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Original Article

# Perspective Study of Elevated First Trimester C-reactive Protein as Predictor of Gestational Diabetes in South Karnataka Population: A Retrospective Study

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## Abstract

**Background:** The C-reactive protein (CRP) derived from the liver is sensitive and systemic biomarker of inflammation and has been associated with increased risk of developing diabetes mellitus in pregnancy.

**Materials and Methods:** Ninety-two pregnant women having gestational diabetes of the first trimester and same number of normal pregnant women of controlled group were studied. Blood sugar (fasting and postprandial) and CRP, body mass index (BMI), age, and period of gestation/weeks of gestation were compared in both groups.

**Results:** The BMI of gestational diabetes group was 26.05 (SD ± 3.40) and 22.80 (SD ± 2.14), *t*-test was 7.75, and *P*-value was highly significant. Laboratory findings, blood glucose (fasting and post-meal), and CRP (mg/l) were higher in gestation diabetes and *P*-values were highly significant ($P < 0.00$). CRP values were more or less constant in blood glucose (fasting and post-meal) hence *P*-value was insignificant ($P > 0.98$).

**Conclusion:** CRP values were higher in gestational diabetes due to inflammation and oxidative stress. These finding are important for obstetrics and gynecologist to treat such patient efficiently to prevent morbidity and mortality of fetus and mother too.

**Key words:** Andhra Pradesh, Blood glucose, C-reactive protein gestational, Type-II diabetes mellitus

## INTRODUCTION

C-reactive protein (CRP) is synthesized by the liver and has been shown to be a sensitive and systemic biomarker of inflammation.[1] It is reported that a number of case–control studies have reported that CRP is associated with increased risk of developing type-II diabetes mellitus (type-II DM).[2] The positive relation in cross-sectional studies or case–control studies could be due to CRP being a consequence of hyperglycemia. Therefore, a prospective study is carried out to ascertain the elevation of to ascertain onset of hyperglycemia in the development of type-2 DM.[3] Hence, attempt is made to study in the first trimester pregnant women; the level of CRP and blood glucose levels is correlated and same parameters are also compared normal (controlled) group. CRP is also synthesized in the adipose tissue and may be present in excessive quantities in patients with abdominal obesity, eventually resulting in insulin resistance and diabetes.[4]

The role of CRP in predicting diabetes in pregnancy is ruled out because, during pregnancy, all endocrine glands are more active than normal life period in females.

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## MATERIALS AND METHODS

Ninety-two patients regularly visiting Obstetrics and Gynaecology Department of Basaveshwara Medical College, Chitradurga-577502, Karnataka, were studied.

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### Inclusion Criteria

Pregnant patients having of gestation and diabetic with above 20 weeks of gestation, multipara were selected. Normal pregnant women above 20 weeks of gestation were studied as control groups.

### Exclusion Criteria

Known diabetics, having history of gestational diabetes mellitus (GDM) in the past pregnancy patients associated with endocrine disease such as thyroid, adrenal, or immune compromised diseases.

### Method

Ninety-two pregnant women having gestational diabetes and same number of normal pregnant women selected as control group. Diagnosis of GDM based on 4th International workshop conference on gestational diabetes which adapts the Carpenter-Coustan criteria.[5] About 8 ml of blood from each patient was collected after an overnight fast (after 12 h) by venipuncture 4 ml is collected in clean plain bulb and remaining in the EDTA and fluoride bulb. Blood was allowed to clot serum which was then separated by centrifugation. Blood sugar was studied by glucose oxidize and peroxidase and point (enzymatic method), and CRP by immune turbidimetric method. The duration of study was October 2018–April 2020.

### Statistical Analysis

The studied parameters in gestational diabetic women and controlled group were compared by *t*-test. The statistical analysis was carried out in SPSS software.

## OBSERVATION AND RESULTS

In Table 1, comparison clinical manifestation (baseline) in GDM patients and controlled group:

1. The age (year) 23.60 (SD ± 2.55) in control group *t*-test was 0.82 and *P*-value was insignificant ($P > 0.07$)
2. Gestation age (weeks) 31.86 (SD ± 2.85) in GDM patients, 31.82 (SD ± 2.82) in controlled group *t*-test was 0.096 and *P*-value was insignificant 0.92
3. Body mass index (BMI) in GDM was 26.05 (SD ± 3.40), 22.80 (SD ± 2.14) in controlled group *t*-test was 14.1 and *P*-value was highly significant ($P < 0.000$).

