***Original Research Article***

**Visceral Adiposity Index and Leptin Levels in Diabetic Subjects in Port Harcourt: Effects of Disease Duration and Sex**

**ABSTRACT**

**Background**: Diabetes mellitus (DM) is a group of endocrine diseases, resulting from insufficient or improper use of insulin. It is characterized by prolonged high blood sugar levels. The exponential increase in DM poses serious public health concerns. Visceral fat previously thought to be inert, has been found to be an endocrine organ, that produces adipokines such as leptin and adiponectin, which regulate several body functions including metabolism, inflammation, appetite, cardiovascular function and immunity. This study evaluated visceral adiposity index (VAI), and leptin levels in diabetic subjects in Port Harcourt, highlighting the effect of disease duration and sex differences.

**Materials and Methods**: A total of 225 subjects were used for the study. This was made up of 125 diabetic (test) subjects and 100 non-diabetic (control) subjects. Subjects observed overnight fast prior to sample collection. Fasting blood sugar (FBS) was determined using the glucose oxidase method. Glycated haemoglobin (HbA1c) was determined using electrochemical piezoelectric sensor method. Fasting insulin and leptin were determined using enzyme–linked immunosorbent assay (ELISA) method. Insulin resistance was determined using the homeostatic model assessment for insulin resistance (HOMA-IR) method. Triglyceride (TG) and high-density lipoprotein cholesterol (HDL-C) were determined by enzymatic method. Body mass index (BMI) was determined with the formula: BMI = weight (kg) / height² (m²). Waist to hip ratio (WHR) was determined with the formula; WHR= Waist circumference (WC) (cm) / Hip Circumference (HC) (cm). Visceral adiposity index (VAI) was determined with the formula: VAI = [WC/39.68 + (1.88 × BMI)] × (TG/1.03) × (1.31/HDL) for men, and [WC/36.58 + (1.89 × BMI)] × (TG/0.81) × (1.52/HDL) for women.

**Results**: HbA1c, FBS, insulin and HOMA-IR were significantly higher (*P*<0.05) in the diabetics, compared to the controls. Leptin levels, VAI, BMI and WHR of the diabetics were significantly higher (*P*<0.05), compared to the controls. Leptin levels were significantly higher (*P*<0.05) in the female diabetics, compared to the male diabetics. There were no significant differences (*P*>0.05) in HOMA-IR and WHR between the male and female diabetics. VAI and BMI were significantly higher (*P*<0.05) in the female diabetics, compared to their male counterparts. There were no significant differences (*P*>0.05) in leptin levels, HOMA-IR, VAI, HbA1c and BMI in subjects with diabetes for 1 – 5 years, 6 – 10 years and those with diabetes for 11 years and above. Leptin was positively correlated with insulin (r=0.363), insulin resistance (r=0.540) and VAI (r=0.210). There was a significant (*P*<0.05) and positive correlation between leptin and WHR (r=0.229), and leptin and BMI (r=0.238). VAI (r=0.698) and WHR (r=0.638) were significantly (*P*<0.05) and positively correlated with insulin resistance (HOMA-IR).

**Conclusion**: Type 2 diabetes is associated with visceral fat accumulation, hyperleptinaemia, and leptin resistance. Sex differences contributed to significantly elevated VAI, BMI and leptin levels in the female diabetics, compared to the male diabetics. Disease duration had no effect on the parameters, as leptin, VAI, BMI, HOMA-IR and HbA1c remained elevated throughout the duration of diabetes. VAI and leptin estimation should be incorporated into routine clinical assessments as reliable biomarkers for insulin resistance and metabolic dysfunction.

*Keywords: Diabetes mellitus, Visceral Adiposity Index (VAI), Leptin, Insulin resistance, Duration of diabetes.*

**1. INTRODUCTION**

Diabetes is a metabolic disease that occurs as a result of insufficient production of insulin or resistance of insulin by the cells of the body, leading to hyperglycaemia. It affects people all over the world. The number of people with diabetes increased from 108 million in 1980 to 422 million in 2014. According to the World Health Organization (WHO), about 537 million people aged 20-79 years across the world had diabetes in 2021, and experts predict this number will rise to 643 million by 2030 and 783 million by 2045 (Magliano et al., 2021). Several studies and epidemiological reports have shown a continuous increase in the prevalence of diabetes among adults in Nigeria. Prevalence values have moved from approximately 2.2% in the 1990s to almost double figures in present day Nigeria. The international diabetes federation (IDF) reported that about 3.9 million adults lived with diabetes in Nigeria in 2019, and it is projected to be almost double (6 million) by 2045 (IDF, 2019; Akinkugbe, 1997; Ajayi *et al*., 2023; Uloko *et al*., 2018; Adeloye *et al.*, 2017; Cookey *et al*., 2022).

