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ASSOCIATION BETWEEN SERUM FERRITIN AND TYPE-2 DIABETES MELLITUS

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ABSTRACT

Background: Diabetes mellitus is one of the most common, chronic, metabolic disorder, in which glucose utilization is reduced, producing hyperglycemia. It is due to absolute or relative insulin deficiency, that leads increased blood glucose level in the body. Along with hyperglycemia, development of debilitating complications like retinopathy, nephropathy, neuropathy, cardiovascular diseases and cerebrovascular diseases also make it as a major public health concern in the world.
Objectives: To determine the association between serum ferritin level and Type-2 Diabetes mellitus status among the patients attending Internal Medicine OP of Govt. Medical College, Trivandrum, Kerala.
Method: The study titled "Association between serum ferritin and Type-2 Diabetes mellitus" was conducted in Internal Medicine OP, MCH, Thiruvananthapuram. This study was completed in Central diagnostic biochemistry laboratory, MCH, Thiruvananthapuram. The study population included 45 cases and 45 controls. Cases were selected based on the inclusion-exclusion criteria. Age and sex matched healthy controls were taken as control. Blood samples were collected after getting written patient consent, for assessing the biochemical parameters such as ferritin, plasma blood glucose, HbA1c, cholesterol and creatinine.
Results:

- The average value of serum ferritin in diabetic patients was 197.36±113.45ng/ml whereas in control group it was 48.87±16.36ng/ml
- The mean value of HbA1c levels in cases was 9.13±1.60% and that of control was 4.59±0.39%
- The serum ferritin level showed a positive correlation with duration of diabetes mellitus.(r=0.975,p=0.000)
- The serum ferritin level have a significant positive correlation with HbA1c levels(.(r=0.916,p=0.000)
- The study showed a positive correlation of serum ferritin levels with FBS and PPBS, with r=0.569, p=0.000 in FBS and r=0.885,p=0.000 in PPBS.
- When correlating serum ferritin levels with total cholesterol and creatinine, shows a positive correlation with r=0.856,p=0.000 in total cholesterol and r=0.830,p=0.000 in creatinine. Serum ferritin level in Type 2 diabetics is significantly elevated than non-diabetics. Ferritin may be used as one of the important biomarkers in predicting diabetes mellitus.

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INTRODUCTION

Diabetes mellitus is one of the most common, chronic, metabolic disorder, in which glucose utilization is reduced, producing hyperglycemia. It is due to absolute or relative insulin deficiency, that leads increased blood glucose level in the body. Along with hyperglycemia, development of debilitating complications like retinopathy, nephropathy, neuropathy, cardiovascular diseases and cerebrovascular diseases also make it as a major public health concern in the world.

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Incidence of Diabetes mellitus is increasing globally and it is the one of the leading cause of death in the world. In India, among the 20 and 79 aged people, 65.1 million have diabetes and by 2035 rise to 109 million is expecIron is the most important and abundant metal in the human body. Ferritin, one of the major intracellular protein complex regulating iron homeostasis and widely used biomarker to assess the iron stores of the body. Although it is an essential mineral necessary for different physiological activities, the problem is that the health hazard because of its high reactivity. As iron can reversibly oxidized and reduced easily and take part in the formation of powerful oxidant species like hydroxyl radical. Iron is stored as Fe^{3+} oxidation state in ferritin. When body needs iron, Fe^{3+} is reduced to Fe^{2+}.The catalytic iron can

convert poorly reactive free radicals like hydrogen peroxide to highly reactive hydroxyl radical and superoxide anion that may lead to oxidative damage. This can change membrane properties and results in severe tissue damage. Ferritin is an effective marker of body iron stores. Iron overload in blood can contributes abnormal glucose homeostasis. As ferritin is a powerful pro-oxidant, it can cause oxidative damage to pancreatic beta cells and there by decreased secretion of insulin. Insulin resistance also seen in systemic iron over load.

AIM: To determine the association between serum ferritin level and Type-2 Diabetes mellitus status among the patients attending Internal Medicine OP of Govt. Medical College, Trivandrum, Kerala.

REVIEW OF LITERATURE

Diabetes mellitus consists of a group of metabolic disorders that share the phenotype of hyperglycemia. It is one of the most challenging health problem in the 21 century and the leading cause of death in many countries. Hyperglycemia is a common effect of uncontrolled diabetes mellitus, it can be caused mainly by, decrease in insulin production by beta cells of the Islet of Langerhans in the pancreas, defect in insulin action, decreased glucose utilization and increased glucose production.

