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Obesity, A Global Threat

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ABSTRACT

In this modern civilization, the society is running through a so-called fast life-style. Under the mask of fast life-style we are continuously struggling to hide our faults in daily lives. As a result of which the entire society have become submerged under the dirty dump of diseases, a non-sense habits. Among these dirts, obesity is one of the major threat to modern society, which is also a principal contributing factors of most of the diseases of this era. But unfortunately in most of the cases it is a self-made condition. Obesity is not only a threatening condition but also a common manifestation of many other systemic diseases. Obesity also increases the risk of other diseases. Therefore 'World Health Organization' (WHO) defined that "obesity is a global problem".

This paper deals with obesity, it causes pathogenesis, prevention and management and differential diagnosis method. This paper will admonish the society how we allow obesity in our life carelessly, how to get relief from it and the fate by this fatal condition.

KEYWORDS: *Obesity, Lifestyle diseases, Fatty, Lifestyle*

INTRODUCTION

In this modern civilization, the society receives a good number of experiences along with many new diseases and defects, which can harm the entire society. Among those, obesity is one of those.

Obesity or abdominal obesity which means deposition of excessive fat around the viscera like stomach, liver, e.t.c. and on the abdominal wall; Central obesity has different multidimensional effects on our health, both positive & negative. The result of central obesity can leads to many systemic diseases, such as Hypertension (HTN), stress, anxiety (psychological disease), Atherosclerosis, NIDDM e.t.c. In many cases central obesity can results into different life threatening conditions, such as Heart block and different chronic disabilities, such as Rheumatism. One can say this is a root of different diseases, but in some cases central obesity is the effects also.

DEFINITION

Central obesity is the excessive deposition or accumulation of fat on the walls of the abdominal viseras and onn the abdominal wall simultaneously or individually.

DEPOSITION OF FAT

Visceral fat:- It is located in the peritoneal cavity along the viscera covered by peritoneum.
Subcutaneous fat:- Located beneath the skin.
Intramuscular fat:- Deposited in the skeletal muscle.
In central obesity fat is also deposited in the gluteal region and on the chest.

Progression of deposition of fat in different gender
In male: Abdomen→ buttocks →chest
In female:- Breast→ Buttocks→ Abdomen

In female oestrogen is responsible for fat accumulation in the buttocks, thighs and hips. When women reach menopause and the oestrogen production declines the progression of fat migrates from their gluteal reagion and thighs to their abdomen. For females, this so-called sex-specific fat appears to be physiologically advantageous, at least during pregnancies. But it lefts a negative effect on the cosmetic values also. Throughout most of their lives females have a higher percentage of body fat than males. This difference according to the gender begins early in life. In the first 72 months after birth, the number and size of fat cells triple in both boys and girls, resulting in a gradual, and similar, increase in body fat. But after about 96 months after birth, girls begin gaining fat mass at a greater rate than boys do. This increase appears to result from a lower female basal fat oxidation rate, and it is accomplished by expanding fat cell size, not number. Between six years of age and adolescent, there is little or no increase in fat cell number, for either boys or girls, in healthy-weight children. In obese children, however, the number of fat cells can increase throughout childhood. During lactation, however, sex-specific fat cells are not so stubborn. They increase their fat-releasing activity and decrease their storage capacity, while at the same time fat storage increases in the mammary adipose tissue. This suggests that there is a physiological advantage to sex-specific fat. The fat stored around the pelvis, buttocks and thighs of women appears to act as reserve storage for the energy demands of lactation.

Males are more susceptible to upper body fat accumulation than female. Mostly in the abdominal cavity due to Structure of fat deposited in body Fat or adipose tissue which is loose connective tissue composed mostly of adipocytes. It also contains the stromal vascular function of cells including fibroblasts, pre-adipocytes, vascular endothelial cells and a variety of immune cells such as adipose tissue macrophages.

ADIPOSE TISSUE AND ITS PHYSIOLOGY:

The fat cell is under multiple influences, including that of autonomous nervous system local blood flow changes and various hormones and factors delivered from plasma or produced locally. Following S NS stimulation, noradrenaline and NPY are released from sympathetic nerve terminals, whereas adrenal medulla secretes adrenaline. The major pathways regulating lipolysis are adrenergic. In human fat c ells, both 1 & 2 adrenergic receptors (A Rs) initiate activation of lipolytic cascade by stimulation of cyclic adenosine monophosphate (cAMP) production, activation of cAMP-dependent protein kinase A (P KA) leading t o phosphorylation of perilipin and hormone-sensitive lipase (HSL), and promotion of lipolysis in vitro. Human fat cells express large number of adrenergic receptors, their stimulation inhibits cAMP production and lipolysis. Rodents possess 3 adrenergic receptors in the white fat cells, whereas in human fat cells the role of the 3 ARs is unclear. Differences exist in the adrenergic regulation of lipolysis in adipose tissues from different sites in normal-weight subjects and in obese subjects. The lipolytic response of isolated fat cells to the catecholamines is weaker in subcutaneous (abdominal/femoral) than in visceral adipose tissue. One possible explanation includes defective signaling pathways. Alterations in expression and function of HS L or other interacting proteins like adipocyte lipid-binding protein (ALBP) m ay also explain these regional differences in lipolysis. Reduced lipid mobilization occurs during exercise in subcutaneous fat of obese subjects.