Diabetes is generally classified into two main classes, type 1 diabetes mellitus (T1DM) and type 2 diabetes mellitus (T2DM). Type 2 diabetes is the most common form, representing 90% to 95% of all diabetes cases. There is no singular cause of type 2 diabetes, as it is often a combination of several factors. One of the main causes of type 2 diabetes is insulin resistance. Insulin resistance occurs when insulin released from the beta cells cannot be utilised by the body (skeletal muscle and adipose tissues). Other contributory risk factors for type 2 diabetes include, obesity, age, family history, lifestyle, etc. (ADA, 2014; Zhang *et al*., 2021).

Obesity is a complex condition with substantial accumulation of excess total body fat (visceral and subcutaneous) that could impact health. Visceral fat, a specific type of fat found around organs deep within the abdominal cavity, is more dangerous to health as research has found that it contributes to insulin resistance by secreting retinol binding protein 4 (RBP4), which increases insulin resistance (Flores-Cortez *et al*., 2022). Also, visceral fat which was previously thought to be inert has been found to be an endocrine organ, as it produces adipokines like leptins and tumor necrosis factor-α (TNF- α). These adipokines regulate several body functions including metabolism, appetite, inflammation, cardiovascular function, immunity, and other physiological functions (Chalupova *et al*., 2016).

Predominantly secreted by adipocytes throughout the body to signal satiation and reduce appetite, leptin is involved in the maintenance of long-term energy balance by regulating food intake and energy expenditure, via signalling of the hypothalamus (Hebebrand *et al*., 2022). Variations in leptin levels is thought to affect the body’s sensitivity to insulin in relation to adiposity in different individuals (Owei *et al*., 2017), as adipokine dysregulation can contribute to obesity-related disorders (Fasshauer & Blüher, 2015). Studies show that the adipocyte-derived peptide hormone leptin plays an important role in linking obesity, inflammation, metabolic syndrome, and cardiovascular diseases (Izquierdo *et al*., 2019), more so that leptin resistance is believed to play a significant role in adiposity, obesity and diabetes.

Visceral adiposity index (VAI) is a gender specific mathematical model that is based on simple anthropometric and metabolic parameters, which are indicative of fat distribution and function (Amato *et al*., 2010). It is a reliable indicator of increased risk for cardiometabolic diseases (Luo *et al*., 2024, Ruiz-Castell *et al*., 2021). VAI is a better indicator for obesity, as opposed to body mass index (BMI) that cannot differentiate between subcutaneous and visceral fat (Ahn *et al*., 2019), making it a valuable tool for assessing metabolic dysfunction in diabetes. This study evaluated visceral adiposity index and leptin levels in type 2 diabetic subjects in Port Harcourt, Nigeria.

**2. Materials and Methods**

**2.1 Study Area**

The study was carried out in Rivers State University Teaching Hospital (RSUTH), formerly known as Braithwaite Memorial Specialist Hospital (BMSH), a government owned hospital facility. It served as the study area where samples were collected from diabetic patients who visit the hospital, after getting approval from the ethics committee on research.

**2.2 Study Design**

A cross-sectional study design was adopted for the study. A total of 225 subjects were used for the study. This was made up of two main groups: 125 diabetic (test) subjects and 100 non-diabetic (control) subjects. Diabetes was confirmed with glycated haemoglobin (HbA1c) levels greater than or equal to 6.5% (ADA, 2018). The sample size for this study was calculated by the Cochran’s sample size model (Cochran, 1977), using a prevalence rate of 7.96% for diabetes in Rivers State (Cookey *et al*. 2022).

