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RESEARCH ARTICLE

A REVIEW OF OBESE AND LEAN DIABETES WITH SPECIAL REFERENCE TO STHULA AND KRUSHA PRAMEHA

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ABSTRACT

According to new data released by The Lancet on World Diabetes Day, the number of adults with diabetes has more than quadrupled since 1990, reaching over 800 million globally. The alarming rise in diabetes over the last three decades can be attributed to increasing obesity rates, poor food marketing, sedentary lifestyles, and financial difficulties. Conversely, malnourished individuals accounted for the majority of hyperglycaemia in another semi-urban/rural Indian study. The key feature of non-obese T2D seems to be a defect in insulin secretion capacity rather than peripheral insulin resistance seen in classical diabetes. Ayurveda emphasizes that the approach to lean diabetic patients should differ from that of obese diabetic patients and identifies *medo-dhatu* (fat metabolism) as the primary pathology in any type of *prameha* (diabetes).

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INTRODUCTION

According to new data released by The Lancet on World Diabetes Day, the number of adults with diabetes has more than quadrupled since 1990, reaching over 800 million globally[1]. In India alone, 77 million adults over the age of 18 have type 2 diabetes, and another 25 million are at high risk of getting the condition soon. Surprisingly, over 50% of these people are ignorant of their illness, which, if untreated, can cause major health issues. The risk of heart attacks and strokes is two to three times higher in adults with diabetes. Additionally, reduced blood flow and nerve damage increase the likelihood of foot ulcers, infections, and even limb amputations. Diabetic retinopathy is a major cause of blindness that arises from chronic damage to the retinal blood vessels. Diabetes is also a leading cause of kidney failure[2].

METHODOLOGY

Comprehensive literature search, including classical texts and various review articles and clinical studies published in online databases, to identify relevant studies on the Ayurvedic management of diabetes mellitus.

RESULTS

The alarming rise in diabetes over the last three decades can be attributed to increasing obesity rates, poor food marketing, sedentary lifestyles, and financial difficulties. Approximately 90% to 95% of diabetic patients have type 2 diabetes, and many are overweight. Despite being traditionally thought of as "adult-onset," type 2 diabetes is becoming alarmingly more common in children and adolescents due to rising obesity rates in these age groups[3]. Type 2 diabetes mellitus is defined by relative insulin deficiency and insulin resistance. In Kerala, central obesity and overweight/obesity were identified as the most significant modifiable risk factors for developing type 2 diabetes[4]. Conversely, malnourished individuals accounted for the majority of hyperglycaemia in another semi-urban/rural Indian study[5]. A recently published systematic review and meta-analysis of prospective cohort studies (minimum 12-month follow-up, over 2.69 million participants from 20 countries) using ethnic-specific BMI categories emphasized the crucial role of obesity. The study demonstrated an increasing risk of Type 2 Diabetes (T2D) with varying body weights: a risk of 0.93 for underweight, 2.24 for overweight,

4.56 for obesity, and a significant 22.97 for severe obesity, compared to those with normal weight. Interestingly, being underweight provided a protective factor against T2D only in non-Asian individuals (RR = 0.68, 95% CI: 0.40–0.99, I^2 = 56.1%, n = 6)[6]. Among underweight and normal-weight categories, deficiencies in insulin secretion and insulin resistance may, respectively, have higher and lower impacts than in obese individuals with diabetes. An early failure of β-cells may lead to the onset of diabetes at lower insulin resistance[7]. The key feature of non-obese T2D seems to be a defect in insulin secretion capacity rather than peripheral insulin resistance seen in classical diabetes. These findings highlight the role of fat accumulation and distribution in causing insulin resistance even in non-obese T2D individuals.
The definition of T2DM in non-obese individuals is still controversial due to limited clinical measurements. The current definition of obesity using Body Mass Index (BMI) is not very helpful since these individuals have BMIs of <25 kg/m², which is considered normal.

