Review Article

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STHAULYA, AMA, AND MEDA DHATU, THE DYSFUNCTIONAL NUTRITIONAL METABOLISM WITH SPECIAL REFERENCE TO OXIDATIVE STRESS: A NARRATIVE REVIEW

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ABSTRACT

Obesity and its accompanying consequences are increasingly linked to oxidative stress, as evidenced by a growing body of research. Several biochemical pathways, including superoxide production from NADPH oxidases and glyceraldehyde auto-oxidation, can cause systemic oxidative stress in obese individuals. Hyperleptinemia, inadequate antioxidant defence, chronic inflammation, and postprandial reactive oxygen species production contribute to oxidative stress. This Oxidative stress is related to or co-related to "*Ama*". *Ama* is generated by improper or insufficient digestion of food particles due to a lack of *Jatharagni* and *Mala* accumulation in the body. Modern physiology has linked *Ama* to potentially harmful Free radicals. It is essential to neutralize free radicals because they have one or more unpaired electrons. Oxygen-free radicals, or "Reactive Oxygen Species," make up most dangerous free radicals. *Ama* is in the same boat as *Avipakam* (incompletely metabolized) due to its metabolism. This Ama brings on many diseases. Many diseases have been linked to free radicals.

Keywords: *Sthualya*, Obesity, Oxidative stress, *Ama*, Free Radicals, NADPH

INTRODUCTION

Obesity is a chronic disease that affects people's physical, financial, and mental well-being, regardless of their cultural, economic, or ethnic background. Excess body fat lowers the quality of life and increases healthcare costs and death risk. Diabetes, cardiovascular disease, cancer, asthma, sleep disorders, hepatic, renal, and infertility are all associated with obesity.[1-4] Overweight is defined as a BMI of 25.0 to 29.9 kg/m^2, while obesity is defined as a BMI of 30 kg/m^2. BMI,[5] on the other hand, does not distinguish between lean and fat mass, nor does it specify fat distribution as a distinguishing factor. Obesity-related risk factors are found to be less dependent on total body weight and more dependent on fat distribution, according to new research.[6-7] As a result, because it promotes pro-oxidant and pro-inflammatory states, abdominal fat is now recognized as a significant risk factor for obesity-related illnesses.[8-10]

This ailment is covered under *Sthaulya* in *Ayurveda*, the science of life. Because of its complicated pathologies and multifactor aetiologies, *Sthaulya* is expected to be extremely difficult to treat. The creation of *Ama* is the fundamental cause of these complexities. Because of the long-term ingestion of *Nidaana* (aetiologies), the body cannot clear the toxins created because of these aetiologies, and these toxins accumulate in the form of *Ama*. This *Ama* produces a layer all over the *Jathargni*, interfering with its function, and creates *Saama Dhatus* by interfering with *Dhatuagni's* function. This causes an excessive accumulation of *Meda Dhatu*, resulting in *Sthaulya* illness. This *Ama* is frequently linked to Free Radicals, ROS, that cause oxidative stress.

Oxidative stress has been associated with obesity in epidemiological, clinical, and animal studies. In cell culture and animal studies, oxidative stress increases pre-adipocyte proliferation, differentiation, and mature adipocyte size.[11] Reactive oxygen species have been proven to impact hypothalamic satiety and hunger neurons (ROS). N-acetyl-glycer-aldehyde auto-oxidation, polyol and hexose-amine routes are all produced by NOXPH oxidases. Obesity causes oxidative stress due to poor antioxidant defence, chronic inflammation, and postprandial ROS generation.[12-16] This paper attempts to cover the role of *Ama* in low-quality *Dhatu* formation, focusing on *Meda Dhatu*.

