Review Article

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The Obesity and Periodontal Disease: A Review Article.

Abstract:
Obesity is multifactorial disease affecting developing and developed countries worldwide. It is systemic condition associated with several diseases with increased rate of morbidity and mortality. It is major public health problem with various comorbidities affecting general as well as oral health. Periodontitis is an inflammatory condition resulting in destruction of supporting structures of teeth, connective tissue, periodontal ligament and alveolar bone resorption. There is possible correlation of obesity and periodontal disease. This article aim to review about the relationship about periodontitis and obesity and recommend some possible measures to detect cases at an early stage .

Keywords: Obesity, Periodontal Disease, Inflammation

Introduction:

World Health Organisation has defined Obesity as abnormal or excessive accumulation of fat in the adipose tissue[1]. Obesity is considered to be major public health problem Worldwide affecting the overall quality of life[2].

Obesity has been considered to be significantly associated with increased adipose tissue, hypertension, Diabetes mellitus, cardiovascular diseases, hyperlipidemia, cerberovascular diseases[3]. Some of the studies have suggested that obesity also had a significant link with periodontitis and prevalence of morbidity and mortality of cardiovascular diseases[4].

Periodontitis is inflammatory condition affecting the supporting structures of teeth, loss of teeth, demolition of periodontal structures and alveolar bone resorption[5]. Various cytokines like tumor necrosis factor, Interleukin-[1] ,Interleukin-[6] are produced by inflammatory diseases. It has been found that there is a similar pathophysiology between obesity and periodontitis where pro inflammatory cytokines and hormones together called as adipocytokines are released by adipose tissue[6].

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Obesity is multifactorial in origin with wide range of etiological causes contributing to obesity are genetics, metabolic factors, biological factors, diet, physical activity, medication which ultimately lead to variation between intake of energy and energy expenditure[7]. Obese patients have increase resistance to insulin which can lead to hyperinsulinemia, hyperlipidemia, hyperglycemia.

BMI Range	Classified as
<18.5	Underweight
18.5 – 24.9	Normal
25.0 – 29.9	Overweight
30.0 – 34.9	Obesity class I
35.0 – 39.9	Obesity class II
>40.0	besity class III

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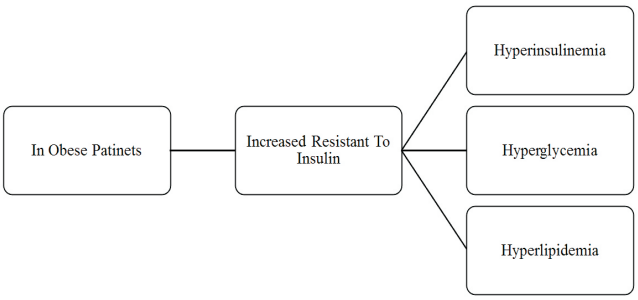
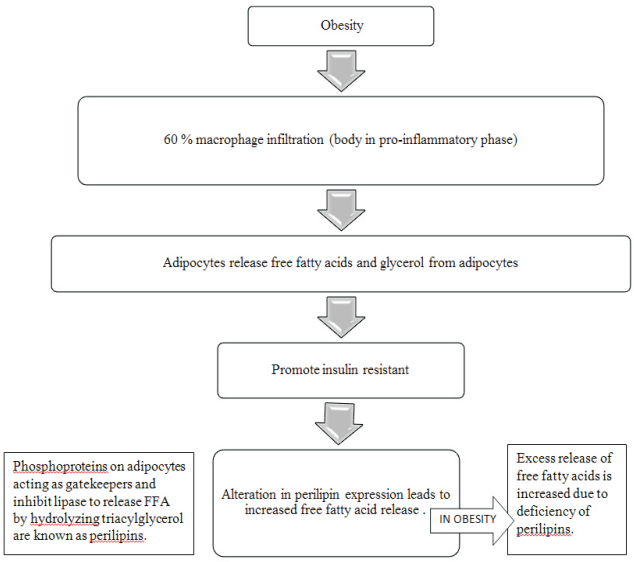
Diabetes Mellitus, hypertension, cardiovascular diseases, ishaemic stroke, obstructive sleep apnea, asthma, Gastrointestinal abnormalities like GERD,Osteoarthritis, Polycystic ovary syndrome in females, impotence and infertility in males, cholelithiasis



PATHOPHYSIOLOGY OF OBESITY



Obesity and Periodontal Disease:

It has been found that Obesity is second strongest risk factor after smoking leading to destruction of periodontal disease. Perlstein et al in 1977 first reported that there is relation of obese people and periodontitis9. They suggested that more bone loss is present in obese rats on comparison with normal one[10].

