***Original Research Article***

**Lipid Accumulation Product Index, Caspase-1 and** **Inflammatory Status of Diabetics in Port Harcourt, Nigeria**

**ABSTRACT**

**Background:** This study evaluated Lipid Accumulation Product (LAP) index, Caspase-1, and the Inflammatory Status of Diabetics in Port Harcourt, Nigeria.

**Materials and Methods:** 165 subjects comprising 90 diabetic (test) subjects and 75 non-diabetic (control) subjects were recruited for the study. Fasting blood samples were collected for biochemical analyses. Fasting blood sugar (FBS) was determined using the enzymatic oxidation method. Glycated haemoglobin (HbA1c) was determined using the fluorescence immunoassay method. Caspase-1, tumor necrosis factor alpha (TNF-α), interleukin-1 beta (IL-1β), and fasting insulin were determined using the enzyme-linked immunosorbent assay (ELISA) method. Insulin resistance was determined using the homeostatic model assessment of insulin resistance (HOMA-IR) method. Albumin (ALB) was determined using the bromocresol green (BCG) dye-binding method. Lipid accumulation product index (LAPI) was calculated with the formula: [WC (cm) – 65] × [TG (mmol/L)] for men, and [WC (cm) – 58] × [TG (mmol/L)] for women.

**Results:** HbA1c, FBS, and HOMA-IR were significantly higher (*P*<0.05) in diabetic subjects compared to the non-diabetic controls. There was no significant difference (*P*>0.05) in insulin levels in the diabetics and controls. LAPI, caspase-1, and IL-1β were significantly higher (*P*<0.05) in diabetic subjects compared to the controls. TNF-α showed no significant difference (*P*>0.05), while ALB levels were significantly lower (*P*<0.05) in diabetics compared to the controls. There were no significant differences (*P*>0.05) in LAPI, caspase-1, TNF-α, IL-1β and ALB levels in the diabetic subjects with varying years of illness, as caspase-1, TNF-α, and IL-1β remained elevated, while ALB levels remained reduced throughout the duration of diabetes. Correlation analysis showed LAPI was positively/significantly (*P*<0.05) correlated with HOMA-IR and IL-1β. HOMA-IR was significantly (*P*<0.05) and positively correlated with TNF-α. There was a significant (*P*<0.05) and positive correlation between Caspase-1 and TNF-α, and Caspase-1 and IL-1β. Albumin was negatively correlated with TNF-α and IL-1β.

**Conclusion:** Type 2 diabetes is a chronic inflammatory disease characterised by hyperglycaemia, insulin resistance and lipid accumulation. Lifestyle and pharmacological interventions are recommended to abate the self-perpetuating cycle of inflammation, lipid accumulation, and metabolic dysfunction in diabetes.

***Keywords****: Diabetes, Lipid accumulation product index, Caspase-1, Inflammatory cytokines, Interleukin-1β, Tumour necrosis factor-α.*

**1. INTRODUCTION**

Diabetes is a chronic metabolic disorder primarily caused by defects in insulin secretion, insulin action, or both. This results in persistent hyperglycaemia that leads to various complications, including microvascular complications like nephropathy and retinopathy, as well as macrovascular complications that contribute to cardiovascular disorders like atherosclerosis, coronary heart disease and stroke [1, 2]. Diabetes is a significant global health concern; due to its increasing prevalence and the profound implications it has for individual health and healthcare systems. It affects millions worldwide, leading to substantial burdens on families and national health sectors in different countries. This global health burden has spurred the development of various preventive and therapeutic approaches. There are two major types of diabetes: type 1 diabetes mellitus (T1DM) and type 2 diabetes mellitus (T2DM). T2DM accounts for over 90% of global diabetes cases and is closely linked to risk factors such as sedentary lifestyle and obesity [3, 4].

Obesity is particularly problematic as it contributes to insulin resistance through the expanded adipose tissue, increased secretion of adipokines and possible systemic inflammation [5]. Insulin resistance, a hallmark of T2DM occurs when cells particularly the liver, muscle, and adipose tissues fail to respond effectively to insulin, resulting in impaired glucose uptake and elevated blood sugar levels [6, 7]. Insulin resistance adversely affects lipid metabolism, inhibiting lipoprotein lipase (LPL) activity, increasing hepatic lipogenesis, and reducing high-density lipoprotein (HDL-C) levels [8, 9].

