OKE SUCCESS OLUWASEYI

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MEDICINE AND SURGERY

1. **SIMPLE OBESITY** is characterized by a normal or increased growth rate with an acceleration of bone age maturation. Despite normal growth, simple obesity is characterized by a reduced GH secretion evaluated by standard provocative tests, the administration of GH-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 characterized by normal growth in the presence of **‘hyposomatotropism’.**
2. **HOW DOES CONGENITAL SYNDROME AND DRUG THERAPY AFFECT OBESITY**

Obesity occur in several multiple congenital anomaly syndromes, including Prader-Willi syndrome, Bardet-Biedl syndrome, Cohen syndrome, Albright hereditary osteodystrophy,, turner syndrome, down syndrome as well as some rarer disorders.

**EFFECTS OF PRADER WILLI’S SYNDROME ON OBEITY**: The obesity associated with this syndrome results from a chronic imbalance between energy intake and expenditure due to hyperphagia, decreased physical activity, reduced metabolic rate and an inability to vomit. Individuals have a lower lean body mass compared with controls contributing to reduced energy expenditure.

**BARDET-BIEDL SYNDROME ON OBESITY**: Patients with this syndrome lack the negative regulatory mechanisms of appetite-regulating hormones with respect to nutritional status and exhibit resistance to anorexigenic leptin. This results in a shift towards the orexigenic effects of this self-regulating system. These alterations may in part be responsible for the disturbed appetite regulation in these patients.

**COHEN SYNDROME**: When obesity is present here, it typically occurs around the torso, with the arms and the legs remaining slender (called truncal obesity).

**TURNER SYNDROME**: Women with Turner syndrome may be at increased risk of developing coronary artery disease as a result of higher frequency of hypertension and obesity. Routine screening for risk factors for ischaemic heart disease is recommended.

**DOWN SYNDROME**: according to a research made, Children with down syndrome were more likely to be overweight or obese than the general population of the youth without down syndrome. Likely determinants of obesity included increased leptin, decreased resting energy expenditure, comorbidities, unfavorable diet, and low physical activity levels. Obesity was positively associated with obstructive sleep apnea, dyslipidemia, hyperinsulinemia, and gait disorder. Interventions for obesity prevention and control were primarily based on exercise-based programs, and were insufficient to achieve weight or fat loss.

**DRUG THERAPY**

It is essential that medications are used in conjunction with healthy eating, physical activity, and behavior modification, as medication usage without such changes are generally ineffective.

The goal of drug therapy for overweight individuals is long-term weight reduction and improvement in overall health.

In short terms (6 to 12 months) clinical trials evaluating drug therapy, weight loss of 4 to 8 percent is typical.

It should be noted that not every drug works for every patient; individual responses vary widely. Also, when the maximal therapeutic effect is achieved, a plateau is reached and weight loss ceases. Finally, when drug therapy is discontinued, weight gain can be expected.

**AETIOLOGY OF CANCER**

Cancer is caused by accumulated damage to genes. Such changes may be due to chance or to exposure to a cancer causing substance. The substances that cause cancer are **CARCINOGENS**. A carcinogen may be a chemical substance, such as certain molecules in tobacco smoke. The cause of cancer may be environmental agents, viral or genetic factors. In majority of the cancer cases, the disease cannot be attributed to a single cause.

Cancer risk factors can be roughly divided into the following groups:

1. Biological or internal factors, such as age, gender, inherited genetic defects and skin type.
2. Environmental exposure, for instance to radon and UV radiation, and fine particulate matter.
3. Occupational risk factors, including carcinogens such as many chemicals, radioactive materials and asbestos.
4. Lifestyle-related factors.

**LIFESTYLE-RELATED FACTORS THAT CAUSE CANCER INCLUDE:**

* Tobacco
* Alcohol
* UV radiation in sunlight
* Some food-related factors, such as nitrites and poly aromatic hydrocarbons generated by barbecuing food, a high-diet fat.