**2.3 Study Eligibility Criteria**

Participants admitted into the study were subjects who are residents of Port Harcourt, who had observed overnight fast prior to sampling. Those with glycated haemoglobin (HbA1c ≥ 6.5%) were classified as test. Females involved in the study were non-pregnant. Those excluded from this study were subjects with other chronic metabolic conditions.

**2.4 Sample Collection and Processing**

A total volume of about 8mls of venous blood was collected via venipuncture using vacutainer needles/bottles, with 2mls collected into Ethylene diamine tetra-acetic acid (EDTA) tubes, another 2mls into fluoride oxalate tubes and then 4mls collected into plain vacutainer tubes. The EDTA samples were analysed immediately after collection for glycated haemoglobin (HbA1c), while the fluoride oxalate samples were centrifuged and plasma analysed for fasting blood sugar (FBS), within 4 hours of sample collection. The blood samples in the plain bottles were centrifuged and serum separated into cryovials. They were stored at -20ᵒC in a refrigerator, until the time of determination of other biochemical parameters. Anthropometric measurements of the subjects such as height, weight, waist and hip circumference were also taken.

**2.5 Reagents and Biochemical Analyses**

All reagents were commercially purchased and the manufacturer’s standard operating procedures were strictly followed. Fasting blood sugar (FBS) was determined using the glucose oxidase method (Barham & Trinder 1972), as described by Randox Laboratories Limited, United Kingdom (UK). Glycated haemoglobin (HbA1c) was determined using electrochemical piezoelectric sensor method (Halamek *et al*., 2007), as described by SD Biosensor Incorporated, Republic of Korea (ROK). Fasting insulin and Leptin were determined using Enzyme–linked immunosorbent assay (ELISA) method (Engvall & Perlmann, 1972), as described by Elabscience Biotechnology Company Limited, China. Insulin resistance was determined using the homeostatic model assessment for insulin resistance (HOMA-IR) method (Mathews *et al*., 1985). Triglyceride (TG) was determined by enzymatic method (Sullivan *et al*., 1985), as described by Randox Laboratories Limited, United Kingdom (UK). High Density Lipoprotein Cholesterol (HDL-C) was determined by enzymatic method (Lopes-Virella et al., 1977), as modified by Randox laboratories limited (UK). Body mass index (BMI) was determined with the formula: BMI = weight (kg) / height² (m²) (Nuttall, 2015). Waist to hip ratio (WHR) was determined with the formula; WHR= Waist circumference (WC) (cm) / Hip Circumference (HC) (cm) (Baioumi, 2019). Visceral adiposity index (VAI) was determined with the formula: VAI = [WC/39.68 + (1.88 × BMI)] × (TG/1.03) × (1.31/HDL) for men, and [WC/36.58 + (1.89 × BMI)] × (TG/0.81) × (1.52/HDL) for women (Amato *et al*., 2010).

**2.6 Statistical Analysis**

Data generated was analysed using GraphPad Prism version 8.0.2 Independent student’s t-test, analysis of variance (ANOVA) and Tukey Post-test were done where necessary. Pearson’s correlation was also used to correlate parameters. Results were considered significant at a 95% confidence interval (P ≤ 0.05). Results are expressed as mean ± standard deviation.

**3. RESULTS AND DISCUSSION**

**Table 1: Glycaemic Parameters of the Subjects**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Subjects | HbA1c (%) | FBS (mmol/L) | Insulin (µIU/L) | HOMA-IR |
| Diabetics (Test)  n=125 | 9.15 ± 2.12 | 8.46 ± 1.61 | 19.31 ± 8.78 | 7.26 ± 1.42 |
| Non-Diabetics (Control)  n=100 | 5.35 ± 0.34 | 4.77 ± 0.63 | 13.95 ± 9.04 | 2.94 ± 0.89 |
| *P*-Value | < 0.0001 | < 0.0001 | 0.0003 | < 0.0001 |
| Summary | S | S | S | S |

*Keys: S – Significant, NS – Not Significant, n – Number of Subjects*

Table 1 shows the glycaemic parameters of the subjects: glycated haemoglobin (HbA1c), fasting blood sugar (FBS), fasting insulin, and homeostatic model assessment for insulin resistance (HOMA-IR). The results show that HbA1c was significantly higher (*P*<0.05) in the diabetics compared to the non-diabetics. FBS was significantly higher (*P*<0.05) in diabetics compared to non-diabetics. The elevated HbA1c and FBS of the diabetic subjects indicate hyperglycaemia and poor glycaemic control. This is as a result of circulating glucose not entering the cells due to insufficient insulin, or insulin resistance, and gets attached to the haemoglobin molecule in red cells overtime. The results are in consonance with the works of Briggs et al., (2016), in which diabetics in Port Harcourt had significantly elevated HbA1c and fasting glucose levels. Onodugo *et al*. (2019), also reported that most diabetics in Nigeria have poor glycaemic control with HbA1c greater than 7%.