Inflammation is thought to play a critical role in the pathophysiology of diabetes, contributing to β-cell dysfunction, insulin resistance, and various diabetes-related complications [10]. Cellular and systemic inflammatory molecules have been linked to the pathogenesis and progression of diabetes, including its complications [11, 12]. Caspase-1, a key regulator of the inflammasome pathway, activates pro-inflammatory cytokines like interleukin 1β, perpetuating systemic inflammation when activated by various stimuli including hyperglycaemia [13].

Lipid accumulation product index (LAPI) is believed to be a superior marker of adiposity when compared to the traditional body mass index (BMI). Unlike BMI, which merely accounts for weight and height without considering fat distribution, LAPI integrates metabolic and anthropometric indices, providing a more accurate assessment of visceral fat accumulation and adiposity. Research has shown that LAPI correlates more strongly with metabolic syndrome and cardiovascular risk factors than BMI, making it a potential tool for evaluating metabolic health in diabetic populations [14, 15]. This study evaluated lipid accumulation product (LAP) index, caspase-1 levels, and various inflammatory cytokines in individuals with type 2 diabetes in Port Harcourt, Nigeria.

**2. Materials and Methods**

**2.1 Study Area**

The study was carried out in Port Harcourt, the capital city of Rivers State, Nigeria. Port Harcourt is positioned along the Bonny River within the Niger Delta. Subjects for the research study were recruited from the Rivers State University Teaching Hospital (RSUTH), and the University of Port Harcourt Teaching Hospital with the approval of their respective ethics committee on research.

**2.2 Study Design and Study Population**

The study adopted a cross-sectional case-control study design. A total of one hundred and sixty-five (165) subjects age and sex matched were used for the study. Ninety (90) were diabetic subjects with glycated haemoglobin (HbA1c) levels greater than or equal to 6.5% [1], while seventy-five (75) were non-diabetic control subjects.

**2.3 Sample Size Determination**

The sample size was determined using Cochran’s sample size model [16]. A prevalence of 4.3% was used according to the World Health Organization 2022 report on the prevalence rate of type 2 diabetes mellitus in Nigeria [17]. The sample size was initially set at 140, accounting for a 10% attrition rate, with 70 subjects in each group; diabetics and non-diabetics. However, the number of subjects was later adjusted upwards, resulting in 90 subjects in the test group and 75 in the control group.

**2.4 Eligibility of Subjects**

The subjects were recruited based on specific eligibility criteria for the study, which specified that subjects must be at least 18 years and above, non-pregnant, resident of Port Harcourt, and willingly provide informed consent. Individuals diagnosed with chronic conditions or serious comorbidities that could impact metabolic markers, such as cardiovascular or immunosuppressive diseases, and liver disease, were not eligible. Structured interviewer-based questionnaires were administered to subjects to get demographic and other data.

**2.5 Sample Collection**

Proper vene puncture technique was employed in the collection of the blood samples. Special care was taken to avoid haemolysis of the blood samples during specimen separation and handling.

**2.6 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 enzymatic oxidation method [18], as described by ELITech Clinical Systems, France. Glycated haemoglobin (HbA1c) was determined using the fluorescence immunoassay method [19], as described by Finecare™, China. Caspase-1, tumor necrosis factor alpha (TNF-α), interleukin-1 beta (IL-1β), and fasting insulin were determined using the enzyme-linked immunosorbent assay (ELISA) method [20], as described by Elabscience Biotechnology Company Limited (China). Insulin resistance was determined using the homeostatic model assessment of insulin resistance (HOMA-IR) method [21]. Albumin (ALB) was determined using the bromocresol green (BCG) dye-binding method [22], as described by ELITech Clinical Systems, France. Lipid accumulation product index (LAPI) was calculated with the formula: [WC (cm) – 65] × [TG (mmol/L)] for men, and [WC (cm) – 58] × [TG (mmol/L)] for women [23].