Iron Metabolism

Iron is one of the most important and essential trace elements in the body. Total ironcontent in a human of 70 kg body weight varies approximately from 2.3 gm to 3.8 gm. Average iron content of adult males is about 3.8 gm and of females about 2.3 gm.

There are different Types of Iron Present in body and are divided into two broadcategories:

- Essential (or functional) iron
- Storage iron.

Essential Iron: Essential or functional iron is one which is involved inthe normal metabolism of the cells. They are mainly divided into three groups:

- Haem Proteins - Haemoglobin, Myoglobin, Catalases, Peroxidases
- Cytochromes
- Iron Requiring Enzymes -Xanthine oxidase, Cytochrome C reductase,

Storage iron: two major compounds.

- Ferritin
- Haemosiderin.

Iron Homeostasis

Human iron metabolism is the set of chemical reactions maintaining human homeostasis of iron at both the systemic and cellular level. The control of this necessary but potentially toxic metal is an important part of many aspects of human health and disease Understanding iron metabolism is also important for understanding diseases of iron overload, such as hereditary hemochromatosis, and iron deficiency, such as iron deficiency anemia.

Absorption of Iron and factors regulating absorption: Normally, the loss of iron from the body of a man is limited to 1 mg per day. Menstruating women lose iron with menstrual blood. Around 10 to 20 mg of Fe is taken in the diet and only about 10 per cent is absorbed. The greatest need of iron is during infancy and adolescence. The only mechanism by which total body stores ofiron is regulated is at the level of absorption. Garnickproposed a “mucosal block theory” for iron absorption.

Iron Transport and Utilisation: Transport of Fe throughout the body is accomplished with a specific protein calledtrans ferrin. Transferrin transports Fe from the GI tract to the bonemarrow for Hbsynthesis and to all other cells as required. Transferrin can transport a maximum of two atoms ofiron as Fe3+ per molecule. Normally, in plasma/serumtransferrin is about 33 per cent saturated with Fe.cell surface specific receptors are available for the iron-transferrin complex. Tissues havinghigh uptake, have a larger number of receptors present, eg. liver,. The number of receptors decreases when a personis replete with iron and increases with depletion. Iron is transported to bone marrow where it is required for Hbsynthesis. Fe2+ is incorporated in protoporphyrin IX withthe help of the enzyme “ferrochelatase”. Iron is also transported into cells where it is used for both oxidative phosphorylation and as an enzyme cofactor.

Ferritin: Ferritin is a universal intracellular protein that stores iron and releases it in a controlled fashion. The protein is produced by almost all living organisms, including algae, bacteria, higher plants, and animals. Ferritin in storage form of Fe occurs in reticuloendothelial system (RES), viz. liver, spleen and bone marrow and also in intestinal mucosal cells. In humans, it acts as a buffer against iron deficiency and iron overload (Lebovitz, 2001). It is found in most tissues as a cytosolic protein, but small amounts are secreted into the serum where it functions as an iron carrier. Plasma ferritin is also an indirect marker of the total amount of iron stored in the body, hence serum ferritin is used as a diagnostic test for iron-deficiency anemia (Owen, 2001). Ferritin is the primary intracellular iron-storage protein in both prokaryotes and eukaryotes, keeping iron in a soluble and non-toxic form. Free iron is toxic and catalyses the conversion of O–2 tohydroxy OH– oxy radicals. Iron bound to ferritin is nontoxic.It is the storage protein of iron and found in blood, liver, spleen, bone marrow and intestine (mucosal cells).

Ferritin and Type-2 Diabetes Mellitus

Ferritin is a complex globular protein that stores iron as soluble and non-toxic component. In oxidative stress, Fe^{2+} enters to cells and then changes to Fe^{3+}, linked to ferritin and then protect cells from oxidative stress (Gu, 2003). Increasing concentration of iron and ferritin in cells could cause resistance to insulin and dysfunction of β cells of pancreases. Hyperinsulinemia due to resistance to insulin may be responsible for increasing serum ferritin. It has been suggested that disturbance of iron metabolism could cause insulin resistance, hyperinsulinemia, dyslipidemia, HTN and central obesity (Farnkvist, 2003 and Wild, 2004). Type 2 Diabetes mellitus is characterized by impaired insulin secretion, insulin resistance, excessive hepatic glucose production, and abnormal

