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Original Article

Biochemistry Section

Correlation of Serum Fetuin A with hs-CRP Levels and Lipid Profile in Assessment of Cardiovascular Risk in Hypothyroid Pregnant Females: A Cross-sectional Study

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ABSTRACT

Introduction: Pregnancy is associated with multiple metabolic and hormonal alterations. In India, hypothyroidism is substantially prevalent endocrinal disorder seen in pregnant women. Fetuin A is a carrier plasma glycoprotein synthesised by the liver and its reduced levels result in increased risk of peripheral arterial disease, Coronary Artery Disease (CAD) in patients with type 2 diabetes mellitus and in dialysis patients. Reduced fetuin A levels have been seen in hypothyroid females. However higher levels of proinflammatory marker high sensitivity C-Reactive Protein (hs-CRP) have been associated with hypothyroidism and also with cardiovascular disease risk.

Aim: To analyse and correlate serum Thyroid Stimulating Hormone (TSH), free triiodothyronine (fT3), free thyroxine (fT4) and Anti-Thyroid Peroxidase antibodies (Anti TPO antibody), lipid profile, hs-CRP and fetuin A level in pregnant females.

Materials and Methods: This cross-sectional study was conducted in Kalpana Chawla Government Medical College, Karnal, Haryana, India, from September 2022 to December 2023. The study included 160 pregnant females without any chronic disease who were investigated for TSH, fT3, fT4 and Anti TPO antibody, Lipid profile, fetuin A and hs-CRP. Statistical analysis was done using t-test, Mann-Whitney U test and Pearson correlation coefficient with p-value <0.05 as significant and p-value <0.001 as highly significant.

Results: The median gestational age was 22 weeks (with IQR 11.50-32.0) in cases and 16 weeks (IQR 11.50-27.0) in controls. Fetuin A levels were lower in case group with median value of 4.98 ng/mL (IQR 2.28-10.35) versus control group with median value 5.44 ng/mL (IQR 2.75-12.24). hs-CRP levels were also higher in pregnant hypothyroid females (case group) with median value 2.89 mg/dL (IQR 1.74-6.10) as compared to controls (median 2.32 IQR 1.74-6.46).

Conclusion: There was no significant correlation of fetuin A with hs-CRP, but triglycerides, Very Low Density Lipoprotein (VLDL) and Atherogenic Index of Plasma (AIP) showed a statistically significant negative correlation with fetuin A.

Keywords: Coronary artery disease, Gestation, High sensitivity C-reactive protein, Inflammatory marker, Thyroid dysfunction

INTRODUCTION

Hypothyroidism is extensively prevalent endocrinal disorder in pregnant women in India [1]. However, its early diagnosis and consequent therapeutic intervention has not kept pace with the magnitude of the disease burden The socio-economic implications of hypothyroidism include its association with accelerated atherosclerosis, increased risk of CAD and low serum T3 levels have also been inversely correlated with calcification of coronary artery [1]. Fetuin A is a heterodimeric plasma glycoprotein which is predominantly expressed in embryonic cells and adult hepatocytes, and to a lesser extent in adipocytes and monocytes. It is involved in the regulation of calcium metabolism, osteogenesis, the insulin signaling pathway and also acts as an ectopic calcification inhibitor, protease inhibitor, inflammatory mediator, atherogenic factor, and adipogenic factor [2].

Various studies have demonstrated an inverse association of plasma fetuin A levels with severity of arterial calcification, presence of CAD and mortality associated with cardiovascular disease [3,4]. Fetuin A has also been shown to promote lipid-induced insulin resistance by the enhancement of free fatty acids binding to Toll-Like Receptor 4 (TLR4). In adipose tissue, fetuin A has been shown to downregulate the expression of adiponectin, thereby suppressing its anti-inflammatory and insulin-sensitising properties [5]. An in-vitro experiment had demonstrated the enhancing impact of T3 on the expression of liver cell line fetuin A. The molecular mechanism involves T3 binding to thyroid receptor 1, which then modulates the transcription of numerous proteins, including fetuin A [6]. Thyroid hormones collaborate with fetuin A to prevent calcification of soft-tissue, as T3, at physiological concentrations, has been shown to enhance the expression of Matrix Gla Protein (MGP), a potent inhibitor of calcification in Vascular Smooth Muscle Cells (VSMC), which prevents vascular calcification, via thyroid hormone nuclear receptors. Low thyroxine (T4) level was associated with a high progression risk for coronary artery calcification [3].

