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Evaluation of Resistin and Its Relationship with Vitamin D3 and Vitamin B12 in Type 2 Diabetes: A Case–Control Study

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Abstract : Background: T2DM, which accounts for over 90% of all cases of diabetes, is characterized by insulin resistance (IR), insufficient insulin secretion by pancreatic islet beta cells, and an inadequate compensatory insulin secretory response. Serum resistin levels in T2DM patients were evaluated, along with their relationship to vitamin D3 and vitamin B12.
Methods: In this case- control, there were 60 participants: group 1 (30 T2DM patients) and group (30 healthy controls) who were matched for age and BMI. Random blood glucose (RBS), HbA1c, resistin, vitamin D3, vitamin B12, liver enzymes, and renal function tests were measured from fasting blood samples. ELISA was used to quantify resistin. SPSS was used for data analysis, independent sample t-tests were used for intergroup comparisons, and Pearson correlation analysis was used within the diabetic group.
Results: Poor glycemic control was indicated by significantly higher RBS and HbA1c levels in T2DM patients when compared to controls ($p<0.001$). Diabetics had significantly higher serum resistin levels (9.66 ± 1.27 ng/mL) than controls (4.13 ± 0.44 ng/mL) ($p<0.001$). While there was no significant difference in vitamin B12 levels, the diabetic group's vitamin D3 levels were significantly lower ($p<0.001$). With the exception of a marked rise in alkaline phosphatase in diabetics, renal and the majority of hepatic parameters were similar across groups. Glycemic, vitamin, and biochemical parameters did not significantly correlate with resistin.
Conclusion: Type 2 diabetes is associated with elevated circulating resistin and reduced vitamin D3 levels, suggesting independent inflammatory and nutritional disturbances. The lack of significant correlations indicates that these biomarkers may contribute separately to T2DM pathophysiology.

Keywords - *Resistin; Type 2 Diabetes Mellitus; Vitamin D3; Vitamin B12; Insulin Resistance; Glycemic Control*

I. INTRODUCTION

Type 2 diabetes mellitus (T2DM) is a common chronic disease that causes hyperglycemia due to insulin resistance, decreased insulin secretion, or both. T2DM is more frequent in males than women and increases with age and fat, especially visceral or abdominal. A family history of diabetes or cardiovascular illness (especially hypertension or dyslipidemia) and inactivity enhance the risk of T2DM [1]. Pre-diabetes is characterized by elevated blood sugar levels that are not yet high enough for a Type 2 diabetes diagnosis. Type 2 diabetes results from insufficient insulin production or cellular resistance to insulin [2].

Triglycerides in adipose tissue are no longer considered passive energy storage. Leptin, adiponectin, and resistin are secreted by this tissue to regulate glucose, lipid, and body weight. In rodents, anti-diabetic

thiazolidinedione down-regulate resistin, a 12.5-kDa cysteine-rich polypeptide that is released from adipose tissue [3].

Human resistin is mostly generated by PBMCs. Although resistin has been linked to obesity and insulin resistance in rats, its significance in causing insulin resistance in humans remains controversial [4].

Earlier research demonstrated resistin is up-regulated in mouse models of obesity and insulin resistance, down-regulated by rosiglitazone, and immune-neutralized to reduce hyperglycemia and increase insulin sensitivity. These findings brought resistin to scientific attention as a potential etiological link between fat and diabetes and a pathogenic factor in insulin resistance. While most human studies examined resistin scenery in type II diabetics or its involvement in obesity, plasma resistin data with humans, especially in type I diabetics or non-obese persons, is limited [5].

This study aimed to evaluate serum resistin levels and to examine their relationship with vitamin D3, vitamin B12, and glycemic control in patients with type 2 diabetes mellitus compared with healthy individuals.

II. METHODS AND MATERIALS

2.1 Study design and patients

The protocol governing this research. (Thirty patients with type II diabetes and thirty individuals in the control group.) Fasting blood samples were collected to assess the circulating levels of fasting glucose (FBS), HbA1c, resistin, renal function tests, liver function tests, and vitamins D3 and B12. Thirty healthy individuals without any medical conditions in the past six months served as the control group. The physical examinations of all participants were conducted by the same physician, and their blood pressure, height, and weight were documented.

2.2 Samples collection and preparations

Five millilitres of fasting blood samples were obtained from all participants. A portion of that (2 ml) was transferred to the EDTA vial. Half of this sample was subjected to centrifugation, and the plasma was utilized for the determination in accordance with the protocol provided by the kit manufacturer, while the entire blood sample was employed to measure HbA1c. The fasting sera separated from the clotted blood samples were also utilized for the assessment of FBS, resistin, and D3, B12.

2.3 Determination of resistin

The serum resistin were performed by enzyme-linked immunosorbent assay (ELISA) kits from BioVendor Laboratory Medicine Industry (Czech Republic). Automated washing of 96-well ELISA plates was performed by a microplate washer (BioTek™ 50TS, USA), and the absorbance was measured at 450 nm using an ELISA reader (BioTek 800TS), The measurements were conducted and results were recorded following the manufacturer's instructions.

