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A Study to Find a Correlation between *Medoroga* and Osteoporosis - A Critical Review

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ABSTRACT

In this most advanced techno-age making the human race is more susceptible to lifestyle-related disorders obesity has become a pandemic affecting the living standards of human beings. Obesity is the precursor of several comorbid conditions. Several recent studies have proposed that obesity is a risk factor for certain fractures indicated a strong contrary association between obesity and bone mineral density. As per the *Saptadhatvatmak PurushSiddhanta* (Principle of 7 body constituents), *Meda* (Fatty tissue) and *Asthi* (Bone tissue) *Dhatu*(body constituents) are interdependent and Health statistics link obesity with an increased risk of fracture which has an important public health implication and emphasizes the need to develop effective strategies to reduce fracture risk in obese subjects. The study is aimed to find the correlation between obesity and osteoporosis through a critical review of classical literature, modern literature, and published research work in PubMed and Google scholar index journals. The recent advances in biochemistry help to recognize the relationship between obesity and osteoporosis. The study shows that consuming a high-fat diet, inactive lifestyle, or lack of physical exercise hampered several metabolic pathways (RANKL/RANK/OPG Pathway), changes the fate of mesenchymal stem cells from their normal destination, and altered bone homeostasis. The above study can answer how the etiopathogenesis of obesity-induced osteoporosis can be interrupted by adopting a healthy lifestyle and proper choice of treatment.

Key Words:*Obesity, Medorog, Osteoporosis, Ashthikshaya,* RANKL/RANK/OPG Pathway

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INTRODUCTION

Advanced technology leads to deprived health due to stagnant lifestyle. Obesity is one such lifestyle disorders which is the precursor of several comorbid conditions. Obesity in urban India is emerging as a major health problem because of changes in lifestyle and food habits. In a study,the prevalence rate of obesity and central obesity in Men in India is2.7%.[1]Health statistics link obesity with an increased risk of fracture which is an alarming issue and suggest the need

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tocreate awareness regarding the same.[2]In *Ayurveda,* it is also called *"Medoroga"or "Sthualya". Acharya Charaka* explained it under *Ashtuaninditiya Adhyaya* (eight condemnable disorders*).*[3]As per *Ayurveda Sthualya* (obesity)is a disorder of *Medovaha Stratos.*[4]Ayurveda describesthe human body as an 'equilibrium' between*Dosha*, *Dhatu,*and *Mala*. Ayurveda's *SaptdhatwatmakPurushSidhhant* is a critical principle for understanding the etiopathogenesis of any disease and assists in disease treatment planning. *Meda* is the fourth *Dhatu* out of seven. [5] Among these *Dhatus, Asthi Dhatu* is in charge of maintaining the body's structural structure. [6] According to *Acharya Charak* in *Chikitsa Sthana*, *Asthi* is produced from *Meda Dhatu*.[7]*Meda Dhatu's* primary role is to nourish *Asthi* (bones) and to strengthen the body.[6]*Strotas* are specialized canals that transport transformed*Dhatus* to replenish subsequent *Dhatu*. *Medovaha Strotas* carries a modified version of *Medodhatu* (1-25 dihydroxycholecalciferol).[8]*Medoroga* is defined in *Ayurveda* as an excessive build-up of *Meda* (fat/adipose tissue) and *Mamsa* (flesh/muscle tissue), resulting in flabbiness of the hips, belly, and breast. [9, 10] Obesity is defined as *Santarpanothha Vikar* based on dietary state (disease due to consumption of excessive calorie intake). [11] It is designated as a condemnable disease [3] because it is difficult to cure since the *Dhatu* (*Meda Dhatu*) and *Dosha* (*Vat Dosha*) involved in the *Samprapti* (etiopathogenesis) of obesity have opposing properties, making *Brimhan* and *Langhan*therapies ineffective. [12]Obesity was thought to protect against osteoporosis because mechanical loading enhances osteogenesis.[13]Recent study, however, shows that fat adults are more prone to bone fractures from ordinary falls. [14] Osteoporosis is a disease that causes declining bone density, making bones prone to fracture, frequently spontaneously, under the mechanical pressures of daily living. *Medoroga* is commonly prevalent in persons who lead sedentary lifestyles, and vigorous exercise is essential for calcium absorption for bone modelling (the act of keeping bone health). Certain lifestyle adjustments and suitable therapies can help *Medoroga* patients avoid osteoporosis. The purpose of this study was to determine the relationship between *Medoroga* and osteoporosis in order to investigate the core notion of "*MedahPushtimasthnam*."[6]

