

Correlation of Vitamin D with Inflammation and Hypertriglyceridemia in Type 2 Diabetes Mellitus in Southeast India

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Abstract:

Aims: Diabetes Mellitus has become an increasing global health problem and is a major health problem in India. Even though lot of research has been done for the treatment and prevention of this disease, still research in Southeast India is limited. Recent studies show a correlation between Vitamin D and diabetes; however other contributory factors also influence this correlation. The present study was carried out with an objective to find out specific correlation between Vitamin D, inflammation, triglyceride level and insulin resistance in type 2 DM excluding all other confabulatory factors.

Methods: A case control study was done comprising of 140 type 2 diabetic patients and 150 healthy control individuals. Biochemical parameters like fasting blood glucose, lipid profile (total cholesterol, triglyceride, HDL and LDL), Vitamin D, hsCRP, Fasting Insulin were measured. Insulin resistance was calculated by HOMA-IR. Statistical Analysis was done by using SPSS Version 19 software.

Results: Hypo-vitaminosis D was found in Diabetic patients and a significant correlation was found between Vitamin D, triglyceride level, severity of inflammation and severity of insulin resistance.

Conclusion: The present study concludes that Vitamin D deficiency is present in type 2 diabetes mellitus and has a cause-effect relationship with inflammation and hypertriglyceridemia.

Key words: Insulin resistance; Vitamin D; Inflammation; Dyslipidemia; Cause-effect relationship.

I. Introduction

Diabetes mellitus (DM) is an increasing endemic global health problem mostly in developing countries like South East Asia and Western Pacific region with India and China as focal points. (1) In India the prevalence has increased from 50.8 million in 2010 to 62.4 million in 2011 (according to Madras Diabetic Research Foundation) (2) and is projected to increase to 69.9 million by 2025 and 100 million by 2030. The disease requires continuous medical treatment and proper education of patient for self-management and frequent follow-up to prevent acute complications and to reduce the risk of long term complications. (3) This is heavily taxing on the individual with the expenditure of care being very high with as much as 1/3 rd of low income household (4) as well as on the country's economy as the doctor- patient ratio is low. The scenario in Southeast India is different because of large population belonging to low and middle income group with no proper diet or proper access to medical care. Thus increased research is important for mainly prevention as well as accurate adequate treatment.

1.1 Diabetes Mellitus And Insulin Resistance

The major risk factors are obesity (BMI > 25 kg/m²), habitual physical inactivity, race/ ethnicity, previously identified impaired fasting glucose or impaired glucose tolerance, hypertension, decreased high density cholesterol (< 35 mg/dl), increased triglyceride (> 250 mg/dl), history of gestational diabetes mellitus or delivery of baby weighing > 4.1 kg polycystic ovary disease). (5). The three cardinal abnormalities seen in type 2 DM are resistance to the action of insulin in peripheral tissues like adipose tissue, skeletal muscle and liver, defective insulin secretion mainly in response to glucose stimulus and increased production of glucose by liver. (6). Initially there occurs hyper-insulinemia to overcome the insulin resistance but eventually β cell dysfunction occurs which is the inability of β cell to adapt to the decreased insulin sensitivity thereby precipitating type 2 DM. (5) The interest in Vitamin D and DM correlation was first generated by animal studies which showed the presence of Vitamin D Receptor (VDR) in pancreatic tissue. (7) This fuelled a lot of speculation in recent decades whether Vitamin D has any role in preventing disease or decreasing its complications.

1.2 Vitamin D

Vitamin D has a major action on β cells of pancreas. It increases the intracellular calcium concentration via non-selective voltage dependant calcium channel activation. It also activates calcium dependant endopeptidases which help in cleavage of proinsulin to insulin thereby increasing the insulin concentration. (8) In peripheral tissues Vitamin D stimulates the expression of insulin receptors and regulates the cytosolic ionised calcium level (Ca^{+2}). Decreased cytosolic ionised calcium level impairs signal transduction causing decreased GLUT 4 activity, thus Vitamin D indirectly regulates the insulin mediated intracellular processes. Vitamin D also mediates the transcriptional activation of human insulin gene; Vitamin D Response Element (VDRE) present in human insulin gene promoter region and stimulates expression of insulin receptor.

