ISSN: 2322 - 0902 (P)
ISSN: 2322 - 0910 (O)

International Journal of Ayurveda and Pharma Research

Review Article

VISCERAL FAT - HEALTH IMPLICATIONS AND AYURVEDIC PURVIEW

Athira P[1]*, M.C. Shobhana[2]

*[1]PG Scholar, [2]Professor & HOD, Dept. of Swasthavritta, VPSV Ayurveda College, Kottakkal, Kerala, India.

Article info

Article History:
Received: 21-12-2024
Accepted: 18-01-2025
Published: 07-02-2025

KEYWORDS:
Visceral fat, Insulin resistance, Type 2 diabetes, Obesity, Fatty liver, Stress.

ABSTRACT

Visceral fat is a type of fat that accumulates around the internal organs in the abdominal cavity. It is having a lot of normal physiological functions within the body and when it exceeds the normal level it leads to many diseases. Visceral fat is associated with several health conditions, including insulin resistance, Type 2 diabetes, obesity and fatty liver. A comprehensive understanding of the impact of visceral fat on the above diseases and detrimental effects of visceral fat on human health and well-being is needed. Stress has a major role in visceral fat accumulation as chronic stress can lead to increased levels of cortisol, which promotes the storage of visceral fat. Hence an integrated module which includes diet, yoga, meditation, and exercise to reduce cortisol levels and prevent visceral fat accumulation is needed. Early detection and prevention of visceral fat accumulation is of prime importance to reduce the risk of developing these health conditions. According to Ayurveda visceral fat accumulation can be considered as a result of *Medodhathudushti* which is the major cause of *Sthoulya* and *Prameha*. For the prevention of excess visceral fat accumulation, Ayurveda recommends certain daily routines, dietary guidelines, ethical principles and lifestyle modifications. Ayurveda emphasizes self-control, non-suppression of natural urges and maintaining a positive outlook to reduce stress levels. This paper unravels the link between obesity, insulin resistance, and fatty liver and highlights the ayurvedic principles to tackle visceral fat and its complications.

INTRODUCTION

Adipose tissue is a complex, multifunctional organ comprising diverse cells that serve various purposes. The impact of adipose tissue on the body varies significantly depending on its type and location. Visceral adipose tissue, a subtype of white adipose tissue, is distinguished by its large cells and association with adverse metabolic health outcomes. Located near vital organs such as the intestines, liver, and pancreas, visceral adipose tissue plays a more active role in metabolic processes. In contrast, subcutaneous adipose tissue, another type of white adipose tissue, is considered more metabolically benign. Characterized by smaller cells situated beneath the skin, subcutaneous adipose tissue stores energy and provides insulation. Brown adipose tissue, the third type, is renowned for its exceptional metabolic health benefits due to its thermogenic properties. The high concentration of mitochondria in brown adipose tissue imparts its distinctive brown hue. Although it is present in limited quantities in specific areas of the body, brown adipose tissue is linked to favorable health outcomes.[1]

The accumulation of visceral fat is closely tied to various metabolic and cardiovascular diseases. The proper functioning of all adipose tissue, particularly subcutaneous adipose tissue (SAT), plays a crucial role in preventing the development of visceral adiposity. If SAT becomes dysfunctional and loses its ability to effectively store energy, the excess energy can spill over into visceral fat depots, leading to their expansion.[2] Both the amount of visceral adipose tissue and overall body fat percentage have been associated with, and may potentially influence, the progression of various diseases. However, the commonly used Body Mass Index (BMI) does not provide a comprehensive assessment of these factors.[3] Individuals with a healthy weight can still exhibit metabolic obesity, highlighting the limitations of weight alone as a health indicator. Visceral fat

Access this article online
Quick Response Code
https://doi.org/10.47070/ijapr.v13i1.3506

accumulation can occur independently of body weight, and assessing visceral fat in normal-weight individuals can reveal crucial information for mitigating metabolic risk factors.[4]

Research has identified several key factors that contribute to the accumulation of visceral fat, including aging, hormonal fluctuations, excessive sugar consumption, and a sedentary lifestyle. The unique metabolic properties of intra-abdominal fat, which exhibits high rates of both fat formation and breakdown, can lead to an increase in free fatty acids in the portal circulation, which can then enter the liver. This excess fatty acid flux can trigger a cascade of metabolic disturbances, including enhanced lipid production, glucose synthesis, and insulin resistance, ultimately resulting in a range of cardiovascular and metabolic disorders, such as high blood pressure, glucose intolerance, and atherosclerosis. Notably, individuals with visceral fat accumulation, regardless of their overall weight status, are more likely to exhibit impaired glucose and lipid metabolism, and are at increased risk of developing coronary artery disease.[5] In the 1980s, Björntorp and his research team proposed a groundbreaking hypothesis, based on extensive studies involving thousands of participants, which highlighted the critical role of psychosocial stress factors in contributing to the adverse health consequences of abdominal obesity and the accumulation of visceral fat.[6,7,8]

MATERIALS AND METHODS

Published articles are used to acquire material on the topic.

