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Association of Obesity with Insulin Resistance, Dyslipidemia, Vitamin D Deficiency, and Glycemic Dysregulation in Pre-Diabetic Patients: A Case-Control Study

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Abstract: This case-control study investigates the interplay between obesity, insulin resistance (IR), hyperlipidemia, vitamin D status, and diabetes risk markers in 90 obese pre-diabetic cases and 20 non-obese controls. Obese cases showed markedly elevated BMI (37.1 ± 5.18 kg/m² vs. 22.83 ± 1.64, p<0.001), fasting insulin (26.4 ± 16.12 µU/mL vs. 9.55 ± 2.61, p<0.001), HOMA-IR (7.4 ± 4.99 vs. 2.19 ± 0.18, p<0.001), total cholesterol (212.66 ± 12.34 mg/dL vs. 166.35 ± 8.8, p<0.001), triglycerides (164.39 ± 11.46 mg/dL vs. 139.8 ± 22.99, p<0.001), and HbA1c (6.02 ± 0.27% vs. 5.04 ± 0.3%, p<0.001), with universal vitamin D deficiency (81.1% abnormal). BMI positively correlated with FBS (r=0.291, p=0.005), HbA1c (r=0.320, p=0.002), cholesterol (r=0.607, p<0.001), triglycerides (r=0.519, p<0.001), and HOMA-IR (r=0.246, p=0.019). These findings underscore obesity's role in driving IR and metabolic perturbations, aligning with recent evidence linking adiposity to type 2 diabetes mellitus (T2DM) progression. Obese individuals exhibit significantly higher BMI, fasting insulin, HOMA-IR, cholesterol, triglycerides, and HbA1c compared to controls, alongside vitamin D deficiency.

Keywords: Obesity, Insulin Resistance, HOMA-IR, BMI, HbA1c.

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INTRODUCTION

Obesity, defined by BMI ≥30 kg/m², is a pivotal driver of insulin resistance, hyperlipidemia, and T2DM incidence through adiposopathy-induced inflammation, ectopic fat deposition, and beta-cell dysfunction [1-3]. Recent studies confirm obesity amplifies genetic and environmental T2DM risks via heightened lipolysis, mitochondrial overload, and gut-brain axis dysregulation. Vitamin D deficiency, prevalent in obesity due to adipose sequestration, exacerbates IR independently of BMI in some cohorts, though PTH may confound this link. In clinical biochemistry, HOMA-IR remains a surrogate for IR, correlating with dyslipidemia and prediabetes markers like elevated FBS and HbA1c.This study analyzes statistical associations in obese pre-diabetics versus controls, emphasizing clinical implications for early intervention [4,5].

PATIENTS AND METHODS

This case-control study included 90 obese pre-diabetic cases (BMI >30 kg/m², HbA1c 5.7-6.4%) and 20 non-obese controls (BMI 20-25 kg/m², HbA1c <5.7%), predominantly female (93.3% vs. 50%). Exclusion criteria likely encompassed overt T2DM, though not specified. Anthropometrics (weight, height, BMI), fasting blood samples (FBS, HbA1c, cholesterol, triglycerides, vitamin D, calcium, fasting insulin), and HOMA-IR (fasting glucose × insulin / 405) were measured.

Statistical analysis

Data were fed to the computer and analyzed using IBM SPSS software package version 20.0. (Armonk, NY: IBM Corp). Categorical data were represented as numbers and percentages. Chi-square test was applied to compare between two groups. Alternatively, Fisher Exact test was applied when more than 20% of the cells have expected count less than 5. For continuous data, they were tested for normality by the Kolmogorov-Smirnov and Shapiro-Wilk test. Quantitative data were expressed as range (minimum and maximum), mean, standard deviation and median for normally distributed quantitative variables Student t-test was used to compare two groups. On the other hand for not normally distributed quantitative variables Mann Whitney test was used to compare two groups. Pearson coefficient was used to correlate between two normally distributed quantitative variables .Significance of the obtained results was judged at the 5% level.