Pathophysiological role of Vitamin D in diabetes and pre-diabetes was evidenced by a randomised controlled trial done by Dutta et al in 2013. (9, 10) It was found that Vitamin D deficiency or insufficiency was present in pre-diabetic patients, individuals with lowest Vitamin D had highest insulin resistance and Vitamin D had statistically significant inverse correlation with insulin resistance. These findings are similar to Calcium and Vitamin D for Diabetes Mellitus (CaDDM) randomized controlled trial by Mitri et al in 2011(11) and a cross sectional study done by Pittas in 2007 which showed inverse relationship between serum Vitamin D concentration and fasting or post glucose load value. (12) However in a double blind randomized trial of cholecalciferol for 12 weeks done by Kampmann in 2014, it was observed that even with improvement of Vitamin D status there was no significant change in insulin sensitivity or inflammation. (13) Thus hypovitaminosis is present in type 2 DM but other confounding factors should be considered to establish the association between Vitamin D and insulin resistance.

1.3 Inflammation

The first study showing the link between inflammation and insulin resistance was demonstrated by Hotamisligil et al in 1993 which showed that TNF α mRNA expression is increased in adipose tissue of obese rats which on neutralization causes improved insulin action on glucose uptake. (14)

The correlation of vitamin D and inflammation sparked interest due to presence of VDR on immune cells like monocytes, macrophages, activated T and B lymphocytes. Experimentally it has been shown that vitamin D decreases proliferation, immunoglobulin synthesis and cytokine production in mixed lymphocyte cell culture. (15). Vitamin D has an immune-modulatory effect by interacting with VDRE in promoter region of cytokine genes and interferes with nuclear transcription factors involved in cytokine generation. It down regulates the activation of NF- $\text{K}\beta$ which regulates genes encoding pro-inflammatory cytokines. Vitamin D also upregulates expression of Calbindin, a cytosolic calcium binding protein which protects against cytokine induced apoptosis by controlling cytosolic free calcium (Ca^{+2}). (8) This effect was demonstrated in a study done by Hartaigh et al in 2013(16) on patients undergoing coronary angiography in a cross sectional findings from Ludwigshafen Risk and Cardiovascular Health (LURIC) study which found that on increasing Vitamin D concentration insulin resistance and pro-inflammatory markers IL 6, CRP decreased considerably. However some studies have found different results. In a double blind randomized trial done by Akbarzadeh et al in 2013, type 2 DM patients received 12 weeks of oral cholecalciferol and hsCRP, IL 6 and IL 18 were assessed at end of study. The authors found that IL 18 and hsCRP had positive associations with insulin resistance but no significant difference in the serum levels was found in between patients and controls after oral supplementation thereby concluding that inflammation was associated with insulin resistance in type 2 DM but no anti-inflammatory effect of vitamin D was detected. (17) This indicates that there could be other factor / factors which have to be considered for the effective action of Vitamin D.

1.4 Diabetic Dyslipidemia

Diabetic dyslipidemia is due to insulin resistance which causes decreased uptake of free fatty acids (FFAs) by striated muscle and adipose tissue. This results in increased levels of FFAs which are delivered to the liver, and cause overproduction of very low-density lipoprotein (VLDL) which manifests as hypertriglyceridemia. There is also decrease in lipoprotein lipase (LPL) activity which leads to an accumulation of triglyceride-rich lipoproteins in the plasma. (18).

In spite of lot of research on Vitamin D there has been no conclusive evidence that supplementation of Vitamin D can improve diabetic status. This study has been taken up to identify which variable (Vitamin D, inflammation or hypertriglyceridemia) has the stronger correlation with insulin resistance and thereby correctly assess type 2 diabetes mellitus for individual management.

II. Materials & Methods

This case control study was undertaken in the Department of Biochemistry, M.K.C.G. Medical College, Brahmapur from November 2013 to July 2014.

Inclusion criteria: The study included 140 patients meeting the criteria of DM (as per National Diabetes Data Group and World Health Organization i.e. symptoms of DM plus random blood sugar >200 mg/dL, fasting plasma glucose >126mg/dL or 2hr plasma glucose >200 mg/dL during oral glucose tolerance test) attending the Endocrinology Outpatient Department at MKCG Medical College and Hospital, Brahmapur, Odisha during the study period. The subjects having recent onset of DM (0-10 years) and having optimal Body Mass Index (BMI) of 20 – 25 were included in the study to eliminate confounding factors, and having voluntarily consented to support the research work. 150 control subjects were chosen from healthy individuals and were age and sex matched.