2.4 Determination of biochemical parameters

The serum glucose was determined using an endpoint glucose oxidase method with an automated chemistry analyzer (RA-1000 Technicon, USA). HbA1c was determined using an automated analyzer base on chromatography method (Drew DS5, UK).

2.5 Statistical analysis

Data were expressed as Mean ± SEM. Pre- and post-hemodialysis levels of the different analytes were compared for the groups of male and female subjects using unpaired t-tests. All statistical analyses were generated by using GraphPad Prism (version 7.0). A confidence level of $p < 0.05$ was considered statistically significant for all analyses.

III. RESULTS

3.1 Demographic characteristics of diabetic patients

The current study examined and contrasted various biochemical parameters between patients diagnosed with type 2 diabetes mellitus (case group) and healthy control subjects. The results are summarized in Tables 1 and 2.

There was no statistically significant difference in age between the case group (33.75 ± 10.99 years) and the control group (36.56 ± 14.33 years) (P = 0.66). Similarly, BMI exhibited no significant difference between diabetic patients (31.88 ± 5.14 kg/m²) and controls (29.50 ± 4.27 kg/m²) (P = 0.31).

Table 1: comparison of age and BMI between Type2 diabetes patients and healthy controls

Parameters	Control group Mean±SD	Case group Mean±SD	P. value
Age	36.56 ± 14.33	33.75 ± 10.99	0.66
BMI	29.50 ± 4.27	31.88 ± 5.14	0.31

Note. Data were expressed as Mean ± SD; Statistical analyses were performed by t test (paired test) for multiple comparisons; ‡ t test significance (2-tailed).

3.2 Comparison of studied biomarkers between diabetic patients and controls

The diabetes group had significantly higher random blood glucose (RBS) (202.88 ± 10.26 mg/dL) than the control group (100.78 ± 13.19 mg/dL) (P < 0.001). Diabetic patients had significantly higher HbA1c levels (8.87 ± 0.83%) compared to controls (5.23 ± 0.43%), indicating poor glycemic control (P < 0.001).

Urea and creatinine levels were similar between groups (P = 0.62 and 0.32, respectively). GOT and GPT liver enzymes did not differ between diabetics and controls (P = 0.75 and 0.67, respectively). The diabetes group had significantly higher alkaline phosphatase (ALK) levels (148.38 ± 40.44 U/L) compared to controls (97.89 ± 18.87 U/L) (P < 0.01). Significantly higher resistin levels (9.66 ± 1.27 ng/mL) were seen in type 2 diabetes patients compared to the control group (4.13 ± 0.44 ng/mL) (P < 0.001).

Diabetics had considerably lower vitamin D3 levels (11.55 ± 4.47 ng/mL) compared to healthy persons (20.77 ± 3.86 ng/mL) (P < 0.001), showing a high prevalence of vitamin D insufficiency. Vitamin B12 levels were not significantly different between the case (314.13 ± 113.75 pg/mL) and control groups (326.33 ± 66.28 pg/mL) (P = 0.78).

Table 2: Biochemical Parameters in Type 2 Diabetes Mellitus Compared to the Control Group.

Parameters	Control group Mean±SD	Case group Mean±SD	P. value
RBS	100.78 ±13.19	202.88 ± 10.26	<0.001
HbA1c	5.23 ± 0.43	8.87 ± 0.83	<0.001
Urea	39.56 ± 2.00	38.75 ± 4.43	0.62
Creatinine	0.82 ± 0.13	0.75 ± 0.15	0.32
GOT	17.89 ± 4.48	17.25 ± 3.69	0.75
GPT	17.44 ± 4.39	16.13 ± 7.93	0.67
ALP	97.89 ± 18.87	148.38 ± 40.44	<0.01
Resistin	4.13 ± 0.44	9.66 ± 1.27	<0.001
Vitamin D3	20.77 ± 3.86	11.55 ± 4.47	<0.001
Vitamin B12	326.33 ±66.28	314.13 ± 113.7	0.78

Abbreviations: RBS, Random Blood Sugar; HbA1c, Glycated hemoglobin; GOT; Glutamic Oxaloacetic Transaminase; GPT, Glutamic-Pyruvic Transaminase; ALP, Alkaline Phosphatase. Note. Data were expressed

as Mean ± SD; Statistical analyses were performed by t test (paired test) for multiple comparisons; ‡ t test significance (2-tailed); p-value less than 5% is significant difference.

3.3 Correlation between resistin and other biochemical parameters

Table 3 illustrations type 2 diabetes patients' Pearson correlation coefficients for biochemical markers. HbA1c did not correlate with any of the measures. Urea had a slight negative connection with creatinine and vitamin B12, but not statistically.

Creatinine did not correlate with liver enzymes (GOT, GPT, ALK) or resistin. No significant correlations were seen between GOT and GPT and glycaemic or inflammatory indicators. Most biochemical indicators, including RBS, HbA1c, liver enzymes, and renal markers, correlated poorly with resistin. Vitamin D3 had mild negative associations with GPT, ALK, and resistin albeit not statistically significant. The correlation matrix shows no statistically significant connections between resistin, vitamin D3, vitamin B12, and other biochemical indicators in diabetics..

