Review Article

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A REVIEW ON CONCEPT OF *ATISTHOULYA* VIS -A-VIS METABOLIC SYNDROME: AN APPROACH TO EXPLORE THE CONVECTIONAL ENTITY

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ABSTRACT

Metabolic Syndrome (MetS) has been introduced in conventional medical system very recently as a rapidly emerging medical problem caused by dysfunctional metabolic state associated with obesity. It is a challenging fact for *Ayurveda* also. *Atisthaulya* is one of the oldest documented metabolic disturbance in *Ayurveda* associated with obesity. As per *Ayurvedic* literature *Atisthaulya* are associated with increased morbidity and mortality. A systemic documentation about risk factors, patho-physiological phenomena, complications and management principle of *Atisthoulya* has been described in *Ayurvedic* literatures. Hence, an understanding of Ayurvedic concept of *Atisthoulya* in conventional parlance may explore the potential field to find out a solution for MetS from *Ayurveda*.

Keywords: *Atisthaulya,* Metabolic Syndrome, Dysfunctional metabolic state

INTRODUCTION

The word *sthaulya* is derived from the word '*sthula'* that means a person having excessive growth of body specially in *udaradi* (Abdominal) region. The state of *sthula* is known as *sthaulya.* As per *Acharya Madhavkar*, often over enhancement of *meda dhatu* in *udara* (Abdomen), leads to *sthoulya*. According to various *Ayurvedic* literatures, excessive enhancement of *meda dhatu* may give rise to a grievous pathological state. Such pathological state has been termed as *atisthaulya* by *Acharya Charak* and *sthaulya* by *Acharya Madhavkar*.

In Ayurvedic literature, *ati-sthaulya* or *sthaulya* has been described not as obese state only rather than a clustering of pathological events induced by obesity.

Obesity are now in the prime focus area of interest in conventional system of medicine also due to it's association with many others diseases like coronary artery disease (CAD), hypertension (HTN), type 2 diabetes mellitus (T_2DM) etc. Obesity especially central obesity is considered potentially harmful for energy homeostasis and may give rise to metabolic disarrangement.

Obesity induced dysfunctional metabolic system are pathologically manifested by a clustering of cardiovascular diseases (CVDs) risk factors such as dyslipidaemia, impaired glucose tolerance, elevated blood presser and are termed as Metabolic Syndrome (MetS). MetS is now considered as a driving force for a new CVD epidemic. Around 20-25% population of world have Mets.[1]

MetS has serious implication on an individual health and healthcare cost. To combat with such upcoming burden is also a challenging fact for *Ayurveda*.

A standardized *Ayurvedic* management protocol is mostly unavailable in most of diseases associated with modern era as well as MetS. But prior to generate any such data, the entity of such diseases in *Ayurveda* need to be explored. The aim of the review is to explore the conventional entity of *Ati-sthoulya* by the fundamental analysis of *Ati-shoulya* and MetS, in respect to risk factors, diagnostic evaluation and patho-physiological phenomena.

Data obtained from various Medical text books, *Ayurvedic* Compendia, published scientific research sources has been collected, analyzed and presented in regard to concern topic. PubMed, Scopus and Google Scholar databases were searched for studies.

CRITICAL ANALYSIS

Diagnostic Evaluation of Atisthoulya

Ati-sthoulya has been described as a clustering of pathological events induced by excessive growth of *meda dhatu.*[2] It is a morbid condition that is associated with several grievous diseases, which even could lead to sudden death.[3] It also has been identified as a cause of reduced life span in *Charak Samhita.*[4]

According to classical Ayurvedic compendia, diagnosis of *Ati-sthoulya* are based on anthropometric, physiologic abnormality. The anthropometric abnormality includes excessive growth of of *Meda-Mamsa* in *sphig* (Gluteal region), *udar* (Abdomen)*, stana* (Breast) and that's are found to lope during walking.[5]

The physiological abnormalities have been described in classical ayurvedic compendia in terms of upadrava (complications) that includes[6] -