AIM

To find the correlation between Medoroga and osteoporosis through critical review.

OBJECTIVES

1. To analyze the fundamental concept of *"MedahPushtimasthnam".*
2. To develop effective strategies to reduce fracture risk in obese subjects.
3. To elaborate the concept of *Dhatuposhan* w.s.r.t. *Asthiposhan* from *Meda Dahtu.*

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MATERIALS AND METHODS

The study was carried out through a careful examination of ancient literature, contemporary literature, and published research work in PubMed and Google Scholar index journals. The most likely relationship between the acquired information has been established and is being presented carefully.There are three sections to the literary review. The first is the *Rachana-Sharir*(Anatomy) of *Medoroga*, followed by a review of Medoroga and osteoporosis, and finally biochemical pathways that reveal the relationship between fati.e.,*Meda* and bone.

***Rachana- Sharir* (Anatomy) of Medoroga:**

Medoroga is characterized as an abnormal and excessive development of *Medo Dhatu* in conjunction with *Mansa Dhatu*, resulting in a pendulous appearance of the buttock, belly, and abdomen. *Medoroga*, also known as *Sthaulya*[9, 10] is a reprehensible disease.[3]Excessive Meda Dhatu in *Medoroga*blocks the *Strotasa* (circulation channel), resulting in successive *Dhatu* undernutrition.It is also regarded as a palliative illness because both nourishing therapy *(Brimhana)* and fasting *(Langhana)* have been shown to be inadequate therapies. [12]To find the root cause, and to develop an effective strategyto reduce fracture risk in obese subjects we must analyze the disease at the molecular level with the help of *Saptadhatwatmak Siddhant*(Law of seven basic constituents of the body) and *Aashrayashayi Bhava* (The Home and Dwellers).

***Saptadhatvatmak Siddhant*:**

Saptadhatvatmak Siddhant is highly important in determining the underlying cause of any condition. According to *Ayurveda*, our body is composed of seven *Dhatus: Rasa* (transformed ingested food undergone digestion), *Rakta* (Blood), *Mansa* (Muscles tissue), *Meda* (Fatty Tissues), *Asthi* (Bone tissue), *Majja* (Bone Marrow) *and Shukra* (reproductive tissues).[15]As these *Dhatus* exist to preserve and support our body's health, they are referred to as *Dhatus.* All of these are nurtured first by the individual's*Jatharagni*(digestive juice in duodenum*)*, which works on the ingested food and converts it into *Ras Dhatu.* This*ahara Rasa* would then be transported into each level of *Dhatu* (bodily tissues) through vessels as mentioned in *KedarkulyaNyaya* to nourish the succeeding *Dhatu*. Each *Dhatu* performs its unique role in order to keep the body healthy.*Medo Dhatu* is the fourth *Dhatu* among the seven.[5] *Meda Dhatu* is responsible for making the body unctuous, sweating, strengthening the body, and nourishingAsthi *Dhatu* and receive its nutrition from the *Mansa Dhatu*.[6]

Pramana of *Meda Dhatu*- 2 *Anjali*[16]i.e., approximately 320ml.*Medodhara Kala Sthana* (Location): *Aacharya Sushruta* pioneered the notion of *Kala* in *Sharirsthana*. *Dhatu* (tissue) and *Ashaya*(Cavity) are separated by this restricting membrane. There is seven *Kala* in all, with *Medodhara Kala* being the third.It is supposed to be found in the *Udara* (abdomen), *Anu Asthi* (short bones), and *Majja* (bone marrow) in the long bones.[17]