DISCUSSION

Visceral Fat and Stress

The growing prevalence of stress and obesity has sparked intense research into their interconnectedness. Emerging evidence suggests that chronic stress triggers the hypothalamic-pituitary-adrenal (HPA) axis, leading to excessive glucocorticoid production, which may contribute to the development of visceral obesity. Interestingly, consuming palatable food can activate the brain's reward centers by releasing endogenous opioids, which can help mitigate the negative effects of stress by dampening HPA axis activity. However, repeated stimulation of these reward pathways, whether through stress, palatable food, or a combination of both, can lead to neurobiological adaptations that foster compulsive overeating. The hormone cortisol may also play a role in enhancing the reward value of food, potentially through interactions with neuroendocrine mediators like leptin, insulin, and neuropeptide Y (NPY). While insulin and leptin can counteract glucocorticoids in the short term, chronic stress can disrupt this delicate balance, potentially driving increased food intake and visceral fat accumulation. As research continues to unravel these complex mechanisms, it appears that the obesity epidemic may be exacerbated by the pervasive presence of chronic stress, unsuccessful dietary restrictions, and their combined effects on amplifying the allure of highly palatable foods.[9] Both short-term and long-term exposure to stress can significantly impact eating habits, affecting not only the amount but also the type of calories consumed. Furthermore, stress-related changes in food intake and energy balance can intersect with emotional states, influencing overall consumption patterns (Epel et al., 2001).[10] There is a significant connection between consuming foods high in fat and/or sugar and the regulation of emotional well-being. Research has shown that eating palatable foods can provide emotional relief by reducing stress and anxiety symptoms, particularly in individuals exposed to chronic stress or unpredictable stressful situations (Finger et al., 2011, 2012; la Fleur et al., 2005; Maniam and Morris, 2010; Pecoraro et al., 2004; Ulrich-Lai et al., 2010).[11-16] Moreover, people experiencing depressive moods tend to exhibit a stronger preference for and consume more "comfort foods" as a way to cope with their negative emotions, suggesting that these foods provide a sense of emotional relief (Macht, 2008).[17] The pleasurable emotional responses to high-calorie, appetizing foods, including their rewarding and hedonic effects, are thought to contribute significantly to excessive eating and the development of obesity, as they can trigger a cycle of overconsumption (Fulton, 2010).[18]

Visceral Fat and Type 2 Diabetes Mellitus

Type 2 diabetes (T2D) is a chronic metabolic condition marked by insulin resistance in multiple tissues. While it is commonly linked to obesity, recent studies suggest that visceral adipose tissue (VAT) plays a more significant role in determining the risk of developing T2D, regardless of an individual's body mass. The high metabolic activity of VAT leads to the release of free fatty acid (FFA) molecules, which in turn induce insulin resistance in nearby tissues. Certain populations, such as older males of Asian descent, are more susceptible to VAT accumulation due to genetic and hormonal factors. However, targeted dietary and lifestyle interventions can help mitigate the growth of VAT, potentially reducing the risk of developing T2D.[19]

Type 2 diabetes accounts for the vast majority of diabetes cases and is distinguished by insulin resistance in multiple tissues, including skeletal muscle, liver, and fat cells, leading to impaired glucose uptake and regulation.[20-23] Prolonged insulin secretion leads to decreased insulin sensitivity, prompting the body to produce even more insulin, a condition known as hyperinsulinemia, in an attempt to prevent blood sugar levels from rising. However, this compensatory mechanism is not sustainable, and over time, the β-cells responsible for insulin production become exhausted, leading to impaired insulin secretion and ultimately, elevated blood glucose levels.[23] The escalation of blood sugar levels can trigger a cascade of complications affecting multiple organs and organ systems. While medication and weight loss may help manage the condition, insulin resistance is generally irreversible. Notably, obesity is a major risk factor for developing type 2 diabetes, underscoring the importance of elucidating the complex relationship between obesity and T2D.[23]