**2.7 Statistical Analysis**

Data generated were 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: Glycemic Parameters of the Subjects**

|  |  |  |  |  |
| --- | --- | --- | --- | --- |
| Subjects | HbA1c (%) | FBS (mmol/L) | Insulin (µIU/L) | HOMA-IR |
| Diabetics (Test) n=90 | 8.86 ± 1.63 | 7.67 ± 0.44 | 9.96 ± 3.32 | 3.17 ± 0.30 |
| Non-Diabetics (Control) n=75 | 5.28 ± 0.20 | 4.37 ± 0.48 | 7.67 ± 2.86 | 1.52 ± 0.65 |
| *P*-Value | < 0.0001 | < 0.0001 | 0.1156 | 0.0005 |
| Summary | S | S | NS | S |

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

Table 1 shows the glycaemic parameters of the subjects, including 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. This implies type 2 diabetes is associated with elevated HbA1c, indicating chronic hyperglycaemia. These findings highlight chronic dysregulation in glucose metabolism and poor glycaemic control in the diabetic subjects, increasing the risk of complications like neuropathy, nephropathy, and retinopathy [24]. Similarly, the significantly elevated FBS levels confirms hyperglycaemia, which is a characteristic of type 2 diabetes. In people living with type 2 diabetes, persistent hyperglycaemia leads to excess glucose binding to haemoglobin in red blood cells (RBCs), forming glycated haemoglobin (HbA1c) [25, 26]. The results are in consonance with the works of Briggs *et al*. [27], in which diabetics in Port Harcourt had significantly elevated HbA1c and fasting glucose levels. Similarly, Rashed *et al*. [28] and Wu *et al*. [29] in their works demonstrated increased HbA1c concentrations in diabetic subjects, further supporting the findings of this research.

Insulin levels were not significantly different (*P*>0.05) between diabetics and non-diabetics. Insulin resistance was significantly higher (*P*<0.05) in the diabetics compared to non-diabetics. This indicates normal production and circulating levels of insulin in the diabetics. The significantly elevated HOMA-IR (insulin resistance) indicates non-responsiveness of the liver, adipose tissue and peripheral cells to the actions of insulin, this would hinder the effective uptake of glucose, and as a consequence result in prolonged hyperglycaemia, leading to persistently elevated HbA1c levels and other metabolic derangements [30, 31]. During the early stages of type 2 diabetes, the pancreatic beta cells often compensate for the reduced effectiveness of insulin by producing an increased amount of insulin [32]. This compensatory mechanism results in normal or elevated insulin levels despite the presence of insulin resistance.

**Table 2: LAPI, Caspase 1 and Inflammatory Parameters of the Subjects**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| Subjects | LAPI | Caspase 1 (pg/ml) | TNF-α (pg/ml) | IL-1β (pg/ml) | ALB (g/L) |
| Diabetics (Test) n=90 | 59.15 ± 6.86 | 316.1 ± 14.87 | 142.3 ± 13.15 | 279.0 ± 16.77 | 36.53 ± 5.05 |
| Non-Diabetics (Control) n=75 | 27.89 ± 9.79 | 213.5 ± 14.39 | 102.9 ± 9.27 | 115.4 ± 10.38 | 40.46 ± 3.93 |
| *P*-Value | < 0.0001 | < 0.0001 | 0.0556 | < 0.0001 | 0.0005 |
| Summary | S | S | NS | S | S |

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

Table 2 shows the results of lipid accumulation product index (LAPI), Caspase-1, tumour necrosis factor-alpha (TNF-α), interleukin-1 beta (IL-1β), and albumin (ALB). The results show that LAPI was significantly higher (*P*<0.05) in the diabetics, compared to the controls. The results indicate lipid overaccumulation and central adiposity in the diabetic subjects. LAPI is a vital marker of central adiposity, metabolic dysfunction and insulin resistance. In type 2 diabetes, insulin resistance promotes lipolysis in adipose tissue, leading to elevated triglyceride (TG) levels, which is a major component of lipid accumulation. Additionally, excess visceral fat and lipolysis releases free fatty acids (FFAs) that impair insulin signalling, further exacerbating insulin resistance [33]. Physical inactivity and a high caloric intake are common risk factors for type 2 diabetes. These factors promote the production, accumulation, and storage of fats [34].

