Comparison of Anthropometric, Glycemic, and Hepatobiliary Biomarkers in Pre - Diabetes and Healthy Adults with Dietary Counselling

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ABSTRACT:

Objectives:
To compare anthropometric, glycemic, blood pressure, and biochemical parameters between prediabetic individuals and healthy controls and to examine whether central adiposity correlates with metabolic dysregulation.

Methods:
A cross-sectional study was conducted among 100 adults, including 50 prediabetic individuals and 50 age matched healthy controls. Anthropometric measures, blood pressure, lipid profile, glycemic indices, liver enzymes, and hepatobiliary markers were recorded. Mann –Whitney U test was applied to compare groups and assess associations.

Results:
Prediabetic participants exhibited significantly higher waist circumference, WHR, WHtR, ABSI, AVI, BMI, systolic and diastolic blood pressure, fasting blood glucose, PPBS, and HbA1c compared with controls. Height and weight did not differ significantly. Elevated triglycerides, along with increased liver enzymes in some participants suggested underlying metabolic disturbance. Correlation analysis demonstrated meaningful associations between central adiposity indices and glycemic and metabolic markers.

Conclusion:

The study demonstrates that individuals with prediabetes exhibit significantly higher central adiposity, elevated blood pressure, and impaired glycemic control compared with healthy individuals. These findings indicate early metabolic dysregulation and highlight the usefulness of simple anthropometric measures in identifying individuals at elevated metabolic risk.

INTRODUCTION:

American Diabetes Association defines prediabetes as a state of intermediate hyperglycemia that places individuals at increased risk for developing type 2 diabetes. Prediabetes is diagnosed by impaired fasting glucose, impaired glucose tolerance, HbA1c between 5.7 – 6.4 [1]. Globally,

the prevalence of prediabetes has been increasing rapidly. As of 2021, an estimated 5.8% of the global adult population aged 20–79 years had prediabetes, with projections suggesting increase to 6.5% by the year 2045 [2.] The burden is particularly high in developing countries like India, where urbanization, sedentary life, and unhealth dietary changes have speeded the prevalence of metabolic diseases.

Obesity especially in abdomen is a well-known risk factor for insulin resistance and prediabetes. Body Mass Index (BMI) have been widely used to assess general adiposity, but they do not distinguish between lean mass and fat mass, nor account for fat distribution [3].

Anthropometric indices serve as important tools to identify individuals at increased risk of metabolic disease. The indices offer many advantages such as non-invasive, inexpensive, and less skills required to use which makes them accessible for large scale screenings and feasible in low resource settings [4]. The use of anthropometric indices lies not only in predicting obesity but also in their ability to measure variations in fat distribution, which is a key determinant of metabolic risk [5].

Several studies have shown that central obesity markers (WC, WHtR, combined indices) correlate more strongly with insulin resistance, dyslipidaemia, and hepatic fat accumulation than BMI [6, 7]. For example, combining BMI with ABSI or WC improved prediction of non-alcoholic fatty liver disease (NAFLD) risk more than using each measure alone [8].

Hepatic insulin resistance is frequently accompanied by fat accumulation in the liver, known as Non-Alcoholic Fatty Liver Disease (NAFLD). NAFLD is now recognized as the hepatic manifestation of metabolic syndrome and is closely linked to pre T2DM and prediabetes [9]. Elevated liver enzymes, such as alanine aminotransferase (ALT) and aspartate aminotransferase (AST), are commonly observed in NAFLD and may serve as indirect markers of hepatic dysfunction [10]. Hypertension commonly coexists with prediabetes. Elevated blood pressure intensifies metabolic and cardiovascular risks, underscoring the need to measure blood pressure alongside anthropometric and biochemical parameters in risk assessment [11].

The hepatic manifestation of metabolic dysregulation, broadly referred to as Non-Alcoholic Fatty Liver Disease (NAFLD), often coexists with insulin resistance and prediabetes [12]. Hepato - biliary markers such as ALT, AST, and GGT reflect hepatic fat accumulation and early liver injury which commonly arise from insulin resistance.

Since abdominal adiposity and altered fat distribution strongly drive hepatic insulin resistance, correlating these biomarkers with anthropometric indices helps identify individuals in the prediabetic stage who are at higher risk of NAFLD and metabolic deterioration.