The study group was divided into two groups: group 1 comprised of patients with DM having normal triglyceride level (< 150 mg/dl) and group 2 comprised of patients having hypertriglyceridemia (> 200 mg/dl) to find the association of dyslipidemia in DM.

Exclusion criteria: Patient with co morbid associations like chronic renal disease, cardiovascular disease, coronary artery disease, recent amputation, autoimmune diseases and smokers were excluded from the study. Patient taking vitamin D supplements, steroids, NSAIDs, antibiotics and multi- vitamin or anti oxidant supplements were also excluded from the study group. Patients who were overweight or obese (BMI > 25) and having long duration (> 10 years) of DM were also excluded from the study.

Approval for this work was obtained from the Institutional Ethical Committee, M.K.C.G. Medical College, Brahmapur.

Biochemical Analysis:

A sample of venous blood (10 ml) was collected in a dry, sterile disposable syringe under aseptic conditions. 2 ml of blood was kept for fasting blood sugar estimation in vials containing fluoride as glycolytic inhibitor. Rest of the sample was placed in a centrifuge tube and serum was separated as soon as possible.

The study comprised of estimation of fasting blood glucose by glucose oxidase method and lipid profile (total cholesterol by cholesterol oxidase- phenol amino antipyrine (CHOD-PAP) method, triglyceride by glycerol phosphate oxidase- amino anti pyrine , dichloro hydroxybenzene sulfonate method and HDL by phosphotungstate method) by commercial kits adapted to EM360 Erba Transasia Auto-Analyzusing commercial kits from Erba Diagnostics adapted to EM360 Erba Transasia Auto analyzer. LDL was calculated using FRIEDEWALD EQUATION= TC- HDL -TG/5. Vitamin D was estimated by commercially available ELISA kit of DIAsource Immunoassays Catalog number KAP1971. Serum hsCRP was estimated using commercially available ELISA kit marketed by Calbiotech Catalog number: CR120C. Fasting Insulin was assessed by commercial ELISA kit made by Diagnostic Automation / Cortes Ciagnostics Inc. Immunodiagnosics, Catalog number: 1606Z. Insulin resistance was calculated by HOMA-IR using the formula: (19)

$$\text{HOMA-IR} = [\text{Fasting insulin in } \mu\text{IU/ ml} \times \text{Fasting glucose in mg/dl}] \div 405$$

Statistical Analysis was done by using SPSS Version 19 software. Statistical analysis of data was done using students t test and correlation was calculated by using the Pearson’s correlation method. A p value of <0.05 was considered significant

III. Results

Table 1: Compares the age, duration of disease, fasting blood glucose, lipid profile, fasting insulin, HOMA-IR, hsCRP, Vitamin D in control group and patient group1 &2.

Serial no.	Parameters	Control	Group 1 (TG<150mg/dl)	Group 2(TG>200 mg/dl)	p value
1.	Age(years)	53.12 ± 11	54.0 ± 8	54.5 ± 8	> 0.05Not significant
2.	Duration of DM (years)	0	4.16 ± 3.16	3.9 ± 3.2	>0.05Not significant
3.	FBS(mg/dl)	96.2 ± 11	135 ± 25	152 ± 27	.000
4.	Cholesterol (mg/dl)	143 ± 21	168 ± 11	271 ± 44	.000
5.	Triglyceride (mg/dl)	108 ± 23	129 ± 13	250 ± 45	.000
6.	HDL (mg/dl)	59 ± 14	43 ± 9	34 ± 3	.000
7.	LDL (mg/dl)	52 ± 23	100 ± 15	182 ± 40	.000
8.	Fasting insulin (μIU/ ml)	6.2 ± 1.2	16.12 ± 6	25.4 ± 8	.000
9.	HOMA-IR	1.46 ± 0.27	5.7 ± 2.8	9.6 ± 3.7	.003
6.	hsCRP (mg/l)	1.5 ± 0.5	6.5 ± 3	8.0 ± 2.5	.000
7.	Vitamin D (ng/ml)	27.2 ± 3.5	16.9 ± 3.37	13.9 ± 2.5	.000