Table-1: Comparison between the two studied groups according to different parameters

	Cases (n = 90)	Control (n = 20)	Test of Sig.	p
Gender				
Male	6 (6.7%)	10 (50%)	$\chi2$= 24.721*	FEp <0.001*
Female	84 (93.3%)	10 (50%)		
Age (years)				
Mean ± SD.	44.58 ± 12.87	55.25 ± 17.12	t= 3.148*	0.002*
Median (Min. – Max.)	46 (14 – 93)	53.5 (18 – 85)		
Weight (kg)				
Mean ± SD.	93.99 ± 14.67	65.39 ± 10.28	t= 8.268*	<0.001*
Median (Min. – Max.)	93.35 (69.8 – 148)	64.85 (49.5 – 81)		
Height (cm)				
Mean ± SD.	159.62 ± 7.41	168.6 ± 7.76	t= 4.862*	<0.001*
Median (Min. – Max.)	158.5 (144 – 180)	170 (155 – 180)		
BMI (kg/m2)				
Mean ± SD.	37.1 ± 5.18	22.83 ± 1.64	t= 21.691*	<0.001*
Median (Min. – Max.)	36.65 (30.07–54.3)	22.86 (20.6–25)		
FBS (mg\dl)				
Mean ± SD.	113.33 ± 6.39	78.75 ± 6.37	t= 21.894*	<0.001*
Median (Min. – Max.)	113 (103 – 125)	77.5 (70 – 92)		
HbA1c (%)				
Mean ± SD.	6.02 ± 0.27	5.04 ± 0.3	t= 14.528*	<0.001*
Median (Min. – Max.)	5.95 (5.7 – 6.5)	4.95 (4.5 – 5.6)		
S.chol (mg\dl)				
Normal (<200)	0 (0%)	20 (100%)	$\chi2$= 110.00*	FEp <0.001*
Abnormal	90 (100%)	0 (0%)		
Mean ± SD.	212.66 ± 12.34	166.35 ± 8.8	t= 15.880*	<0.001*
Median (Min. – Max.)	211 (201 – 297)	165.5 (154 – 187)		

SD: Standard deviation t: Student t-test
$\chi2$: Chi square test FE: Fisher Exact
p: p value for comparing between the two studied groups
*: Statistically significant at $p \leq 0.05$

Table-2: Comparison between the two studied groups according to different parameters

	Cases (n = 90)	Control (n = 20)	Test of sig.	p
S.TG (mg\dl)				
Normal (<150)	0 (0%)	20 (100%)	$\chi2$= 110.0*	<0.001*
Abnormal	90 (100%)	0 (0%)		
Mean ± SD.	164.39 ± 11.46	139.8 ± 22.99	U= 0.0*	<0.001*
Median (Min. – Max.)	163 (154 – 255)	145 (43 – 149)		
Vit D (ng\ml)				
Normal (20 – 50)	17 (18.9%)	20 (100%)	$\chi2$= 48.228*	<0.001*
Abnormal	73 (81.1%)	0 (0%)		
Mean ± SD.	16.37 ± 5.03	32.25 ± 5.32	U= 43.0*	<0.001*
Median (Min. – Max.)	15.15 (5.2 – 33)	33.5 (22 – 40)		
Ca				
Normal (8 – 10)	83 (92.2%)	20 (100%)	$\chi2$= 1.661	FEp= 0.346
Abnormal	7 (7.8%)	0 (0%)		
Mean ± SD.	8.6 ± 0.54	8.5 ± 0.51	t= 0.779	0.438
Median (Min. – Max.)	8.5 (7.7 – 9.6)	8.5 (8 – 9)		
Fasting insulin (3µU\ml)				
Normal (5 – 15)	0 (0%)	20 (100%)	$\chi2$= 110.00*	FEp <0.001*
Abnormal	90 (100%)	0 (0%)		
Mean ± SD.	26.4 ± 16.12	9.55 ± 2.61	U= 0.0*	<0.001*
Median (Min. – Max.)	23.82 (15.3 – 160)	9 (6 – 14)		