Later , after 21 years the first human study was conducted by Saito et al. on the Japanese population, revealed that obese subjects have [8.6] times higher probability of suffering from periodontitis than the non -obese ones[11]. Some of the epidemiological increase in production of pro-inflammatory cytokines like Interleukin, TNF-a, Interleukin-[1] and 6 and various bioactive substances like bioactive reactive species which may affect the periodontal tissues directly[12]. Individual is called as obese person when his or her BMI is more than or equal to [30].

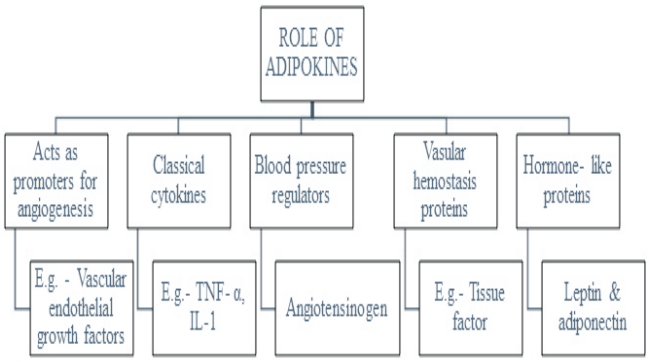
Quetelet's Index is known as Body Mass Index, the most common tool to calculate Obesity. The calculation of BMI is done by dividing a person's body weight (kg) by squaring height in metres(m2)[13].

The exact mechanism for association of obesity and periodontitis are not yet investigated. However , Hormones and adipokines play an major important role.

Recent advances in inflammatory responses such as periodontitis focus on adiponectin, leptin and resistin[14].

Adipokines:

Adipokines altogether consists of adipocytes, pre-adipocytes, macrophages which produce more than 50 bioactive molecules



Leptin:

Leptin was first discovered adipokine in 1994 .It has main pro and anti-inflammatory action. It is secreted by adipocytes15. The stimulation of monocyte and macrophage function leads to anti-inflammatory action. It main role is to increase the life span of osteoblasts by stimulation of bone formation.

Adiponectin:

Adipocytes produces adiponectin. It is a hormone which is secreted by adipose tissue and plays an important role in the

metabolism of glucose and lipid[16]. In addition to this it also possesses anti-inflammatory properties.

Resistin:

Resistin is secreted by adipocytes which belong to family of resistin like molecules. It causes insulin obstinacy during inflammation[17]. Levels of Resistin rises with increase in age.

Tumor Necrosis Factor –Alfa:

It is secreted from macrophages and accumulated in adipose tissue. It enhance the periodontal degradation[18]. It is considered to be important anti-inflammatory adipokine. Insulin resistance is increased which further induce production of C-reactive peptide and general systemic inflammation. It is believed that TNF promote hepatic dyslipidaemia and decreases insulin level.

Polyphenols and Periodontitis:

Production of crevicular fluid is increased after inflammatory stimulation and chemotaxis of polymorphonuclear lymphocytes which further inactivate the pathogens of periodontitis and which release oxygen species and hypochrolus acid[19]. External factors such as smoking, diabetes, obesity and metabolic syndrome increases local oxidative stress. Periodontal destruction occurs after disequilbrium between oxidative stress and antioxidant activity.

Interleukin – 6

Interleukin – 6 is produced by adipose tissue mainly by abdominal fat. IL-6 is found to be produced by cells than adipocytes[20]. It is pro-coagulant which increases plasma concentration of fibrinogen ,plasminogen,C-reactive peptide. Increase level of Interleukin – 6 thought to be increased in cardiovascular diseases.

Reactive Oxygen Species:

It is believed that reactive oxygen particles are the result of cellular metabolism but overproduction of reactive oxygen species induces destruction by DNA, lipids and proteins[21]. Some studies have concluded that there is close correlation of obesity and periodontitis with chronic inflammation[22]. This mechanism can lead to Oxidative damage of gingival and progression of periodontitis.

Conclusion:

Obesity is a multifactorial disease and its correlation with dental status has been realized by researchers in the last few years. Oral health professionals must be aware of increase obese people and its relation with multiple risk factor syndrome for general as well as oral health. There is multifaceted association among periodontitis ,obesity and chronic diseases[23]. Studies have suggested that adipokines like leptin, resistin and adiponectin plays an important role in inflammatory process. However exact mechanism is yet to be investigated[24]. Dentists must counsel the persons with obesity regarding the complications and severity of this disease. Weight of individuals must be examined along with the oral examination on regular basis. Promotion of healthy lifestyles ,physical activity should be recommended to reduce the burden of disease[25].

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