The Mean ± SD age in years of the control and patient groups 1, 2 were 53.2 ± 12, 54.0 ± 8 and 54.5 ± 8 respectively. The duration of disease was 4.16 ± 3.16 and 3.9 ± 3.2 years for diabetic case groups 1 and 2. A significant difference was observed in fasting blood sugar (FBS) between diabetic patients group 1 and group 2 (135 ± 25 and 152 ± 27 mg/dl) and controls (96.2 ± 11mg/dl). A significant difference was found in total cholesterol level in controls (143 ± 21 mg/dl) and in diabetic case group 1 and group 2 (168 ± 11 and 271 ± 44 mg/dl) respectively. Similar significant differences were found in TG level (controls - 108 ± 23 mg/dl and diabetic case group 1 and case group 2 -129 ± 13 and 250 ± 45 mg/dl respectively; HDL level (control - 59 ± 14

mg/dl , diabetic case group1 and case group2 - 43 ± 9 and 34 ± 3 mg/dl respectively) ; and LDL level (control - 52 ± 23 and diabetic case group 1 and group 2 - 100 ± 15 and 182 ± 40 mg/dl respectively).A significant difference was also found in fasting insulin in controls (6.2 ± 1.2 μ IU/ ml and diabetic patients group 1 and group 2(16.12 ± 6 and 25.4 ± 8 μ IU/ ml respectively). A significant difference was found for HOMA-IR in controls 1.46 ± 0.27 and diabetic patients group 1 and group (5.7 ± 2.8 and 9.6 ± 3.7) respectively. The Mean \pm SD for hsCRP in mg/l was 1.5 ± 0.5 for control and 6.5 ± 3 and 8.0 ± 2.5 for diabetic case group1 and group 2 respectively. The Mean \pm SD for Vitamin D in ng/ml was 27.2 ± 3.5 for control and 16.9 ± 3.37 and 13.9 ± 2.5 for diabetic case group1 and case group2 respectively.

Table 2: Comparison of fasting blood glucose, lipid profile, fasting insulin, HOMA-IR, hsCRP, Vitamin D in control group and patient groups 1 with normal triglyceride level.

	Parameter	Control	Diabetic Group 1	P value
1.	FBS(mg/dl)	96.2 ± 11	135 ± 25	0.00
2.	Triglyceride (mg/dl)	108 ± 23	129 ± 13	0.00
3.	Fasting insulin (μ IU/ ml)	6.2 ± 1.2	16.12 ± 6	0.00
4.	HOMA-IR	1.46 ± 0.27	5.7 ± 2.8	0.00
5.	hsCRP (mg/l)	1.5 ± 0.5	6.5 ± 3	0.00
6.	Vitamin D (ng/ml)	27.2 ± 3.5	16.9 ± 3.37	0.00

A significant difference was observed in fasting blood glucose (FBS) between diabetic patients group 1 and group 2 (135 ± 25 and 152 ± 27 mg/dl) and controls (96.2 ± 11 mg/dl). A significant difference was found in TG level (controls - 108 ± 23 mg/dl and diabetic case group 1 - 129 ± 13 mg/dl respectively). A significant difference was also found in fasting insulin in controls (6.2 ± 1.2 μ IU/ ml and diabetic patients group 1 (16.12 ± 6) μ IU/ ml respectively). A significant difference was found for HOMA-IR in controls 1.46 ± 0.27 and diabetic patients group 1 (5.7 ± 2.8) respectively. The Mean \pm SD for hsCRP in mg/l was 1.5 ± 0.5 for control and 6.5 ± 3 for diabetic case group1 respectively. The Mean \pm SD for Vitamin D in ng/ml was 27.2 ± 3.5 for control and 16.9 ± 3.37 for diabetic case group1respectively.

Table 3: Pearson Correlation in between the triglyceride level, insulin resistance, inflammation and Vitamin D in diabetic patients

	R ²	P	R ²	p	R ²	P	R ²	p
TG	1		.712	.000	.604	.000	-.616	.000
HOMA-IR			1		.731	.000	-.761	.000
hsCRP					1		-.733	.000
VITAMIN D							1	

	TG	HOMA-IR	hsCRP	VITAMIN D

We observed positive correlation between TG level and HOMA-IR ($R^2=0.712$, p value<0.01).There was significant positive correlation between TG level and hsCRP ($R^2= 0.604$, p value<0.01) and has significant negative correlation with vitamin D ($R^2= -0.616$, p value<0.01). HOMA-IR level has significant positive correlation with hsCRP level ($R^2= 0.731$, p value<0.01) and significant negative correlation with vitamin D level ($R^2= - 0.761$, p value<0.01) .Vitamin D level has significant negative correlation with hsCRP ($R^2= -0.733$, p value<0.01)

