e-ISSN: 2456-4435
Sept 2017 | Vol. 01 | Issue : 04
International Journal of Research in Indian Medicine

"Patho – physiology of obesity & it's ayurvedic management"

Gapat Vikas Nivrutti*[1], Divekar Ujwala M.[2]

1. PG scholar
2. Guide

Department Samhita & Siddhant

C.S.M.S.S. Ayurved Mahavidyalaya, Kanchan Wadi, Aurangabad, Maharashtra, India

*Corresponding Author: **drvikasgapat@gmail.com**

Abstract:

Ayurveda is the Science of life deals with the Preventive and curative aspect. According to Ayurveda any disorder Tridosha's are responsible.

Obesity is due to Vata & Kapha dosha vaishamya.

The multidimensional process of biological transformations in the body are controlled and regulated by AGNI.

Hence Agni (Dhatwagni) are disturbed in Obesity. BMR is regulated by this Agni.

Obesity is described as "Medoroga" or Sthoulya roga in Ayurveda. Continues indulgence in high fat food, fried food items etc. along with sedentary lifestyle leads to excess accumulation of body fat which gets deposited in the numerous body channels.

It is defined by body mass index (BMI) and further evaluated in terms of fat distribution via the waist--hip—ratio and total cardiovascular risk factors.

Diet, Life style and panchakarma management plays a important role in the management of Obesity.

Aim :- The aim of article is to discuss the pathogenesis and management of Obesity.

Objectives :- To create awareness of treatment of Obesity according to Ayurveda.

Type of study :- Descriptive and conceptual study.

Literary review :- According to ayurveda (Charaksamhita) our sharir is made up of dosha, dhatu and malas. All these responsible for maintaining body. Roga (disease) is the effect of diseqilibrium of dhatus and health is the results of

equilibrium of doshas & dhatus. (Dhatusamya kriyachokta tantrasya aasya prayojanam)

Key words:

Patho physiology, Obesity, Ayurvedic & Management.

INTRODUCTION:

In the modern era day to day Obesity is the commonest problem. More than 12 million cases per year are diagnosed in India.

Obesity is a medical condition in which excess body fat has accumulated to the 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, and it is calculated as follows.

Where m and h are the subject's weight and height respectively.

BMI is usually expressed in kilograms per square meter, resulting when weight is measured in kilograms and height in meters. To convert from pounds per square inch multiply by 703 (kg/m2)/(lb/sq in).

The range 25–30 kg/m2 defined as overweight. Some East Asian countries use lower values. Obesity increases the likelihood of various diseases and conditions, particularly cardiovascular diseases, type 2 diabetes, obstructive sleep apnea, certain types of cancer, osteoarthritis and depression.

Classification:

BMI (kg/m2) Classification[18]

BMI	Classification
18.5	underweight
18.5 - 25.0	normal weight
25.0 - 30.0	overweight
30.0 - 35.0	class I obesity
35.0 - 40.0	class II obesity
40.0	class III obesity

A BMI of > 45 or 50kg / m2 is super obesity.

As Asian populations develop negative health consequences at a lower BMI than Caucasians, some nations have redefined obesity; Japan have defined obesity as any BMI greater than 25 kg/m2 while China uses a BMI of greater than 28 kg/m2.

Effects on health

Excessive body weight is associated with various diseases and conditions, particularly cardiovascular diseases, diabetes mellitus type 2, obstructive sleep apnea, certain types of cancer, osteoarthritis[2] and asthma. As a result, obesity has been found to reduce life expectancy.

Obesity increases the risk of many physical and mental conditions. These co morbidities are most commonly shown in metabolic syndrome, a combination of medical disorders which includes: diabetes mellitus type 2, high blood pressure, high blood cholesterol, and high triglyceride levels.

Health consequences fall into two broad categories: those attributable to the effects of increased fat mass (such as osteoarthritis, obstructive sleep apnea, social stigmatization) and those due to the increased number of fat cells (diabetes, cancer, cardiovascular disease, non-alcoholic fatty liver disease). Increases in body fat alter the body's response to insulin, potentially leading to insulin resistance.