The exact cause of hyperglycemia in lean T2DM patients remains unclear. A popular concept is sarcopenic obesity, where metabolic obesity from excess adiposity combines with decreased muscle mass. In such cases, failing beta cells cannot handle even minimal insulin resistance conferred by a lean body. Thus, it is reasonable to suggest that the percentage of body fat is more critical than total body weight itself. This raises the question of whether the goal should be reducing body weight or body adiposity to prevent diabetes, with the latter being crucial but challenging to achieve[8]. Additionally, evidence on the benefits of weight loss for this group is conflicting. Some studies show that weight loss significantly reduces diabetes risk in both obese and non-obese individuals, while others report adverse effects of weight loss in lean patients[9]. The primary pathophysiology seems to be rapid beta-cell failure due to the higher prevalence and early initiation of insulin use, supported by numerous studies[10].

In Ayurveda, the pathogenesis of *prameha* is attributed to *kapha vardhakaahara* and *vihara*, which lead to excessive *kapha dushti*. This excessive kapha then attains *sthana samsraya* in the *medo dhatu*, particularly *abaddhamedas*, due to their similar properties such as *madhura* (sweet) and *snigdha* (unctuous). It further combines with *kleda* and *mamsa bhava*, vitiating *mamsa*, and all these *dushyas* localize in the *vasti*, eventually being excreted through urine, thus manifesting *prameha*[11].

When discussing *poorvarupa* (initial symptoms), it is emphasized that if a patient presents to a *vaidya* with the *poorvarupa* of *prameha*, even without *prabhutamutrata* (excessive urination), they should be considered as *pramehi*[12]. This underscores the importance of recognizing *poorvarupa*, which are essentially the signs of *medodushti*.In terms of *sadhyasadhyata* (prognosis), it is noted that *pittapramehi* without *medodushti* is considered *sadhya* (curable)[13]. Regarding treatment, *acaryas* have described 20 types of *prameha*, but the primary consideration is whether the patient is *sthula* (obese) or *krusha* (lean). This is attributed to *medovahasrotovikara* (disorders of the fat metabolism channels).Regardless of *dosha* (*vata*, *pitta*, or *kapha*), a *sthulapramehi* should undergo *shodhanachikitsa* (purification therapy), followed by *brmhana* (nourishment). In contrast, a *krushapramehi* should receive *brmhana* directly[14].

DISCUSSION

Although modern science has not yet reached a definitive conclusion on the role of adipose tissue in diabetes among lean individuals, Ayurveda offers a clear understanding. Ayurveda emphasizes that the approach to lean diabetic patients should differ from that of obese diabetic patients and identifies *medo dhatu* (fat metabolism) as the primary pathology in any type of *prameha* (diabetes). If a patient does not present symptoms indicating *medodushti* (impaired fat metabolism), it suggests that there is still potential for diabetes reversal. However, if *medodushti* is present, treatment becomes more challenging. The *samprapti* (pathogenesis) of *medovahasrotodushti* can be understood as adipose insulin resistance followed by muscular insulin resistance. This is explained by the vitiation of *medo dhatu* by vitiated kapha, which then mixes with *mamsadhatu*. Acharya states that once *samprapti* progresses, everything consumed by the person will convert to *medas* (fat). This mirrors insulin resistance, where excess glucose is stored as fat in the body. Therefore, *medovahasrotas* (fat metabolism channels) are given utmost importance in *pramehachikitsa* (diabetes treatment).

In *sthulapramehi* (obese diabetic patients), *apatarpanachikitsa* (depletion therapy) including *shodhana* (purification) helps to improve insulin sensitivity and reduce chronic inflammation caused by impaired adipose tissues. In *krushapramehi* (lean diabetic patients), *santarpanachikitsa* (nourishment therapy) may improve muscle mitochondria, enhancing muscular insulin sensitivity and the health of pancreatic beta cells, thereby improving insulin secretion. Addressing obese and lean diabetic patients with different treatment principles, as explained by Acharya, is crucial. Identifying the severity of *medodushti* and applying *krsha-sthulapramehachikitsa* (treatment for lean and obese diabetes) may yield significant results in diabetes management.

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