Table 4: Comparison of fasting blood glucose, lipid profile, fasting insulin, HOMA-IR, hsCRP and Vitamin D in patient groups 1 and group 2

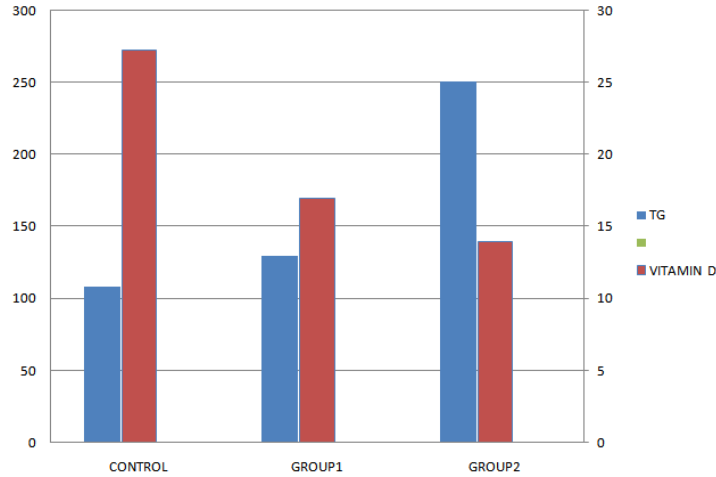
	Parameter	Diabetic group 1	Diabetic group 2	P value
1.	FBS(mg/dl)	135 ± 25	152 ± 27	0.00
2.	Triglyceride (mg/dl)	129 ± 13	250 ± 45	0.00
3.	Fasting insulin (μ IU/ ml)	16.12 ± 6	25.4 ± 8	0.00
4.	HOMA-IR	5.7 ± 2.8	9.6 ± 3.7	0.00
5.	hsCRP (mg/l)	6.5 ± 3	8.0 ± 2.5	0.00
6.	Vitamin D (ng/ml)	16.9 ± 3.37	13.9 ± 2.5	0.00

A significant difference was observed in fasting blood glucose (FBS) between diabetic patients group 1 and group 2 (135 ± 25 and 152 ± 27 mg/dl) respectively. A significant difference was found in TG level in diabetic case group 1 and case group 2 - 129 ± 13 and 250 ± 45 mg/dl respectively. A significant difference was also found in fasting insulin in diabetic patients group 1 and group 2(16.12 ± 6 and 25.4 ± 8 μ IU/ ml) respectively. A significant difference was found for HOMA-IR in diabetic patients group 1 and group (5.7 ± 2.8 and 9.6 ± 3.7) respectively. The Mean \pm SD for hsCRP in mg/l was 6.5 ± 3 and 8.0 ± 2.5 for diabetic case

group1 and group 2 respectively. The Mean \pm SD for Vitamin D in ng/ml was 16.9 ± 3.37 and 13.9 ± 2.5 for diabetic case group1 and case group2 respectively.

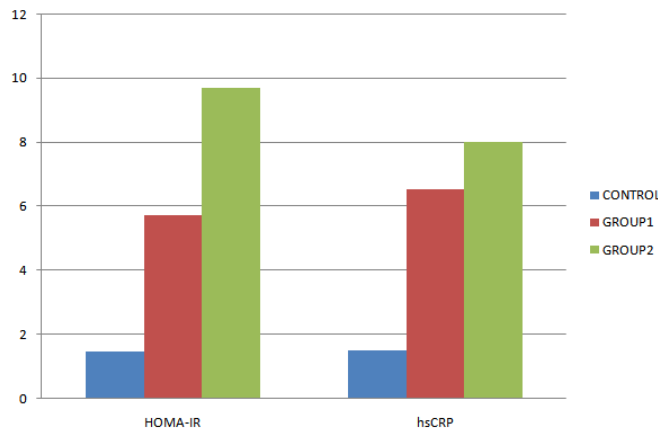
Graph 1: Comparison of Vitamin D and triglyceride levels in controls and in case groups 1 and 2.