Medical field Condition Medical field Condition

Cardiology

- coronary heart disease: angina and myocardial infarction
- congestive heart failure
- high blood pressure
- abnormal cholesterol levels
- deep vein thrombosis and pulmonary embolism

Dermatology

- stretch marks
- acanthosis nigricans
- lymphedema
- cellulitis
- hirsutism
- intertrigo

Endocrinology and Reproductive medicine

- diabetes mellitus
- polycystic ovarian syndrome
- menstrual disorders
- infertility
- complications during pregnancy
- birth defects
- intrauterine fetal death

Gastroenterology

- gastroesophageal reflux disease
- fatty liver disease
- cholelithiasis (gallstones)

Neurology

- stroke
- meralgia paresthetica
- migraines
- carpal tunnel syndrome
- dementia
- idiopathic intracranial hypertension
- multiple sclerosis

Oncology

- esophageal
- colorectal
- pancreatic
- gallbladder,
- endometrial
- kidney
- Leukemia
- Hepatocellular carcinoma
- malignant melanoma

Psychiatry

- depression in women
- social stigmatization

Respirology

- obstructive sleep apnea
- obesity hypoventilation syndrome
- asthma
- increased complications during general anaesthesia

Rheumatologyand Orthopedics

- gout
- poor mobility
- osteoarthritis
- low back pain

Urology and Nephrology

- erectile dysfunction
- urinary incontinence
- chronic renal failure
- hypogonadism
- buried penis

Sign and symptoms of Obesity by Ayurveda:-

1. Excessive Hunger
2. Excessive Thirst
3. Excessive Sweating
4. Excessive Sleep
5. Breathlessness on mild exertion
6. Difficulty in perform heavy work
7. Sluggishness
8. Decreased body strength
9. Foul odor of body

10. Short life span

Causes :-

At an individual level, a combination of excessive food energy intake and a lack of physical activity is thought to explain most cases of obesity. A limited number of cases are due primarily to genetics, medical reasons, or psychiatric illness. In contrast, increasing rates of obesity at a societal level are felt to be due to an easily accessible and palatable diet, increased reliance on cars, and mechanized manufacturing.

A 2006 review identified ten other possible contributors to the recent increase of obesity: (1) insufficient sleep, (2) endocrine disruptors (environmental pollutants that interfere with lipid metabolism), (3) decreased variability in ambient temperature, (4) decreased rates of smoking, because smoking suppresses appetite, (5) increased use of medications that can cause weight gain (e.g., atypical antipsychotics), (6) proportional increases in ethnic and age groups that tend to be heavier, (7) pregnancy at a later age (which may cause susceptibility to obesity in children), (8) epigenetic risk factors passed on generationally, (9) natural selection for higher BMI, and (10) assortative mating leading to increased concentration of obesity risk factors (this would increase the number of obese people by increasing population variance in weight).

Diet :-

The widespread availability of nutritional guidelines has done little to address the problems of overeating and poor dietary choice.

The primary sources of these extra carbohydrates are sweetened beverages, which now account for almost 25 percent of daily food energy in young adults in America, and potato chips. Consumption of sweetened drinks such as soft drinks, fruit drinks, iced tea, and energy and vitamin water drinks is believed to be contributing to the rising rates of obesity and to an increased risk of metabolic syndrome and type 2 diabetes. Vitamin D deficiency is related to diseases associated with obesity.

As societies become increasingly reliant on energy-dense, big-portions, and fast-food meals, the association between fast-food consumption and obesity becomes more concerning.

Sedentary lifestyle:- A sedentary lifestyle plays a significant role in obesity. Worldwide there has been a

large shift towards less physically demanding work, and currently at least 30% of the world's population gets insufficient exercise. This is primarily due to increasing use of mechanized transportation and a greater prevalence of labor-saving technology in the home. In children, there appear to be declines in levels of physical activity due to less walking and physical education.

Genetics :- Like many other medical conditions, obesity is the result of an interplay between genetic and environmental factors.