Obesity significantly increases the risk of developing type 2 diabetes (T2D) due to several factors. One key reason is that obese individuals tend to produce excessive amounts of insulin in response to glucose consumption, leading to hyperinsulinemia. This can put a strain on pancreatic β-cell function, making them more susceptible to dysfunction and insulin resistance, ultimately contributing to the development of T2D.[24,25] In individuals with obesity, the excess adipose tissue releases a multitude of bioactive molecules, including fatty acids, glycerol, hormones, and pro-inflammatory cytokines. These molecules can disrupt insulin signaling pathways, leading to decreased insulin sensitivity and ultimately, insulin resistance. The chronic inflammation triggered by these adipose tissue-derived factors contributes to the development of metabolic dysregulation, further increasing the risk of type 2 diabetes.[26,27] While obesity is not a definitive predictor of type 2 diabetes (T2D), it does increase the likelihood of developing the condition. Interestingly, research suggests that the relationship between obesity and T2D is bidirectional, with T2D also contributing to obesity. Studies have found that individuals with a genetic predisposition to T2D may be more likely to become obese over time due to their inherent susceptibility to insulin resistance, which leads to abnormal glucose and insulin levels in the bloodstream, promoting adipose tissue accumulation.

Furthermore, evidence suggests that dysfunction of the hypothalamic-pituitary-adrenal (HPA) axis plays a significant role in the development of insulin resistance. Specifically, elevated cortisol levels and decreased levels of sex steroids and growth hormone are associated with the accumulation of visceral fat, which in turn contributes to increased circulating free fatty acids. These factors are thought to contribute to the development of insulin resistance, highlighting the complex interplay between metabolic hormones, adiposity, and T2D.[28]

Visceral Fat and Obesity

Visceral obesity, also known as abdominal or central obesity, is characterized by an accumulation of adipose tissue around the internal organs in the abdominal cavity. This type of obesity has been strongly linked to various metabolic disorders, including disrupted glucose and lipid metabolism, as well as insulin resistance, which can increase the risk of developing type 2 diabetes, cardiovascular disease, and other health complications,[29,30] increased predisposition to cancers of the colon,[31] breast [32] and prostate,[33] and it is associated with prolonged hospital stays, increased incidence of infections and non-infectious complications, and increased mortality in hospital.[34] Visceral obesity itself is an independent component of metabolic syndrome and the magnitude of obesity directly relates to the prognosis of this condition.[29,35,36] Visceral adipose tissue (VAT) is a metabolically active tissue that secretes a range of bioactive molecules and hormones, including adiponectin, leptin, tumor necrosis factor, resistin, and interleukin 6 (IL-6). Notably, adiponectin plays a crucial role due to its anti-inflammatory and anti-angiogenic properties, which have protective effects against various diseases. Interestingly, the levels of circulating adiponectin have been found to be inversely related to the amount of VAT, suggesting that individuals with higher amounts of visceral fat tend to have lower levels of this beneficial hormone,[37] while decreased concentrations of adiponectin are associated with Type 2 diabetes, elevated glucose levels, hypertension, cardiovascular disease and certain malignancies.[29,37]

Visceral Fat and Non-Alcoholic Fatty Liver Disease

Non-Alcoholic Fatty Liver Disease (NAFLD) represents a continuum of liver damage, ranging from mild steatosis (fatty liver) to more severe forms, including non-alcoholic steatohepatitis (NASH), which can progress to advanced liver fibrosis, cirrhosis, and ultimately, hepatocellular carcinoma (liver cancer). This spectrum of disease highlights the potential for NAFLD to evolve into a serious and life-threatening condition, emphasizing the importance of early diagnosis and intervention.[38] The prevalence of NAFLD is increasing worldwide with each passing year, given the current trends in dietary irresponsibility and preponderance of a sedentary lifestyle.[39] Moreover, there has been a linear rise of NAFLD with that of diabetes and metabolic syndrome.[40] NAFLD could be largely due to unhealthy diets such as overeating or overconsumption of fats and sugar. With numerous factors that can lead to NAFLD, high visceral fat, also known as visceral adipose tissue (VAT), is one of the prominent observations present in a subset of NAFLD patients.[41] It is also well established that beyond a certain threshold, excess VAT may result in a malfunction of adipose tissue and cardiometabolic disturbances. Insulin plays a pivotal role in the development of non-alcoholic fatty liver disease (NAFLD). When insulin resistance occurs, the pancreas compensates by increasing insulin production to enhance peripheral glucose uptake and suppress hepatic glucose production. However, the liver is exposed too much higher concentrations of insulin due to the direct delivery of insulin from the pancreas into the portal vein, which drains into the liver. As a result, when the liver continues to produce glucose at high rates despite elevated insulin levels, it indicates the presence of hepatic insulin resistance, a key factor in the pathogenesis of NAFLD.[42] Insulin's primary role in the liver is to inhibit glycogenolysis, the breakdown of glycogen to glucose, rather than gluconeogenesis, the production of new glucose. As long as the liver's autoregulatory mechanisms are intact, fasting glucose levels remain within normal limits. However, when hepatic autoregulation is disrupted, both glycogenolysis and gluconeogenesis increase, leading to elevated hepatic glucose production. This, in turn, contributes to the development of fasting hyperglycemia and ultimately, type 2 diabetes.[43]