Cause

1. **Habits and lifestyle:-**
   - Sleepless night lead / lower duration of sleep
   - Junk food intake
   - Lack of physical exercise
   - Addiction of tobacco and alcohol
2. **Disease and syndrome**
   - Sleeping disorder
   - Hyperphagic tendency
   - Type2 Diabetes mellitus
   - Fatty liver
   - Hypertension(CVD)
3. **Hormonal disorder**
   - Leptin
   - Ghrelin
   - Cholecystokinin
   - Insulin
   - Thyroid hormone
4. **Genetics**
5. **Drugs**

Pathogenesis

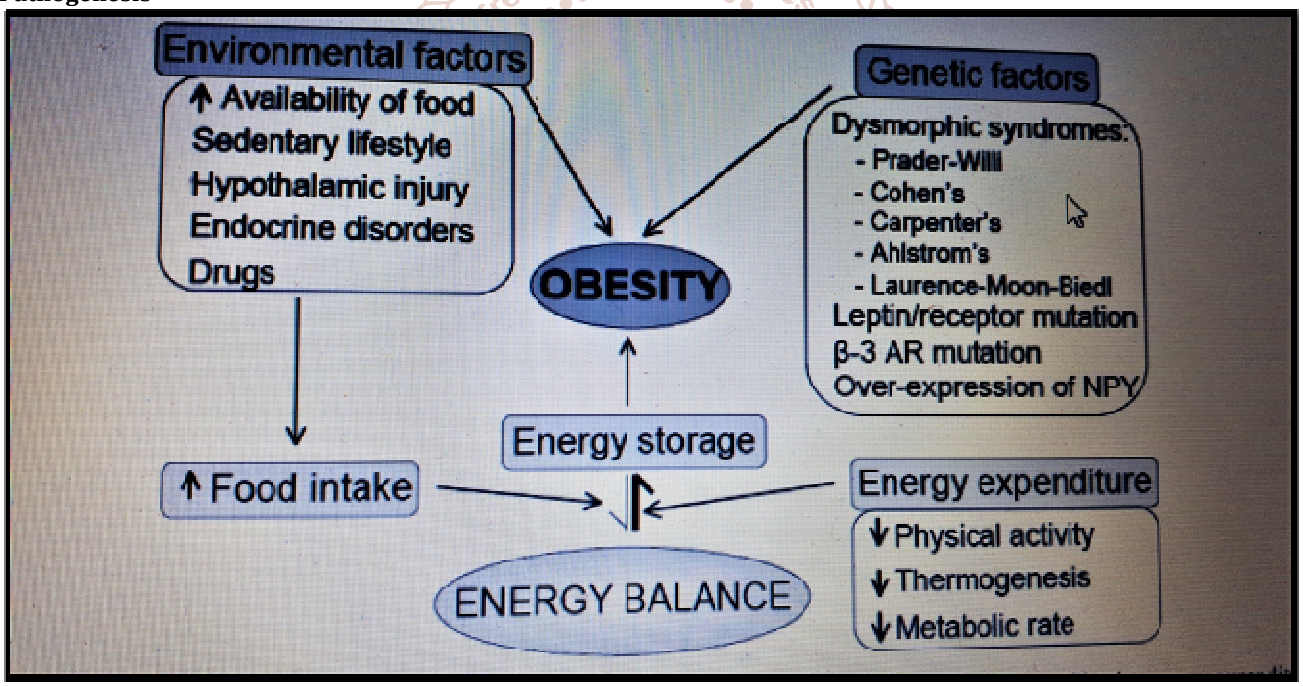

Figure 01

Obesity as a D isorder of th e Homeostatic Control of Energy Balance

Although it is known that a disturbance of the homoeostatic mechanisms controlling energy balance causes obesity, it is less clear how the balance is disturbed, since the mechanisms are very complex and involve numerous systems in the body. Soon after the first demonstration of leptin deficiency and leptin receptor dysfunction in subjects, it was thought that alterations in leptin kinetics might provide a simple explanation of how energy balance was disturbed in obese subjects. But most of information on leptin was derived from rodent experiments. Plasma leptin is higher in obese subjects compared with normal weight individuals. In fact, leptin concentrations are proportional to body fat mass in both obese and lean subjects. Thus, obesity is not due to the deficiency in circulating leptin. Resistance to leptin might be one of factors in development of obesity. Such resistance could be at the level of carriage of leptin in the circulation or its transport into the central nervous system (CNS). Defects in the leptin receptor or in the transducing system – decreased expression of C RF or overexpression of NPY could represent other disturbances in leptin system.

Dysfunctions of mediators other than leptin are implicated in obesity. TNF, another cytokine that relays information from fat to brain, is increased in the adipose tissue of insulin-resistant obese individuals. It has been suggested that UCP-2, a protein uncoupling oxidative phosphorylation in white fat cells is dysfunctional in obese individuals. Alterations in PPAR transcription factors Alpha, Beta and Gamma may have a role in obesity. These transcription factors promote lipogenesis and regulate gene expression of enzymes associated with lipid and glucose homeostasis. PPAR is preferentially expressed in adipose tissue and has a synergistic action with another transcription factor C /EBP alpha, to promote conversion of pre-adipocytes to adipocytes. The gene for UCP in white adipose tissue has regulatory sites for PPAR and C/EBP-alpha.