Like many other medical conditions, obesity is the result of an interplay between genetic and environmental factors. Polymorphisms in various genes controlling appetite and metabolism predispose to obesity when sufficient food energy is present. As of 2006, more than 41 of these sites on the human genome have been linked to the development of obesity when a favorable environment is present. People with two copies of the FTO gene (fat mass and obesity associated gene) have been found on average to weigh 3–4 kg more and have a 1.67-fold greater risk of obesity compared with those without the risk allele. The differences in BMI between people that are due to genetics varies depending on the population examined from 6% to 85%.

Obesity is a major feature in several syndromes, such as Prader–Willi syndrome, Bardet–Biedl syndrome, Cohen syndrome, and MOMO syndrome. (The term "non-syndromic obesity" is sometimes used to exclude these conditions.)

The thrifty gene hypothesis postulates that, due to dietary scarcity during human evolution, people are prone to obesity.

Other illnesses :- Certain physical and mental illnesses and the pharmaceutical substances used to treat them can increase risk of obesity. Medical illnesses that increase obesity risk include several rare genetic syndromes (listed above) as well as some congenital or acquired conditions: hypothyroidism, Cushing's syndrome, growth hormone deficiency,

The risk of overweight and obesity is higher in patients with psychiatric disorders than in persons without psychiatric disorders.

Certain medications may cause weight gain or changes in body composition;

these include insulin, sulfonylureas, thiazolidinediones, atypical antipsychotics, antidepressants, steroids, certain anticonvulsants (phenytoin and valproate), pizotifen, and some forms of hormonal contraception.

Social determinants :- While genetic influences are important to understanding obesity, they cannot explain the current dramatic increase seen within specific countries or globally. Though it is accepted that energy consumption in excess of energy expenditure leads to obesity on an individual basis, the cause of the shifts in these two factors on the societal scale is much debated. There are a number of theories as to the cause but most believe it is a combination of various factors.

In the developing world, women, men, and children from high social classes had greater rates of obesity.

In the developing world urbanization is playing a role in increasing rate of obesity.

Gut bacteria:- The study of the effect of infectious agents on metabolism is still in its early stages. Gut flora has been shown to differ between lean and obese humans. There is an indication that gut flora in obese and lean individuals can affect the metabolic potential.

An association between viruses and obesity has been found in humans and several different animal species. The amount that these associations may have contributed to the rising rate of obesity is yet to be determined.

Patho-physiology :- There are many possible pathophysiological mechanisms involved in the development and maintenance of obesity. This field of research had been almost unapproached until the leptin gene was discovered in 1994 by J. M. Friedman's laboratory. These investigators postulated that leptin was a satiety factor. In the ob/ob mouse, mutations in the leptin gene resulted in the obese phenotype opening the possibility of leptin therapy for human obesity. However, soon thereafter J. F. Caro's laboratory could not detect any mutations in the leptin gene in humans with obesity. On the contrary Leptin expression was increased proposing the possibility of Leptin-resistance in human obesity. Since this discovery, many other hormonal mechanisms have been elucidated that participate in the regulation of appetite and food intake, storage patterns of adipose tissue, and

development of insulin resistance. Since leptin's discovery, ghrelin, insulin, orexin, PYY 3-36, cholecystokinin, adiponectin, as well as many other mediators have been studied. The adipokines are mediators produced by adipose tissue; their action is thought to modify many obesity-related diseases.

Leptin and ghrelin are considered to be complementary in their influence on appetite, with ghrelin produced by the stomach modulating short-term appetitive control (i.e. to eat when the stomach is empty and to stop when the stomach is stretched). Leptin is produced by adipose tissue to signal fat storage reserves in the body, and mediates long-term appetitive controls (i.e. to eat more when fat storages are low and less when fat storages are high). Although administration of leptin may be effective in a small subset of obese individuals who are leptin-deficient, most obese individuals are thought to be leptin resistant and have been found to have high levels of leptin. This resistance is thought to explain in part why administration of leptin has not been shown to be effective in suppressing appetite in most obese people.

While leptin and ghrelin are produced peripherally, they control appetite through their actions on the central nervous system. In particular, they and other appetite-related hormones act on the hypothalamus, a region of the brain central to the regulation of food intake and energy expenditure. There are several circuits within the hypothalamus that contribute to its role in integrating appetite, the melanocortin pathway being the most well understood.