Caspase-1 was significantly higher (*P*<0.05) in diabetics, compared to controls. TNF-α levels were not significantly different (*P*>0.05) between diabetics and non-diabetics. IL-1β was significantly higher (*P*<0.05) in diabetics, compared to non-diabetics. This implies that type 2 diabetes is associated with the release of pro-inflammatory cytokines that cause chronic low-grade inflammation in diabetes. Elevated Caspase-1 levels suggest increased inflammasome activities, indicating heightened systemic inflammation and a pro-inflammatory state in type 2 diabetes. Caspase-1 activates pro-inflammatory cytokines such as IL-1β and IL-18, via the NOD-like receptor protein 3 (NLRP3) inflammasome pathway [35]. In diabetes, chronic low-grade inflammation usually occurs due to hyperglycaemia, hyperlipidaemia, and metabolic stress. The persistent hyperglycaemia triggers the activation of Caspase-1 via the NLRP3 inflammasome, leading to the release of pro-inflammatory cytokines. On the other hand, oxidative stress and mitochondrial dysfunction can further amplify Caspase-1 activation, increasing its circulating levels [36]. Additionally, the expansion of visceral adipose tissue as indicated by the increased LAPI, leads to the secretion of pro-inflammatory cytokines and activates the inflammasome, leading to increased Caspase-1 production [37].

Furthermore, interleukin-1β (IL-1β) a pro-inflammatory cytokine plays a crucial role in beta-cell dysfunction, insulin resistance, and systemic inflammation in diabetes [38]. The significantly elevated IL-1β levels reflect enhanced immune activation and sustained inflammation in the diabetics. Aside from its protective role in the immune system, IL-1β plays a significant role by directly impairing beta-cell function through apoptosis. Also, an increased IL-1β activity contributes to insulin resistance by interfering with insulin receptor signalling [39]. This may have also contributed to the insulin resistance seen in the diabetics. In contrast, Tumour necrosis factor-alpha (TNF-α) is not a primary driver of inflammation and beta cell destruction in diabetes. TNF-α’s effects are non-specific and more indirect, contributing to insulin resistance and inflammation in the adipose tissue [40, 41]. The interplay between these cytokines underscores the complexity of inflammatory processes in diabetes, highlighting how individual cytokines like IL-1β can have specific roles [39], while others like TNF-α may reflect broader inflammatory states without directly correlating to diabetic pathology. The results are in agreement with the works of Zhang *et al*. [42], in which caspase-1 was significantly increased in diabetics compared to controls. Qian & Huang [43], in their study also revealed that the serum levels of IL-1β and TNF-α were significantly higher in the observational group compared to the comparison group, indicating elevated inflammatory responses in patients with type 2 diabetes.

Albumin was significantly lower (*P*<0.05) in diabetics, compared to non-diabetics. This implies type 2 diabetes is associated with reduced albumin synthesis and/or increased urinary excretion. Type 2 diabetes mellitus is a chronic metabolic and inflammatory condition characterised by systemic inflammation that suppresses albumin synthesis [44]. This suppression is mainly caused by the production and release of high levels of pro-inflammatory cytokines, such as IL-1β, IL-18, and TNF-α. As a result of these cytokine activities, individuals with type 2 diabetes often experience reduced albumin production and lower circulating levels [45]. Albumin is synthesised in the liver, and liver function is affected in diabetes, negatively impacting protein metabolism [46]. Furthermore, chronic hyperglycaemia can damage kidney filtration, leading to albumin loss in the urine (albuminuria) [47]. Nazki *et al*. [48] reported significantly decreased plasma albumin levels in diabetic subjects compared to control subjects. This aligns with the results of our study. In an earlier study carried out by Rodríguez-Segade *et al*. [49], when investigating plasma albumin concentration as a predictor of HbA1c among patients with type 2 diabetes mellitus, their findings showed that albumin levels were reduced in diabetic subjects. Also, they found that there was a significant negative correlation between HbA1c levels and albumin levels. HbA1c levels were higher in patients with lower albumin levels.