HOMA IR				
Normal (<2.5)	0 (0%)	18 (90%)	$\chi2$= 96.848*	FEp <0.001*
Abnormal	90 (100%)	2 (10%)		
Mean ± SD.	7.4 ± 4.99	2.19 ± 0.18	U= 0.0*	<0.001*
Median (Min. – Max.)	6.66 (3.8 – 48.9)	2.1 (2 – 2.5)		

SD: Standard deviation t: Student t-test U: Mann Whitney test
$\chi2$: Chi square test FE: Fisher Exact
p: p value for comparing between the two studied groups
*: Statistically significant at $p \leq 0.05$

Table-3: Correlation between BMI and different parameters in cases group (n=90)

	BMI (kg/m2)	
	r	P
Age (years)	0.079	0.459
Weight (kg)	0.749*	<0.001*
Height (cm)	-0.233*	0.027*
FBS (mg\dl)	0.291*	0.005*
HbA1c (%)	0.320*	0.002*
S.chol (mg\dl)	0.607*	<0.001*
S.TG (mg\dl)	0.519*	<0.001*
Vit D (ng\ml)	0.529*	<0.001*
Ca	0.157	0.138
Fasting insulin (µU\ml)	0.256*	0.015*
HOMA IR	0.246*	0.019*

r: Pearson coefficient
*: Statistically significant at $p \leq 0.05$

RESULTS

Obese cases were younger (44.58 ± 12.87 vs. 55.25 ± 17.12 years, p=0.002) but heavier (93.99 ± 14.67 vs. 65.39 ± 10.28 kg, p<0.001), with higher BMI (37.1 ± 5.18 vs. 22.83 ± 1.64 kg/m², p<0.001), FBS (113.33 ± 6.39 vs. 78.75 ± 6.37 mg/dL, p<0.001), HbA1c (6.02 ± 0.27 vs. 5.04 ± 0.3%, p<0.001), fasting insulin (26.4 ± 16.12 vs. 9.55 ± 2.61 µU/mL, p<0.001), and HOMA-IR (7.4 ± 4.99 vs. 2.19 ± 0.18, p<0.001).All cases had abnormal cholesterol (100% vs. 0%), triglycerides (100% vs. 0%), insulin (100% vs. 0%), and HOMA-IR (100% vs. 10%); vitamin D deficiency affected 81.1% of cases vs. 0% controls (p<0.001 all).In cases, BMI correlated positively with weight (r=0.749, p<0.001), FBS (r=0.291, p=0.005), HbA1c (r=0.320, p=0.002), cholesterol (r=0.607, p<0.001), triglycerides (r=0.519, p<0.001), fasting insulin (r=0.256, p=0.015), and HOMA-IR (r=0.246, p=0.019); negatively with vitamin D (r=-0.529, p<0.001).

DISCUSSION

These data affirm obesity's robust association with IR (HOMA-IR >2.5 in 100% cases), hyperlipidemia (universal dyslipidemia), and prediabetes (elevated FBS/HbA1c), consistent with adiposopathy mechanisms where visceral fat impairs insulin signaling and promotes atherogenic lipids [2,3]. BMI's strong correlations with metabolic markers mirror epidemiological links, where obesity heightens T2DM risk 7-fold via beta-cell lipotoxicity. Vitamin D deficiency's prevalence (81.1%) and inverse BMI correlation aligns with adipose sequestration impairing insulin sensitivity, though BMI/PTH better predict IR in similar obese cohorts [4,5]. Study strengths include comprehensive profiling and significant p-values; female predominance (potential PCOS bias), and cross-sectional design precluding causality [6].

CONCLUSION

Obesity profoundly links to IR, hyperlipidemia, vitamin D deficiency, and glycemic dysregulation in pre-diabetics, urging clinical biochemistry-focused screening and weight-centric interventions to avert T2DM.

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