 **CANCER CAUSING FACTORS RELATED TO WORK AND LIVING ENVIRONMENTS INCLUDE:**

* Asbestos fibers
* Tar and pitch
* Polynuclear hydrocarbons (e.g benzopyrene)
* Some metal compounds
* Some plastic chemicals (e.g Vinyl chloride)
* Pesticides, fertilizers and power lines have been researched for a direct link to childhood cancers.

**BACTERIA AND VIRUSES CAN CAUSE CANCER:**

* Helicobater pylori (H. pylori, which causes gastritis)
* HBV, HCV (hepatitis viruses that cause hepatitis)
* HPV (human papilloma virus, papilloma virus, which causes changes e.g Cervical cells)
* EBV (Epstein-Barr virus, the herpes virus that causes inflammation of the throat lymphoid)

**RADIATION CAN CAUSE CANCER:**

* Ionising radiation (e.g X-ray radiation, soil radon)
* Non-ionised radiation (the sun’s ultraviolet radiation)
* In some cases, children who have been exposed to some form of high-dose chemotherapy and radiation may develop a second malignancy later in life. These strong anticancer agents can alter cells and/or the immune system. A second malignancy is a second malignancy is a cancer that appears as a result from treatment of a different cancer.

**SOME DRUGS MAY INCREASE THE RISK OF CANCER:**

* Certain antineoplastic agents
* Certain hormones
* Medicines that cause immune deficiency

**FAMILY HISTORY, INHERITANCE AND GENETICS MAY PLAY AN IMPORTANT ROLE IN SOME CHILDHOOD CANCERS**

* It is possible for cancer of varying forms to be present more than once in a family.
* It is unknown in these circumstances if the disease is caused by a genetic mutation, exposure to chemicals near a family’s residence, a combination of these factors, or simply coincidence.

**SOME GENETIC DISORDERS**

* For example, **Aldrich and Beckwith-Wiedemann syndrome** are known to alter the immune system.
* The bone marrow produces cells that later mature and function as part of the immune system.
* One theory suggests that the cells in the bone marrow, stem cells, become damaged, so when they reproduce to make more cells, they make abnormal cells or cancer cells. The cause of the defect in the stem cells could be related to an inherited genetic defect or exposure to a virus or toxin.

 In 5-10 per cent of breast cancer genetic predisposition plays an important role in the emergence of the disease.

**MOLECULAR BASIS OF CANCER**

 Normal cells tend to replicate normally and when they get old, they get removed and replaced by APOPTOSIS. The molecular basis of this is seen in the shortening of **TELOMERES** on the chromosome in normal cells. Cancer cells are able to escape apoptosis of the normal cell cycle. They accomplish this by the action of the enzyme **TELOMERES** **POLYMERASE** which helps elongate the telomeres, prolonging the cell life.

In this way, apoptosis is prevented and thus cancer cells are immortalized. All normal cells receive signal for apoptosis, chemicals that cause cancer destroy these signals, hence cells continue to multiply uncontrollably.

Molecular studies have shown that cancer is a paradigm of acquired genetic disease. The process of protein production involves a cascade of several different steps, each with its attendant enzymes, which are also encoded by DNA and regulated by other proteins.

Most steps in the process can be affected, eventually leading to an alteration in the amount or structure of proteins, which in turn affects cellular function. However, whereas cellular function may be altered by disturbance of one gene, malignant transformation is thought to require two or abnormalities occurring in the same cell. Although there are mechanisms responsible for DNA maintenance and repair, the basic structure of DNA and the order of the nucleotide basis can be mutated. These mutations can be inherited or can occur sporadically, can be present in all cells or only in tumor cells. At nucleotide level, these mutations can be substitutions, additions or deletions. Several oncogenes including p53, c-fms and Ras genes, can be activated by point of mutations that lead to amino acid substitution in critical portions of the protein.