fat metabolism. Although the exact mechanism of iron-induced diabetes is uncertain, it is likely to be mediated by three key mechanisms: 1) insulin deficiency, 2) insulin resistance, and 3) hepatic dysfunction. An understanding of the pathogenic pathways of iron-induced diabetes is derived mainly from studies on animal models of hemochromatosis. The crucial role of iron in the pathophysiology of disease is derived from the easiness with which iron is reversibly oxidized and reduced. This property, while essential for its metabolic functions, makes ironpotentially hazardous because of its ability to participate in the generation of powerful oxidant species such as hydroxyl radical. The catalytic iron converts poorly reactive free radicals like hydrogen peroxide (H_2O_2) into highly reactive ones such as hydroxyl radical and superoxide anion that can initiate and propagate the cascades leading to oxidative damage. These free radicals arepowerful pro-oxidants which cause lysis of the lipid cellular membrane, damage the configurationalharmony of proteins, and displace nucleic acids in genes (InternationalDiabetesFederation, 2005; Amos, 2010 and King, 1995). Thus, the catalytic action of freeiron is instrumental to insulin resistance in the beginning and later on to reduced insulin release,which subsequently results in the development of T2DM (Harris, 1988; TheDiabetesEpidemiology, 1998; Sicree, 2003; Gu, 2003 and Farnkvist, 2003). Emerging scientific evidence hasdisclosed that the relationship is bidirectional, wherein glucose metabolism also encroaches upon diverse iron-related pathways (Wild, 2007). Oxidative stress-triggered inflammatory cytokines influencesuch alliances, replicating and reinforcing the initiated phenomenon (Wallum, 1992). Long-standing diabeticco-morbidities are also moderated by iron-mediated deterioration (Lefebvre, 1995). Elevated iron stores could enhance oxidation of lipids, especially of free fatty acids; through accelerated production of free radicals. The complex process of advanced glycation end product formation produces reactive oxygen species (ROS) by metal catalyzed reactions. Advanced glycation end products themselves bind transition metals, potentiating their toxic effects, including insulin resistance. ROS interfere with insulin signaling at various levels, impairing insulin uptake through a direct effect on insulin receptor functions and inhibiting the translocation by GLUT4 in the plasma membrane. Iron through Fenton's reaction participates in the formation of highly toxic free radicals such as hydroxide andformation of highly toxic free radicals such as hydroxide and the superoxide anion that are capable of inducing lipid peroxidation. Hydroperoxides react with transition metals to form stable aldehydes, such as malondialdehyde (MDA). ROS can stimulate vascular smooth muscle cell growth and proto-oncogeneexpression. In patients with type 2 diabetes mellitus, higher levels of MDA, a marker of lipid peroxidation is found. Oxidative stress induces both insulin resistance by decreasing internalization of insulin and increased ferritin synthesis.

MATERIALS AND METHODS

Reagent kits

Ferritin : CLIA Method
Glucose : GOD-POD Method
HbA1c : Nephelometry Methodology
Creatinine : Modified Jaffe's Method
Cholesterol : CHOD-POD Method

The study was completed in central diagnostic biochemistry laboratory, MCH, Thiruvananthapuram, department of MLT Medical College Thiruvananthapuram. Study was conducted on samples collected from Internal Medicine OP and processed at Central diagnostic biochemistry laboratory, MCH.

Study period

A period of 6 months after approval of ethics committee

Study population

Case: The study population includes patients with Type-Diabetes mellitus, age ranges from 40-60, whose blood samples are sent to the Central diagnostic biochemistry laboratory, Medical college Hospital, Thiruvanathapuram.

Control: Non Diabetic patients in the similar age group.

Exclusion criteria

- Anemia
- Liver diseases
- Glucocorticoid administration
- Pregnancy

Study design: Cross sectional study with case control comparison.

Study setting

- Internal Medicine OP, Medical College Hospital,l, Thiruvananthapuram.
- Central Diagnostic Biochemistry Laboratory, Medical College Hospital, Thiruvananthapuram.