COMPARISON OF VITAMIN D AND TRIGLYCERIDE LEVEL IN CONTROLS AND CASES

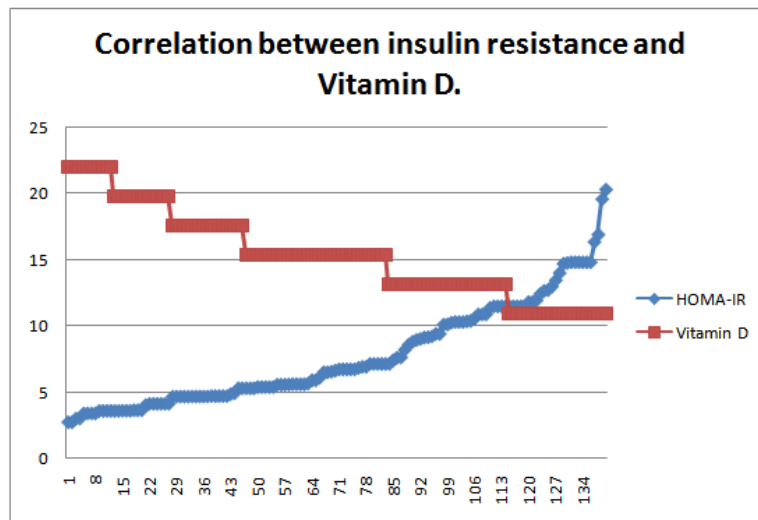


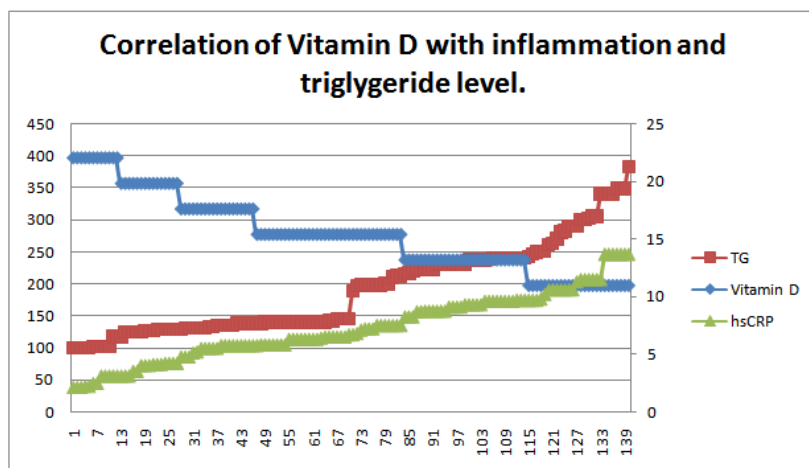
Graph 2: Comparison of insulin resistance and inflammation in controls and case groups 1 and 2.

COMPARISON OF INSULIN RESISTANCE AND INFLAMMATION IN CONTROLS AND CASES



Graph 3: Correlation of insulin resistance and Vitamin D.





Graph 4: Correlation of Vitamin D with inflammation and triglyceride level.

IV. Discussion

In our study, 140 type 2 DM patients were included and divided into two equal age and sex matched groups according to their triglyceride levels: group 1 having normal triglyceride level < 150 mg/dl (Mean ± SD = 129 ± 13 mg/dl) and group 2 having increased triglyceride level >200 mg/dl (Mean ± SD = 250 ± 45mg/dl); both groups having approximately equal duration of diabetes Mean ± SD = 4.16 ± 3.16 years and 3.9 ± 3.2 years respectively. The fasting plasma sugar and fasting insulin of group2 was significantly higher than group1; therefore the insulin resistance as calculated by HOMA-IR was significantly higher in group2 (Mean ± SD = 9.6 ± 3.7) as compared to group1 (Mean ± SD = 5.7 ± 2.8) and controls (Mean ± SD = 1.46 ± 0.27). The Vitamin D level was significantly low in diabetic patients group 2 with Mean ± SD = 13.9 ± 2.5 ng/ml compared to that of group 1 (Mean ± SD = 16.9 ± 3.37 ng/ml) and controls who had Mean ± SD = 27.2 ± 3.5 ng/ml.

These findings indicate that hypo-vitaminosis D is present in DM; and it is assumed that Vitamin D supplementation could have therapeutic role in DM. This is in concurrence with a study conducted by Talaei et al in 2013 (20) in which 100 type 2 DM patients were recruited and serum insulin, vitamin D concentration were measured, HOMA-IR was calculated at baseline. Then on supplementation of oral vitamin D 50,000 IU per week for 8 weeks significant improvement was found in serum fasting plasma glucose, insulin and HOMA-IR.