Table 3: Correlation of IL-6 and IL-10 with the parameters examined in the pre-dialysis group.

Variables	Coefficient correlation	RBS	HbA1c	Urea	Creatinine	GOT	GPT	ALP	Resistin	Vit. D3
HbA1c	r	0.061								
	p	0.886								
Urea	r	-0.395	-0.041							
	p	0.333	0.924							
Creatinine	r	0.093	-0.453	-0.213						
	p	0.826	0.260	0.612						
GOT	r	0.314	0.651	-0.380	-0.333					
	p	0.449	0.080	0.354	0.421					
GPT	r	0.220	0.438	0.334	-0.661	0.428				
	p	0.600	0.277	0.419	0.074	0.291				
ALP	r	0.453	-0.098	0.355	-0.153	-0.435	0.149			
	p	0.259	0.818	0.388	0.717	0.282	0.726			
Resistin	r	-0.080	-0.232	0.301	0.148	0.023	0.464	-0.335		
	p	0.851	0.581	0.469	0.726	0.957	0.247	0.417		
Vit. D3	r	0.401	-0.242	-0.909**	0.368	0.196	-0.262	-0.349	0.029	
	p	0.325	0.563	0.002	0.369	0.642	0.530	0.397	0.947	
Vit B12	r	0.258	-0.405	-0.213	-0.020	0.189	-0.220	-0.103	-0.054	0.164
	p	0.538	0.319	0.613	0.963	0.654	0.601	0.808	0.898	0.699

IV. DISCUSSION

This study examined type 2 diabetes biochemical changes, focusing on serum resistin, vitamin D3, and vitamin B12. The results showed significant differences between diabetics and healthy controls, supporting the idea that inflammatory and nutritional indicators are associated to metabolic dysregulation in diabetes [6].

Poor glycemic control was shown by a substantial increase in RBS and HbA1c in the diabetic group. This supports prior research that chronic hyperglycemia is a key hallmark of type 2 diabetes and a major cause of metabolic and inflammatory problems [7].

This study's result that diabetics had higher serum resistin levels than healthy people is notable. Resistin, an inflammatory adipokine, causes insulin resistance, endothelial dysfunction, and systemic inflammation. Diabetics have elevated resistin levels, supporting its function in glucose dysregulation and type 2 diabetes etiology. The elevated resistin levels in diabetics indicate its independent role in their inflammatory profile, even though the correlation study indicated no statistically significant association between resistin and other biochemical markers [8].

In type 2 diabetes, vitamin D3 levels dropped significantly. Vitamin D insufficiency is common among diabetics and linked to insulin resistance, decreased insulin production, and inflammation. Hypovitaminosis D

may lead to diabetes metabolic imbalance, according to these studies. Although this study identified no significant link between vitamin D3 and resistin, the simultaneous increase in resistin and reduction in vitamin D shows that both indicators may participate in independent but related pathways affecting glucose metabolism and systemic inflammation [9].

Blood levels of vitamin B12 were somewhat lower in diabetics but not significantly different from controls. Dietary changes or metformin use, which reduces vitamin B12 absorption, may explain this. Lack of significant difference suggests vitamin B12 status is less affected in this study population or that other factors may modulate it [10].

The two groups had similar liver enzymes (GOT, GPT), indicating that diabetes patients had no severe hepatic impairment. Alkaline phosphatase was considerably higher in the diabetic group, which may indicate subclinical cholestasis, bone turnover changes, or vitamin D deficiency, which is essential for bone metabolism [11].

Urea and creatinine levels were unchanged, indicating maintained renal function in the population. This matches participants' lack of severe diabetic nephropathy [12]. The study shows that type 2 diabetes causes metabolic and inflammatory changes, including higher resistin and decreased vitamin D3. Lack of substantial correlations between variables may imply that each biomarker contributes independently to illness progression or that a larger sample size is needed to discover minor interactions [13].

V CONCLUSION

This case–control study demonstrates that type 2 diabetes mellitus is associated with significant metabolic, inflammatory, and nutritional alterations. Patients with T2DM exhibited markedly elevated serum resistin levels alongside significantly reduced vitamin D3 concentrations compared with healthy controls, highlighting the coexistence of inflammatory activation and micronutrient deficiency in diabetes. In contrast, vitamin B12 levels and most renal and hepatic parameters remained comparable between groups, indicating preserved organ function in the studied population. The absence of significant correlations between resistin, glycemic indices, vitamin D3, and vitamin B12 suggests that these biomarkers may contribute independently to the pathophysiology of T2DM rather than through direct interrelated mechanisms. Overall, elevated resistin and vitamin D deficiency may represent parallel but distinct pathways involved in metabolic dysregulation in type 2 diabetes. Further large-scale and longitudinal studies are warranted to clarify their mechanistic roles and potential clinical utility as biomarkers or therapeutic targets in diabetes management.

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