Sample size calculation: Formula for calculating Sample size.

$$\text{Sample size } N = \frac{2\left(z_{1-\frac{\alpha}{2}} + z_{1-\beta}\right)^2 s^2}{\delta^2}$$

$$S^2 = \frac{s_1^2 + s_2^2}{2}$$

S_1= standard deviation in the first group
S_2= standard deviation in the second group
δ =Mean difference between the groups
α= Significance level
1-β= Power
S_1= 53.46
S_2= 54.9
δ =32.4
α= 5%
1-β= 80%

$$S^2 = \frac{s_1^2 + s_2^2}{2}$$

δ= m_1 - m_2
Type I error (α) = 5%
Type II error (β) = 20%
Power = 1- β = 80%

$$\left(z_{\propto} + z_{1-\beta}\right)^2 = 7.849$$

Sample size N= $$\frac{2\left(z_{1-\frac{\propto}{2}}+z_{1-\beta}\right)^2 s^2}{\delta^2}$$

Sample required for the study N= 45 in each group

Sample collection and storage: After getting written consent, blood samples for estimation of different parameters were collected. Serum samples were used to estimate ferritin, FBS, PPBS, creatinine, cholesterol and EDTA blood sample were used for HbA1c.

Materials needed for the study: Reagents were stored in refrigerator. Estimations were done by standard techniques in fully automated Biochemistry analyzer EM-360,

Estimation of Serum Ferritin

Method: CLIA.

Principle: The Access Ferritin assay is a two-site immune enzymztic (Sandwich) assay. A sample is added to a reaction vessel with goat-anti-ferritin-alkaline phosphatase conjugate, and paramagnetic particles coated with goat anti-mouse: mouse anti-ferritin complexes. Serum or plasma (heparin) ferritin binds to the immobilized monoclonal anti-ferritin on the solid phase, while the goat anti-ferritin enzyme conjugate reacts with different antigenic sites on the ferritin molecules. After incubation in a reaction well , materials bound to the solid phase are held in a magnetic field while unbound materials are washed away. Then, the chemiluminescent substrate Lumi-Phos 530 is added to the vessel and light generated by the reaction is measured with a luminometer. The light production is directly proportional to the concentration of ferritin in the sample. The amount of analyte in the sample is determined from a stored, multi-point calibration curve.

Estimation of Glucose

Method: GOD-POD method

Calculation

$$\text{Conc. of glucose} = \frac{\text{Absorbance of test}}{\text{Absorbance of std}} \times 100$$

ESTIMATION OF HbA1c

Method: Nephelometry methodology

ESTIMATION OF CHOLESTEROL

Method: CHOD/POD Method

Calculation: Cholesterol concentration (mg/dl) = OD sample/ OD Std x 200. Linearity will get upto cholesterol concentration 700 mg/dl. If there concentrations is above it dilute with 0.9 % normal saline and multiply the result with dilution factor.

Estimation of Serum Creatinine

Method: Modified Jaffe's method

Calculation:

$$\text{Creatinine conc.(mg/dl)} = \frac{\text{(T2-T1) of sample}}{\text{(T2-T1) of std}} \times 2$$

OBSERVATION AND RESULTS

The Study population consists of 45 cases and 45 control, in the age group of 40-60 attending Inernal medicine OP, Govt. Medical College Trivandrum.

Table 1. Distribution according to gender

Gender	Category				Total	
	Case		Control			
	N	%	N	%	N	%
Male	20	44.4	20	44.4	40	44.4
Female	25	55.6	25	55.6	50	55.6
Total	45	100.0	45	100.0	90	100.0

Table 2. Distribution According to Age

Age	Category				Total	
	Case		Control			
	N	%	N	%	N	%
40-45	4	8.9	4	8.9	8	8.9
46-50	7	15.6	10	22.2	17	18.9
51-55	14	31.1	11	24.4	25	27.8
56-60	20	44.4	20	44.4	40	44.4
Total	45	100.0	45	100.0	90	100.0

Table 3. Mean and SD of FBS among study population

	N	FBS(mg/dl)		t	p
		Mean	sd		
Case	45	162.56	25.14	18.360	<0.001
Control	45	90.51	7.80		

Table 4. Mean and SD of PPBS among study population

	N	PPBS(mg/dl)		t	p
		Mean	sd		
Case	45	250.78	64.48	12.365	<0.001
Control	45	130.56	9.83		

Table 5. Mean and SD of HBA1C among study population

	N	HbA1C		t	p
		Mean	sd		
Case	45	9.13	1.60	18.532	<0.001
Control	45	4.59	0.39		

Table 6. Mean and SD of FERRITIN among study population

	N	FERRITIN (ng/dl)		t	p
		Mean	sd		
Case	45	197.36	113.45	8.690	<0.001
Control	45	48.87	16.36		