Techniques for Measuring Visceral Adiposity

Several methods have been developed to evaluate visceral fat, with a focus on techniques that are quick, easy to perform, and require minimal technical expertise. Anthropometric measurements and Bioelectrical Impedance Analysis (BIA) are examples of rapid, albeit approximate, assessments of body composition. However, these approaches only provide indirect estimates of Visceral Adipose Tissue (VAT). In contrast, Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) are the only methods that can directly measure cross-sectional areas or volumes of VAT. Meanwhile, Body Mass Index (BMI) remains the most widely used diagnostic tool for assessing generalized obesity, despite its limitations in distinguishing between different types of body fat.[44] A BMI greater than 25 kg m−2 is defined as overweight while a BMI over 30 kg m−2 is characterised as obese (world health organization).[45] visceral fat cross-sectional area, measured by ct imaging, correlated well (males: r=0.813; females: r=0.825) with normal BMI ranges (18.5–24.9 kg m−2).[44] Despite the frequent use of BMI, it cannot distinguish between lean and fat body mass and it certainly does not appreciate differences between subcutaneous and visceral fat compartments. Waist-to-hip ratio (WHR), waist circumference (WC) or sagittal abdominal diameter (the height of the abdomen when the patient is in the supine position) are additional measures used in clinical practice to derive estimates of fat distribution.[46]

Bioelectrical Impedance Analysis

Bioelectrical Impedance Analysis (BIA) is a non-invasive, radiation-free, and cost-effective technique that has been extensively used to assess body composition in various clinical settings. Its accessibility, safety, and affordability make it an attractive alternative for evaluating body composition, particularly in situations where other methods, such as CT or MRI, may not be feasible or practical.[47,48] Although bioelectrical impedance analysis (BIA) is a convenient and non-invasive method, its accuracy and specificity are limited due to its reliance on the differential resistance of electrical currents passing through fat and lean tissues. As a result, prediction equations are employed to estimate fat-free mass. While BIA can provide a reasonable estimate of whole-body fat content, its ability to accurately assess abdominal subcutaneous and visceral fat is more challenging. However, recent studies have shown promising correlations between BIA estimates and precise imaging techniques, such as computed tomography (CT), when assessing visceral fat, suggesting that BIA may still be a useful tool in certain contexts.[49,50]

Dual Energy X-Ray Absorptiometry and Air Displacement Plethysmography

The whole-body imaging technologies of DXA and air displacement plethysmography have received increased attention for their precision and rapid assessment. While they are not necessarily clinically accessible, these modalities are progressively used by researchers. However, this can only provide estimations of visceral adiposity as they cannot distinguish between different adipose tissue deposits.

CT and MRI

Computed Tomography (CT) and Magnetic Resonance Imaging (MRI) are currently considered the gold standard methods for quantitatively assessing intra-abdominal adipose tissue, also known as visceral fat. These imaging modalities provide highly accurate and detailed measurements of visceral fat area and volume, allowing for precise evaluation and monitoring of this critical health indicator.[51] Computed Tomography (CT) offers a direct and highly accurate method for assessing visceral fat deposition in both adults and children, thanks to its exceptional resolution of adipose tissue. Interestingly, research has shown that visceral fat volume exhibits a stronger correlation with Body Mass Index (BMI) compared to visceral fat cross-sectional area in both males and females. This suggests that visceral fat volume may be a more sensitive indicator of BMI-related health risks,[52] suggesting that when CT or MRI analysis is not available, BMI may provide a surrogate measure of vat.

Magnetic Resonance Imaging (MRI) remains a highly valuable tool for investigating fat deposition, particularly in the abdominal region. Its ability to accurately assess abdominal fat distribution makes it an essential method for researchers and clinicians seeking to understand the complex relationships between fat accumulation, metabolic health, and disease risk.[45] In practice, multiple-slice MRI is one of the preferred options for volume calculation, but its use is limited by accessibility and cost. Accuracy is often high with MRI, but defining different adipose tissue deposits depends on the settings of the MRI scanner. Along with CT imaging, multislice volume MRI is generally considered a gold standard reference for measuring total and regional adipose tissue volumes.[53,54]