OBJECTIVES:

- To evaluate the association of Body Mass Index (BMI), Waist Circumference (WC), Waist to Hip Ratio (WHR) and other anthropometric measures with liver enzymes (ALT, AST, GGT) and hepatobiliary function indicators.
- To investigate whether the relationship between anthropometric indices, hepatobiliary function in healthy participants and pre-diabetic risk differ based on age or gender.

- To counsel the patients which will in turn improve both their anthropometric and biochemical parameters.

METHODOLOGY:

This is a cross - sectional study held at a tertiary care hospital. Two groups were assigned. The sample size of the study was 100, 50 participants in each group. The study participants were selected based on:

Inclusion Criteria:

Case- Healthy individuals of 18 to 60 years

Control- Prediabetic patients from 30 to 60 years with no known history of endocrine disorders and chronic illness visiting the medicine OPD.

Exclusion Criteria:

Patients with liver disease, chronic illness, hemolytic anemia, any infection and pregnant woman will be excluded from the study.

Anthropometric Measurements were done by the following procedure:

Weight - Measured using a digital weighing scale, with participants in light clothing and no footwear.

Height - Measured with a stadiometer while standing erect, without shoes.

Waist Circumference (WC) - Measured at the midpoint between the lower rib margin and iliac crest.

Hip Circumference (HC) - Measured at the widest point of the buttocks.

Neck circumference (NC) - Neck circumference is measured using a non-stretchable measuring tape placed horizontally around the neck just below the Adam's apple (or at the midpoint for females) not compressing the skin while the participant is looking straight ahead.

The anthropometric indices measured for both groups in the study are :

Body Mass Index – weight (in kg) / height (in m^2)

Abdominal Volume Index – $2 \times (WC\ (cm))^2 + 0.7 \times (WC\ (cm) - HC\ (cm))^2 / 1000$

Body Shape Index (ABSI) – WC (in metre) / $BMI^{2/3} \times$ height $(metre)^{1/2}$

Biochemical Parameters:

Biochemical parameters were measured using appropriate technique. Liver enzymes such as albumin, total bilirubin, ALT, AST, ASP along with routine biochemical parameters like FBS, PPBS HbA1c and lipid profile were also recorded. Serum triglycerides were quantified through an enzymatic colorimetric method where triglyceride molecules undergo complete hydrolysis to free fatty acids and glycerol under the catalytic action of lipase. The liberated glycerol is

subsequently processed in a coupled enzymatic reaction system to generate a measurable chromogenic end product. Total cholesterol was determined using an enzymatic assay incorporating a selective blocking reagent. This reagent inactivates apolipoprotein B (ApoB)–containing lipoproteins, preventing their reactivity with the cholesterol assay reagents under analytical conditions. High-density lipoprotein cholesterol (HDL-C) was measured using a direct homogeneous enzymatic method. In this procedure, non-HDL (ApoB-containing) lipoproteins are rendered non-reactive by a selective masking reagent, allowing specific quantification of HDL-C. Very-low-density lipoprotein cholesterol (VLDL-C) and low-density lipoprotein cholesterol (LDL-C) concentrations were estimated indirectly using the Friedewald equation.

Fasting blood glucose was measured using the glucose oxidase method. In the glucose oxidase reaction, glucose is oxidized to gluconic acid with concurrent formation of hydrogen peroxide, which reacts with a chromogen to yield a quantifiable colorimetric signal. Glycated hemoglobin (HbA1c) was analysed by High-Performance Liquid Chromatography (HPLC), enabling separation and quantification based on charge or structural differences between glycated and non-glycated hemoglobin fractions.