Literature

Literature on *Ama* (Free radicals), *Sthaulya* (Obesity), and *Meda Dhatu Poshana* (Adipose tissue nourishment and replenishment) was gathered from various *Ayurvedic* Compendia. Current literature and recent research publications were also searched online from scientific sites such as PubMed, Google Scholar, and Medline plus using keywords such as *Ama*, Free Radicals in *Ayurveda, Meda dhatu,* and *Sthaulya.* Analysing the collected descriptions allowed researchers to determine the applicability and relevance of *Dhatu Poshana* dysfunction to *Sthaulya.*

DISCUSSION

Ayurveda's core principle is keeping *Dosha, Dhatu*, and *Mala* balanced. The potency of each Jatharagni initially nourishes these three, and then productive nutrients (*Ahara Rasa*) are passed into each level of *Dhatu* (body tissues). One pool provides all the nutrients required for tissue creation and growth.[17] Among the three *Upasthambhas* (*Ahara, Nidra, Brahmacharya), Ahara* is the most important. Traditional texts explain that usually, the body follows three laws to fulfil the nutritional needs of the body by converting the food into such form so that it is absorbable (*Ksheera Dadhi Nyaya* – transformation of nutrients, *Khale Kapota Nyaya* - selective uptake of nutrients, *KedariKulya Nyaya* – transportation of nutrients via channels) but in obesity, such mechanism is deeply affected

Kshira Dadhi Nyaya (Metabolic transformation of nutrients)

Kshira (milk) is distinct from *Dadhi* (curd). Milk undergoes a complete change before becoming curd. According to this hypothesis, a certain tissue undergoes differentiation into subsequent tissues. *Rasa Dhatu*, like milk, transforms into *Rakta Dhatu* following this law. Like *Mamsa* and *Meda*, *Asthi* and *Majja* and *Shukra* are generated by the gradual stage of transformation, and *Shukra* is accountable for the cause of pregnancy. *Shukra* (garbha). *Karma Parinama Paksha* is another name for this.[18-19]

Khale Kapota Nyaya (Selective Uptake of Nutrients)

After harvesting, the grain is stored in the *Khale* (granary or bran), and *Kapot* is a pigeon or bird symbol. For example, Pigeons visit a grain pile to pick up the requisite and return to their nest. Energy is used in this procedure by *Kapotas* to meet their needs. As with pigeons (*Kapotas*), the *Rasa Dhatu* contains the nutrient components for all the *Dhatus* in the body, just like the *Rasa Dhatu* contains the grains for the *Dhatus*. [18,20,21]

Kedari Kulya Nyaya (Transporation of nutrients via channel)

In this context, *Kulya* refers to a little waterway in a paddy field called *Kedari* (*Kyari*). Water from the reservoir is delivered to tiny fields (*Kedari*) via canals as part of the irrigation process. Water moves in *Kulya* (canals) in the direction of gravitational pull. The water reaches the first section of the field (*Kedari*) and then goes on to the second half of the field via a pressure gradient (considering the first *Kedari* is filled with water, then water moves to the next *Kedari*). This mechanism explains passive diffusion through cell membranes. *Meda Dhatu* and Obesity.[18,20,22]

Dysfunction

Dysfunction of *Kshira Dadhi nayaya* in *Ama* and *Sthaulya*
In the case of *Ama* formation, the formed *Ahara Rasa* is coated with *Ama,* known as *"Sama Anna Rasa",* because the subsequent *Dhatus* made from it, i.e. *Rasa, Rakta, Mamsa, Meda Dhatu* is also covered with *Ama* and creates *Sama Dhatus*.

Dysfunction of "*Khale Kapot Nayaya*" in *Ama* and *Sthaulya*
Similarly, the consecutive *Dhatus* formed is of the lowest nutritional Quality and high in *Kapha, Guru, Snigdha* and similar properties. Therefore, the *Dhatus – Rasa, Rakta, Mamsa,* and *Meda* cannot perform their optimum.

Dysfunction of *"Kedari Kulya Nyaya"* in *Ama* and *Sthaulya*
However, in this situation, the *Dhatus* up to the point of *Meda* has been somewhat produced. Still, the nutrient pool is depleted, and the next *Dhatu* cannot pick up essential attributes required for nourishment. The *Meda Dhatuagni* has been transformed into *Manda- Medadhatwagni*, preventing further transformation.