Genetics and Obesity

Genetic determinants can either play a major role in the pathogenesis of obesity or enhance susceptibility to its development. The dysmorphic forms of obesity in which genetics play a major role include the Prader-Willi syndrome, Ahlstrom's syndrome, the Laurence-Moon-Biedl syndrome e.t.c. A growing number of studies indicate associations between DN A sequence variation in specific genes and the occurrence of obesity. Interestingly, the involvement of 22 such genes w as reported in at least five separate studies. The obesity gene map shows putative loci on all chromosomes except Y. The gene defect called tub results in a defective phosphatase and causes retinitis pigmentosa and obesity in mice, making it similar to the Laurence-Moon-Biedl syndrome in humans. Linkage of human obesity to other factors related to energy balance has been reported. For instance, the Trp/64/Arg mutation of the human 3-adrenergic receptor (3-AR) gene is associated with an earlier age o f onset of NIDDM and characteristics of insulin resistance as well as weight gain in patients with morbid obesity. However, such findings have not been consistent in different ethnic populations. It has been reported that plasma IL-8 levels are increased in obese subjects. IL-8 is related to fat mass and TNF system. Elevated circulating IL-8 could be one of the factors that link obesity to greater cardiovascular risks. Most of genomic studies in humans, demonstrated substantial genetic heterogeneity influencing BMI regulation.

As for the work done by Dr. Kumara Swami Thangaraj of Hyderabad based centre for cellular and molecular biology obesity in the Indian population is largely dependent on the genetic tendencies. They analysed nearly about 1 million SNP (Single nucleotide polymorphism) markers distributed throughout the genomes. After proper analysis one SNP marker of THSD7A was significantly associated with obesity.

Environmental Factors and Obesity

Environmental factors interact with genetic susceptibility in the pathogenesis of obesity. For example, hypothalamic injury from trauma or surgery and destructive lesions in the region of the ventromedial or the paraventricular nuclei can produce obesity. The two major f actors i n hypothalamic obesity are hyperphagia and a disturbance in the ANS activity. One explanation for this is altered secretion of NPY, which is produced in arcuate nucleus and stimulates eating. Other possible explanations are impairment in re productive function, decrease in sympathetic and increase in parasympathetic activity – other key features of hypothalamic obesity.

Drugs: All the drugs metabolised by liver and kidney. Therefore excessive dose of drug, treatment, intake of drug etc. can cause hepatocyte resistance which leads to metabolic dysfunction. Therefore fat metabolism is also disturbed and as a result accumulation o fat occurs in abdominal cavity. Drgs which are known to cause Obesity are: Phenothiazines; such as chlorpromazine, antidepressants; amitriptyline, antiepileptics; valproate, steroids; glucocorticoids, antihypertensive agents; terazosin e.t.c.

Food Intake and Obesity

A typical obese subject has usually put on 20 kg over 10 years. This means that there h as been a daily excess of energy input over output of 30-40 kcal initially, increasing gradually to maintain the increased body weight. The type of food eaten can play a role in disturbing the energy balance. Fat has more calories per gram compared to carbohydrates or proteins. There are 9 cal/gm of dietary fat, whereas caloric value of carbohydrates and proteins are only 4 calories. It is possible that the mechanisms regulating appetite react more slowly to fat than to protein and carbohydrate, so satiety systems come into the picture too late. Increase in density of foods, portion size, better palatability of food, increase in availability and low cost promote obesity. Obese people try to diet to lose weight. But when a subject reduces calorie intake, there is a shift into negative energy balance. An individual loses weight but, in parallel, the resting metabolic rate decreases, and there is a concomitant reduction in energy expenditure. Probably, the system is trying to return the body weight to the "set-point", which implies maintenance of energy balance is dependent on numerous metabolic feedback loops that are tuned by an individual's susceptibility genes. Thus, an individual who was previously obese and is now of normal weight, generally needs fewer calories for maintaining that weight than an individual who has never been obese. The decrease in energy expenditure appears to be largely due to an alteration in the conversion efficiency of chemical energy to mechanical work in skeletal muscle. This adaptation to the caloric restriction contributes to t he difficulty of maintaining weight loss by diet.

Physical Activity and Obesity

Physical activity can be broadly divided into exercise and non-exercise activities. Non-exercise activities include employment related work and the activity of daily living, which one can termed as Sedentary habit. It is difficult to measure the energy expended in non-exercise activity. In general, an increase in sedentary behaviour, and a decrease in activity of daily living and employment physical activity promotes obesity. It is now recognized that increased energy expenditure by physical activity has a more Obesity Treatment Current Medicinal Chemistry, 2009 Vol. 16, No. 17 positive role in reducing fat stores and adjusting energy balance in the obese, especially when it is combined with modification of the diet. Native population study gives an example. M any years ago, a tribe of Pima Indians was divided into two groups: one of them settled in Mexico and continued with simple life, eating frugally and spending most of time in hard physical work. They are usually lean and have low incidence of NIDDM. Another group moved to the USA – an environment with easy access to calorie rich food and less need for hard physical work. They are on average 57 pounds heavier than the Mexican group and have a higher incidence of early onset NIDDM.