Serum albumin concentrations were measured using either the Bromocresol Purple (BCP) dye-binding method in which albumin-dye complexes produce a colour proportional to concentration. Total and direct bilirubin were determined based on the diazo reaction principle. Bilirubin reacts with diazotized sulfanilic acid to form azobilirubin, which is detected photometrically. Conjugated bilirubin reacts directly, whereas unconjugated bilirubin requires an accelerator to facilitate solubilization. Alkaline phosphatase (ALP) activity was measured using a kinetic method employing p-nitrophenyl phosphate as substrate. ALP catalyses hydrolysis of p-nitrophenyl phosphate to p-nitrophenol, producing a yellow chromophore detectable spectrophotometrically under alkaline conditions. Aspartate aminotransferase (AST) activity was quantified using a coupled enzymatic system, wherein AST transfers an amino group from aspartate to α-ketoglutarate to generate oxaloacetate and glutamate. Oxaloacetate is then reduced to malate by malate dehydrogenase with simultaneous oxidation of NADH to NAD^+ recorded at 340 nm. Alanine aminotransferase (ALT) activity was measured similarly, with ALT catalysing the transfer of an amino group from alanine to α-ketoglutarate to form pyruvate and glutamate. Pyruvate is subsequently reduced to lactate by lactate dehydrogenase, and the rate of NADH oxidation at 340 nm is proportional to enzyme activity. γ-Glutamyltransferase (GGT) activity was estimated by monitoring the transfer of the γ-glutamyl group from γ-glutamyl-p-nitroanilide to glycylglycine. The liberated p-nitroaniline is quantified spectrophotometrically and reflects enzymatic activity.

Dietary Counselling:

All clinical, biochemical, and anthropometric data was integrated to assess each participants pre-diabetic risk status. Based on the findings, individualized dietary advice was provided with emphasis on:

- Reduction in refined carbohydrates

- Increase in fibre - rich foods
- Adequate protein intake
- Reduction in trans and saturated fats

RESULTS:

The data was analysed using SPSS 19 and JASP 9 software. For all qualitative and quantitative variables, mean ± SD was calculated. The Mann - Whitney U test was used to check differences in median values between the two groups.

There were no significant differences were observed for height ($p = 0.914$) or weight ($p = 0.174$). Pre-diabetic participants had significantly higher measures of central adiposity and cardiometabolic risk; waist circumference (mean 85.8 vs 80.5 cm; $p = 0.003$), neck circumference (34.4 vs 33.2 cm; $p = 0.003$), and BMI (27.1 vs 25.2 kg/m²; $p = 0.015$). Blood pressure was also higher in the pre-diabetic group: systolic BP (mean 126.9–132.4 vs 119.3–122.7 mmHg; $p = 0.002$–0.001) and diastolic BP (84.5–82.2 vs 77.7–79.6 mmHg; $p = 0.001$–0.003).Glycemic markers showed the largest and most consistent differences: fasting blood glucose (mean ≈108.6–110.2 vs 90.1–91.2 mg/dL; $p < 0.001$), post-prandial blood sugar (mean ≈147.6–150 vs 113.5 mg/dL; $p < 0.001$), and HbA1c (mean ≈5.79–6.0% vs 5.09–5.3%; $p < 0.001$).

Table 1: Demographic And Biochemical Characteristics of Participants in Healthy and Pre-Diabetics

Parameter		N	Mean	Standard Deviation	Median	25th Percentile	75th percentile	Mann Whitney	p-value
Height	Healthy	50	44.72	12.246	34	44	55	1186	0.661
	Prediabetic	50	46.1	11.85	35	48.5	55		
Weight	Healthy	50	64.144	6.181	60.125	65.05	68	105.2	0.174
	Prediabetic	50	67.514	9.631	59.1	66.95	77.9		
Waist circumference	Healthy	50	78.976	10.533	68.972	78.811	86.683	940	0.033
	Prediabetic	50	83.227	8.485	77.387	81.717	88.584		
Neck circumference	Healthy	50	40.067	3.223	36.3	39.7	44	838	0.007
	Prediabetic	50	41.62	3.241	39.5	42.75	44.5		
BMI	Healthy	50	23.513	2.815	21.462	22.87	25.052	978.5	0.062
	Prediabetic	50	25.11	4.424	21.927	24.59	28.047		
SBP	Healthy	50	119.96	6.21	115	119.5	125	1284	0.817
	Prediabetic	50	119.62	6.344	115	118.5	124.75		
DBP	Healthy	50	77.68	4.573	74	78	81	1345	0.514
	Prediabetic	50	77.02	5.239	72	77	82		
FBG	Healthy	50	89.2	6.465	83	88.5	95	0	< .001
	Prediabetic	50	111.04	7.406	105	110	117		
PPBS	Healthy	50	123.24	9.501	116	122	131.75	2	< .001
	Prediabetic	50	150.38	5.656	147	150	154.75		
HbA1c	Healthy	50	5.05	0.351	4.725	5.1	5.4	0	< .001
	Prediabetic	50	6.05	0.212	5.9	6.1	6.2		
Cholesterol	Healthy	50	179.1	15.542	166.5	180	193	1578	0.024