Adopting a healthy diet and engaging in physical fitness can promote reductions in body weight and visceral fat.[55] The Mediterranean diet, rich in fiber from fruits, vegetables, legumes, and whole grains while limiting foods high in saturated and trans fats (e.g., fatty meats and highly processed foods), has been associated with lower abdominal fat gain over 10 years in the Spanish population.[56] However, the impact of the Mediterranean diet on the incidence of abdominal obesity remains inconclusive. The AdolesHealth study on European adolescents indicated that adherence to the Mediterranean diet did not influence waist circumference or body fat changes based on bioimpedance measurements.[57] A systematic review assessing the effects of the Mediterranean diet on central obesity demonstrated that 13 out of 18 studies showed a significant reduction in central obesity.[58] The explanation for the inconsistent results of healthy diet on VAT among many studies may be related to the characters and age group of study population, type of intervention, and degree of physical activity. Intermittent fasting, has been recognized as a beneficial dietary method for addressing obesity and type 2 diabetes, offering a unique approach to weight management and metabolic regulation.[59,60] The advantages of intermittent fasting are closely tied to its efficacy in promoting weight loss, although sustaining this regimen over an extended period can be challenging. A meta-analysis revealed that intermittent fasting yields significant reductions in body weight and Body Mass Index (BMI) when compared to both control groups and calorie-restricted diets.[59]

Ayurvedic Purview

In Ayurveda, the generation of *Ama dosha* occurs at multiple levels, beginning from the *Jataragni Pachana* state due to *Jatharagnimandya* resulting in *Dhatwagnimandya* and formation of *Sama medo dhatu* (as excessive visceral fat). Excessive consumption of sweet tastes (*Madhuratara annarasa*) leads to *Ama dosha* production, which in turn disrupts *Agni's* functioning. This condition is contributing to the vitiation of *Jatharagni* and *Dhatwagni*, particularly *Mdedodhatwagni*. As a result, *Dhatuparinama* (digestion and metabolism) is impaired, hindering the proper formation of *Medo dhatu*. In essence, *Medodhatwagnimandya* causes an overproduction of *Sama medo dhatu*, leading to *Medo roga*. *Sama medas* in turn deposits at various *Srotases*, causing *Srotorodha* and other diseases. According to the *Dhatu parinama sidhanta,* proper formation of *Annarasa* due to the normal functioning of *Jataragni* leads to the *Utharothara dhatu vridhi* (*Dhatuposhana*). *Ativridhi* of a particular *Dhatu* is happening because of the *Jataragnimandya* followed by the *Dhatwagni mandya* of that particular *Dhatu*.

Ayurvedic Principles for Preventing Visceral Fat

Ayurveda's dietary principles, including *Ashta Ahara vidhi* and *Dwadasha ashana pravichara,* offer a holistic approach to preventing diet-related diseases. By incorporating these principles into daily life, individuals can reduce their risk of various health issues. The *Ashta Ahara* principles, such as *Prakriti* and *Karana*, provide a foundation for healthy eating, while *Dwadasha Ashana pravichara* focuses on therapeutic dietary interventions, considering factors like *Vrtyartham, Sushka, Ushna,* and *Ruksha*. This knowledge enables personalized dietary advice to be tailored to specific health needs.

In terms of preventing visceral fat accumulation, Ayurvedic lifestyle practices, such as Dinacharya, are crucial. This includes following a daily routine, avoiding suppression of natural urges, and cultivating Sadvritta. Among the various aspects of Dinacharya, Vyayama and Udwartana are particularly important for preventing excess fat accumulation. Ayurveda also emphasizes the importance of avoiding suppression of natural urges, which can contribute to various diseases. Additionally, Sadvritta and Achararasayana, which relate to mental well-being, play a key role in managing stress and its associated complications, promoting overall health and well-being.

CONCLUSION

Visceral fat, plays a pivotal role in maintaining the body's homeostasis. Nevertheless, excessive accumulation of these fats can have severe consequences, including an increased risk of cardiovascular disease and liver cirrhosis. A sedentary and unhealthy lifestyle is a primary cause of visceral fat deposition, which can lead to the development of non communicable diseases. Ayurvedic concept of *Medojanya vikara* needs to be considered here. Dietary and daily regimen mentioned in Ayurveda under the broad headings of principles of dietetics and *Dinacharya* has to be followed for preventing this kind of health issues and resulting complications. *Swasthavritta*, the preventive principles in Ayurveda has to be adopted by each individual for acquiring an ultimate state of well-being.

REFERENCES

1. An S.M., Cho S.H., Yoon J.C. Adipose Tissue and Metabolic Health. Diabetes Metab. J. 2023; 47: 595–611. doi: 10.4093/dmj.2023.0011.