Table 7. Mean and SD of total cholesterol among study population

	N	Total.cholesterol(mg/dl)		t	p
		Mean	sd		
Case	45	210.20	40.25	7.716	<0.001
Control	45	159.38	18.23		

Table 8. Mean and Sd of Creatinine among study population

	N	Creatinine(mg/dl)		t	p
		Mean	sd		
Case	45	1.11	0.36	4.624	. <0.001
Control	45	0.83	0.18		

Table 9. Distribution according to FBS- control

FBS (Control)	Frequency	Percentage
<90	22	48.9
90-100	19	42.2
101-150	4	8.9
Total	45	100

Table 10. Distribution according to FBS- case

FBS (cases)	Frequency	Percentage
100-150	17	37.8
151-200	27	60
>200	1	2.2
Total	45	100

Table 11. Distribution according to PPBS- case

PPBS (cases)	Frequency	Percentage
<200	10	22.2
201-300	28	62.2
301-400	4	8.9
>400	3	6.7
Total	45	100

Table 12. Distribution according to PPBS- control

PPBS(Control)	Frequency	Percentage
100-140	37	82.2
141-200	8	17.8
Total	45	100

Table 13. Distribution according to HbA1c- case

Criteria	HbA1c (%)	Case		Control		Total	
		N	%	N	%	N	%
Normal	4.2-6.2	0	0.0	45	100.0	45	50.0
Diabetic , good control	6.3-6.8	1	2.2	0	0.0	1	1.1
Action suggested	6.9-7.6	6	13.3	0	0.0	6	6.7
Poor Control	>7.6	38	84.4	0	0.0	38	42.2
Total		45	100.0	45	100.0	90	100.0

Table 14. Distribution according to FERRITIN- case

Ferritin	Frequency	Percentage
<100	2	4.4
101-200	27	60.0
201-300	12	26.7
301-400	1	2.2
>500	3	6.7
Total	45	100.0

Table 15. Distribution according to ferritin-control

Ferritin (Control)	Frequency	Percentage
20-40	15	33.3
41-60	18	40.0
61-80	11	24.4
>80	1	2.2
Total	45	100.0

Table 16. Distribution according to cholesterol-case

Total cholesterol (Cases)	Count	within Category
<200	23	51.1
201-300	18	40
>300	4	8.9
Total	45	100

Table 17. Distribution according to cholesterol-control

Total cholesterol (Control)	Count	within Category
<150	12	26.7
150-180	28	62.2
>180	5	11.1
Total	45	100

FERRITIN	Pearson Correlation r	p
HbA1c	.916**	<0.001
FBS	.569**	.000
PPBS	.885**	.000
Creatinine	.830**	.000
Cholesterol	.856**	.000
Duration of DM(years)	.975**	.000

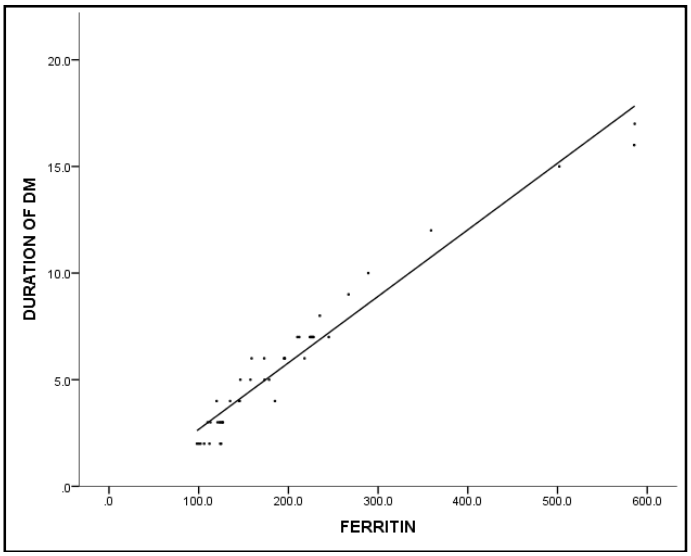
Table 18. Distribution according to creatinine- case and control group

Creatinine	Category				Total	
	Case		Control			
	N	%	N	%	N	%
<0.7	1	2.2	17	37.8	18	20
0.7-1.4	41	91.1	28	62.2	69	76.7
>1.4	3	6.7	0	0	3	3.3
Total	45	100	45	100	90	100