Sleepless night lead/ lower duration of sleep

Now-a-days sleepless night lead or lower sleep duration is a common habit or a necessity of our daily lifestyle. It is seen that these type of people are victim of anxiety, stress, etc. psychological disorder. In a survey it is seen that the people with lower sleep duration have 15.5% decreased leptin levels and 14/9% increased ghrelin level. Therefore energy homoeostasis is disturbed, increase appetite and hunger is seen and regulation of fat stores is higher than normal. Then frequency and amount of food intake is increased. As a result excessive fat stores in the abdominal cavity which initiates central obesity.

Addiction of tobacco and alcohol

Ethyl alcohol stimulate the centre of brain to take more sugar, salt and fat. Therefore the alcoholic people are seen desperate, lazy, stressed, anxious in many surveys. They do not think about healthy life rather they led irregular and unhealthy life. Therefore, they keep themselves away from physical exercise or any physical activity. This cause too lead to central obesity. And by abusing tobacco many metabolic syndrome takes place, which may lead to central obesity.

Hormonal disorder & Obesity

- **Leptin:-** Leptin is hormone which secretes from adipose tissue and small intestine. It helps in energy homoeostasis. It acts on receptors in the arcuate nucleus of the hypothalamus. When leptin resistance occur , energy homoeostasis is disrupted. Therefore central obesity occurs.
- **Ghrelin:-** Ghrelin or lenomorelin is a peptide hormone. It is secreted by ghrelinogenic cells in the gastrointestinal tract. It regulates the appetite and energy homoeostasis. Therefore much secretion of ghrelin cause disturbed energy homoeostasis and increased appetite. It cause frequent intake of food in large quantity. This excessive fat stores in the body and results obesity.
- **Cholecystokinin:-** Cholecystokinin is a peptide hormone of gastrointestinal tract. It stimulates the digestion of fat and protein and also act as an hunger suppressant. Therefore less secretion of cholecystokinin hamper the digestion of protein and fat and increase the hunger. Therefore food intake is increase and excessive fat deposited in body cavity. This hormone secretes from enteroendocrine cells in the duodenum.
- **Insulin:-** Insulin is a peptide hormone secrets from beta-cells of islets of Langerhans of pancreas. It helps in metabolism of carbohydrate, protein, fat. In insulin resistance this metabolism is hampered and excessive fat cells deposited in adipose tissue which leads to central obesity.
- **Thyroid hormone:-** these are two hormone produced by triiodothyronine (T3) and thyroxine (T4). These hormones regulates metabolism. Therefore increased secretion of thyroid hormones increase the metabolism rate. As a result hypophagic condition takes place and frequency and amount of intake is increased. Therefore excessive fat deposited in abdominal cavity. Thus central obesity takes place.

Pathophysiology of Obesity

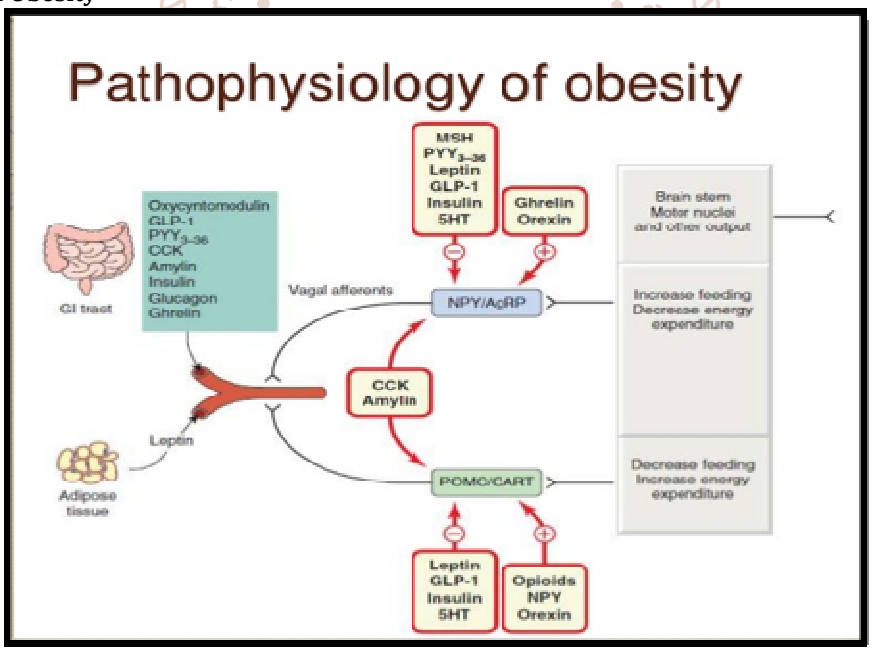

Figure-02

Role of Obesity and the Metabolic Syndrome in CKD Initiation

The question still remains whether obesity and the metabolic syndrome can, indeed, initiate renal injury. Kincaid-Smith was first suggested that the metabolic syndrome and insulin resistance could be the real enemies in glomerulosclerosis that is attributed to "hypertensive nephrosclerosis." This is supported by the fact that there are no rigorous clinical or pathologic studies to provide strong evidence of a cause-and-effect relationship between hypertension per se and ESRD, especially in white individuals. Several studies hinted that CKD is related to obesity, independent of hypertension. For instance, obesity was shown to affect independently the progression of preexisting renal diseases, such as in IgA nephropathy, in patients with unilateral renal agenesis, or after unilateral nephrectomy . Furthermore, kidneys that were obtained from obese donors (BMI >30) were more likely to exhibit a lower GFR and a higher rate of allograft dysfunction over several years than kidneys that were obtained from lean individuals (BMI <25). These data suggest that obesity contributes to and perhaps even initiates CKD.