- *Ayuhras* (Reduced life span)
- *Jaboporodh* (Restricted movement or Hypokinesia)
- *Kriccha-vyavaya* (Sexual dysfunction)
- *Dourbalya* (Fatigue)
- *Dourgandha* (Bromhidrosis)
- *Swedabadha* (Hyperhydrosis)
- *Atikhuda* (Polyphagia)
- *Atipipasa* (Polydypsia)

Risk Factors for Atisthoulya

The following etiological factor is responsible behind the development of *sthlouya* :
1. *Ati-sampuran* (Excessive dietary habit) and *Avyam* (Sedentary lifestyle)[7]
2. Bija-swabhaba[8] (Genetic predisposition)

A wide distribution of *meda dhatu* in obese individual favor the more enhancement of *meda* in body from their dietary sources.[9]

Patho-physiological Phenomena in Atisthoulya

Two important patho-physiological events have been mentioned in regard to *Ati-sthoulya* –
1. Excessive enhancement of *meda dhatu* and *Vishama-dhatuposhana*[10] (derangement in normal physiology of *dhatu*)
2. *Meda-dusti*[11] (Vitiated *meda dhatu*)

In *Ati-sthoulya*, there is excessive enhancement of *meda dhatu* as well as impaired nourishment in others *dhatu* is found in individual. These two phenomena lead to development of various complications. Vata is the considered as a key *dosha* behind the development of pathological consequences occurs in *Ati-sthoulya*.[12] Vitiated *vayu* are responsible for vitiation of *agni* in *Ati-sthoulya*.

Diagnostic Evaluation of Metabolic Syndrome (MetS)

MetS is a state of dysfunctional metabolic system induced by obesity and characterized by impaired glucose tolerance, dyslipidaemia and elevated blood presser level. All this increases the risk for CVD in a normal human subject. Central obesity plays a key role behind the whole event. Mets severely effect on morbidity and mortality.[13] MetS is a clinical condition composed of anthropometric, physiologic, and biochemical abnormalities predisposing affected individuals to the development of type 2 diabetes mellitus (T2DM) and cardiovascular disease (CVD).

Multiple sets of diagnostic criteria for MetS were initially created to identify insulin-resistant subjects or to predict clinical events, such as cardiovascular disease. At present, 5 separate definitions for MetS exist:
- The World Health Organization (WHO) definition (1999),
- The European Group for the Study of Insulin Resistance definition (1999),
- The American Association of Clinical Endocrinologists position statement (2003),
- The Adult Treatment Panel III (ATP III) guideline (2005)
- The definition from the International Diabetes Federation Consensus Group (2005)

Each of these definitions is based on both anthropometric and biochemical abnormality as well as have share certain common elements, such as anthropometric criteria relating to obesity biochemical abnormality such as hyperglycemia, dyslipidemia, and hypertension. But the laboratory value thresholds and the number of positive criteria required for diagnosis differ according to definition.[14]

Diagnostic criteria of MetS According to International Diabetes Federation includes[15] :
Central obesity (defined as waist circumference, male >90 cm and female >80 cm In case of South Asian).

With Any of following two-
1. Raised triglyceride>1.7 mmol/L or specific treatment for this lipid abnormality.
2. Reduced HDL cholesterol<1.03mmol/L (in male), 1.29mmol/L (in female) or specific treatment for this lipid abnormality.
3. Raised Blood Pressure: SBP≥130mmHg or DBP≥85mmHg
4. Raised fasting plasma glucose≥5.6mmol/L or previously diagnosed Diabetes Mellitus

Risk Factors for MetS

Obesity specially associated with visceral adiposity is impotent risk factors for MetS. MetS are explained by the complex interaction between
a) Multiple genetic factors and
b) Environmental predisposing factors like food intake and the degree of physical activity

Recently, two loci have been identified, which influence on central adiposity.[16]

