International Journal of Allied Medical Sciences and Clinical Research (IJAMSCR)

IJAMSCR |Volume 3 | Issue 3 | July-Sep- 2015
www.ijamscr.com

Research article ***Medical research***

Serum nitric oxide (NO) production in preeclampsia

Dr.Asha.N.S,[1] Dr.Anju Varghese.[2]

[1]*Department of Biochemistry, Government Medical College, Thiruvananthapuram, Kerala.*

[2]*Department of Biochemistry, MOSC Medical College, Kolenchery, Kerala.*

Corresponding author: Dr.Asha.N.S

ABSTRACT

The present study was undertaken to observe the levels of Serum nitric oxide (NO) in preeclampsia. 50 preeclamptic patients in the third trimester and 50 normal healthy subjects in the third trimester with normal blood pressure and no complications were recruited for the present study by convenient sampling technique. Data was analyzed by SPSS 20.0. Values are expressed as mean ± SD. P-value <0.05 was taken as significant. In the present study, we have observed lower S.nitrite levels in patients when compared with control group. Our data provides further evidence that support that diminished nitric oxide synthesis contributes to the pathophysiologic changes seen in preeclampsia

Key words: Preeclampsia, Serum nitric oxide.

INTRODUCTION

Pre-eclampsia, occurs prior to eclampsia (Greek word “eklampsis” meaning sudden flashing) [1] , is a systemic syndrome characterized by hypertension, proteinuria and oedema, often complicated by renal failure, pulmonary oedema and coagulopathy. Consequences of these could be a retarded growth of the foetus or mortality preceded by seizures and coma. Pre-eclampsia can occur in early pregnancy termed as “early onset pre-eclampsia” at However, endothelial dysfunction is common in both early and late onset, responsible for the symptoms like proteinuria and hypertension. Failure to control these symptoms may result in foetal prematurity and premature delivery [2]. Pre-eclampsia is a cause for about one-third of maternal mortality in developing countries with limited access to health care [3]. Pre-eclampsia and eclampsia are seen in around 4.6 per cent of all deliveries5 and the neonatal mortality rate is around 43 per 1000 live births in India. Therefore, early detection or prediction of PE is imperative6 and non-invasive diagnostic methods based on biomarkers hold the promise. The exact cause for the pathogenesis of pre-eclampsia remains unclear. However, several studies have reported that this condition results due to abnormal placenta rather than the foetus [4, 5]. Pre-eclampsia occurs only in the presence of a placenta and almost always remits after its delivery. NO is a very unstable, short half-life gas that breaks down rapidly into the stable products nitrate and nitrite [6]. NO is a potent vasodilator which modulates pulmonary and systemic vascular tone [7]. At relatively high concentrations NO causes tissue injury and at lower concentrations in presence of oxygen, superoxide and other reactive oxygen species, NO can be converted into a range of potent oxidants such as nitrogen dioxide and peroxy nitrite which may amplify and exacerbate the harmful effects of lipid peroxidation [8, 9] . It was reported that Nitric oxide production was increased with preeclampsia. The biologic significance of increased production is unknown, but it might be compensation

for the vasoconstriction of preeclampsia [10]. Maternal and fetal serum NO levels are increased significantly in pre-eclampsia and eclampsia, which possibly represents a compensatory/protective mechanism to maintain blood flow and limit platelets aggregation in the fetal-maternal circulations. The increase in NO production is directly related to the severity of pre-eclampsia; this would be of diagnostic significance for the prediction of the severity of this syndrome [11].

The present study was undertaken to observe the levels of Serum nitric oxide (NO) with preeclampsia.

MATERIALS AND METHODS

The study was approved by Institutional Ethics Committee. A written, informed consent was obtained from all the participants. The study was performed in accordance with the "Ethical Guidelines for Biomedical Research on Human Participants, 2006" by the Indian Council of Medical Research and the Declaration of Helsinki, 2008.