Mechanisms of Renal Failure in Obesity and Obesity-Initiated Metabolic Syndrome

The mechanisms by which obesity/metabolic syndrome may initiate and exacerbate CKD remain elusive and largely speculative. In addition to hemodynamic factors that are related to obesity, inflammatory and metabolic effects that are related to obesity and obesity-initiated metabolic syndrome have been implicated.

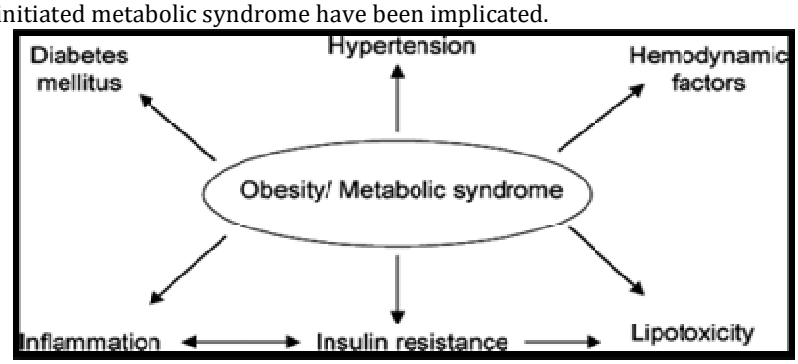

Figure- 03

Association of Inflammation with CKD in Obesity and Obesity-Initiated Metabolic Syndrome

The hallmark of the metabolic syndrome is insulin resistance. Insulin is an anti-inflammatory hormone; therefore, resistance to its action may explain why obesity/metabolic syndrome is a proinflammatory state. Furthermore, inflammatory mediators such as TNF-α have been shown to mediate insulin resistance. Plasma concentrations of some proinflammatory adipokines (cytokines secreted by adipose tissue) such as IL-6, TNF-α, C-reactive protein (CRP), and resistin are elevated in patients with the metabolic syndrome, whereas the levels of other anti-inflammatory adipokines such as adiponectin are reduced, which may contribute to insulin resistance. Thus, insulin resistance potentiates chronic inflammation and vice versa. Which is the chicken and which is the egg in the initiation of the metabolic syndrome is difficult to ascertain. Several studies have shown that adipose tissue, especially visceral adipose tissue, is a major source of cytokine secretion in the metabolic syndrome and that inflammatory cells, especially mature bone marrow–derived macrophages, invade adipose tissue early in obesity. These cytokines could be produced by the adipocyte (e.g., leptin), macrophages infiltrating the adipose tissue (e.g., TNF-α), or both (e.g., IL-6). Visceral adipose tissue is now considered an endocrine organ and a site for elaboration and secretion of hormones and cytokines.

Inflammation is a major risk factor for atherosclerosis in the general population and has been strongly associated with the metabolic syndrome. More recently, inflammation was linked to obesity and the metabolic syndrome in patients with CKD. Ramkumar et al. found a strong association between inflammation as defined by a CRP level >3 mg/dl and a high BMI in patients with CKD. The same investigators also found that, in the NHANES III cohort, the presence of the metabolic syndrome was associated with greater odds for inflammation for various levels of creatinine clearance. Wu et al. found increased expression of genes that are related to lipid metabolism (LDL receptor, fatty acid binding protein-3, and sterol regulatory element binding protein [SREBP-1]), inflammatory cytokines (TNF-α and its receptors, IL-6 signal transducer, and IFN-γ), and insulin resistance (glucose transporter-1 and vascular endothelial growth factor) in glomeruli of patients with obesity-related glomerulopathy compared with gender- and age-matched glomeruli of control donor kidneys. These findings somewhat strengthen the speculation that inflammatory cytokines and lipid by products affect renal function in obese patients, but this is yet to be proved definitively. In the following sections, we focus on the specific roles of the likely "nephrotoxins" in obesity/metabolic syndrome.

