pressure, diabetes, high blood cholesterol) that put them at risk for developing heart disease. Medications should not be used for cosmetic reasons.

• Medications should only be used as an adjunct to diet modiﬁcations and an exercise program.

• Like diet and exercise, the goal of medication treatment has to be realistic. With successful medication treatment, one can expect an initial weight loss of at least 5 pounds during the ﬁrst month of treatment, and a total weight loss of 10%-15% of the initial body weight. It is also important to remember that these medications only work when they are taken. When they are discontinued, weight gain often occurs. medications also decrease appetite and create a sensation of fullness. Hunger and fullness (satiety) are regulated by brain chemicals called neurotransmitters. Examples of neurotransmitters include serotonin, norepinephrine, and dopamine. Anti-obesity medications that suppress appetite do so by increasing the level of these neurotransmitters at the junction (called synapse) between nerve endings in the brain.

Various pharmacologic agents, referred to as anorectic drugs, are used as adjuncts to behavioral therapy in weight reduction programs. The two classes of anorectic drugs currently available are the noradrenergic and the serotonergic agents.

1. Noradrenergic drugs affect weight loss through action in the appetite center.Phenylpropanolamine (Dexatrim), a sympathomimetic drug and a synthetic derivative of ephedrine, is available as an over-the-counter appetite suppressant and decongestant. In studies lasting 14 weeks, the subjects who took phenylpropanolamine had a greater weight loss than those who took placebo, although the difference was minimal.

2. The serotonergic drugs partially inhibit the reuptake of serotonin and release serotonin into the synaptic cleft, thus acting on the hypothalamus to decrease satiety.

Fluoxetine (Prozac) is a highly selective serotonin reuptake inhibitor (SSRI) that has been studied in the treatment of obesity. Fluoxetine may increase energy expenditure by raising basal body temperature; however, weight loss has not been consistent among subjects in clinical trials. In a three-month study, fluoxetine did not significantly reduce weight when compared with placebo. In a longer clinical trial, significantly greater weight loss was achieved in the subjects taking fluoxetine at 20 weeks, compared with the subjects taking placebo.

HOW CONGENITAL SYDROME AFFECT OBSEITY

The negative impact of overweight and obesity is potentially greater in children affected by a congenital heart disease (CHD) and is associated with high systolic blood pressure levels. More than one quarter of this population is already overweight. Two main causes have been described: physical activity restrictions and interventions for weight gain in infancy, when many lesions cause undernutrition. These interventions often include consumption of increased calories and foods with high fat and sodium content. Although nutritional requirements and physical functional capacity change as these children grow older and their heart lesions are successfully treated, the inappropriate dietary behaviors and physical inactivity are frequently maintained across childhood. The presence of modifiable risk factors for ischemic heart disease in this population, such as an abnormal lipid profile (high total cholesterol/LDL/triglycerides, low HDL) and excess weight may lead individuals with congenital heart disease to have a combination of risks that may persist into adulthood.In this population, factors inherent to the heart disease can be added to other traditional risk factors for the development of ischemic heart disease in the future. Changes in the lifestyle are necessary to change these risk factors and its comorbidities in the adult life of these people who are living longer.

1. • Cancer arises from cancer within cells and external factors within the environment Example : inherent mutation or mutation due to metabolism and environment.

• Aetiology of cancer is multifactorial, physical chemical, hormonal, metabolic, genetic and environment and all have a role in development of cancer.

• They all have replication of genes/mutation.

• Thus carcinogens are mutagens and vice versa.

• In summary, carcinogens whether physical or chemical cause DNA damage which leads to mutation and mutation leads to cancer

• Every normal cell has DNA repair gene mechanism that corrects defects that occur during replication.

• Carcinogens and hereditary mutations affect the repair genes and hence cancer occurs.

• About 50% of human cancer is due to mutation or deletion of this repair gene called anti oncogens or oncosuppressor gene.

MOLECULAR BASIS - Normally, normal cells replicate and remove from the body through apoptosis, a natural process - The molecular basis of this is seen in the shortening of the telomeres on the chromosomes on the normal cells - Cancer cells are able to escape apoptosis of the normal cell cycle. They accomplish this by production of the enzyme, TELOMERE POLYMERASE, which tends to lengthen the telomere which prolongs life. In this way cancer cells are immortalised because they escaped apoptosis - All normal cells receive signal for apoptosis, chemicals that cause cancer destroy signals hence cells continue to multiply uncontrollably