The hs-CRP is an acute phase reactant that represents a marker of subclinical inflammation. Corona G et al., found significantly higher levels of hs-CRP in patients with hypothyroidism as compared to euthyroid controls [7]. There is a lacunae in knowledge with regard to hs-CRP assessment during pregnancy with hypothrodism. Pregnancy, by itself, is a low-grade inflammatory state and pregnant hypothyroid patients show several signs and symptoms suggestive of inflammation. This interaction may result from the elevated CRP in hypothyroidism [8]. TSH induces synthesis of TNF-α and signs and symptoms of hypothyroidism are thought to be the result of an interaction of IL-6 with TNF-α and IL-1. In addition, the lack of thyroid hormones leads to slowing down the overall metabolic rate, reduced rate of CRP clearance and CRP uptake in target cells may result in increased levels of CRP [9,10]. Ridker PM et al., and

Koyyada A and Orsu P, have concluded that raised hs-CRP is a marker of an enhanced inflammatory reaction and is associated with the increased incidence of CAD [11,12]. However, studies regarding the association between fetuin-A levels and hypothyroidism in pregnant females are scarce.

The present study was conducted to evaluate the interrelationship between fetuin A with thyroid profile, lipid profile and hs-CRP level among pregnant hypothyroid females.

MATERIALS AND METHODS

This cross-sectional study was conducted at Kalpana Chawla Government Medical College, Karnal, Haryana, India, from September 2022 to December 2023. The study was commenced after ethical approval from the Institutional Ethical Committee (IEC approval no: KCGMC/IEC/2022/39).

Inclusion criteria: In this study, 160 pregnant women aged 20 to 40 years, visiting for their first antenatal check-up to the obstetrics OPD within the study duration, were included by convenient sampling after informed consent.

Exclusion criteria: Non pregnant women, pregnant women with pre-existing thyroid disorders, patients with liver and kidney disorders, patients with diabetes mellitus were excluded from this study.

Data collection: The diagnosis of hypothyroidism was based on history and thyroid profile analysis. On the basis of thyroid profile results, the subjects were divided into two groups:

1. Group 1 consisting of 80 pregnant women, in age group 20-40 years, diagnosed with hypothyroidism during pregnancy.
2. Group 2 consisting of 80 gestational age matched euthyroid pregnant women in the age group of 20-40 years.

Study Procedure

Thyroid profile including TSH with cut-off during pregnancy ≥2.5 µIU/mL [13], fT3 with reference range for adult 1.4-4.2 pg/mL [14], fT4 with reference range during pregnancy 0.76- 2.24 ng/dL [15], Anti TPO positive at cutoff >55 IU/mL [16] and fetuin A with normal average value 234.5 ng/mL in non pregnant adult [17], were done by Enzyme Linked Immuno Sorbent Assay (ELISA) on Transasia Elisa reader, lipid profile and hs-CRP with cutoff value >5 mg/dL [18] were done on Roche Cobas 501 autoanalyser. Patient samples were stored at -80°C and batch analysed by ELISA.

STATISTICAL ANALYSIS

The data collected was analysed using Statistical Package for the Social Sciences (SPSS), Analysis of Variance (ANOVA), International Business Machines (IBM) software version 20.0. Numerical data were summarised as mean and SD (median and interquartile range, IQR when necessary). Kolmogorov-Smirnov test was used to test whether numerical data conformed to normal distribution. Intergroup comparison was carried out with Independent groups t-test when the assumptions were met, and with the Mann-Whitney U test when assumptions were not met. The correlation between fetuin A with TSH, fT3, fT4, anti-TPO, fT3:fT4 ratio, AIP were analysed with the Pearson correlation coefficient.