In obese subjects, especially those with visceral adiposity, the urgency to metabolize the overload of nutrients from excessive intake and low energy expenditure associated to poor physical activity, subjects the cells to metabolic stress that initiates and perpetuates oxidative and inflammatory cascades, leading to damage in insulin signaling and resistance of tissues to hormone action. Also, the physical location of visceral fat permits liberating free fatty acids (FFA) and other metabolites of the adipose tissue directly onto the portal circulation and from there its direct ingress to nearby organs like the liver and pancreas, which are exposed to lipotoxicity.[17]

Patho-physiological Phenomena in MetS

Two patho-physiological phenomena are thought to play an important role in the underlying mechanism of MetS.[18] It includes
a. Impaired normal metabolic pathway of carbohydrate, fat, protein due to insulin resistance.[19]
b. Dysfunctional adipose tissue.[20]

These all leads to a constellation of metabolic risk factors and is associated with the development of atherosclerotic cardiovascular disease and type 2 diabetes (T_2DM) in adults. Insulin resistance and hyperinsulinemia are thought to be central to the development of MetS. Although not all individuals with insulin resistance proceed to develop MetS, suggesting that other factors may contribute to the pathogenesis of MetS.[21]

DISCUSSION

Concept of Dhatu & Dhatuposhana

Concept of *dhatu* is a unique speculation in *Ayurveda*. According to *Ayurvedic* literature, The *dhatu* is a component of human body which is associated with structural support as well as nourishment in human body.[22] *Meda* is considered as a *dhatus* in *Ayurveda*.
According to various classical text *meda dhatu* are potentially found in following site –
- *Sphig* (Gluteal region)
- *Udara* (Abdomen)
- *Vapabaha* (Omentum)
- *Vrikka* (Kidney)
- *Asthi* (Inside the bones)

Acharya Madhavkar mentioned that *'Snehat medo janayati'* that means *meda dhatu* is derived from *sneha* or fatty food intake[23].

According to *Ayurveda*, *Dhatus* are fundamental physiological components in human body. Every *dhatus* are consist of two portion –

- *Sar ansa* – This portion of *dhatu* are deposited in a particular site.
- *Prasada ansa* – This portion of *dhatu* are utilized for the nourishment of others *dhatu*.

Fundamental concept of *meda dhatu* in *Ayurveda*, correspond to adipose tissue in anatomical aspect as well as derivational aspect. Fundamental concept of *meda dhatu* in *Ayurveda*, correspond to adipose tissue in anatomical aspect as well as derivational aspect. Homeostasis in the transformation of *dhatu* is a basic essentiality to maintain normal physiology. It is termed as *dhatugati-sama* that is maintained by *vayu*.[24]

Concept of *dhatu-gati* have a similarity with metabolism which is defined as a chemical process that makes it possible for the cell to continue living[25]. There are two types of metabolic pathways –

- Catabolic pathway – That is responsible for breakdown of complex molecules, energy release and energy storage.
- Anabolic pathway - That is responsible for synthesis of complex molecules from smaller molecules with the storage of energy

Catabolic pathway has three phases[26]

a) Primary phase - Convert macromolecule to small unit
b) Secondary phase - Related to absorption, catabolism toward a smaller component and final common oxidative pathway
c) Tertiary phase – Related to energy release through electron transport chain (ETC) in the mitochondria and chemiosmosis or energy store.

Chiefly secondary and tertiary phase of catabolism as well as anabolic pathway has a similarity with the concept of *dhatugati or dhatu-poshana*.

Insulin resistance is thought to be central to the development of MetS and may play a role in the pathogenesis of its individual metabolic components. Altered physiological function of *vayu* in context to *Ati-sthoulya* has a fundamental similarity with the state of insulin resistance in MetS.