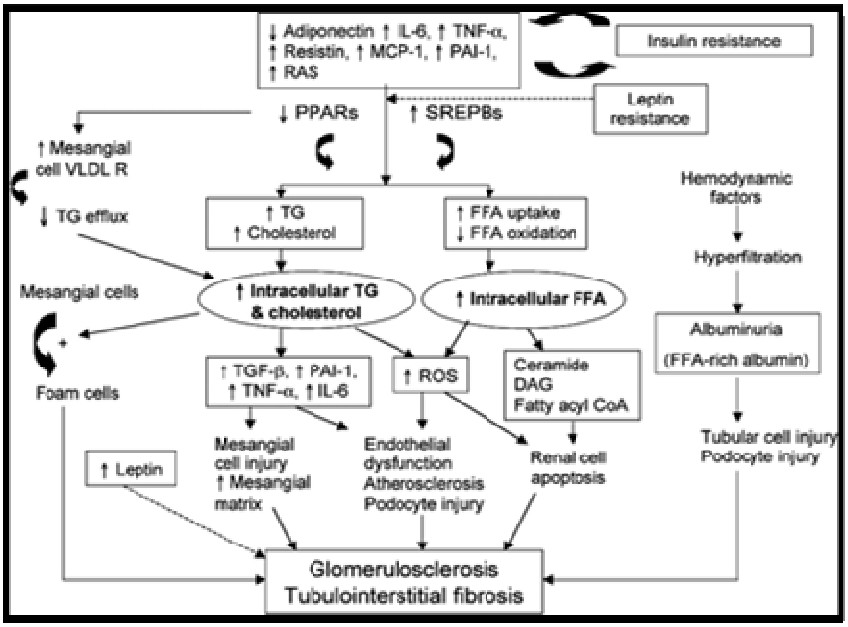

Figure 04

Obesity & Hypertension:

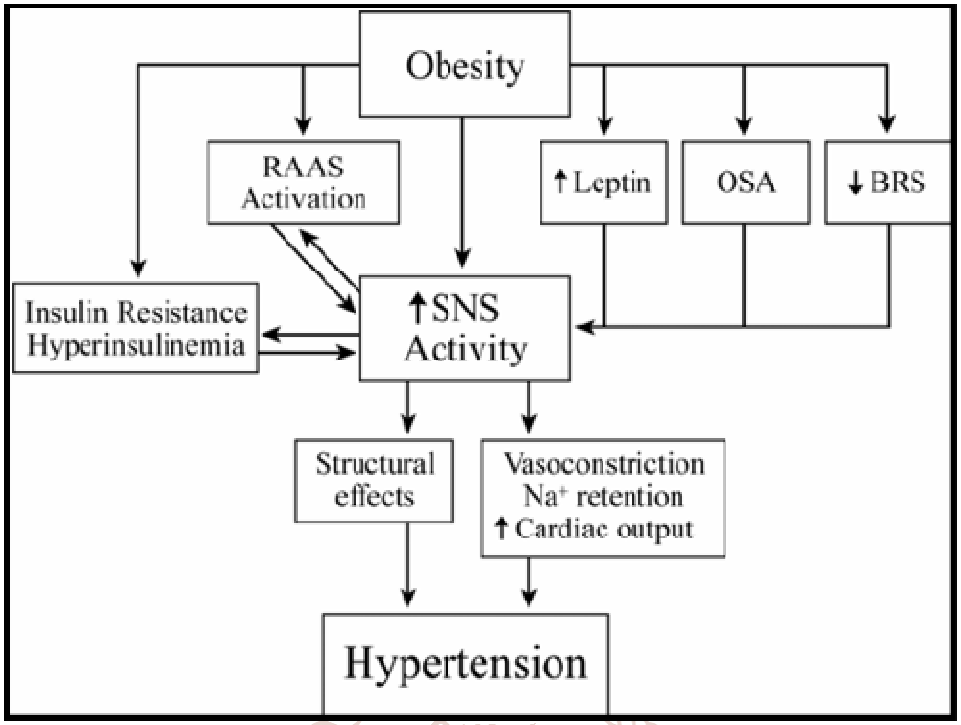

Figure 05

Obesity & Respiratory System:

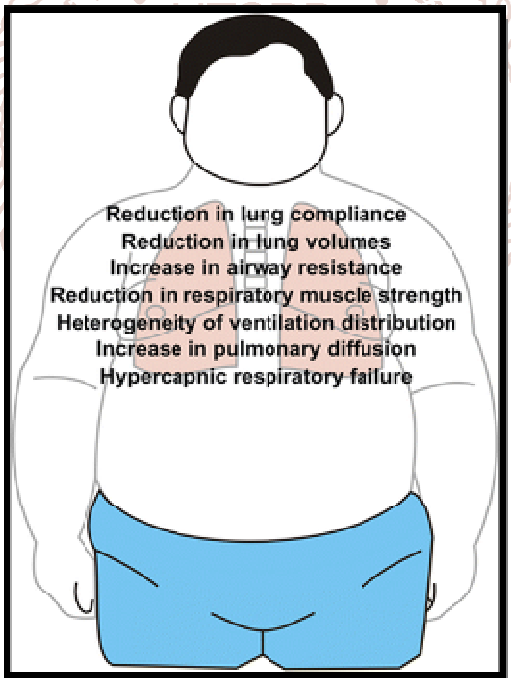

Figure 06

Biochemical and molecular changes

Obesity is associated with a state of chronic systemic inflammation that is driven predominantly by the action of substances released by adipose tissue. Chronic inflammation is caused by activation of the innate immune system, which promotes a pro-inflammatory state and oxidative stress (OS) and a consequent systemic acute-phase response. Systemic inflammation may play a crucial role in the pathogenesis of various obesity-related complications, including metabolic syndrome, T2DM, cardiac disease, liver dysfunction, and cancer.