PARTICIPANTS, INCLUSION AND EXCLUSION CRITERIA

50 preeclamptic patients in the third trimester and 50 normal healthy subjects of the third trimester with normal blood pressure and no complications were recruited for the present study by convenient sampling technique. The following criteria were used to recruit the patients.

INCLUSION CRITERIA

1. Willing participants

Exclusion criteria

1. Preeclamptic patients with hypertension
2. Patients with kidney or liver diseases
3. Patients with GDM or other antenatal complications.

Blood for S.nitrite was collected in plain bottles by venepuncture. One hour after collecting the sample, blood was centrifuged at 3000 rpm for 15 minutes for serum separation. Serum was kept at -20^{0}C.

DETERMINATION SERUM NO PRODUCTION

NO production was estimated by using a rapid method based on the Griess reaction involving a shortened incubation period of nitrate with cadmium [12, 13].

DATA ANALYSIS

Data was analyzed by SPSS 20.0. Values are expressed as mean ± SD. P-value <0.05 was taken as significant.

RESULTS

Results are presented in figure no: 1. The mean value of S.nitrite in the patient group is 12.564μmol/L and in the control group is 18.481μmol/L. S.nitrite levels are significantly lower in patients when compared with control group.

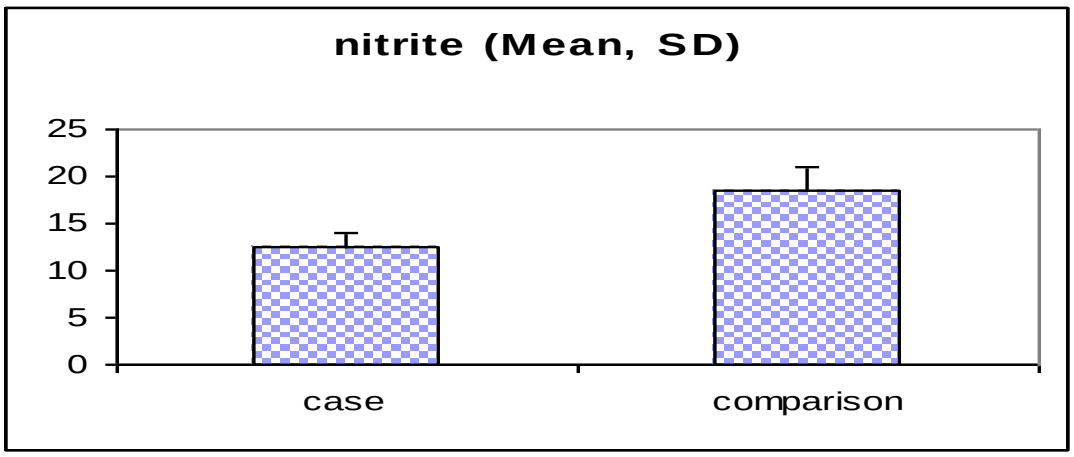

Figure: 2 Serum nitrate levels in cases and controls

DISCUSSION

It was reported that Maternal and fetal serum NO levels are increased significantly in pre-eclampsia and eclampsia [11]. In contrast, it was reported that serum NO levels decreased [14-19]. However, in some studies NO levels were not altered significantly during pre eclampsia [20]. The evidence indicates that NO can have either a prooxidant or an

antioxidant effect on lipid peroxidation depending on a variety of contingent factors. In the present study, we have observed lower S.nitrite levels in patients when compared with control group.

Our data provides further evidence that support that diminished nitric oxide synthesis contributes to the pathophysiologic changes seen in preeclampsia.