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Dosha: it is the *Sthana* of *Kapha Dosha*.[18]

Strotas: It is the *Moolasthana* of *Asthivaha Strotas and Sweadavaha Strotas*.[19]

Medovaha Strotas: The *Strotas* carry transformed tissues to their destined organ.[8] *Medovaha Strotas* carry *Dhatu* which provides nutrition to *Asthi Dhatu*. Its *Moolasthanas*are*Vrukkau*(Kidneys) and *Vapavahanam* (omentum) [19]and *Kati* (Waist). [20]

Ashraya-Ashrayi Bhava:Meda Dhatu gives shelter to *Kapha Dhatu.*[18]

Function: *Sneha* (Greasy Skin), *Sweda* (Perspiration), *Drudhatva* (Strength), and *Asthipushti*(Nourishment of bones) [6] and also maintaining the *Snigdhata of Netra* (Eyes) and *Gatra* (Body parts). [21]

***Ashraya-AshrayiBhava*:**[18]

The terms *ashrayashrayi* bhava refer to the places (*Ashraya*) where tissue live and the tissue(*Ashrayi*) who live there.[29]*Asthi Dahtu* gives shelter to *Vata Dosha*, *Rakta Dhatu* and *Sweda* gives shelter to *Pitta Dosha*, and the rest of the *Dhatu* and*Upadhatu* gives shelter to *Kapha Dosha*.[18] This principle of *Ashraya-AshrayiBhava* helps us to understand the etiopathogenesis of the disease and also to plan the line of treatment of any disorders. This principle proposes that if the *Ashraya Dhatu* get vitiated the *Ashrayi Dosha* also get vitiated or vice versa, like vitiated *Medo Dhatu*, leads to vitiation of *Kapha Dosha* and these vitiated *Meda Dhatu* and *Kapha Dosha* cause *Strotorodh* i.e., obstruction of channels and resulted into *Margavarodhjanya Vata Vrudhhi* (vitiation of *Vata* Dosha). But the exception is *Vata Dosha* and *Asthi Dhatu*. This signifies that a vitiated *Vata Dosha* will result in *Kshaya*of(diminished) *Asthi Dhatu*. [22]

***Dhatuposhan Nyaya*:**

To comprehend the idea of *Agni*, Charaka introduced the theory of nutrition transit in *Chikitsasthana. Kshirdadhi Nyaya* (Law of Transformation), *Kedarikulya Nyaya (Law of Transportation)*, and *KhalekapotNyaya* (Law of Selection) are the three.[23]According to *Kshirdadhi Nyaya*, the entire preceding Dhatu is turned into the next Dhatu. According to *Kedarikulya Nyaya*, all Dhatu acquire sustenance through channels, however according to *KhalekapotNyaya*, each Dhatu selects essential nutrients from the *Aahar Rasa*. According to *Ayurveda,Ahar rasa* is initially changed into *Prasad Bhaga* (Nutri-rich part) and *KittaBhaga* (waste part). *Prasad Bhaga* is separated again into *Sthul* (bulky part) and *Sukshma*(essence parts). *ShtulAnsha* nurtured that Dhatu, and *SukmaAnsha* fed the subsequent Dhatu. And once again, this has all been dependent on *Agni* of that *Dhatu* itself.Following meal consumption, digestion converts it to *Aahar Rasa*, which is delivered to the liver via portal circulation from intestinal blood capillaries.This nutrient-rich blood is processed in the liver to filter out the poisonous material and make the nutrient appropriate for absorption.This processed blood is delivered from the liver to the heart via inferior vena cava and thereafter to all of the body's cells.Upto this part nutrition is to delivered via

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KedarikulyaNyaya.Each organ receive blood from the arteries through small arterioles, where they receive their nutrition through the process of selective absorption i.e.,*Khalekapot Nyaya*.