	Prediabetic	50	172.72	13.635	161.75	175	183		
HDL	Healthy	50	51.28	6.014	47	53	56	1355	0.471
	Prediabetic	50	50.6	5.229	46.25	50.5	55		
LDL	Healthy	50	111.88	10.804	102.25	111.5	122	1258.5	0.956
	Prediabetic	50	111.62	11.628	102	112	121		
Triglyceride	Healthy	50	109.3	20.587	92	105.5	123.5	969	0.046
	Prediabetic	50	118.26	22.234	100	122	136.5		
VLDL	Healthy	50	21.86	4.117	18.4	21.1	24.7	969	0.047
	Prediabetic	50	23.652	4.447	20	24.4	27.3		
Albumin	Healthy	50	4.264	0.49	3.8	4.3	4.6	1365.5	0.426
	Prediabetic	50	4.192	0.437	3.8	4.2	4.5		
Total Bilirubin	Healthy	50	0.819	0.25	0.595	0.835	1.045	1251.5	0.994
	Prediabetic	50	0.816	0.253	0.61	0.82	1.055		
AST	Healthy	50	25.72	9.223	17.25	27.5	34	1274	0.871
	Prediabetic	50	25.38	8.285	20	26	31.75		
ALP	Healthy	50	87.68	24.244	70.25	87.5	109	1198	0.723
	Prediabetic	50	89	27.996	64.5	88	14.75		
ALT	Healthy	50	26.8	10.868	17	27.5	35.75	1337.5	0.548
	Prediabetic	50	25.48	10.142	16	25	33		
GGT	Healthy	50	33.92	11.19	25.75	35.5	42.75	1582.5	0.022
	Prediabetic	50	28.76	11.045	20	29.5	37.75		
BSI	Healthy	50	0.267	0.074	0.231	0.266	0.293	1368	0.418
	Prediabetic	50	0.261	0.087	0.185	0.265	0.313		
AVI	Healthy	50	22.536	4.157	19.1	21.9	26.75	913	0.020
	Prediabetic	50	24.47	4.419	21.075	25.85	28.225		

BMI - Body mass index; SBP - systolic blood pressure; DBP - Diastolic blood pressure ; FBG- Fasting blood glucose; PPBS-post prandial blood sugar ; HbA1c - Glycated Haemoglobin ; LDL ,Low density lipoprotein ; HDL ,High density lipoprotein; TAG ,Triacylglyceride; TC ,Total cholesterol ; VLDL , Very low-density lipoprotein ;ALT ,Alanine Aminotransferase ; AST ,Aspartate Aminotransferase ; GGT , Gamma-Glutamyl Transferase ;ALP ,Alkaline Phosphatase ; AVI ,Abdominal Volume Index.

$p \leq 0.005$; $p \leq 0.01$ significance.