In Table 2, comparison of laboratory findings:

1. Blood glucose (fasting) 123.25 (SD ± 18.42), 83.60 (SD ± 4.90) in controlled group *t*-test was 19.9 and *P*-value was highly significant ($P < 0.000$)
2. Blood glucose (post meal) 228.46 (SD ± 12), 110.69 (SD ± 6.68) in controlled group *t*-test was 82.2 and *P*-value was highly significant ($P < 0.00$)

**Table 1: Comparison of clinical manifestation (baseline) in both groups**

| Parameter | Gestation DM 92 | Control group 92 | *t*-test | *P*-value |
|---|---|---|---|---|
| Age (year) | 23.60 (SD±2.55) | 23.29 (SD±2.55) | 0.82 | *P*>0.07 |
| Gestational age (weeks) | 31.86 (SD±2.85) | 31.82 (SD±2.62) | 0.096 | *P*>0.92 |
| BMI | 26.05 (SD±3.40) | 22.80 (SD±2.14) | 7.75 | *P*<0.000 |

*P*<0.000 = Highly significant, DM: Diabetes mellitus, BMI: Body mass index

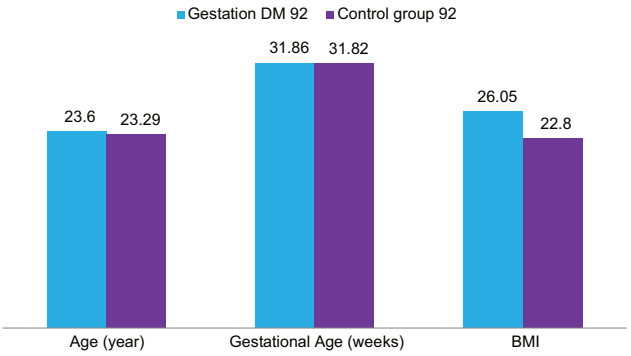
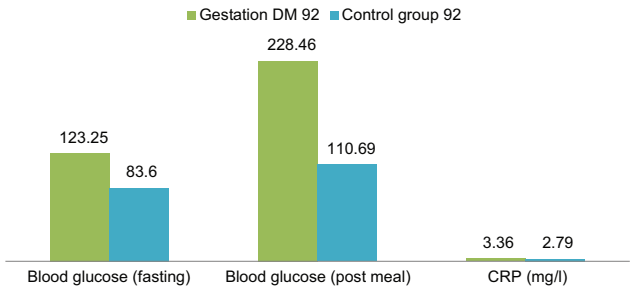



**Table 2: Comparison study of laboratory findings in both groups**

| Parameter | Gestation DM 92 | Control group 92 | *t*-test | *P*-value |
|---|---|---|---|---|
| Blood glucose (fasting) | 123.25 (SD±18.42) | 83.60 (SD±4.90) | 19.9 | *P*<0.000 |
| Blood glucose (post meal) | 228.46 (SD±12) | 110.69 (SD±6.68) | 82.2 | *P*<0.000 |
| CRP (mg/l) | 3.36 (SD±0.37) | 2.79 (SD±0.11) | 14.1 | *P*<0.001 |

*P*<0.001 = Highly significant, DM: Diabetes mellitus, CRP: C-reactive protein



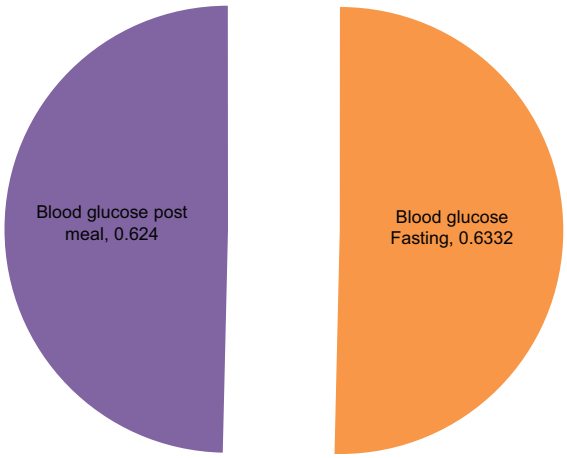

3. CRP (mg/l) 3.36 (0.37) in GDM and 2.79 (SD ± 0.11) in controlled group *t*-test was 14.1 and *P*-value was highly significant ($P < 0.001$).