Ayurvedic perspective of *Medoroga*:

Medoroga is characterized as an abnormal and excessive development of *Medo Dhatu* in conjunction with *Mansa Dhatu*, resulting in a pendulous appearance of the buttock, belly, and abdomen. *Medoroga*, also known as *Sthaulya*[10], is a reprehensible disease [3] since it lowers the body's immune system, rendering it susceptible to a number of other ailments. *Avyayamat*(no exercise), *Diwaswap*(daytime sleep), *Shlesma-Ahara Sevan* (heavy, fatty, excessive food), *Madhur Ahar*(sweet diet), (high-fat diet), *Achintanat*(carefree or unstressed lifestyle), and *Swabhavat*(obvious) have all been identified as factors of *Medo Dhatu* vitiation.[24, 25]When *Meda Dhatu*gets vitiated, because of its *Snigdha* (unctuous) and *Picchhil* (Viscid) qualities, it obstructs the channel of circulation of tissue fluid, making the succeeding *Dhatu*, *Asthi Dhatu*, to be deprived, as well as other *Dhatus* such as *Rakta* and *Mansa* to be deprived by obstructing channels. Again, blockage causes *Vata* vitiation (*Margavarodhjanya Vata Prakopa*) and stymies the digestive process. Vitiated *Vata Dosha*, make *Jathargni*(digestive Power more compelling, leading to increased appetite and thirst. [26]*Dhatu- Mansa* and *Meda*, *Dosha- Kapha* and *Vata*, and *Mala- Sweda* are the *SamraptiGhatak* (etiopathogenesis) involved in the manifestation of *Medoroga*.It is also regarded as a palliative illness because both nourishing therapy *(Brimhana)* and fasting *(Langhana)* have been shown to be inadequate therapies. [12] To find the root cause, we must analyze the disease at the molecular level.

Symptoms of *Medoroga*:

The *Medoroga* patients show eight inherent defects in them: reduced lifespan, constricted or limited movement (hampered due to loose, tender, and heavy fats), reduced sexual activities or impotence (due to small quantity of semen produced and obstruction of the channel of semen by *Medas*), debility (due to *Dhatu* imbalance), emit bad smell (due to the inherent nature of fatty tissues as well as excessive sweating), excess appetite and thirst (due to vitiated Vayu in the body causing excessive digestive fire (*Agni*). [27]

Treatment:

As per *Arundatta*, *Brimhana*, and *Langhana*, both types of *Chikitsa* do not play a significant role in the management of *Medoroga*. As *Brihan* causes more vitiation of *Meda* and *Kapha* whereas *Langhan* causes vitiation of *Vata Dosha*.[12]So, sidestepping from above mentioned causes and adopting active lifestyle along with *Guru-Apatarpan*[28] like use of *Shaljatu Kalpa*, *TriphalaLauha*, etc. medicinal therapy is prescribed in *Ayurveda* for the management of *Medoroga*.[29]

Osteoporosis:

Osteoporosis is described as a decrease in bone mass that raises the risk of fracture. The World Health Organization (WHO) defines osteoporosis as a bone mineral density that is 2.5 standard

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deviations or more below the mean peak bone mass (average of young, healthy people) as determined by dual-energy X-ray absorptiometry. [30]Primary and secondary osteoporosis are the two kinds of osteoporosis. In women, primary osteoporosis is caused by ageing and oestrogen deficiency following menopause. Secondary osteoporosis, on the other hand, can arise at any age as a result of chronic predisposing factors such as medical conditions, long-term treatment, and so on.[31]In such cases, body cells attempt to preserve homeostasis by recycling nutrients and energy via the autophagy process.[32] Bone remodelling is one such process. This process of bone remodelling is regulated by osteoblasts, which are bone-forming cells, and osteoclasts, which are bone-degrading cells. Pathological problems may result from an imbalance in bone remodelling.