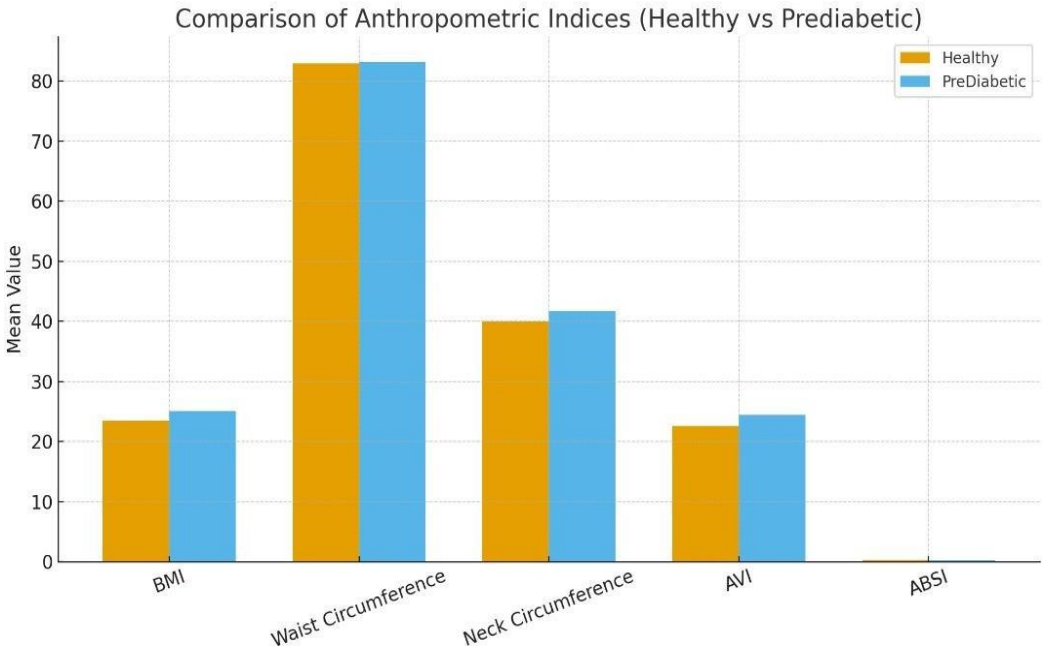

Fig. 1 – Comparison of different Anthropometric Measures and Indices between both groups

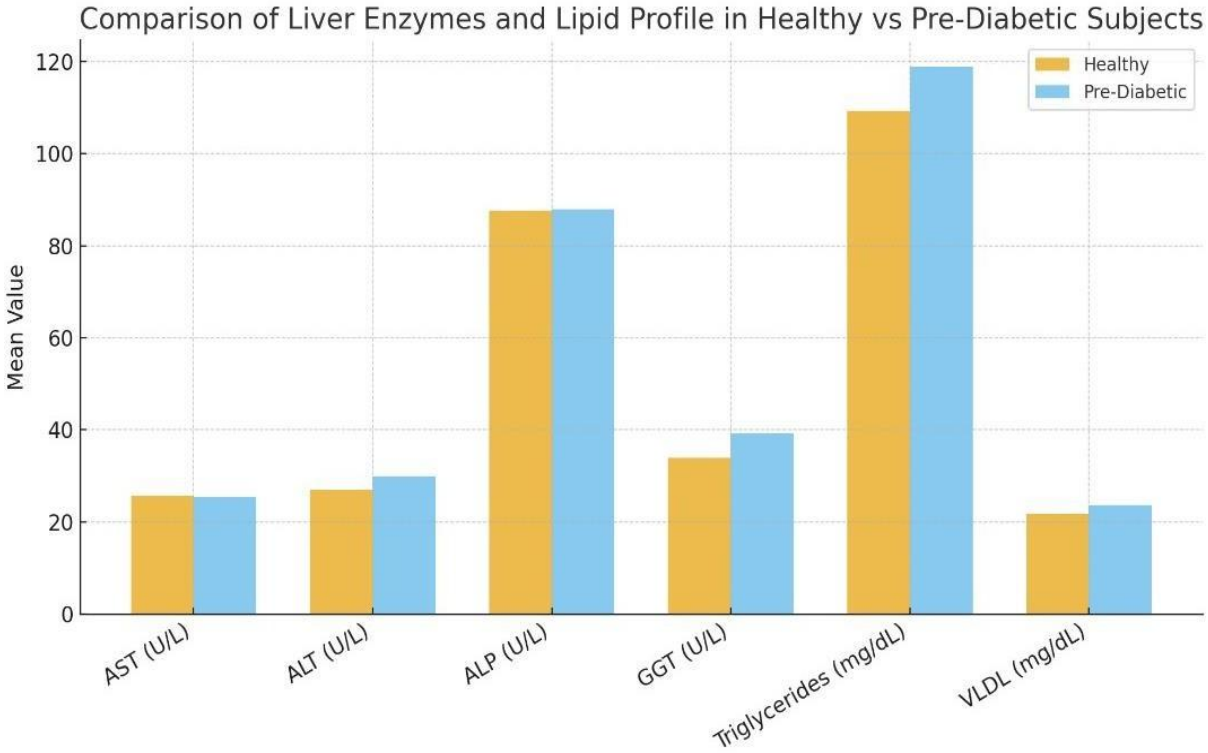

Fig. 2 – Comparison of Liver enzymes between both groups

Prediabetic subjects have increased levels of anthropometric indices (BMI, NC, WC, AVI) and liver enzymes (GGT, ALT, AST, ALP) and lipid profile parameters (VLDL, triglycerides) compared to Healthy controls

DISCUSSION:

This study explored the association between anthropometric indices and hepatobiliary function in prediabetic subjects. The findings demonstrated that increased anthropometric measures such as BMI, waist circumference (WC), and waist-hip ratio (WHR) were significantly associated with alterations in liver enzymes and markers of hepatobiliary dysfunction, particularly alanine aminotransferase (ALT), aspartate aminotransferase (AST), alkaline phosphatase (ALP), and gamma-glutamyl transferase (GGT). These results suggest that anthropometric indices, especially those reflecting central adiposity, may serve as accessible predictors of early hepatobiliary impairment in prediabetic individuals.