The mode of action and the probable reason behind this process needs to be understood at the level of *Rasa Dhatu* and then *Meda Dhatu*.

RASA DHATU CAN BE COMPARED TO PLASMA

Intracellular antioxidant defences must be adequate to maintain the balance of tissues' antioxidant–pro-oxidant. Vitamin and mineral deficiencies can also impair antioxidant defence in the pathogenesis of obesity. Plasma levels of a-tocopherol or b-carotene per unit of plasma low-density lipoprotein (LDL) are well-known antioxidant protection biomarkers. Low carotenoids, vitamin C, and vitamin E levels have increased BMI. The CARDIA study found an inverse connection between BMI and total serum carotenoids (a-carotene, b-carotene, a-cryptoxanthin, and zeaxanthin/lutein). Obese children had lower serum b-carotene levels than normal control children in the National Health and Exanimation Survey. In one study, obese girls had lower plasma levels of atocopherol/LDL and b-carotene/LDL than non-obese girls. According to Aasheim and Bohmer, most obese patients have low vitamin levels, particularly A, B6, C, D, and E. Dietary vitamin C intake was inversely associated with central fat distribution in the European Prospective Population Study (EPIC). People with BMI >50 had lower plasma levels of vitamin E/triglycerides than people with BMI 30. Obese children, especially those with central obesity, had more deficient selenium and zinc levels. Obese people also have magnesium, selenium, iron, and zinc deficiency. These findings imply that low vitamin and mineral concentrations in obese people promote reduced antioxidant defence. [23-34]

MEDA DHATU CAN BE CORRELATED TO WHITE ADIPOSE TISSUE

Plasma-free fatty acids (FFA) rise in obesity, as does fat storage in the waist and hips (WAT). The mitochondrial electron transport chain generates O2 - by blocking the translocation of adenine nucleotides. At that point, the plasma FFA levels are elevated. FFA activates NOX via PKC in cultured vascular cells to produce reactive intermediates. As the conjugated fatty acids encounter oxygen, they become sensitive to oxidation, which causes the generation of free radicals and the accumulation of oxidative by-products. Fats are more susceptible to oxidative alteration due to greater 4-hydroxynonenal (4-HNE) concentrations per unit of intramuscular triglycerides in obese individuals. Obese individuals may also have a larger target for ROS oxidation because of the higher amounts of lipid molecules in their bodies. Excessive fat builds up in WAT was found to enhance lipid peroxidation within the WAT itself, according to research using animal models of obesity by Furukawa and colleagues. A decrease in the activity of antioxidant enzymes such as Superoxide Dismutase (SOD), catalase (CAT), and Glutathione Peroxidase (GPx) in WAT was found in animal experiments to be linked to obesity. The consumption of lipids in the diet also contributes to the development of chronic oxidative stress in the body. In middle-aged men with abdominal obesity, conjugated linolenic acid consumption elevated urine concentrations of 8-epi PGF2a. [35-39]

CONCLUSION

It is concluded from the above discussion and review *Ama* is the first and main culprit. This causes the *Sthualya*. There is also a clear imbalance between *Ama* formations and their removal or

neutralization; in terms of free radicals, we can say that free radical load is more, and antioxidant defence is not enough to remove these free radicals. It can be either due to excessive free radical formation or a weakened antioxidant defence system leading to Oxidative stress. It is also concluded from the above discussion that *Ama* disrupts the *Dhatu Poshan Nayayas* and creates *Sama Dhatus,* and maximum disruption is at the *Meda Dhatu*. The lowest antioxidant activity occurs in the adipose tissue, causing high oxidative stress and leading to inflammatory and pro-inflammatory states of hyperplasia and hypertrophy. This explains the complex pathology of *Sthaulya* disease.

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