Because it is a subtle condition, it requires special care because a simple fall can cause spinal fracture or femoral neck fracture can result in significant body deformity, which is related with morbidity and increased mortality. [33, 34, 35] BMD either peripheral or central DEXA scanning is used to detect osteoporosis, and the BMD test result is defined by T-score. The ICMR research suggested 0.624, 0.428, and 0.717 gm/cm^2 cut-off values for osteoporosis in women and 0.661, 0.48, 0.714 gm/cm^2 in males at the hip, forearm, and spine, respectively. [36]

Bone metabolism:

Bone is a mineralized connective tissue that is extremely vascular. Bone is a dynamic and vital tissue in our bodies since it builds the basis of our skeleton. Bone modelling and remodelling occur throughout life and are critical to skeletal homeostasis. Bony cells are classified into four types: osteoprogenitor cells, osteoblasts, osteocytes, and osteoclasts. Osteoblasts are cells that produce bone. It secretes organic matrix and alkaline phosphatase, which help to mineralize the fabric matrix and turn it into the osteoid matrix. And the osteoblast was encased in the osteoid matrix and labelled as an osteocyte. Osteoblasts develop from osteoprogenitor cells, which are mesenchymal stem cells found in bone marrow. Depending on conditions such as signalling pathways, transcription factors, age, and metabolism, this mesenchymal stem cell can develop into either an adipocyte or an osteoblast. [37] Adipogenesis of MSCs rather than osteogenesis is also aided by altered metabolism. Obesity has been shown in numerous studies to modify the differentiation potential of BMSCs in favour of enhanced adipocyte development and impaired osteoblast and chondrocyte differentiation, contributing to a discrepancy in bone homeostasis.[38, 39]

Vitamin D insufficiency in obese individuals:

Vitamin D is a lipid-soluble nutrient. Cholecalciferol, a precursor of cholesterol, is the natural form of vitamin D. Vitamin D activity can be obtained from ergocalciferol and cholecalciferol. During the production of cholesterol, 7-dehydrocholesterol is generated as an intermediary. Vitamin D is produced in the skin as a result of the action of UV radiation on

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7-dehydrocholesterol. The production of vitamin D3 in the skin is proportional to the amount of sunshine received. A particular hydroxylase found in the liver first hydroxylates cholecalciferol at the 25th position to become 25-hydroxycholecalciferol (25-OH D3). The most common storage and circulation form of vitamin D is 25-OH D3.The active form of Vitamin D is 25-hydroxycholecalciferol (25-OH D3), which is further hydroxylated into 1-25 dihydroxycholecalciferol (1,25 DHCC) by the activity of the enzyme 25 dihydroxycholecalciferol 1-hydroxylase found in the kidney. 1-25 dihydroxycholecalciferol (1,25 DHCC) increased calcium absorption from the stomach and helped to preserve bone health. Calcitriol is derived from 1,25 DHCC, which has three hydroxy groups (1,3, and 25 carbon). Calcitriol controls calcium and phosphate levels in the blood at three separate levels. [40]

Calcitriol boosts calcium and phosphate absorption in the intestine. Second, it enhances calcium absorption by osteoblasts for calcium phosphate deposition and, in conjunction with parathyroid hormone, increases calcium and phosphate mobilization from bone. Finally, it helps to reduce calcium and phosphate excretion via the kidney by increasing reabsorption.These events occur in reaction to low plasma calcium concentrations. According to one study, serum vitamin D and excessive fat gain are inversely related.[41]

Lipid metabolism: Lipids were once thought to be our body's primary source of energy. It has been established that lipids are an important component in maintaining our body's equilibrium. Because lipids such as triglycerol, phospholipids, glycolipids, and cholesterol make up the majority of the cell membrane, they operate as a mediator in numerous signalling pathways. Triglycerides make up the majority of dietary lipids. The activity of pancreatic lipolytic enzyme and bile salt in the small intestine digests almost all lipids. The pancreatic enzyme can digest lipid only after it gets emulsified by the bile salt which acts as a detergent and breaks down the lipid into small lipid droplets. Bile salt is an end product of cholesterol degradation. Bile salt plays important role in lipid digestion. Free fatty acid, cholesterol, and monoglycerides are the final product of lipid digestion and absorbed by the intestinal mucosa from the lumen, where they are re-esterified to form triglycerides and are unified into chylomicrons; the chylomicrons are then released into intestinal lymph, secreted into blood circulation through the thoracic duct. Lipoproteins are the key protein thatis responsible for the transport of lipid to and from the tissues. The fatty acids penetrate through the capillary endothelial cells and are either stored in adipose cells or oxidized in skeletal muscle cells. [42]