RESULTS

The study included 80 pregnant females with hypothyroidism which constituted the case group and another 80 healthy euthyroid pregnant females were included in the control group. In present study, majority of the subjects were aged between 21-30 years in both the groups. There was no significant association between case and control group with regards to age, weight, height and Body Mass Index (BMI) [Table/Fig-1].

Parameter	Controls (n=80) n (%)	Cases (n=80) n (%)	p-value
Age (years)			
18-20	11 (13.8)	5 (6.3)	0.087
21-25	40 (50.0)	38 (47.5)	
26-30	22 (27.5)	20 (25.0)	
>31	7 (8.8)	17 (21.3)	
Weight (kg)	56.61±9.76	57.78±9.78	0.450
Height (m)	1.58±0.08	1.59±0.08	0.769
BMI (kg/m^2)	22.65±3.67	23.03±3.81	0.528

[Table/Fig-1]: Comparison of demographic and clinical characteristics of case and control groups.
** p-value <0.05 is statistically significant and p-value <0.001 is highly significant

There was highly significant difference between the median values of TSH between the case and control group (p-value=0.001). The median values of fT3 also showed statistically significant difference between case and control group (p-value=0.021). Also, there was statistically significant difference between the median values of serum cholesterol between case and control group (p-value=0.010). Fetuin A levels were found to be lower in case group than in control group. Median serum fetuin A levels in case group were 4.98 (2.28-10.35) ng/mL compared to 5.44 (2.75-12.24) ng/mL in control group not statistically significant (p-value=0.662) [Table/Fig-2].

However, a statistically significant negative correlation was identified between fetuin A and gestational age with p-value 0.043 (r-value=-

Variables	Cases			Controls			p-value
	Median	Quartile- I	Quartile- III	Median	Quartile- I	Quartile-III	
Gestational age (years)	22.00	11.50	32.00	16.00	11.50	27.00	0.118
Systolic BP (mmHg)	120.0	118.00	122.00	120.00	116.50	120.00	0.12
Diastolic BP (mmHg)	80.00	78.00	80.00	80.00	74.00	80.00	0.564
Fetuin A	4.98	2.28	10.35	5.44	2.75	12.24	0.662
hs-CRP (mg/dL)	2.89	1.74	6.10	2.32	1.07	6.46	0.236
TSH (µIU/mL)	2.90	2.70	3.45	0.60	0.30	1.00	0.0001**
Ft3 (pg/dL)	3.60	2.65	4.00	3.70	3.10	4.65	0.021*
Ft4 (ng/dL)	0.80	0.50	0.90	0.80	0.70	0.90	0.163
Ft3/Ft4	4.19	3.10	6.59	4.71	3.53	6.86	0.487
Anti-TPO (IU/mL)	0.09	0.02	0.31	0.12	0.02	0.26	0.597
Cholesterol (mg/dL)	204.0	170.50	254.50	173.50	149.50	213.50	0.010**
Triglyceride (mg/dL)	178.50	122.50	240.0	163.50	110.00	222.50	0.374
HDL (mg/dL)	54.00	45.50	66.50	55.00	46.50	61.00	0.152
LDL (mg/dL)	114.50	83.00	154.00	100.50	79.50	132.00	0.085

VLDL (mg/dL)	36.0	25.50	48.00	32.50	22.00	44.00	0.519
AIP (LnTG/HDL-C)	1.16	0.76	1.41	1.09	0.70	1.42	0.88

[Table/Fig-2]: Comparison of median values of case and control group.
*p-value <0.05 significant, **p-value <0.001 highly significant; BP: Blood pressure; hS-CRP: High sensitivity C reactive protein; TSH: Thyroid stimulating hormone; HDL: High density lipoprotein; LDL: Low density lipoprotein; VLDL: Very low density lipoprotein; AIP: Atherogenic index of plasma

0.227). Similarly statistically significant negative correlation was identified between fetuin A and fT3 (p-value=0.048, r-value=-0.222), and fetuin A and fT3/fT4 ratio (p-value=0.022, r-value=-0.257). There was no significant correlation between fetuin A and hs-CRP (p-value=0.882, r-value=0.017) [Table/Fig-3].