In Table 3, CRP parameter in blood glucose fasting and blood glucose post-meal 0.032 (SD ± 0.2) in blood glucose

**Table 3: CRP parameter in fasting and post-meal blood glucose**

| CRP | Blood glucose fasting | Blood glucose post-meal | *t*-test | *P*-value |
|---|---|---|---|---|
| Parameter | 0.6332 | 0.6240 | 0.017 | *P*>0.98 |

CRP: C-reactive protein




is 0.6240 (SD ± 0.4) blood glucose post meal, *t*-test 0.017, $P > 0.98$, *P*-value insignificant.

## DISCUSSION

The present study of elevated C-reactive Protein in first trimester as a predictor of GDM in the Karnataka population. The BMI in GDM group was 26.05 (SD ± 3.40) and 22.80 (SD ± 2.14) in controlled group, *t*-test was 7.75, and *P*-value was highly significant ($P < 0.000$) [Table 1]. The comparative study of laboratory findings – Blood glucose level (fasting) 123.2 (SD ± 18.42) in gestation DM group and 86.60 (SD ± 4.90) in controlled group The t-test was 19.9 and *P*-value was highly significant ($P < 0.00$). In Blood glucose (post meal) study 228.4 (SD ± 12) in gestation DM group and 110.69 (SD ± 6.68) in controlled group. The t-test was 82.2 and ($P < 0.00$) *P*-value was highly significant. CRP value in was 3.36 (SD ± 0.37) in gestation DM group and 27.9 (SD ± 0.11) in controlled group. The t-test was 14.11 and *P*-value was highly significant ($P < 0.00$) [Table 2]. The CRP value was insignificant or more or less same 0.6232 (SD ± 0.2)/0.6240 (SD ± 0.4) in both fasting and post-meal blood glucose studies [Table 3]. These findings are more or less in agreement with the previous studies.[6-8]

GDM is the most common medical complication during pregnancy prevalence of such cases varies between 12% and 18% globally.[9] The increase in BMI seems to play a role in significant increase of CRP serum level. The gain during pregnancy and nutrition factors such as intake of saturated fatty acids is among other risk factors associated with FGDM.[10] The cornerstone of management is glycemic control and poor control during pregnancy has been associated with miscarriage, preterm birth, stillbirth, macrosomia, urinary tract infection, polyhydramnios shoulder dystocia, operative delivery neonatal hyperbilirubinemia, and hypocalcemia.[11] In normal pregnancy, there is an increase of lipid peroxidation products in serum with advancing gestation, which is balanced by an adequate antioxidative response. In GDM, increased blood glucose levels cause auto-oxidation of unsaturated lipids in plasma and membrane proteins which is responsible for generation of free radicals. Hence, this cycle of tissue damage and cell death, leading to increased free radical production and compromised free radical scavenger, exaggerates the oxidative stress.

The first trimester markers may help to predict this complication and improve the management of such cases. Hence, the first trimester of pregnancy is known as insulin sensitive period. Insulin resistance increases during the second trimester of pregnancy.[12] In the management GDM, treatment modalities aimed to improve insulin sensitivity may be useful. It is hypothesized that high sensitivity CRP may cause insulin resistance by increasing insulin receptor substrate-1 (IRS-1) phosphorylation at ser307 and ser612 through Jun N-terminal kinesis and extracellular signal regulated kinases I and II, respectively, leading to impaired insulin, stimulated glucose transporter translocation, and glycogen synthesis.[13] Controlling weight gain during pregnancy reduces the incidence of GDM.

## SUMMARY AND CONCLUSION

The present study of elevated first trimester CRP as a predictor of gestational diabetes will be helpful for obstetrician and gynecologist, radiologist, neonatal physician to predict complications to fetus, and mother as well. This study demands further hormonal, pathophysiological, genetic, and nutritional studies because exact pathogenesis of gestational diabetic is still unclear.

This research paper is approved by Ethical Committee Basaveshwara Medical College, Chitradurga-577502, Karnataka.

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