Concept of Meda & Medadusti

Two different state of *meda dhatu* has been described in *Ayurveda*[27]

- *Baddha meda* (Stored in a particular site)
- *Abaddha meda* (Circulating in nature)

The area where over-growth of *meda* occurs are [28]

a) *Sphig* (Gluteal region)
b) *Udar* (Abdomen)
c) *Stana* (Breast)

As per *Acharya Madhavkar*, often over enhancement of *meda dhatu* occurs in *udara* (Abdomen).[29] All these are the site of adipocity. Specially *udar* (Abdomen) is the site of central obesity that plays the most important role in MetS. Hence, *meda* may be considered as adipose tissue. Adipose tissues are endocrine and immunologically active organs with numerous effects on regulation of systemic energy homeostasis, inflammatory responses and are rich in immune cells. A huge variety of hormones, cytokines, complement and growth factors, extracellular matrix proteins, and vasoactive factors collectively termed adipokines are synthesized in adipose tissue and are released.[30]

There are major structural as well as functional differences of visceral or subcutaneous adipose tissue. Structurally visceral adipose tissues are more cellular, vascular, innervated and contains a larger number of inflammatory and immune cells than subcutaneous adipose tissue. Visceral adipocytes are more metabolically active and has a greater capacity to generate free fatty acids and to uptake glucose than subcutaneous adipose tissue.[31]

Meda-dusti refers to vitiation of *meda* both anatomically and functionally.

The adipose tissue is a central metabolic organ in the regulation of whole-body energy homeostasis. Adipose tissues secrete various hormones, cytokines, and metabolites (termed as adipokines) that control systemic energy balance. In response to changes in the nutritional status, the adipose tissue undergoes dynamic remodeling, including quantitative and qualitative alterations in adipose tissue-resident cells. Adipose tissue remodeling in obesity is closely associated with adipose tissue function. Changes in the number and size of the adipocytes affect the microenvironment of expanded fat tissues, accompanied by alterations in adipokine secretion, adipocyte death, local hypoxia, and fatty acid fluxes. Concurrently, stromal vascular cells in the adipose tissue, including immune cells, are involved in numerous adaptive processes, such as dead adipocyte clearance, adipogenesis, and angiogenesis, all of which are dysregulated in obese adipose tissue remodeling. Chronic overnutrition triggers uncontrolled inflammatory responses, leading to systemic low-grade inflammation.[32]

In obesity, a pro-inflammatory state of adipose tissue, as well as dysfunctional state of adipose tissue has a significant role towards an abnormal metabolic state.[33]

Rather than total adiposity, the core clinical component of the syndrome is visceral and/or ectopic fat.[34]

Ayuhras (Reduced life span)

Aging refers to the deterioration of the biological functions after an organism has attained its maximum reproductive potential. It is generally considered that metabolic syndrome induces precocious aging although the mechanisms that account for this are incompletely known. Important genes in extending lifespan include kinase mammalian target of rapamycin (mTOR), AMP-activated protein kinase (AMPK), sirtuins and insulin/insulin like growth factor 1 (IGF-1) signaling. These genes integrate longevity pathways and metabolic signals in a complex interplay in which lifespan appears to be strictly dependent on substrate and energy bioavailability. Abnormalities in the insulin signaling pathway generate age-related diseases and increased mortality. It is becoming clear that longevity genes might be involved.[35]

Jaboporodh (Restricted movement or Hypokinesia)

Obesity is associated with physical inactivity, which exacerbates the negative health consequences of obesity. Obesity have implied that the functional limitations imposed by the additional loading of the locomotor system in obesity result in aberrant mechanics and the potential for musculoskeletal injury.[36] Also chronic exposure to obesogenic diets is associated with changes in both physical activity levels and dopaminergic function. Diet-induced changes in the dopamine system may be sufficient to explain the development of physical inactivity in people with obesity.[37]

Kriccha-vyavaya (Sexual dysfunction)

Erectile dysfunction (ED) is defined as the recurrent or consistent inability to obtain and/or maintain an erection sufficient for satisfactory sexual performance. ED is the most common male sexual dysfunction and shares many risk factors with systemic conditions including cardiovascular disease (CVD) and the metabolic syndrome (MetS). Erection results from coordinated

communication of hormonal, neural, and vascular systems as well as psychological inputs. Release of nitric oxide (NO) from the cavernous nerves and endothelial cells are an important pathway for smooth muscle relaxation, along with decreased peripheral arteriolar resistance to promote blood inflow into the corporal tissues. Hence, endothelial integrity is crucial to this process.[38]
Numerous metabolic abnormalities found in the metabolic syndrome, including hyperglycemia, excessive fatty acids and insulin resistance, cause an endothelial cell dysfunction by affecting nitric oxide synthesis or degradation. [39]
Men with the metabolic syndrome, often have low total and free testosterone and low sex hormone–binding globulin (SHBG).[40]