Table 19. Correlation chart

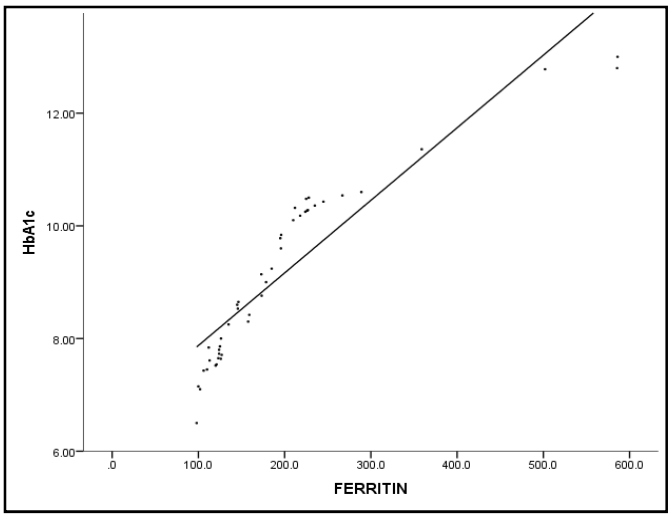
FERRITIN	Pearson Correlation r	p
HbA1c	.916**	<0.001
FBS	.569**	.000
PPBS	.885**	.000
Creatinine	.830**	.000
Cholesterol	.856**	.000
Duration of DM(years)	.975**	.000

Correlation of Ferritin with duration of DM



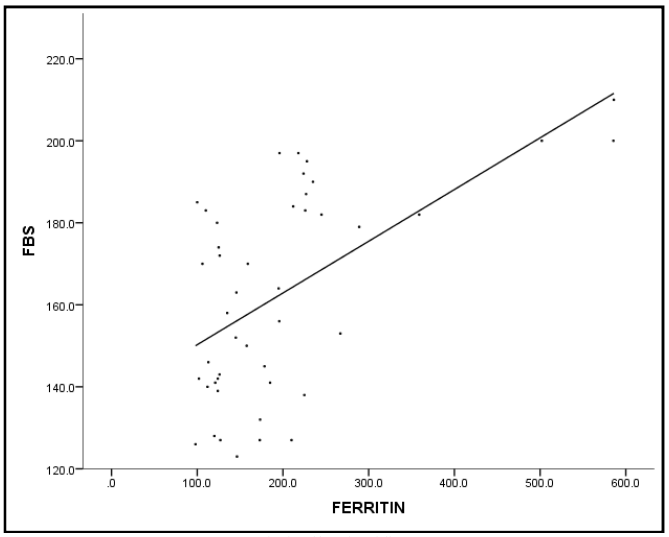
For cases, r =.975, p= .000,statistically significant

Correlation of Ferritin with HbA1c



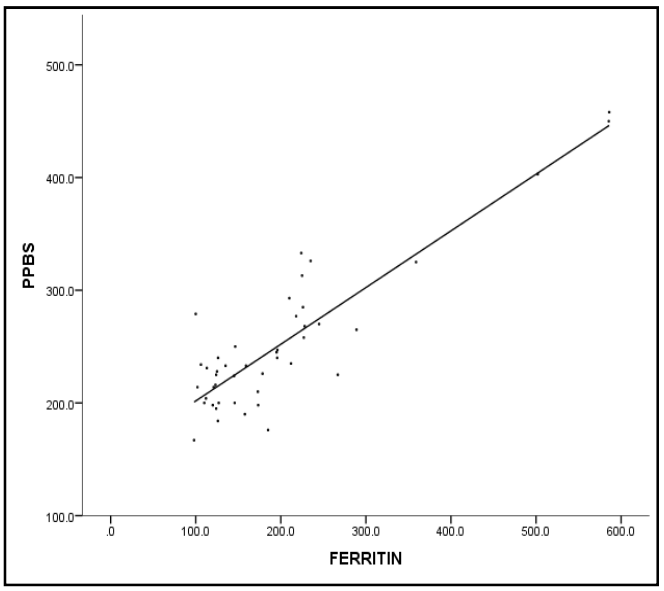
For cases, r =.916, p= <0.001, statistically significant.