2. N.N. Wijayatunga, E.J. Dhurandhar Normal weight obesity and unaddressed cardiometabolic health risk- a narrative review Int J Obes, 45 (2021), pp. 2141-2155, 10.1038/s41366-021-00858-7
3. A.K. April-Sanders, C.J. Rodriguez Metabolically healthy obesity redefined JAMA Netw Open, 4 (5) (2021), Article e218860, 10.1001/jamanetwor kopen.2021.8860
4. A. García-Sánchez, J.I. Gámez-Nava, E.N. Díaz-de la Cruz, E.G. Cardona-Muñoz, I.N. Becerra-Alvarado, J.A. Aceves-Aceves, ..., A.G. Miranda-Díaz The effect of visceral abdominal fat volume on oxidative stress and proinflammatory cytokines in subjects with normal weight, overweight and obesity Diabetes, Metab Syndrome Obes, 13 (2020), pp. 1077-1087,
5. Matsuzawa Y, Shimomura I, Nakamura T, Keno Y, Kotani K, Tokunaga K. Pathophysiology and pathogenesis of visceral fat obesity. Obesity research. 1995 Sep; 3(S2): 187s-94s.
6. Björntorp P. The associations between obesity, adipose tissue distribution and disease. Acta Med Scand Suppl. 1988; 723: 121–34.
7. Krotkiewski M, Björntorp P, Sjöström L, Smith U. Impact of obesity on metabolism in men and women. Importance of regional adipose tissue distribution. J Clin Invest 1983; 72: 1150–8.
8. Björntorp P. Hypothesis: visceral fat accumulation: the missing link between psychosocial factors and cardiovascular disease? J Intern Med 1991; 230: 195–201.
9. Adam TC, Epel ES. Stress, eating and the reward system. Physiol Behav. 2007 Jul 24; 91(4): 449-58. doi: 10.1016/j.physbeh.2007.04.011. Epub 2007 Apr 14. PMID: 17543357.
10. Epel E, Lapidus R, McEwen B, Brownell K. Stress may add bite to appetite in women: a laboratory study of stress-induced cortisol and eating behavior. Psychoneuro endocrinology. 2001; 26: 37–49. doi: 10.1016/s0306-4530(00)00035-4.
11. Finger BC, Dinan TG, Cryan JF. High-fat diet selectively protects against the effects of chronic social stress in the mouse. Neuroscience. 2011; 192: 351–360. doi: 10.1016/j.neuroscience. 2011. 06.072
12. Finger BC, Dinan TG, Cryan JF. The temporal impact of chronic intermittent psychosocial stress on high-fat diet-induced alterations in body weight. Psychoneuroendocrinology. 2012; 37: 729–741. doi: 10.1016/j.psyneuen.2011.06.015.
13. la Fleur SE, Houshyar H, Roy M, Dallman MF. Choice of lard, but not total lard calories, damps adrenocorticotropin responses to restraint. Endocrinology. 2005; 146: 2193–2199. doi: 10.1210/en.2004-1603.
14. Maniam J, Morris MJ. Palatable cafeteria diet ameliorates anxiety and depression-like symptoms following an adverse early environment. Psychoneuroendocrinology. 2010; 35: 717–728. doi: 10.1016/j.psyneuen.2009.10.013.
15. Pecoraro N, Reyes F, Gomez F, Bhargava A, Dallman MF. Chronic stress promotes palatable feeding, which reduces signs of stress: feedforward and feedback effects of chronic stress. Endocrinology. 2004; 145: 3754–3762. doi: 10.1210/en.2004-0305.
16. Ulrich-Lai YM, Christiansen AM, Ostrander MM, Jones AA, Jones KR, Choi DC, Krause EG, Evanson NK, Furay AR, Davis JF, Solomon MB, de Kloet AD, Tamashiro KL, Sakai RR, Seeley RJ, Woods SC, Herman JP. Pleasurable behaviors reduce stress via brain reward pathways. Proc Natl Acad Sci U S A. 2010; 107: 20529–20534. doi: 10.1073/pnas. 1007740107.
17. Macht M. How emotions affect eating: A five-way model. Appetite. 2008; 50: 1–11. doi: 10.1016/ j.appet. 2007.07.002.
18. Fulton S. Appetite and reward. Front Neuroendocrinol. 2010; 31: 85–103. doi: 10.1016/j.yfrne.2009.10.003.
19. American Diabetes Association Diagnosis and classification of diabetes mellitus. Diabetes Care. 2014; 37 (Suppl. S1): S81–S90. doi: 10.2337/dc14-S081.
20. American Diabetes Association 2. Classification and Diagnosis of Diabetes: Standards of Medical Care in Diabetes- 2018. Diabetes Care. 2018; 41: S13–S27. doi: 10.2337/dc18-S002
21. Ndisang J.F., Vannacci A., Rastogi S. Insulin Resistance, Type 1 and Type 2 Diabetes, and Related Complications 2017. J. Diabetes Res. 2017; 2017: 1478294. doi: 10.1155/2017/1478294.
22. Galicia-Garcia U., Benito-Vicente A., Jebari S., Larrea-Sebal A., Siddiqi H., Uribe K.B., Ostolaza H., Martin C. Pathophysiology of Type 2 Diabetes Mellitus. Int. J. Mol. Sci. 2020; 21: 6275. doi: 10.3390/ijms21176275.
23. Klein S., Gastaldelli A., Yki-Jarvinen H., Scherer P.E. Why does obesity cause diabetes? Cell Metab. 2022; 34: 11–20. doi: 10.1016/j.cmet.2021.12.012.
24. Wondmkun Y.T. Obesity, Insulin Resistance, and Type 2 Diabetes: Associations and Therapeutic Implications. Diabetes Metab. Syndr. Obes. 2020; 13: 3611–3616. doi: 10.2147/DMSO.S275898.
25. Al-Goblan A.S., Al-Alfi M.A., Khan M.Z. Mechanism linking diabetes mellitus and obesity. Diabetes Metab. Syndr. Obes. 2014; 7: 587–591. doi: 10.2147/DMSO.S67400.
26. Malone J.I., Hansen B.C. Does obesity cause type 2 diabetes mellitus (T2DM)? Or is it the opposite?