Dourbalya (Fatigue)

One of the defects in metabolic syndrome and its associated diseases is excess cellular oxidative stress and oxidative damage to mitochondrial components, resulting in reduced efficiency of the electron transport chain. Recent evidence indicates that reduced mitochondrial function is related to fatigue, a common complaint of MetS patients.[41]

Dourgandha (Bromhidrosis)

Body odours are a result of the combination of hundreds of emitted odorous volatile organic compounds (VOCs) VOCs that are originally secreted from various cells inside the body via metabolic pathways. The components of VOCs usually reflect the metabolic condition of an individual. Therefore, metabolic disease often results in a change in body odour. VOCs emitted from the skin surface are mainly derived from sweat and sebum. Although some of these VOCs result from internal hormonal or metabolic changes, many VOCs appear to be derived from symbiotic bacteria that live on the skin surface and metabolize and transform secreted compounds in sweat and sebum.[42]

Swedabadha (Hyperhydrosis)

Obese patients have larger skin folds and sweat more profusely after becoming overheated because of thick layers of subcutaneous fat, thereby increasing both the frictional and moisture components.[43] However, currently there are no specific published data on the structure and function of apocrine and eccrine sweat glands in obesity.

Atikhuda (Polyphagia)

Leptin and ghrelin are two hormones that have been recognized to have a major influence on energy balance. Leptin is anorexigenic hormone, whereas ghrelin is orexigenic hormone. Leptin is mainly produced by white adipose tissue and to a certain extent by gastric mucosal cells. Ghrelin is produced from ghrelinergic cells in gastrointestinal tract. Leptin induces weight loss by suppression of food intake and by stimulation of metabolic rate. In case of adiposity, leptin secretion is stimulated. Now it has been established that obesity is associated with leptin resistance and that leads to failure of anorexigenic mechanism.[44]

Atipipasa (Excessive thrust)

Thirst is a subjective perception that leads to desire for fluid intake to maintain body fluid homeostasis in response to deficits in either intracellular or extracellular fluid volume. Osmotic and hormonal stimuli from circulation are detected by lamina terminalis and that information are integrated with other neural signals to generate thirst. Increased water intake is associated with loss of body weight produced via two mechanisms, decreased feeding and increased lipolysis. Mild, but chronic, hypohydration is correlated with increased body weight and its attendant dysfunctions. The common denominator likely is angiotensin II, the principal hormone of body fluid regulation.[45]

An overactive renin-angiotensin system (RAS) has been shown to be involved in MetS. Angiotensin II induces adipogenesis (differentiation into adipocytes) and lipogenesis (triglyceride storage in adipocytes) in vitro. The effects of Angiotensin II on adipose tissue are mediated by Angiotensin II type 1 and type 2 receptors. [46]
Currently there are no specific published data on the clinical evidence of polydipsia in MetS.

CONCLUSION

Both MetS and *Ati-sthoulya* is a clustering of pathological event induced by obesity, which have a serious effect on morbidity and mortality. The diagnosis of *Atisthoulya*, are chiefly based on anthropometric abnormalities as well as various clinical manifestations, which are appeared as complication of *Atisthoulya.* As now a day various biochemical parameter is available, MetS are diagnosed considering both anthropometric and biochemical abnormalities. The complications mentioned in *Ati-sthoulya,* has been found to develop in MetS patients. The concept of *vishama-dhatuposhana* and *meda-dusti* in context to *ati-sthoulya* are also fundamentally equivalent to impaired normal metabolic pathway and dysfunctional adipose tissue respectively.

As there is fundamental equivalency in MetS and *Ati-sthoulya* in respect to risk factors, diagnostic evaluation and patho-physiological phenomena, the therapeutics that has been described in context to *Ati-sthoulya* may be evaluated against MetS.

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