Variables	Correlation coefficient	p-value
Age	0.081	0.476
Gestational age	-0.227*	0.043
Weight	-0.057	0.615
Height	-0.153	0.175
BMI	-0.014	0.902
Systolic BP	-0.052	0.644
Diastolic BP	-0.088	0.439
Hs-CRP	0.017	0.882
TSH	-0.141	0.212
Ft3	-0.222*	0.048
Ft4	0.119	0.291
Ft3/Ft4	-0.257*	0.022
Anti-TPO	0.037	0.744

[Table/Fig-3]: Correlation between serum fetuin A levels and baseline characteristics in pregnant females with hypothyroidism.
*Correlation is significant at the 0.05 level (2-tailed)

Among cardiovascular risk factors, statistically significant negative correlation was established between fetuin A and triglyceride, VLDL, AIP {Ln(TG/HDL)} with p-value 0.029 (r-value=-0.244), 0.021 (r-value=-0.257), 0.046 (r-value=-0.223) [Table/Fig-4].

Parameter	Correlation coefficient	p-value
Cholesterol	-0.102	0.366
Triglyceride	-0.244*	0.029
HDL	-0.053	0.642
LDL	-0.055	0.628
VLDL	-0.257*	0.021
Ln (TG/HDL)	-0.223*	0.046

[Table/Fig-4]: Correlation between serum fetuin A levels and cardiovascular risk factors in hypothyroid pregnant females.
*Correlation is significant at the 0.05 level (2-tailed)

DISCUSSION

Pregnancy is accompanied with complex hormonal changes. Physiology of thyroid is modified during pregnancy, which helps prepare the maternal thyroid gland to cope with metabolic demands. During gestation, the increased physiological demands of growing foetus leads to augumenation of thyroid hormone production and consequently, there is 2-3-fold rise in production of hepatic thyroxine (T4)-binding globulin (TBG), which, may alter equilibrium between bound and free T4 (fT4). Free T4 is utilised by the body, so reduced fT4 can increase TSH levels by feedback mechanism which, in turn leads to hypothyroidism. In addition, during pregnancy, placental production of human Chorionic Gonadotropin (hCG) reaches a peak of 50,000-75,000 IU/L at 8-11 weeks. This rise in hCG causes thyroid stimulation by binding to TSH receptors due to structural analogy with TSH; there is a decrease in serum TSH during the first trimester [2].

In the present study, a statistically significant negative correlation was established between serum fetuin A levels and fT3 (p-value=0.048) and between fetuin A and fT3:fT4 ratio (p-value=0.022), thus indicating possible involvement of fetuin A in pathophysiology of hypothyroidism in pregnant women.

In this study, it was found that, fetuin A was lower in pregnant hypothyroid females (median 4.98, range 2.28-10.35 ng/mL) as compared to healthy pregnant females (median 5.44, range 2.75-12.24 ng/mL) but the difference was not statistically significant (p-value=0.662). Fetuin A is a glycoprotein synthesised predominantly in the liver. A study has reported that alteration in thyroid function affects the liver's protein synthesis ability by modulating RNA polymerase activity and that results in decreased protein synthesis in hypothyroidism and increased protein synthesis in hyperthyroidism [10].

Also, the low levels of fetuin A in hypothyroid pregnant females may cause enhanced calcification in arteries and thus could contribute to CAD in future, as thyroid hormones works in conjunction with fetuin A to prevent calcification of soft-tissue. Fetuin A counteracts vascular calcification by directly binding to the VSMCs and thereby limits oxidative stress, inflammation, and vascular damage. It protects against aberrant calcification by stabilising mineral complexes in the calciprotein particles and mediating their transport and clearance [19].