Studies have highlighted the strong relationship between visceral adiposity and hepatic steatosis, commonly known as non-alcoholic fatty liver disease (NAFLD), which is frequently observed in individuals with prediabetes and insulin resistance [13 & 14]. Similar to our findings, a study reported that waist circumference and BMI were strongly correlated with elevated ALT and GGT levels, indicating subclinical liver injury even in the absence of overt diabetes [15 & 16]. The pathophysiological basis may involve excessive free fatty acid (FFA) flux from abdominal fat depots into the liver, leading to hepatic lipid accumulation, oxidative stress, and hepatocellular damage [17]. Studies have shown that abdominal obesity indices such as WC and WHtR are not only associated with elevated liver enzymes but also with hepatic fat infiltration severity. Similar to our findings, few more studies demonstrated that WHtR correlated more strongly with ultrasonography-confirmed fatty liver than BMI, suggesting that central obesity is a more reliable predictor of NAFLD progression in pre diabetic populations [14 & 18]. This further supports the concept that adipose distribution, rather than overall adiposity, plays a pivotal role in hepatic dysfunction.

Pre-diabetic participants exhibited significantly greater central adiposity (waist, neck, BMI; $p \leq 0.015$), higher blood pressure (SBP and DBP; $p \leq 0.002$), and markedly worse glycemic control (FBG, PPBS, HbA1c; $p < 0.001$) compared with healthy controls, while height and weight did not differ between groups.

Our results also revealed that WHtR was a more sensitive predictor of abnormal hepatobiliary function compared to BMI. This supports the argument that central obesity indices are superior to general obesity measures in predicting hepatic dysfunction. A few study findings demonstrated that WHtR and WC showed stronger associations with hepatic fat content and transaminase elevation than BMI. This highlights the importance of using anthropometric indices beyond BMI when evaluating metabolic and hepatic risk. WHR has been highlighted as a strong anthropometric marker of hepatic insulin resistance. A cohort study by Wagenknecht et al. revealed that individuals with higher WHR exhibited greater hepatic insulin resistance as assessed by clamp studies, along with elevated ALT levels. This supports the notion that fat deposition in visceral depots directly influences hepatocellular injury and impaired glucose handling, creating a bidirectional relationship between obesity and hepatobiliary dysfunction [19]. The association of prediabetes with hepatobiliary dysfunction has been well documented.

Impaired glucose tolerance and insulin resistance promote hepatic de novo lipogenesis, reduce lipid clearance, and enhance oxidative stress, which together contribute to hepatocellular injury and biliary dysfunction. Lonardo et al., study findings reported that NAFLD is considered the hepatic manifestation of metabolic syndrome and is frequently observed in prediabetic and obese subjects [20]. Furthermore, ALT and GGT have been proposed as surrogate markers for insulin resistance, linking hepatobiliary dysfunction directly to impaired glucose metabolism.

Our study found that individuals with higher WHR and WC not only had elevated ALT and AST but also displayed altered ALP and bilirubin levels, suggesting that both hepatocellular and biliary pathways are affected. This observation is supported by research from Fraser et al., who demonstrated that GGT and ALP levels were predictive of metabolic disturbances and incident type 2 diabetes [21]. Such findings suggest that hepatobiliary markers may serve as early indicators of progression from prediabetes to overt diabetes and cardiovascular risk GGT has been implicated in the prediction of metabolic syndrome and type 2 diabetes. Lee et al. reported that individuals with elevated GGT were more likely to develop impaired glucose tolerance and diabetes over time, independent of BMI [22]. This finding aligns with our results, emphasizing that hepatobiliary markers can serve as dynamic tools for identifying prediabetic individuals at higher metabolic risk Gender-specific differences have also been observed in the relationship between anthropometric indices and hepatobiliary dysfunction. For example, Fan et al. reported that WC and WHtR were stronger predictors of liver enzyme abnormalities in men, while BMI showed weaker associations [23]. Hormonal factors, fat distribution patterns, and lifestyle behaviour may underlie these differences. These findings suggest that gender should be considered when interpreting the predictive value of anthropometric indices for hepatic dysfunction in prediabetic individuals [24 & 25].