**Table 3:** **Effects of Duration of Diabetes on Lipid Accumulation Product Index, and Inflammatory Parameters in the Diabetic Subjects**

|  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- |
| Duration (Yrs) | LAPI | Caspase 1 (pg/ml) | TNF-α (pg/ml) | IL-1β (pg/ml) | ALB (g/L) |
| 1 – 5 (n=36) | 57.41 ± 5.05 | 318.3 ± 20.86 | 140.7 ± 19.51 | 265.0 ± 26.48 | 37.71 ± 3.83 |
| 6 – 10 (n=21) | 65.42 ± 7.87 | 318.8 ± 31.45 | 150.1 ± 26.58 | 320.7 ± 37.72 | 35.50 ± 5.50 |
| 11 and above (n=33) | 57.06 ± 5.97 | 312.0 ± 28.0 | 139.0 ± 24.26 | 267.6 ± 26.23 | 35.91 ± 5.85 |
| *P*-value | 0.6158 | 0.9791 | 0.9473 | 0.3964 | 0.3355 |
| F-Value | 0.4890 | 0.02114 | 0.05419 | 0.9404 | 1.113 |
| Summary | NS | NS | NS | NS | NS |

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

Table 3 shows the results of the effects of duration of diabetes on LAPI and the inflammatory parameters. LAPI showed no significant difference (P>0.05) in subjects with diabetes for 1 – 5 years, 6 – 10 years and 11 years and above. Caspase-1 levels were not significantly different (P>0.05) in subjects with diabetes for 1 – 5 years, 6–10 years, and 11 years and above. There was no significant difference (P>0.05) in TNF-α levels in subjects with diabetes for 1 – 5 years, 6 – 10 years, and those with diabetes for 11 years and above. There was no significant difference (P>0.05) in IL-1β levels in subjects with diabetes for 1 – 5 years, 6 – 10 years, and those with diabetes for 11 years and above. There was also no significant difference (P>0.05) in albumin levels in subjects with diabetes for 1 – 5 years, 6 – 10 years, and those with diabetes for 11 years and above. This implies that lipid accumulation product index (LAPI), and inflammatory markers (Caspase-1, TNF-α, IL-1β) remained consistently elevated across different durations of diabetes without significant variations. This persistently elevated inflammatory marker levels across the years of diabetes, reinforces the chronic inflammatory nature of diabetes regardless of disease duration. Albumin, a negative acute phase protein was also low across the years of diabetes, indicating chronic inflammation. Tang *et al*. [50], in researching the interrelationship between LAPi in diabetic kidney disease in patients with type 2 diabetes, reported that the duration of diabetes did not play a significant role in the elevation of LAPI. Spranger *et al*. [51], in their study on the critical role of inflammatory cytokines in the development of type 2 diabetes, did not find a significant relationship between time duration in the onset of diabetes and the activities of inflammatory cytokines in the progression of type 2 diabetes mellitus. However, they reported that subjects who developed type 2 diabetes mellitus had elevated levels of IL-6 and TNF-α, indicating a link between these cytokines and the onset of diabetes.

**Table 4: Correlation between Study Parameters**

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
| Correlation | LAPI | HOMA-IR | Caspase1 (pg/ml) | TNF-α (pg/ml) | IL-1β (pg/ml) | ALB (g/L) |
| LAPi | 1 |  |  |  |  |  |
| HOMA-IR | 0.296(*P*=0.022) \* | 1 |  |  |  |  |
| Caspase1 (pg/ml) | 0.133(*P*=0.805) | 0.024(*P*=0.853) | 1 |  |  |  |
| TNF-α (pg/ml) | 0.192(*P*=0.142) | 0.344(*P*=0.041) \* | 0.346(P=0.007) \* | 1 |  |  |
| IL-1β (pg/ml) | 0.412(*P*=0.028) \* | 0.098(*P*=0.458) | 0.778(*P*=2.52e-13) \* | 0.208(*P*=0.111) | 1 |  |
| ALB (g/L) | 0.114(*P*=0.385) | 0.109(*P*=0.406) | 0.038(*P*=0.771) | -0.153(*P*=0.243) | -0.219(*P*=0.046) \* | 1 |