Thus a deficiency in leptin signaling, either via leptin deficiency or leptin resistance, leads to overfeeding and may account for some genetic and acquired forms of obesity.

Management :-

The main treatment for obesity consists of dieting and physical exercise. Diet programs may produce weight loss over the short term, but maintaining this weight loss is frequently difficult and often requires making exercise and a lower food energy diet a permanent part of a person's lifestyle.

In the short-term low carbohydrate diets appear better than low fat diets for weight loss. In the long term; however, all types of low-carbohydrate and low-fat

diets appear equally beneficial. A 2014 review found that the heart disease and diabetes risks associated with different diets appear to be similar. Promotion of the Mediterranean diets among the obese may lower the risk of heart disease. Decreased intake of sweet drinks is also related to weight-loss. Success rates of long-term weight loss maintenance with lifestyle changes are low, ranging from 2–20%. Dietary and lifestyle changes are effective in limiting excessive weight gain in pregnancy and improve outcomes for both the mother and the child. Intensive behavioral counseling is recommended in those who are both obese and have other risk factors for heart disease.

Orlistat use is associated with high rates of gastrointestinal side effects and concerns have been raised about negative effects on the kidneys. There is no information on how these drugs affect longer-term complications of obesity such as cardiovascular disease or death.

The most effective treatment for obesity is bariatric surgery.

Management by Ayurveda :-

In Charaka special programs are included in obesity therapy in the purpose to cut down excess fat to correct the irregular metabolism and to clear the body channels.

Deep dry udwarthan with herbal pwdors and pastes synchronized Abhyang with specific oils, steam bath, Dhanyaamla Dhara etc. mobilize the accumulated fat whereas specialy designed panchakarma procedures for detoxification clears and prevent its further accumulation.

Diet and life style management plays a important role in the management of obesity.

Management by Ayurveda: Line of treatment -

1. Nidanparivarjan
2. Apatarpan Chikistha
3. Sanshodhan Chikistha – Vaman, Virechan, Lekhanbasti etc.
4. Samshaman Chikistha

1. Diet and exercise
2. Wat-Kapha Nashak Aahar and Vihar
3. Mutra and purish virechaniya Aushadhi

Diet Management :-

Diet should be taken – Puran Shali, Rice, Mooge, Kuluth, Yava (Barli),

Fleseed, Bajra, Kodo, Brinjal, Sarpentgaurd, takra (Butter Milk), Amla, Mustard oil, Hote Water & drinking water before food.

Avoid food: Newly Shalidhanya, Wheat, Rice, Blackgram, potato, milk products, curd, non-veg, eggs, ghee, jaggary & sugar products.

Life style management: Chinta (passive stress), Shrum (work load), night sleep, maithun, lep, langhan, hot water bath, walking, running & exercise daily.

Avoid life style :- Cold water bath, wearing flowers mala, day time sleep, and sedentary life style.

Aasna's :- Do Aasna's like Suryanamaskar – 12 to 24 daily

Sarvangasan, Halasan, Mayurasan, Shirshyasan, Dhanurasan, Shalbasan, Padmasan, Chakrasan etc.

Pranayama like – Bhrastrika, Kapalbhati etc. are use full for obesity

It should be don daily, regularly for long time.

Shamshan Chikista:

1. Arogyavardhini Vati – 2TDS before food.
2. Triphala Churna – 1 TSF – HS
3. Phalatrikadi Quath – 2 TSF – TDS or
4. Vidangarishtha – 2 TSF – TDS
5. Navakguggulu – 2 TDS – with warm water. or
6. Medohar Guggulu – 2 TDS – with warm water.
7. Shilajeet Rasayan – 1 to 2 gram with honey
8. Lep – Shirishadi pralep or Harikyadi Lepam

Reference:

1. https://en.wikipedia.org/wiki/Obesity
2. Kayachikista III khanda Dr. Ajaykumar Sharma – Choukamba Orantalia Edition; 2013.

Cite this article:

"Patho – physiology of obesity & it's Ayurvedic management"

Gapat Vikas Nivrutti, Divekar Ujwala M.

Ayurline: International Journal of Research In Indian Medicine 2017; 1(4) : 1-10