However in a randomised control trial done by Strobel et al in 2014, after 6 month vitamin D supplementation of 1904 IU to 86 diabetic patients, they found that fasting insulin positively correlated with vitamin D concentration but had no effect on HOMA index. (21) In a pilot study done by Parekh D, (22) a randomised double blind study was conducted in 28 Asian Indian patients. They found that after 4 weeks of vitamin D supplementation there was no significant difference in fasting plasma glucose and serum insulin. The authors thereby concluded that short term improvement in vitamin D status was not associated with improvement in glucose tolerance, insulin secretion or insulin sensitivity. In a similar study done by Patel et al (23) where 4 months of 400IU (group1) and 1200 IU (group2) oral calciferol was given to type 2 DM subjects, a rise of serum vitamin D did not improve euglycemia or insulin sensitivity nor effected any change in lipid profile.

The above studies indicate that even though hypo-vitaminosis D is present in type 2 DM, only vitamin D supplementation does not cause significant improvement on glycemic indices but has effect on insulin. This suggests that other confounding factors could be present in DM which needs to be identified.

Another constant factor found in type 2 DM is a chronic low grade inflammation which may cause insulin resistance. In our study hsCRP was measured as surrogate marker of inflammation. We observed that in type 2 DM patients the level of hsCRP was higher in group 2 i.e cases with hypertriglyceridemia (Mean ± SD = 8.0 ± 2.5 mg/l) as compared to cases with normal triglyceride level group1 (Mean ± SD = 6.5 ± 3mg/l) and control group (Mean ± SD = 1.5 ± 0.5 mg/l). This indicates that inflammation is significantly more present in type 2 DM patients with hypertriglyceridemia. On statistical analysis hsCRP showed a significant positive correlation ($R^2 = 0.731$, $p < 0.005$) with insulin resistance as measured by HOMA-IR and a significant negative correlation with vitamin D levels ($R^2 = -0.733$, $p < 0.005$). This indicates that inflammation is detrimental in causing insulin resistance and Vitamin D has a role in decreasing inflammation.

These findings are similar to a multivariate path analysis done by Kabadi et al in 2013 where weak association of vitamin D and HbA1c was found attributable to CRP. (24) Similar finding was seen in MONICA/KORA study in 2011 by Thorand et al in which 416 type 2 DM were selected from the source population of Monitoring of trends and determinants in cardiovascular disease (MONICA) / Cooperative health research in region of Augsburg (KORA) study. They observed significant inverse correlations between vitamin

D and soluble intercellular adhesion molecule -1, IL-6, interferon γ inducible protein-10 / CXCL-10 and C reactive protein. The authors thereby concluded that these four markers of inflammation are potential mediating factors and relationship between vitamin D and type 2 DM is partially mediated by subclinical inflammation. (25) But in a study done by Luo et al in 2009 among Chinese type 2 DM patients, it was seen that hypovitaminosis D was found in patients but there was no association of vitamin D on glycemic control or on markers of systemic inflammation hsCRP, IL-6. (26) This suggests that apart from hypovitaminosis D and inflammation there are other factors involved in the etiopathogenesis of type 2 diabetes mellitus.

Diabetic dyslipidemia is also a common feature of type 2 DM. In our study we observed that in type 2 DM patients triglyceride level showed a significant positive correlation ($R^2 = 0.712$, $p < 0.005$) with insulin resistance as measured by HOMA-IR, a significant positive correlation with inflammatory marker hsCRP ($R^2 = 0.604$, $p < 0.005$) and a significant negative correlation with Vitamin D levels ($R^2 = -0.616$, $p < 0.005$). This indicates that hypertriglyceridemia is detrimental in causing inflammation and insulin resistance and Vitamin D has a role in decreasing the triglyceride levels.

V. Conclusion

In this present study 140 type 2 DM patients and 150 age and sex matched controls were screened. The diabetic cases were divided into two groups based on their triglyceride levels. The level of vitamin D was significantly lower in patients having higher triglyceride levels as compared to patients having normal triglyceride levels and controls. Decrease of vitamin D correlated to triglyceride level, severity of inflammation and severity of insulin resistance. The surrogate inflammation marker hsCRP was detected to be higher in type 2 DM group having higher triglyceride levels. In the present study hypovitaminosis D was detected in diabetes mellitus which indicates that Vitamin D deficiency can cause inflammation and hypertriglyceridemia. However controversy still exists regarding therapeutic role of Vitamin D in type 2 diabetes mellitus. Hence more multicentric and multifactorial large scale research is needed before establishing a beneficial therapeutic role of Vitamin D in diabetes mellitus.

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