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Lipid Peroxidation in Gestational Diabetes

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Summary

We studied lipid peroxidation (MDA), vitamin C and E levels and superoxide dismutase (SOD) activity in 25 gestational diabetic and 25 healthy pregnant women. An increase in MDA was observed in gestational diabetics as compared to control ($p<0.05$), Likewise, a significant fall in vitamin C & E levels was observed in gestational diabetics as compared to control ($p<0.001$). Also, SOD activity was increased in gestational diabetics as compared to that in controls ($p<0.001$). These findings suggest that lipid peroxidation has a role in the pathogenesis of gestational diabetes.

Introduction

Gestational diabetes is one of the most prevalent complications of pregnancy and causes a large fetal wastage. These pregnancies are at increased risk of fetal anomalies, fetal macrosomia, growth retardation, respiratory distress and platelet hyperagregability in the newborn (Nasrat et al, 1996). High blood glucose levels induce oxidative stress and decrease antioxidant defences, thus leading to increased free radical formation (Hunt & Wolff, 1991). Uotila et al (1991) reported that lipid peroxidation is controlled by an adequate antioxidative response during normal pregnancy. Oxygen free radicals have been implicated in diabetic pathogenesis (Oberlay, 1988). The present study was planned to evaluate the oxidative stress and antioxidant defence system in gestational diabetes and normal pregnancy

Material and Methods

This study was carried out in 50 women attending /admitted in Obstetric and Gynecology Department at Pt. B.D. Sharma PGIMS, Rohtak (Haryana), from January to September 1997. Informed consent was obtained. The control group included 25 women having normal healthy pregnancies (HPW). The study group consisted of 25 pregnant women who were diagnosed as having gestational diabetes (G-DM) after undergoing oral glucose tolerance test with 100 g of glucose (Carone et al, 1991). Maternal age ranged between 28-36 years and gestational age ranged from 32-49 weeks. All the women had a negative history of diabetes mellitus, hypertension and obesity. None were taking any vitamin supplment or drugs. Venous heparinised blood samples were collected after overnight fasting and plasma was separated. Malonaldehyde (MDA), was estimated by thiobarbituric acid (TBA) test and vitamin E levels were estimated spectrofluorometrically (Kharb et al, 1998). Vitamin C levels were assayed spectrophotometrically (Roe, 1961) and superoxide dismutase (SOD) levels were estimated by method of Misra & Fridovich (1972). Statistical evaluation was performed by Student's t-test.

Results

The mean fasting, 1 hour, 2 and 3 hours blood

Table I. Glucose tolerance in various groups (mean ±SD, mg%)

	Fasting	1 hour	2 hour	3 hour
HPW	72.8 ± 11.17	108.0 ± 25.4	110.0 ± 24.0	90.92 ± 15.15
G-DM	76.68 ± 14.18	119.48 ± 28.0	110.8 ± 28.7	89.24 ± 26.49

Table II. Parameters of oxidative stress (mean±SE)

	HPW	GDM
MDA (mmol/ul)	1.04 ± 0.22	1.54 ± 0.26[a]
Vitamin C (umol/L)	62.18 ± 12.27	45.69 ± 5.58*
Vitamin E (ug/ul)	9.90 ± 0.38	6.64 ± 0.27*
SOD (EU/L)	184.7 ± 6.12	227 ± 47.66*

[a] as compared to HPW $p<0.5$
* as compared to HPW $p<0.001$

glucose levels in gestational diabetics are given in Table I.

MDA was enhanced in G-DM as compared to that in controls ($p<0.05$), (Table II). Significant fall in vitamin C and E levels were observed in gestational diabetics as compared to that in controls ($p<0.001$).

On the other hand, significantly increased level of SOD were found in G-DM as compared to that in HPW.

Discussion

In the present study, we observed an increase in MDA levels in G-DM as compared to that in controls (Table II). This is in agreement with those reported by Carone, et al (1991) and Kamath et al (1998). While others have found no evidence of increased oxidative stress or antioxidant depletion in diabetic mothers in early pregnancy (Bates et al, 1997). These women had carefully planned pregnancy in respect of diabetic control and this is likely to be the main explanation for the absence of increased lipid peroxidation and good antioxidant levels in these subjects. Noborasco et al (1991) reported a positive correlation between lipid peroxidation and markers of glycaemic controls.

Substantial evidence from animal models suggests that oxidative stress in diabetic pregnancy might contribute to increased risk of fetal abnormality and that this can be prevented by chain-breaking antioxidants, including tocopherol (Eriksson and Borg, 1991).

In the present study, we observed significantly lowered levels of antioxidant vitamin C and E in gestational diabetes as compared to those in healthy pregnant women. In contrast, Bates et al (1997) reported an increase in vitamin C and E levels in diabetic women which they attributed to higher dietary intake of these vitamins.

We also observed high activity of superoxide dismutase (SOD) in gestational diabetes. The increased oxidative stress causes induction of antioxidant enzyme activities and this increase suggests a role of SOD in the protection of embryonic development against free radical damage. This is in accordance with that reported by Carone et al (1991).

In conclusion, gestational diabetes induces a condition of oxidative stress and further study should investigate the correlation between glucose control, free radical damage and embryonic development in normal and complicated pregnancy.

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