Obesity and bone metabolism:

"Obesity and overweight are defined as abnormal or excessive fat build-up that poses a health concern," according to the World Health Organization. Overweight is defined as a BMI of 25 or more, while obesity is defined as a BMI of

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30 or more.[43] Overweight and obesity are identified as abnormal or excessive fat accumulation that can lead to impaired health. Obesity is a systemic disease of the white adipose tissue (WAT). Adipose tissue is anatomically distributed in diverse proportions throughout the human body, and the pattern of distribution is dependent upon many factors including sex, age, race, ethnicity, genotype, diet, physical activity, hormone levels and medication. In humans, adipose tissue is located beneath the skin (subcutaneous fat), around internal organs(visceralfat),Inbonemarrow(yellowbonemarrow), intermuscular (Muscular system), andinthe breast (breasttissue).In a mammal, adipose tissues are white adipose tissue (WAT) and brown adipose tissue (BAT). According to the site, adipose tissues are classified as parietal/ subcutaneous adipose tissues (SAT), and Visceral adipose tissues (VAT). Two distinct phenotypes of obesity are generalized obesity and central or abdominal obesity. Of this abdominal obesity is responsible for developing different comorbid conditions like diabetes, cardiovascular disorders, etc.[44]The researcher explored that both visceral adipose tissue (VAT) and subcutaneous adipose tissues (SAT) possess osteogenic potential.[45]The osteogenic differentiation capacity of visceral adipose tissue (VAT) is more as compared to subcutaneous adipose tissues (SAT). The osteogenic differentiation of visceral adipose tissue (VAT) and subcutaneous adipose tissues (SAT) depends on the tissue vasculature, better blood supply inducesa higher population of osteoprogenitor cells. Obesity resulted in reduction of adipocyte tissue vasculature leading to tissue hypoxia which can affects the process of osteogenesis.[46]

Obesity is a metabolic disorder. Metabolism refers to the collection of diverse and complicated processes that entail the intake and storage of macro- and micronutrients assimilated from food or metabolites generated de novo, as well as the elimination of unwanted byproducts by the organism. This intricate process involves every cell in the body, not just the major organs [47]Adipose tissue is an endocrine organ, that produces hormones that regulate body metabolism.[48]An increase in fat accumulation in adipose tissue ultimately leads to an imbalance of various enzymes like leptin, adiponectin, etc. which leads to hampered various metabolic pathways which are responsible for the maintenance of health. One such metabolic pathway which gets affected by obesity is the RANKL/ RANK/OPG pathway which is useful for the maintenance of bone health.[49,50]Adipokines such as leptin, adiponectin, and TNF ∝affectsbone metabolism by disturbing the osteoblast and osteoclast signalling.[51]Adiponectin increased osteoclast formation indirectly through stimulating RANKL and inhibiting OPG production in osteoblasts.[52] The pro-inflammatory cytokine which increases in inflammatoryconditions leads to the overexpression of RANKL by T cells that correlate with the lower bone mass density.[53]

RANKL/ RANK/ OPG pathway:

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TNF family members play an important role in cell proliferation, survival, and differentiation signalling pathways. The TNF family member, receptor activator of NF-B ligand (RANKL), receptor activator of nuclear factor kappa-B (RANK), and Osteoprotegerin OPG, govern bone modelling in osteoblasts.[54] The OPG/RANKL system has been discovered to regulate osteogenesis and osteoclastogenesis. RANKL promotes osteoclast development and activation, which influences remodelling. [55, 56]RANKL promotes osteoclastogenesis by binding to the receptor activator of nuclear factor kappa-B (RANK) on the surface of osteoclasts. Osteoprotegerin (OPG) is a cytokine receptor that serves as a decoy for the RANKL receptor. By binding to RANKL, OPG inhibits RANK-mediated osteoclast growth and maturation. As a result, the OPG/RANKL ratio is an important determinant of bone mass and skeletal firmness. [57] This shows that RANKL promotes bone resorption by multiplying osteoclasts, whereas the antagonist Osteoprotegerin (OPG) inhibits osteoclast proliferation and aids in bone loss prevention or maintenance (BMD). Obese people have lower levels of OPG in their blood. [58]The naturally occurring inhibitor of RANKL, Osteoprotegerin (OPG), was discovered to protect against bone loss.[59,60]

According to some recent studies, an obese person's impeded adipose tissue vasculature produces an increase in TNF-, IL-1, and IL-6 levels, while adiponectin levels decrease, implying that obesity is an inflammatory condition. Through the RANKL/RANK/OPG pathway, which are capable of regulating osteoclastogenesis induced by the receptor activator of nuclear factor B (NF-B) ligand (RANKL). [61, 62] Visceral fat accumulation, as opposed to subcutaneous fat accumulation, is related with greater levels of pro-inflammatory cytokines, which stimulate nuclear kappa B ligand receptor activators, which promote bone resorption, and hence abdominal obesity is linked to lower BMD.

DISCUSSION

Excessive calorie intake and less calorie expenditure led to excessive accumulation of fat in the adipocyte causing its hypertrophy. Depending on the site i.e., abdominal or gluteofemoral hypertrophy of adipocyte, the severity of obesity-related complication observed. *Dhatus* are the primary elements of the body, according to *Ayurveda*. *Dhatu* is in charge of the body's upkeep and development. *KedarkulyaNyaya, Khalekapot Nyaya, and Ksheerdadhi Nyaya* were the three modes of conveyance used to transport food from previous *Dhatus*. [18] This transfer of *DhatuposhakAnsha* (nutrients) is carried out by Strotas, according to *Kedarkulya Nyaya*. However, *Agni* (digestive fire) is extremely important for that particular *Dhatu's* sustenance. Again, there are thirteen forms of digestive fire: *Jatharagni,* 7 *Dhatvagnies,* and 5 *Bhutagni.* Whatever *AharDravyas* (meal) a person consumes is first

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digested by *Jatharagni*, who then converts it into *rasa Dhatu*. If any impediment, tumor, or other deformity occurs in the passage of these *Strotasa*, the *Uttarottar Dhatu* will *Kshaya*(declining).

As per Ayurveda *Dhatus* are the basic constituents of the body. *Dhatu*is responsible for the maintenance and growth of the body. Each Dhatu received itsnutrition from previous *Dhatu* by three means of transportation i.e.,*KedarkulyaNyaya,KhalekapotNyaya, and KsheerDadhiNyaya.*[18] As per *KedarkulyaNyaya,* this transportation of *DhatuposhakAnsha* (nutrients) is carried out with the help of Strotas. But for the nutrition of that particular *Dhatu*, Agni (Digestive fire) plays a very vital role. This digestive fire is of again thirteen types *Jatharagni*, 7 *Dhatvagnies*, and 5 *Bhutagni*. Whatever *AharDravyas* (food) consumed by the person is first digested by *Jatharagni* which transforms this digested *ahara* into rasa Dhatu. If any kind of deformity like obstruction, tumor, etc. occurs in the path of these Strotasa it will lead to *Kshaya* (declining) of the *UttarottarDhatu*. Every Dhatu has its digestive power called *Dhatvagni*. If the respective *Dhatvagni* gets deranged it causes*Vridhhi*of that particular Dhatu. *Medo-dhatvagnimandya* causes*MedaDhatuvriddhi* called *Medoroga*. As *Meda* lies at the middle in the sequence of *SaptaDhatu,Medovriddhi*disturbs the homeostasis of the body. And that's why *Medoroga* or obesity is a metabolic syndrome as the various metabolic pathways get affected due to hypertrophied adipocytes.