Fasting insulin levels were significantly higher (*P*<0.05) in the diabetics, compared to the controls. HOMA-IR was significantly higher (*P*<0.05) in the diabetics, compared to the controls. The results indicate hyperinsulinaemia and significant insulin resistance in the diabetic subjects. Insulin resistance is associated with hyperinsulinaemic states, as the beta cells try to compensate for resistance and hyperglycaemia, thus produce more insulin. This is central to the development and progression of type 2 diabetes and the associated complications (Briggs et al., 2019; Briggs et al., 2021). The results agree with the work of Thomas *et al*., (2019), which implicates hyperinsulinemia as an important precursor to metabolic diseases associated with obesity. In a similar study, Abubakar et al., (2025), reported significant insulin resistance in type 2 diabetic subjects in Port Harcourt, as HOMA-IR was significantly elevated in the diabetics compared to the controls.

**Table 2: Leptin, Visceral Adiposity Index (VAI) and Anthropometric Parameters of the Subjects.**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Subjects | Leptin (ng/ml) | VAI | BMI (Kg/m2) | WHR |
| Diabetics (Test) n=125 | 49.10 ± 11.48 | 1.68 ± 0.81 | 30.12 ± 6.76 | 0.93 ± 0.07 |
| Non-Diabetics (Control)  n=100 | 42.08 ± 8.57 | 1.03 ± 0.30 | 28.05 ± 5.29 | 0.84 ± 0.06 |
| *P*-Value | < 0.0001 | < 0.0001 | 0.0378 | < 0.0001 |
| Summary | S | S | S | S |

*S – Significant, NS – Not Significant, n – Number of Subjects*

Table 2 shows leptin levels, visceral adiposity index (VAI), body mass index (BMI), and waist to hip ratio (WHR) of the subjects. Leptin levels were significantly higher (*P*<0.05) in the diabetics, compared to the controls. The results indicate hyperleptinaemia in the diabetic subjects, which suggests increased fat stores or obesity in the diabetics. Elevated leptin in diabetes is indicative of leptin resistance, where the body becomes less responsive to the satiety signals from leptin, leading to increased food intake and expansion of the adipose tissue. Leptin resistance is also associated with insulin resistance (as seen in the diabetic subjects), and plays a role in the development and progression of type 2 diabetes. The finding agrees with the works of Ambad et al., (2020) who reported higher leptin levels in diabetic subjects than healthy controls.

Visceral adiposity index (VAI), body mass index (BMI), and waist to hip ratio (WHR) of the diabetic subjects were significantly higher (*P*<0.05) than those of the controls. The results indicate increased accumulation of visceral fat, obesity and central adiposity in the diabetic subjects. With central obesity and the accumulation of visceral fat, there would be an increase in the secretion of pro-inflammatory adipokines, which interfere with insulin signalling pathways and contribute to insulin resistance. Also, increased leptin secretion from visceral fat cells would increase leptin resistance, leading to the excessive consumption of more calories, abdominal obesity, and eventually exacerbate insulin resistance. This gives rise to a vicious cycle of metabolic dysfunction, that worsen diabetes. The results agree with the work of Hulkoti et al., (2022), who reported that VAI was significantly raised in T2DM patients and also seen to be significantly associated with microvascular complications. They stated it could be used as a screening tool for T2DM patients. Obesity is a chronic progressive condition characterized by excessive and abnormal fat accumulation in the body, resulting from the consumption of more calories than the body can use, with a BMI ≥ 30 kg/m2 (Chandrasekaran & Weiskirchen, 2024). These findings are consistent with works of Abubakar et al., (2025), in which they found excessive lipid accumulation and elevated inflammatory cytokines in diabetics, compared to the controls.