Adipose tissue is an endocrine and energy storage organ composed of adipocytes, fibroblasts, endothelial cells, and immune cells. These cells secrete hormones and cytokines (adipokines) that exert endocrine, paracrine, and autocrine functions. Under physiological and pathological conditions, adipokines induce the production of reactive oxygen species (ROS), which trigger OS; this, in turn, leads to increased production of other adipokines. During this process, immune cells produce free oxygen radicals that promote a systemic proinflammatory state.

Excess adipose tissue is associated with the production of various proinflammatory cytokines, including tumor necrosis factor-α (TNF-α), interleukin-1-β (IL-1β), and interleukin-6 (IL-6). TNF-α plays a critical role in the inflammatory response of the immune system as well as in the apoptosis of adipose cells, lipid metabolism, hepatic lipogenesis, and the induction of OS. Increased levels of TNF-α promote a response via the release of IL-6, another proinflammatory molecule, and the reduction of levels of anti-inflammatory cytokines such as adiponectin. TNF-α also increases the interaction of electrons with oxygen, generating superoxide anions. TNF-α levels are elevated in obese individuals and decrease with weight loss.

IL-1β is a pyrogenic cytokine that is released primarily by monocytes in response to tissue damage or infection. It has recently been proposed that IL-1β is also associated with the proinflammatory response in obesity via the increased production of other cytokines, including IL-6. IL-6 is secreted by adipocytes, endothelial cells, pancreatic cells, macrophages, and monocytes and participates in the regulation of energy homeostasis and inflammation. IL-6 influences the transition from acute to chronic inflammation by stimulating the synthesis of pro-inflammatory cytokines and the down-regulation of anti-inflammatory targets. Visceral adipose tissue secretes two or three times more IL-6 than subcutaneous adipose tissue via the production of other pro-inflammatory molecules. In humans, high levels of IL-6 are associated with glucose intolerance, T2DM, SAH, and especially obesity. This cytokine may also suppress the activity of lipoprotein lipase and modulate central appetite control at the hypothalamic level.

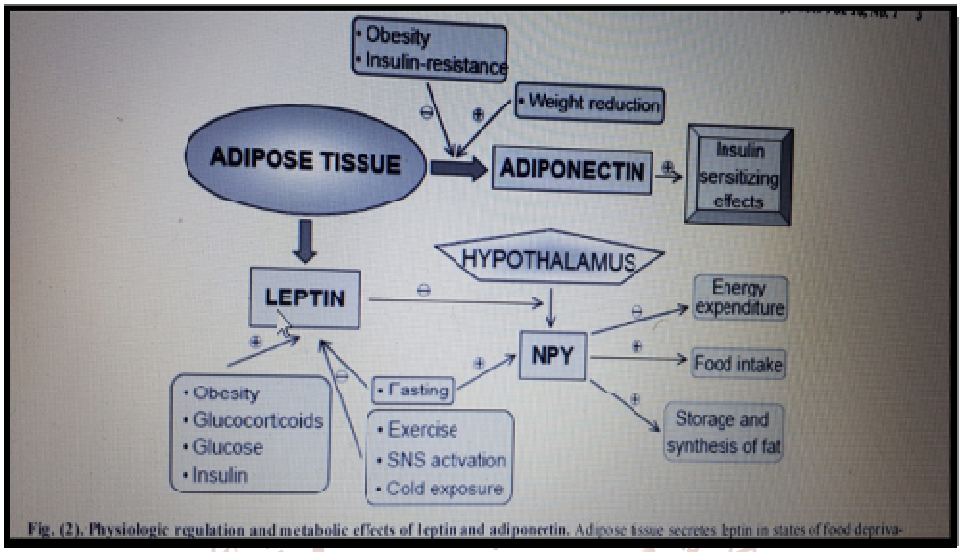

Figure -07

Obese individuals are also more susceptible to oxidative damage. The accumulation of adipose tissue, particularly visceral adipose tissue, induces the synthesis of proinflammatory cytokines, including TNF-α, IL-1, and IL-6. These cytokines promote the generation of reactive oxygen and nitrogen species by macrophages and monocytes, which may lead to increased OS. ROS induce the release of pro-inflammatory cytokines and the expression of adhesion molecules, including connective tissue growth factor, insulin-like growth factor I, platelet-derived growth factor, and vascular cell adhesion molecule I, all of which trigger OS and appear to accelerate aging and cell death, with numerous systemic consequences.

Another mechanism involved in the increased susceptibility of obese individuals to oxidative damage is the depletion of enzymes that are active in antioxidant pathways, including superoxide dismutase (SOD), glutathione peroxidase, and catalase. Antioxidant pathways associated with vitamins A, C, and E and beta-carotene also seem to be depleted. Compared with normal-weight individuals, SOD activity is significantly decreased in obese subjects. Oxidative damage leads to the increased production of free radicals, OS, mitochondrial DNA damage, and depletion of adenosine triphosphate, culminating in damage to cellular structures. The cellular damage caused by this lipotoxic state is a direct consequence of the cascade of proinflammatory cytokines released by adipose tissues.

Adipose tissue is a source of several bioactive adipokines, including leptin, adiponectin, visfatin, resistin, apelin, and type I plasminogen activation inhibitor (PAI-I). These adipokines are directly associated with physiological and pathological processes involving OS.