Strotas are specialized canals that transport *Dhatus* in their transformed state to feed subsequent Dhatus. [8]*Asthi Dhatu* is nourished by the converted form of fat carried by *Medovaha Strotas*. The *Moolasthana* of *Medovaha Strotas* is supposed to be *Vrikkau* (kidneys) and *Vapavahan* (omentum). According to the literature, the active form of cholesterol, 7-cholecalciferol, is produced under the skin in the form of vitamin D and is only changed into active form following hydroxylation in the kidney. And 1-25 dihydroxycholecalciferol (1,25 DHCC), the active form of vitamin D, is responsible for calcium absorption from the intestines or gut. [40] Adipocyte is the storehouse of energy in the form of lipid. Whenever our body needs excess energy this lipid release energy for the functioning of different tissues. Adipocyte is metabolized to acetyl-CoA by utilizing glucose which is used for *de novo* fatty acid synthesis. This free fatty acid is the lipid source for osteoblast.[39]Means Asthi means bones made up of osteocytesi.e., bone tissuesareformed from osteoblasts. This osteoblast required energy from fatty tissue i.e., adipocyte (*Meda Dhatu*) to get transformed into osteocyte i.e., *Asthi Dhatu*. And this process is governed by the metabolic pathway RANKL/RANK/OPG pathway with which is very much influences by the hormones secreted by adipocytes.[49, 50]Obesity,an inflammatory condition that secretes pro-inflammatory cytokines like TNK∝, RANKL, OPG, adiponectin, leptin which modulate the process of bone remodelling and can lead to acondition of

porous bone called osteoporosis. Mesenchymal stem cells are the common precursor of both adipocytes and osteoblasts. The fate of this mesenchymal stem cell whether to form adipocyte or osteoblast is dependent on the functioning of the adipocyte.[40] Through various researches, it is proved that the BMSCs differentiation capacity is altered by obesity in favour of increased adipocyte differentiation and impaired osteoblast and chondrocyte differentiation, which contributes to a disparity of bone homeostasis.[41]

Ahigh-fat diet or inactive lifestyle,altered the bone marrow microenvironment and mesenchymal stem cell differentiation, aidsthe production of adipocytes over osteoblasts (bone-forming cells),and makes bone fragile.

Adipocyte hypertrophy has two negative effects. First is adipose tissue blood flow reduction (ATBP reduction) which lessens the osteogenic differentiation and second Vitamin D remains stagnant in the adipocytes, lowering the serum vitamin D level.[43]This reduces calcium absorption from the intestine, lowering serum calcium, which is one of the triggering factors for bone resorption. Regular exercise causes adipocytes to release stored Vitamin D into the bloodstream.However, in the management of obesity, *Aushadhi Dravya* (drug therapeutics) and diet must also be considered. One should prefer drugs with *Lekhaniya* properties that do not vitiate *Vata Dosha*, such as *Shilajutu Kalpa*, *Guggul Kalpa*, *Ushnambupana*(intake of warm water), and so on.

Obesity is a condition in which long-term consumption of Madhur Aahar combined with a sedentary lifestyle affects the *Jatharagni*, causing improper digestion of ingested food, which then affects the *Dhatwagni*, or selective absorption process, and finally the *Bhutagni*, or enzymes that control metabolic pathways. As a result, successive Dhatu became malnourished.

CONCLUSION

Obesity is on the rise as a result of lifestyle changes, which can lead to a variety of diseases such as osteoporosis and make human life suffer from the lowest living conditions. Understanding the fundamental phenomenon behind the link between obesity and osteoporosis will help us develop an effective strategy for reducing the incidence of fractures in obese people. Furthermore, a high-fat diet, an inactive lifestyle, or a lack of physical activity impeded various metabolic pathways, changed the fate of cells from their normal destination, and affected bone homeostasis, according to the study. The aforesaid study can shed light on how the etiopathogenesis of obesity-induced osteoporosis can be halted by adopting a healthy lifestyle and selecting the appropriate treatment.

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