**Table 3: Effects of Sex on Leptin, Insulin Resistance, Visceral Adiposity Index and the Anthropometric Parameters of the Diabetic Subjects.**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| Sex | Leptin (ng/mL) | HOMA-IR | VAI | BMI (Kg/m2) | WHR |
| Male  n= 48 | 45.61 ± 10.40 | 7.90 ± 2.51 | 1.10 ± 0.29 | 27.64 ± 6.17 | 0.92 ± 0.07 |
| Female  n=77 | 51.25 ± 11.69 | 6.87 ± 1.59 | 2.04 ± 0.82 | 31.64 ± 6.71 | 0.93 ± 0.07 |
| *P*-value | 0.0365 | 0.3251 | < 0.0001 | 0.0113 | 0.3878 |
| Summary | S | NS | S | S | NS |

*Keys: S – Significant, NS – Not Significant, n – Number of Subjects.*

Table 3 shows the effect of sex on the levels of leptin, HOMA-IR, VAI, BMI and WHR of the diabetic subjects. Leptin levels were significantly higher (*P*<0.05) in the female diabetics, compared to the male diabetics. There were no significant differences (*P*>0.05) in HOMA-IR and WHR between the male and female diabetics. VAI and BMI were significantly higher (*P*<0.05) in the female diabetics, compared to their male counterparts. The results indicate sex differences play a role in the accumulation of visceral fat, obesity and secretion of leptin in diabetes. Female sex hormones, particularly estradiol exert anti-obesity effects, modulating subcutaneous and visceral fat distribution, thus conferring cardioprotective effects on pre-menopausal women. However, in diabetes there is a disruption in hormonal balance, and this cardio-protection vanishes. There is dysregulation in lipoprotein metabolism and triglyceridaemia. These metabolic changes alter visceral fat mass and distribution, and have been associated with the higher risk of cardiovascular complications and mortality diabetic women exhibit in comparison to diabetic men (Norhammar, 2018; Goossens et al., 2021). With this dysregulation, the increased visceral fat mass would lead to increased secretion of leptin, leptin resistance and other proinflammatory adipokines. Thus, the protective role of estrogens is absent or could even become detrimental in diabetic pre-menopausal women. The results agree with the study by Jiayu *et al*., (2022) who reported high leptin levels in female diabetics than male diabetics. The results also agree with the study by Alawaini & Abugila, (2020) who reported that obesity was more prevalent in females than males. However, Akhlaq et al., (2024) reported that females have significantly higher HOMA-IR, compared to males.

**Table 4: Effects of Duration of Diabetes on Leptin, Insulin resistance, Visceral Adiposity**

**Index, HbA1c and BMI.**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| Duration (Years) | Leptin (ng/mL) | HOMA-IR | VAI | HbA1c (%) | BMI (Kg/m2) |
| 1 – 5 years  n=46 | 50.32 ± 12.85 | 6.86 ± 1.87 | 1.70 ± 1.01 | 9.09 ± 2.04 | 31.17 ± 7.15 |
| 6 – 10 years  n=35 | 45.83 ± 10.61 | 7.76 ± 1.63 | 1.70 ± 0.57 | 9.45 ± 2.43 | 29.62 ± 7.93 |
| 11 years  and above  n=44 | 50.37 ± 10.50 | 7.29 ± 1.85 | 1.64 ± 0.76 | 9.98 ± 2.02 | 29.40 ± 5.32 |
| *P*-value | 0.3134 | 0.7860 | 0.9465 | 0.7860 | 0.5853 |
| F-Value | 1.179 | 0.2416 | 0.05502 | 0.2998 | 0.5395 |
| Summary | NS | NS | NS | NS | NS |