*Key: \* - Significant correlation*

Table 4 shows the correlation between the study parameters. LAPI was positively and significantly (*P*<0.05) correlated with insulin resistance (HOMA-IR) and IL-1β. HOMA-IR was significantly (*P*<0.05) and positively correlated with TNF-α. There was a significant (*P*<0.05) and positive correlation between Caspase-1 and TNF-α, and Caspase-1 and IL-1β. Albumin was negatively correlated with TNF-α and IL-1β. The results indicate that lipid accumulation is associated with greater insulin resistance. Insulin generally prevents lipolysis by inhibiting the activities of hormone-sensitive lipase (HSL). However, in type 2 diabetes mellitus there is a dysregulation in insulin function leading to the breakdown of stored fats, resulting in high triglyceride levels which can accumulate, thereby increasing the LAPI [52]. A study by Bermúdez *et al*. [53], on the relationship between LAPI and insulin resistance, conducted in the Maracaibo city population in Venezuela, showed that LAPI levels are elevated among diabetic patients. They found that the median LAPI was significantly higher in diabetic patients with insulin resistance, compared to those without insulin resistance. The correlation between LAPI and IL-1β indicates that LAPI is associated with chronic inflammation. The accumulation of visceral fats leads to the release of free fatty acids (FFAs), which in turn causes the production of IL-1β. Adipocytes and immune cells in the adipose tissue produce and release inflammatory cytokines like IL-1β [54]. HOMA-IR correlates with TNF-α indicating that inflammation contributes to insulin resistance. TNF-α has been noted to interfere with insulin receptor signalling, thereby reducing glucose uptake and promoting hyperglycaemia [55]. In a study by Tanase *et al*. [56], on the intricate relationship between type 2 diabetes, insulin resistance, and non-alcoholic fatty liver disease (NAFLD), they indicated the involvement of TNF-α in the development of insulin resistance.

Caspase-1, also known as interleukin-1β converting enzyme is responsible for activating IL-1β, so the correlation indicates increased inflammasome activation in diabetes. The correlation between Caspase-1 and TNF-α suggests that multiple inflammatory pathways such as the Nod Like Receptor Protein-2 (NLRP3 inflammasome pathway contribute to diabetes-related inflammation [57, 58]. Furthermore, IL-1β stimulates Caspase-1 activation, leading to a chronic inflammatory loop, and creating a self-perpetuating cycle of inflammation and metabolic dysfunction [59]. This cycle contributes to worsening insulin resistance, beta-cell destruction, and metabolic deterioration in diabetes. In a similar study, Tang *et al*. [60], stated that an increase in Caspase-1 levels facilitates the production and activation of IL-1β through the NLRP3 inflammasome pathway.

The negative correlation between plasma albumin levels and the pro-inflammatory cytokines, TNF-α and IL-1β, indicate that as the levels of these inflammatory cytokines increase, albumin levels decrease. Albumin plays an essential role in maintaining oncotic pressure, transporting various substances, and reducing proinflammatory. Diabetes is a chronic inflammatory disease and several studies have reported positive correlations of pro-inflammatory cytokines with increased urine albumin excretion (UAE). Proinflammatory and fibrogenic cytokines synthesized and secreted by cells in the local microenvironment directly damage kidney architecture. This is in addition to the impact of the cytokines and insulin resistance on synthesis of albumin by the liver, compounding to reduced plasma levels [61, 62, 63].

**4. CONCLUSION**

The findings in this study revealed significantly elevated lipid accumulation product index, caspase-1 levels, interleukin-1β, tumour necrosis factor-α, and reduced albumin levels in the diabetics compared to the apparently healthy non-diabetics. Inflammation was persistent as caspase-1 levels and the pro-inflammatory cytokines remained elevated, while albumin levels also remained reduced throughout the duration of diabetes. Lipid accumulation positively correlated with insulin resistance and interleukin-1β. Insulin resistance positively correlated with tumour necrosis factor-α. Caspase-1, an activator of pro-inflammatory cytokines also positively correlated with the inflammatory cytokines, which however showed a negative correlation with albumin. Diabetes is a chronic inflammatory disease characterised by hyperglycaemia, insulin resistance and lipid accumulation. Lifestyle and pharmacological interventions are recommended to abate the self-perpetuating cycle of inflammation, lipid accumulation, and metabolic dysfunction in diabetes.

Consent

As per international standards or university standards, patient(s) written consent has been collected and preserved by the author(s).

Disclaimer (Artificial intelligence)

Option 1:

Author(s) hereby declare that NO generative AI technologies such as Large Language Models (ChatGPT, COPILOT, etc.) and text-to-image generators have been used during the writing or editing of this manuscript.

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