Pediatr. Diabetes. 2019; 20: 5–9. doi: 10.1111/pedi.12787.

27. Björntorp P, Rosmond R. Visceral obesity and diabetes. Drugs. 1999 Sep;58:13-8
28. Ritchie SA, Connell JM. The link between abdominal obesity, metabolic syndrome and cardiovascular disease. Nutr Metab Cardiovasc Dis 2007; 17: 319–26
29. Fox CS, Massaro JM, Hoffmann U, Pou KM, Maurovich-Horvat P, Liu CY, et al. Abdominal visceral and subcutaneous adipose tissue compartments: association with metabolic risk factors in the Framingham Heart Study. Circulation 2007; 116: 39–48
30. Oh TH, Byeon JS, Myung SJ, Yang SK, Choi KS, Chung JW, et al. Visceral obesity as a risk factor for colorectal neoplasm. J Gastroenterol Hepatol 2008;23:411–17
31. Schapira DV, Clark RA, Wolff PA, Jarrett AR, Kumar NB, Aziz NM. Visceral obesity and breast cancer risk. Cancer 1994; 74: 632–9
32. Von Hafe P, Pina F, Pérez A, Tavares M, Barros H. Visceral fat accumulation as a risk factor for prostate cancer. Obes Res 2004; 12: 1930–5
33. Tsujinaka S, Konishi F, Kawamura YJ, Saito M, Tajima N, Tanaka O, et al. Visceral obesity predicts surgical outcomes after laparoscopic colectomy for sigmoid colon cancer. Dis Colon Rectum 2008; 51: 1757–67
34. Mathieu P. Abdominal obesity and the metabolic syndrome: a surgeon's perspective. Can J Cardiol 2008; 24: 19D–23D
35. Busetto L. Visceral obesity and the metabolic syndrome: effects of weight loss. Nutr Metab Cardiovasc Dis 2001; 11: 195–204
36. Pinthus JH, Kleinmann N, Tisdale B, Chatterjee S, Lu JP, Gillis A, et al. Lower plasma adiponectin levels are associated with larger tumor size and metastasis in clear-cell carcinoma of the kidney. Eur Urol 2008; 54: 866–73
37. Elhence A, Bansal B, Gupta H, Anand A, Singh TP, Goel A. Prevalence of non- alcoholic fatty liver disease in India: a systematic review and meta-analysis. Journal of Clinical and Experimental Hepatology. 2022 May 1; 12(3):818-29.
38. Sayiner M, Koenig A, Henry L, Younossi ZM. Epidemiology of Nonalcoholic Fatty Liver Disease and Nonalcoholic Steatohepatitis in the United States and the Rest of the World. Clin Liver Dis. 2016; 20: 205–214. doi: 10.1016/j.cld.2015.10.001.
39. Calzadilla Bertot L, Adams LA. The Natural Course of Non-Alcoholic Fatty Liver Disease. Int J Mol Sci. 2016; 17: pii: E774. doi: 10.3390/ijms17050774.
40. Albhaisi, S.; Chowdhury, A.; Sanyal, A.J. Non-alcoholic fatty liver disease in lean individuals. JHEP Rep. Innov. Hepatol. 2019, 1, 329–341.
41. Machado, M.V. Nonalcoholic fatty liver disease in lean subjects: Is it all metabolic-associated fatty liver disease? Hepatoma Res. 2020, 6, 84.
42. Gastaldelli A., Cusi K., Pettiti M., Hardies J., Miyazaki Y., Berria R., Buzzigoli E., Sironi A.M., Cersosimo E., Ferrannini E., et al. Relationship between hepatic/visceral fat and hepatic insulin resistance in non-diabetic and type 2 diabetic subjects. Gastroenterology. 2007; 133: 496–506. doi: 10.1053/j.gastro.2007.04.068.
43. Kvist H, Chowdhury B, Grangard U, Tylén U, Sjöström L. Total and visceral adipose-tissue volumes derived from measurements with computed tomography in adult men and women: predictive equations. Am J Clin Nutr 1988; 48: 1351–61
44. World Health Organization (Switzerland) Global strategy on diet, physical activity and health. Geneva: WHO; 2004
45. Ball SD, Swan PD. Accuracy of estimating intraabdominal fat in obese women. J Exerc Physiol Online 2003; 6: 1–7
46. Mourtzakis M, Prado CM, Lieffers JR, Reiman T, McCargar LJ, Baracos VE. A practical and precise approach to quantification of body composition in cancer patients using computed tomography images acquired during routine care. Appl Physiol Nutr Metab 2008; 33: 997–1006
47. Kyle UG, Bosaeus I, De Lorenzo AD, Deurenberg P, Elia M, Manuel Gómez J, et al. Bioelectrical impedance analysis-part II: utilization in clinical practice. Clin Nutr 2004; 23: 1430–53
48. Nagai M, Komiya H, Mori Y, Ohta T, Kasahara Y, Ikeda Y. Development of a new method for estimating visceral fat area with multi-frequency bioelectrical impedance. Tohoku J Exp Med 2008;214:105–12
49. Shoji K, Maeda K, Nakamura T, Funahashi T, Matsuzawa Y, Shimomura I, et al. Measurement of visceral fat by abdominal bioelectrical impedance analysis is beneficial in medical checkup. Obes Research Clin Pract 2008; 2: 269–75
50. Seidell JC, Bakker CJ, Van der KK. Imaging techniques for measuring adipose-tissue distribution- a comparison between computed tomography and 1.5-T magnetic resonance. Am J Clin Nutr 1990; 51: 953–7
51. Van der KK, Seidell JC. Techniques for the measurement of visceral fat: a practical guide. Int J Obes 1993; 17: 187–96