*Keys: S – Significant, NS – Not Significant, n – Number of Subjects,*

Table 4 shows the results of the effects of duration of diabetes on leptin, HOMA-IR, VAI, HbA1c and BMI of the diabetic subjects. There were no significant differences (*P*>0.05) in leptin levels, HOMA-IR, VAI, HbA1c and BMI in subjects with diabetes for 1 – 5 years, 6 – 10 years and those with diabetes for 11 years and above. This implies leptin levels/leptin resistance, BMI and the index of visceral fat accumulation were persistently elevated across the different durations of diabetes without significant variations. The metabolic derangements that occur prediabetes and in type 2 diabetes ensure continuous dyslipidaemia, fat accumulation and secretion of pro-inflammatory molecules. Thus, insulin resistance and glycaemic control as depicted by HOMA-IR and HbA1c also remained elevated. Also, with leptin resistance, there would be increased food/calorie intake and continued weight gain. Nan et al., (2022) reported no significant differences in leptin, glucose and HbA1c levels s in T2DM patients with duration of disease. Leptin levels in female patients were positively correlated with the duration of disease according to their study. Other studies have reported variable effects of diabetes duration on VAI, with some indicating that longer durations of diabetes might be associated with more significant increases in VAI, while others have found no significant impact of duration (Jiang et al., 2022; Zhao et al., 2025).

**Table 5: Correlation between Study Variables in the Diabetic Subjects.**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| Correlation | Leptin (ng/mL) | Insulin (µiU/L) | HOMA-IR | BMI (Kg/m2) | WHR | VAI |
| Leptin (ng/mL) | 1 |  |  |  |  |  |
| Insulin (µiU/L) | 0.363  (*P*=0.158) | 1 |  |  |  |  |
| HOMA-IR | 0.540  (*P*=0.734) | 0.614  (*P*=3.69e-9)\* | 1 |  |  |  |
| BMI (Kg/m2) | 0.238  (*P*=0.038)\* | 0.550  (*P*=0.670) | 0.483  (*P*=0.113) | 1 |  |  |
| WHR | 0.229  (*P*=0.046)\* | 0.232  (*P*=0.254) | 0.638  (*P*=0.038)\* | 0.135  (*P*=0.243) | 1 |  |
| VAI | 0.210  (*P*=0.069) | 0.662  (*P*=0.162) | 0.698  (*P*=0.028)\* | 0.447  (*P*=0.074) | 0.514  (*P*=0.905) | 1 |

*Key: \* - Significant correlation*

Table 5 shows the results of correlation between the study variables. Leptin was positively correlated with insulin, insulin resistance and VAI. There was a significant (*P*<0.05) and positive correlation between leptin and WHR, and leptin and BMI. VAI and WHR were significantly (*P*<0.05) and positively correlated with insulin resistance (HOMA-IR). The results indicate that as VAI, BMI, and WHR increases, so do leptin levels. The results also indicate an increase in VAI would increase insulin resistance, as will an increase in leptin also increase insulin resistance. Leptin is primarily produced by fat cells, so increased body visceral fat mass and central adiposity is associated with increased leptin production. Also, the increase in visceral fat mass would increase the secretion of free fatty acids and pro-inflammatory cytokines, which are known to interfere with insulin signalling resulting in insulin resistance. This agrees with the works by Kumar *et al*., (2015) and Zulfania *et al*., (2020), that reported increased leptin levels were significantly correlated with BMI and type 2 DM. Hence, increased levels of serum leptin can be used as risk factor in the development of type 2 DM. Wang *et al*., (2020) reported that leptin was positively correlated with WHR. The results also agree with the study by Amato *et al*., (2014) who reported a strong positive correlation between leptin and VAI. Leptin was strongly and positively correlated with insulin and HOMA-IR. Upon regression analysis, leptin contributed to over 20% of the variability in insulin and HOMA-IR, independent of BMI (Jois et al., 2015).

**4. CONCLUSION**

There was poor glycaemic control, as glycated haemoglobin, fasting blood sugar, insulin and insulin resistance were significantly elevated in the diabetics, compared to the non-diabetic control. The study revealed significantly elevated visceral adiposity index (VAI), body mass index (BMI), waist to hip ratio and leptin levels in the diabetics. Sex differences contributed to significantly elevated VAI, BMI and leptin levels in the female diabetics, compared to the male diabetics. There were no significant differences in respect to disease duration, as leptin, VAI, BMI, HOMA-IR and HbA1c remained elevated throughout the duration of disease. VAI was positively correlated with leptin, insulin, HOMA-IR, WHR and BMI, indicating an increase in visceral fat mass is associated with hyperleptinaemia, leptin resistance and insulin resistance. VAI and leptin estimation should be incorporated into routine clinical assessments as reliable biomarkers for insulin resistance and metabolic dysfunction.

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