Correlation of Ferritin with FBS



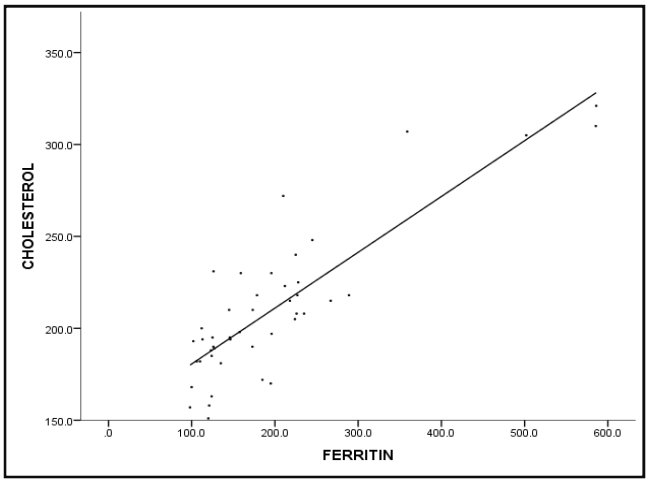
For cases, r =.569, p= .000,statistically significant.

Correlation of Ferritin with PPBS



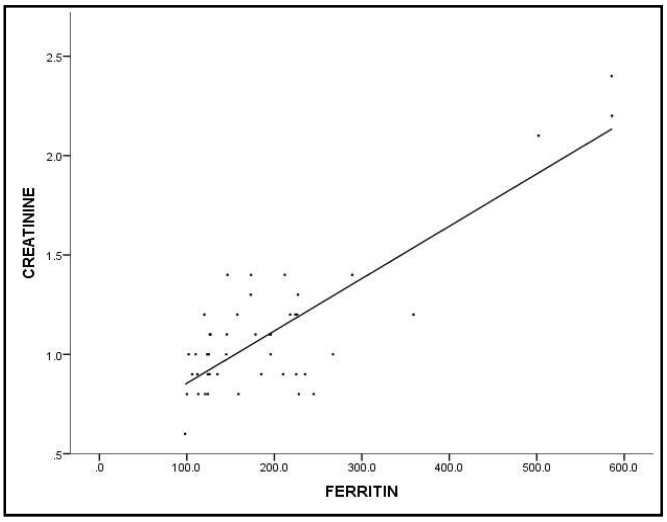
For cases, r =.885, p= .000,statistically significant.

Correlation of Ferritin with Cholesterol



For cases, r =.856, p= .000,statistically significant.

Correlation of Ferritin with Creatinine



For cases, r =.830, p= .000,statistically significant.

DISCUSSION

The study titled “Association between serum ferritin and Type-2 Diabetes mellitus” was conducted in Internal Medicine OP, MCH, Thiruvananthapuram. This study was completed in Central diagnostic biochemistry laboratory, MCH, Thiruvananthapuram. The study population included 45 cases and 45 controls. Cases were selected based on the inclusion-exclusion criteria. Age and sex matched healthy controls were taken as control. Blood samples were collected after getting written patient consent, for assessing the biochemical parameters such as ferritin, plasmablood glucose, HbA1c, cholesterol and creatinine. In the present study 45 diabetic cases are included and the majority of the patients were between the age group >55(44.1%) followed by the age group 51-55(31.1%). In the control group 44.1% population having age >55. The mean age of diabetic patients were 53.89 and in the control group were 53.69.This is similar to the study by Meghna Borah, were the mean age of the patients with diabetic was 53.72. In the present study the mean FBS was 162.36±25.14mg/dl in diabetic casegroup and 90.51±7.80mg/dl in the control group. Sangappa Virupaxappakashinakunti et al found that FBS was 202.39±89.786mg/dl and 90.85±9.608mg/dl, in case and control group respectively.

In this study, mean HbA1c level in case group was 9.13± 1.60and that in control group was 4.59±0.39 this is similar to the study by Anjana v et al, was found HbA1c level in case group was 9.10% and 5.39% in control group. In the current study shows mean serum ferritin level was 197.36±113.45ng/ml in diabetic group and 48.87±16.36ng/ ml in the control group, the study by Pratik H. Raghavan et al found thatmean serum ferritin level in the case group was 192±32 ng/ml and in control group 65±14 ng/ml. In the present study mean value of PPBS in case group was 250.78±64.48 mg/dl and 130.56±9.83mg/dl in the control group, whereas a study by Amitkumar Virji Maheswari et al found that PPBS level 260±95.37 mg/dl in the diabetic group and 105.5±9.83mg/dl in the control group. In this study serum total cholesterol was found as 210.±40.25mg/dl in individuals with diabetic and 159 ±18.23mg/dl, study by Pratik H. Raghavan et al found that 232±40mg/dl and186±28mg/dl, in case and control group respectively. Mean Serum creatinine values are 1.11±0.36mg/dl in patients with diabetic and 0.83±0.18mg/ dl in control group, a stuby done by Meghna Borah et al shows that mean serum creatinine level in diabetic group was 1.01±0.19mg/dl and in control group, 0.98±0.17mg/dl. In a 45 cases of diabetic patients group, mean serum ferritin value was 197.36±113.45ng/ml and in the control group it was 48.87±16.36.ng/ml. Asimiliar study by Padmaja et al shows a mean serum ferritin value of 143.34 ±59.23ng/ml in diabetic case group and that of control was 63.87±27.33 ng/ml. Another study by Sumeetsmotra et al shows average ferritin values of 226.70 ±53.46ng/ml and 174± 54.90ng/ml, in case and control group respectively.