The variability of associations across anthropometric indices may be influenced by lifestyle factors such as physical activity, diet, and alcohol consumption. Several studies have noted that physical inactivity and high-fat diets exacerbate the relationship between obesity and hepatic dysfunction [26]. In our setting, urbanization, sedentary lifestyle, and dietary transitions may have contributed to the observed associations [27 & 28]. Taken together, our findings reinforce the concept that simple anthropometric measurements can provide valuable insights into hepatobiliary health in prediabetic individuals. Among these, central obesity indicators such as WC, WHR, and WHtR appear to be more reliable predictors of hepatic dysfunction than BMI alone [29 & 30]. Identifying these associations at the prediabetes stage is crucial, as it provides an opportunity for early lifestyle interventions to prevent the progression to type 2 diabetes and advanced liver disease.

A study from Puducherry reported that serum total bilirubin levels might prove to be an early biochemical as well as clinical marker in the prediction of the progression of insulin resistance in type 2 diabetics with subclinical thyroid comorbidity [31].

Genetic factors may further influence the metabolic patterns observed. Another study by Jayanthi et al shows that the SNP +45 T>G adiponectin gene polymorphism is associated with reduced adiponectin levels, insulin resistance, and higher BMI, reinforcing the role of visceral adiposity in metabolic dysregulation seen in prediabetes [32]. Subtle alterations in thyroid function, reduced adiponectin activity, and increased NADPH oxidase–driven oxidative stress may underlie worsening anthropometric measures, early hepatic dysfunction, insulin

resistance, and elevated blood pressure. These interconnected pathways reflect a shared mechanism of metabolic deterioration, even before overt type 2 diabetes develops [33].

The metabolic disturbances seen in prediabetes such as altered liver markers, impaired glycemic control, and central adiposity may be further aggravated by tobacco exposure, which independently affects liver and thyroid function. Considering tobacco use as an additional modifier may strengthen risk prediction and support earlier preventive strategies [34].

The biochemical disturbances seen in prediabetes, including elevated glycemic markers and liver enzymes, reflect early insulin resistance linked to central adiposity. Since HOMA-IR is influenced by micronutrients such as magnesium and zinc, metabolic regulation in prediabetes may involve micronutrient-dependent pathways. Integrating these biochemical markers with anthropometric indices therefore strengthens early identification and supports more targeted metabolic intervention [35].

LIMITATIONS OF THE STUDY:

First, the sample size was relatively small which may reduce the statistical power and limit the generalizability of the results. Second, important confounding factors such as medication use, educational background, and socio - economic status were not recorded and may have influenced the clinical and dietary outcomes. Finally, there was a notable loss to follow-up after dietary counselling which makes it challenging to assess long term adherence and the sustained effects of the intervention.

CONCLUSION:

Pre-diabetic participants exhibited significantly greater central adiposity, higher blood pressure and markedly worse glycemic control compared with healthy controls while height and weight did not differ between groups. These differences are directionally consistent across summary statistics and confirm the biochemical classification of the prediabetic group.

The pattern of results such as elevated central adiposity, higher blood pressure, and worse glycemic indices in prediabetic patients are consistent with early cardiometabolic dysregulation and insulin resistance. The magnitude of the glycemic difference and the HbA1c gap are clinically meaningful and support the validity of the pre-diabetes classification in this sample. Elevated waist, ABSI, and AVI along with higher triglycerides in some datasets suggest visceral adiposity may be a key driver of the observed metabolic differences. Despite these limitations, the findings provide valuable preliminary insights and highlight the need for future studies with larger sample sizes and comprehensive data collection.

FUTURE SCOPE:

Future research with a larger and more diverse sample size is recommended to validate the findings and improve generalizability. Incorporating additional variables such as dietary patterns, physical activity levels, medication use, and socioeconomic status may provide stronger insights into the associations observed. Long-term follow-up studies are also needed to assess sustained lifestyle changes and their impact on biochemical and anthropometric outcomes.

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CONFLICT OF INTEREST:

The authors declare there is no conflict of interest.

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