CONFLICTS OF INTEREST: NIL

CONCLUSION

REFERENCES

[1]. Aernout Luttun, Peter Carmeliet. Soluble VEGF receptor Flt1: the elusive preeclampsia factor discovered? J Clin Invest 2003; 111 : 600-2.
[2]. Mutter WP, Karumanchi SA. Molecular mechanisms of preeclampsia. Microvasc Res 2008; 75 : 1-8.
[3]. Noronha Neto C, de Souza AS, Amorim MM. Pre-eclampsia treatment according to scientific evidence. Rev Bras Ginecol Obstet 2010; 32 : 459-68.
[4]. Maynard SE, Karumanchi SA. Angiogenic factors and preeclampsia. Semin Nephrol 2011; 31 : 33-46..
[5]. Lakshmi Tanuja Petla, Rosy Chikkala, K.S. Ratnakar, Vijayalakshmi Kodati & V. Sritharan. Biomarkers for the management of pre-eclampsia in pregnant women. Indian J Med Res 138, July 2013, pp 60-67.
[6]. Palmer RMJ, Ferrige AG, Moncada S. NO release accounts for the biological activity of endothelium derived relaxing factor. Nature 1987; 327: 524-526.
[7]. Singh S, Evans TW. Nitric oxide the biological mediator of the decade: Fact or fiction? Eur Resper J 1997; 10: 699-707.
[8]. D'Ischia M, Palumbo A, Buzzo F. Interactions of nitric oxide with lipid peroxidation products under aerobic conditions: inhibitory effects on the formation of malondialdehyde and related thiobarbituric acid reactive substances. Nitric Oxide: Biology and Chemistry 2000; 4: 4-14.
[9]. O'Donnell VB, Chumley PH, Hogg N et al. Nitric oxide inhibition of lipid peroxidation: Kinetics of reaction with lipid peroxyl radicals and comparison with ∝-tocopherol.Biochem 1997; 36: 15216-15523.
[10]. Ranta V, Viinikka L, Halmesmäki E, Ylikorkala O. Nitric oxide production with preeclampsia. Obstet Gynecol. 1999 Mar;93(3):442-5.
[11]. Shaamash AH, Elsnosy ED, Makhlouf AM, Zakhari MM, Ibrahim OA, EL-dien HM. Maternal and fetal serum nitric oxide (NO) concentrations in normal pregnancy, pre-eclampsia and eclampsia. Int J Gynaecol Obstet. 2000 Mar; 68(3):207-14.
[12]. Griess P. Ber Deutsh Chem Ges 1879; 12: 426.
[13]. Shinn MB. Ind Engng Chem Analyt Edn 1941; 13: 33.
[14]. Zeeman CG, Dekkar GA, Van Geijn HP, et al. Endothelial function in normal and preeclamptic pregnancy: A hypothesis. Eur J Obstet Gynecol Reprod Biol 1992; 43: 113-122.
[15]. Pinto A, Sorrentino R, Sorrentino P, et al. Endothelial cell relaxing factor released by endothelial cells of human umbilical vessels and its impairment in pregnancy induced hypertension. Am J Obstet Gynecol 1991; 164: 507-513.
[16]. Aydin S, Benian A, Madazli R, et al. plasma malondialdehyde, SOD, sE-selectin, fibronectin, endothelin-1 and NO levels in women with preeclampsia. Eur J Obstet Gynecol Reprod Biol 2004; 113: 21-25.
[17]. Van Buren GA, Yang D, Clark KE. Estrogen induced uterine vasodilatation is antagonized by L-nitro [1].arginine methyl ester, an inhibitor of NO synthesis. Am J Obstet Gynecol 1992; 167: 828-833.
[18]. Norris LA, Higgins JR, Darling MRN, et al. Nitric oxide in the uteroplacental, fetoplacental and peripheral circulations in preeclampsia. Obs & Gyn 1999; 93: 958-963.
[19]. Di Iorio R, Marinoni E, Coacci F, et al. Amniotic fluid NO and uteroplacental blood flow in pregnancy complicated by IUGR. Br. J Gynecol 1997; 104: 1134-1139.
[20]. Curtis NE, Gudi NM, King RG, et al. Nitric oxide metabolites in normal human pregnancy and preeclampsia. Hypertens pregnancy 1995; 14: 339-349.