Leptin is a hormone that is secreted by adipocytes in amounts that are directly proportional to adipose tissue mass and triglyceride levels. The function of leptin is primarily anorexigenic; it binds to proteins, circulates in the plasma, reaches the central nervous system, and promotes satiety. However, it has been postulated that obesity is associated with increased levels of leptin and that a decrease in leptin's anorexigenic effect via resistance mechanisms occurs in obese patients. The mechanism by which leptin promotes OS has not been determined. However, one hypothesis is that hexamethylene bis-acetamide inducible-1 (Hexim1) is involved in maintaining whole-body energy balance. These hormones may act by inducing the synthesis of cytokines such as TNF-α, interleukin-2 (IL-2), and interferon-γ and can exert their functions in various cell types, including T cells, monocytes, neutrophils, and endothelial cells. Studies have also shown that leptin increases serum levels of C-reactive protein (CRP), confirming its pro-inflammatory effect.

In contrast to leptin, adiponectin, which is secreted by differentiated adipocytes, has anti-inflammatory and anti-atherogenic effects. It inhibits the adhesion of monocytes to endothelial cells, the transformation of macrophages into foam cells, and the activation of endothelial cells. Adiponectin also decreases TNF-α and CRP levels and increases the release of nitric oxide (NO) from endothelial cells. A deficiency in this hormone results in decreased levels of NO and reduced leukocyte adhesion, leading to chronic vascular inflammation. It has also been observed that TNF-α and IL-6 are potent inhibitors of the synthesis of adiponectin and other adipokines, including visfatin. Exposure of adipocytes to high levels of ROS also suppresses the production of adiponectin. These mechanisms explain why low levels of adiponectin are found in obese individuals.

Visfatin, a recently discovered adipokine, has been positively correlated with the accumulation of adipose tissue. In addition, the level of this hormone decreases with weight loss. Visfatin has pro-oxidant and pro-inflammatory activity and is elevated in obese individuals compared with normal-weight individuals. It stimulates leukocytes and the production of pro-inflammatory cytokines (IL-1, IL-6, and TNF-α) and promotes the generation of ROS.

Resistin, a compound present at low levels in adipocytes and at high levels in circulating monocytes, was initially described as an adipokine that is involved in the regulation of appetite, energy balance, and insulin resistance. However, other studies have shown that resistin is associated with an increase in the incidence of cardiovascular disease in obese individuals. The mechanisms involved are directly related to OS and involve the activation of endothelial cells and the upregulation of adhesion molecules and pro-inflammatory cytokines in vascular walls.

Complications of Obesity:

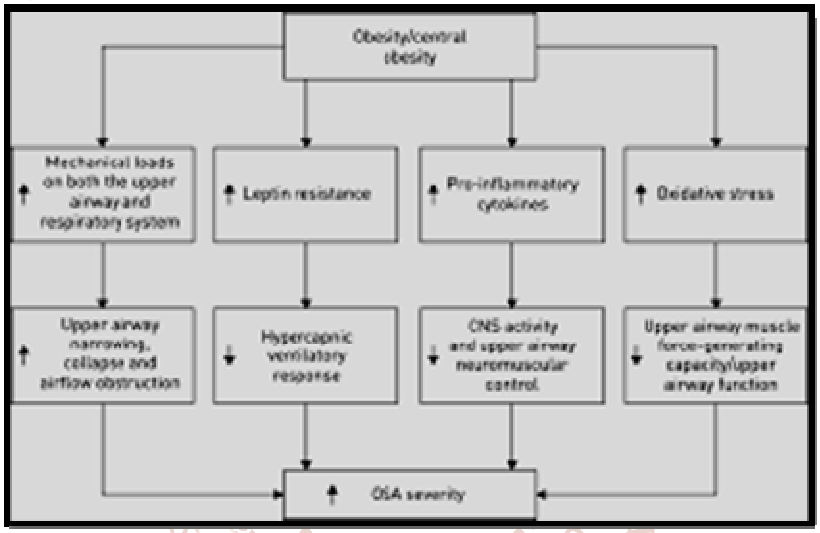

Figure-08

Prevention and management of central obesity

A. Modified and healthy life style:- Lower duration of sleep and sleeplessness night lead should be avoided and every adult person should sleep 6-8 hours every day and that must be a quality sleep. A regular physical exercise should be done everyday at least for 45 in min - 1 hours.

A balanced diet with all component of nutrition in proper ratio should be taken and unhygienic and junk food must be avoided. And always the meal of breakfast should be heavy and meal of dinner should be light.

Any type of abusing substances in nature like alcohol, tobacco etc. must be avoided.

B. Treatment of disease and syndrome:- Any kind of psychological syndrome like stress, anxiety, sleep disorder like insomnia, etc, hyperphagic tendency should be treated with proper counselling and meditation.

Any kind of metabolic disease like type 2 diabetes mellitus, insulin resistance, fatty viscera should be treated with medications, exercise and hormonal treatment

Surgery:- central obesity ca be cured by lypo suction.

Conclusion:

From the above discussion, one can understand easily about the threats of obesity, which is a resultant of unhealthy lifestyle. Ignorance to the obesity may results into several life-threatening outcomes, which can leads to even death. So, it is better to understand basic concepts of life style modification and act accordingly.

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