Summary

Type-2 diabetes mellitus is an endocrinological disease associated with hyperglycemia characterized by both insulin resistance and defective insulin secretion. Ferritin is a complex globular protein that stores iron as a soluble and non-toxic component. Increased concentration of iron and ferritin in cells could cause resistance to insulin and dysfunction of β cells of pancreas. The present study was conducted for six months period in central diagnostic biochemistry laboratory, Medical college hospital, Thiruvananthapuram. The study was mainly carried out in serum and EDTA blood samples which are collected from diabetic patients ,attended in Internal Medicine OP. Cases having anemia ,liver disease, glucocorticoid administration and pregnancy were excluded from the study.

Total of 45 cases and 45 controls were included in this study. Their age, gender, duration of diabetes, other associated diseases were assessed. Biochemical parameters like, FBS, PPBS, HbA1c, serum ferritin, Total cholesterol and creatinine were carried out. For the measurement of FBS and PPBS, fluoride bottles were used. EDTA samples were used for HbA1c measurement using Nephelometry method. Serum ferritin is estimated by CLIA. In diabetic case group and control group 25 patients were females and 20 were males. In the present study the average value of FBS for cases was 162.56±25.14 mg/dl and for controls 90.51±7.80 mg/dl. The PPBS mean value of diabetic cases was 250.78± 64.48mg/dl and that of control was 130.56±9.83mg/dl. The mean HbA1c levels among diabetic cases were 9.13± 1.60 and that in control group was 4.59±0.39. HbA1c levels >7.6 was reported in 84.4% of the diabetic cases and 13.3% have HbA1c level in the range of 6.9-7.6 .The average value of serum creatinine in diabetic group was 1.11± 0.36mg/dl and that of control was 0.83± 0.18mg/dl. Serum cholesterol have a mean value of 210± 64.48mg/dl in case group and that of control was 159.38±18.23mg/dl. The current study shows mean serum ferritin level was 197.36±113.45ng/ml in diabetic group and that of control was 48.87±16.36 ng/ml. Serum ferritin levels shows a good positive correlation with HbA1c with r=0.916 and p value <0.001. The correlative studies of ferritin with duration of diabetes shows a positive correlation with r=0.975 and p=0.000.When considering the correlation of ferritin with FBS and PPBS, shows r=0.569,p=0.000 and r=0.885,p=0.000, respectively. Ferritin have a positive correlation with creatinine, r=0.830,p=0.000 and also with cholesterol, r=0.856,p=0.000.The present findings demonstrate the imbalance in levels of serum ferritin levels among patients with Type 2 Diabetes in comparison to controls. These changes may play an important role in the pathogenesis of Type 2 DM by the involvement of oxidative damages to the pancreatic beta cells.

Conclusion

- The average value of serum ferritin in diabetic patients was 197.36±113.45ng/ml whereas in control group it was 48.87±16.36ng/ml
- The mean value of HbA1c levels in cases was 9.13±1.60% and that of control was 4.59±0.39%
- The serum ferritin level showed a positive correlation with duration of diabetes mellitus.(r=0.975,p=0.000)
- The serum ferritin level have a significant positive correlation with HbA1c levels(.(r=0.916,p=0.000)
- The study showed a positive correlation of serum ferritin levels with FBS and PPBS, with r=0.569,p=0.000 in FBS and r=0.885,p=0.000 in PPBS.
- When correlating serum ferritin levels with total cholesterol and creatinine,shows a positive correlation with r=0.856,p=0.000 in total cholesterol and r=0.830,p=0.000 in creatinine.
- Serum ferritin level in Type 2 diabetics is significantly elevated than non-diabetics.
- Ferritin may be used as one of the important biomarkers in predicting diabetes mellitus.

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