Balamurugan V et al., provided compelling evidence that premenopausal women with hypothyroidism are more predisposed to develop cardiovascular disease than their euthyroid counterparts and advocate that all newly diagnosed female hypothyroid patients need to be screened for markers like homocysteine and atherogenic indices to predict the risk of cardiovascular disease [20].

Hypothyroidism itself predisposes to systolic and diastolic cardiac dysfunction, increased peripheral vascular resistance, endothelial dysfunction, altered coagulopathy, and dyslipidaemia resulting in atherosclerosis. Thyroid hormones can influence homocysteine metabolism by regulating the methylenetetrahydrofolate reductase. They regulate lipogenesis and lipolysis through their receptors (α and β) on the liver and adipose tissue and these functions are deranged during thyroid dysfunction [21].

Dyslipidaemia was a prominent finding in the present study case group with median value of total cholesterol of cases (204 mg/dL, range 170.5 -254.5 mg/dL) being higher as compared to controls (median 173.5 mg/dL, range 149.5-213.5 mg/dL), the difference being statistically significant (p-value=0.010). The median values of triglycerides, LDL, VLDL and AIP were higher in cases as compared to controls but were not statistically significant. Zhou J et al., in their study observed significantly elevated plasma TC and LDL-c levels in pregnant women with hypothyroidism compared to those with pre pregnancy hypothyroidism, with evidence of hepatic TG accumulation [22].

Serum fetuin A levels has a statistically significant negative correlation with cardiovascular risk factors such as triglycerides (p-value=0.029), VLDL (p-value=0.021) and atherogenic index (p-value=0.046) in hypothyroid pregnant females. Patients with hypothyroidism are at increased risk of developing atherosclerosis. This is related to the increase in the concentration of hs-CRP and homo-cysteine in the blood and tissues, which increase expression of adhesion molecules like plasminogen activator inhibitor-1, increase production of oxidised LDL and change in the macrophage engulfing process of oxidised LDL [12]. In a similar study done on hypothyroid females by Albayati AF and Hussein AR, fetuin A was significantly decreased in cases while, hs-CRP, LDL-c and atherogenic index were significantly higher in the same group compared with control [23].

The results of present study depicted the augumentation of risk factors of cardiovascular disease were in hypothyroidism during pregnancy, indicated by, reduced fetuin A, rise in cholesterol, triglyceride, LDL and atherogenic index in the case group. Prasad NS et al., have reported that hypothyroidism is associated with increased total cholesterol, triglycerides, LDL-c and higher AIP levels thus identifying them as at an increased risk of developing cardiovascular disease [24]. Thus the interplay of decreased fetuin A levels, altered thyroid profile, and dyslipidaemia in hypothyroidism during pregnancy might increase the risk of cardiovascular disease in such patients, as evident by this study.

Limitation(s)

In this study, since convenient sampling was done, therefore larger study with proper sample size is more desirable. Moreover, the study was conducted over a short period, making it difficult to assess long-term effects. Samples were collected from only one institution, which may not represent all economical and geographic regions. Further studies with a long-term follow-up involving fetuin A together with lipid profiles and cardiovascular monitoring in patients with thyroid dysfunction during pregnancy will be beneficial in terms of protection against cardiovascular diseases that may occur in the future. Moreover multicentric studies involving larger population are also desirable to confirm the findings of present study.

CONCLUSION(S)

Pregnant women with clinical hypothyroidism during pregnancy are at a greater risk of dyslipidaemia, which can subsequently increase the incidence of post pregnancy complications. The mechanism by which thyroid dysfunction leads to dyslipidaemia during pregnancy, needs to be further inquired into. The significant negative correlation between fetuin A and fT3, fT3/fT4 ratio, triglyceride, VLDL and AIP in the present study points towards a possible role of fetuin A in pathogenesis of cardiovascular disease in hypothyroid pregnant females.

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