52. Heymsfield SB, Wang Z, Baumgartner RN, Ross R. Human body composition: advances in models and methods. Annu Rev Nutr 1997; 17: 527–8 5
53. Ross R, Leger L, Morris D, de Guise J, Guardo R. Quantification of adipose tissue by MRI: relationship with anthropometric variables. J Appl Physiol 1992; 72: 787–95.
54. Elhence A, Bansal B, Gupta H, Anand A, Singh TP, Goel A. Prevalence of non- alcoholic fatty liver disease in India: a systematic review and meta-analysis. Journal of Clinical and Experimental Hepatology. 2022 May 1; 12(3): 818-29.
55. Sayiner M, Koenig A, Henry L, Younossi ZM. Epidemiology of Nonalcoholic Fatty Liver Disease and Nonalcoholic Steatohepatitis in the United States and the Rest of the World. Clin Liver Dis. 2016; 20: 205–214. doi: 10.1016/j.cld.2015.10.001.
56. Calzadilla Bertot L, Adams LA. The Natural Course of Non-Alcoholic Fatty Liver Disease. Int J Mol Sci. 2016; 17: pii: E774. doi: 10.3390/ijms17050774.
57. Albhaisi, S.; Chowdhury, A.; Sanyal, A.J. Non-alcoholic fatty liver disease in lean individuals. JHEP Rep. Innov. Hepatol. 2019, 1, 329–341.
58. Machado, M.V. Nonalcoholic fatty liver disease in lean subjects: Is it all metabolic-associated fatty liver disease? Hepatoma Res. 2020, 6, 84.
59. Gastaldelli A., Cusi K., Pettiti M., Hardies J., Miyazaki Y., Berria R., Buzzigoli E., Sironi A.M., Cersosimo E., Ferrannini E., et al. Relationship between hepatic/visceral fat and hepatic insulin resistance in non-diabetic and type 2 diabetic subjects. Gastroenterology. 2007; 133: 496–506. doi: 10.1053/j.gastro.2007.04.068.

Cite this article as:
Athira P, M.C. Shobhana. Visceral Fat - Health Implications and Ayurvedic Purview. International Journal of Ayurveda and Pharma Research. 2025;13(1):206-213.
https://doi.org/10.47070/ijapr.v13i1.3506
Source of support: Nil, Conflict of interest: None Declared

***Address for correspondence**
Dr. Athira P
PG Scholar,
Dept. of Swasthavritta,
VPSV Ayurveda College, Kottakkal,
Kerala, India,
Email: dr.Athira.96@gmail.com