Research Article

iMedPub Journals
www.imedpub.com

Journal of Biological and Medical Sciences

2022
Vol.6 No.4:001

Oxidative Stress Marker and Antioxidants in Amniotic Fluid in PIH

Kalpana Lohiya* **and KH Deshpande**

Department of Biochemistry, SRTR Govt. Medical College, Ambajogai, India

***Corresponding author:** Kalpana Lohiya, Department of Biochemistry, SRTR Govt. Medical College, Ambajogai, India, Tel: 9404297766; E-mail: kalpanalohiya2016@gmail.com

Received date: January 24, 2022, Manuscript No. JBMS-22-12384; **Editor assigned date:** January 27, 2022, Pre QC No. JBMS-22-12384 (PQ); **Reviewed date:** February 11, 2022, QC No. JBMS-22-12384; **Revised date:** March 28, 2022, Manuscript No. JBMS-22-12384 (R); **Published Date:** April 05, 2022, DOI: 10.36648/ipjbms/22.6.001

Citation: Lohiya K, Deshpande KH (2022) Oxidative Stress Marker and Antioxidants in Amniotic Fluid in PIH. J Biol Med Sci Vol:6 No:4

Abstract

Aim: This study was to evaluate the status of antioxidants and TBARS in amniotic fluid in PIH and compared with normal healthy pregnancy.

Materials and method: This study included 30 cases of healthy pregnant women as a control and 30 cases of pregnant women with PIH admitted in the hospital. Amniotic fluid samples of all patients subjected for estimation of different parameters.

Results: There was significant increase in blood pressure ($p<0.01$) and TBARS levels ($p<0.05$) in the study group when compared to control group. There was significant decrease in Vitamin C ($p<0.01$), Vitamin A ($p<0.05$) and SOD ($p<0.001$) levels in study group when compared to control group. There was no significant difference in age group.

Conclusion: Imbalance between TBARS (product of lipid peroxidation) and antioxidants favor lipid peroxides with the increasing severity of PIH and pre-eclampsia.

Keywords: Amniotic fluid; Li pid peroxidation; Pregnancy induced hypertension

Introduction

Birth of a mature and innocent baby is one of the amazing creatures of the nature. Power of creativity makes mother strong and satisfied.

Though safe and secure pregnancy is a dream of every woman, hypertension occurs in 7-9% of all pregnancies. Pre-Eclampsia (PE) accounts for about 8% of these cases Pregnancy is a physiological stressful condition with an increased requirement of tissue oxygen because of a rapidly developing embryo and a subsequent fetal growth [1]. The fetus is surrounded by Amniotic Fluid (AF). AF plays an important role in fetal development and growth. It originates from maternal and fetal tissues. It reflects the fetal situation. AF is an aquatic and sterile environment where fetus cans easily move. It provides protection to the fetus against external injury. Provides a constant temperature to protect from heat and loss. It gives cushioning effect to fetus [2-5]. One of the most common and potential life threatening complications of pregnancy is Pregnancy Induced Hypertension (PIH). Frequency is 5%-15% in general population of pregnant women [6]. Pregnancy Induced Hypertension (PIH) is a syndrome of hypertension in pregnancy, with or without proteinuria and edema [7]. Hypertensive disorders are the most common medical complication of pregnancy and the major cause of maternal and infant disease and death worldwide. Small vessel spasm is considered the most important cause for hypertension in pregnant and non-pregnant state. The major risk to the fetus results from decreased placental perfusion leading to decreased supply of oxygen and nutrients necessary for fetal growth and wellbeing [8]. The etiology of this disease remains unclear, but pathophysiology occur during pregnancy are vasoconstriction and coagulation which leads to endothelial dysfunction and immune-activation with the development of hypertension during pregnancy, which is the primary cause of Pre Eclampsia (PE) [9].

The onset of labor is associated with pain, fear, anxiety and hypoxia. Uterine contraction reduces blood flow leading to tissue ischemia and reperfusion which induces the production of Free Radicals (FR) [10]. Pregnancy represents a complex state in reproductive biology in which the mother and the fetus may both contribute to the oxidative stress [8]. During pregnancy, there is increased cardiac output due to increased blood volume, which results in oxidative stress [10]. If production of Reactive Oxygen Species (ROS) overwhelms total antioxidant capacity of cell, condition is known as oxidative stress, leading to the generation of FR [11]. ROS include molecules like hydrogen peroxide; ions likes hypochlorite ion; radicals like the hydroxyl radical; and the superoxide anion which is both ion and radical.

ROS are products of the body's incomplete reduction of oxygen molecules. It is assumed that numerous anatomical, physiological and metabolic changes occur in mother's body during pregnancy and they support the production of ROS. Placenta filled with mitochondria is the main source of ROSp [12]. Free radicals are cluster of atoms that contain an unpaired electron in their outermost orbit of electrons [11]. The free radicals can have deleterious effects during pregnancy by triggering pre-eclampsia and PIH [13].

In recent days, lipid peroxidation has drawn much attention. The increase in lipid peroxidation has been related to the pathogenesis of many degenerative disorders such as oxidative

damage to DNA [14]. PIH is a state of extremely increased oxidative stress and increased lipid peroxidation due to decrease of antioxidant capacity in comparison to normal pregnant women [6].

Reactive radicals (such as NO^{-2}, OH^{-}, CCL^{-3}, O^{-2}) abstracts atoms of hydrogen from Polyunsaturated Fatty Acid (PUFA) side chain in membrane or lipoproteins. This leaves unpaired electron on carbon which reacts with O_2, resulting peroxyl radical attaches to adjacent fatty acid side chain to generate new carbon radical and chain reaction will continue [15].

A great attention is being paid to lipid peroxidation, which actually is oxidative damage of lipids and increased creation of lipid peroxides, whose final product is Malondialdehyde (MDA). Oxidative marker, which bonds to MDA fast and strongly is Thiobarbituric Acid Reactive Substance (TBARS). Overall attack of one reactive free radical can oxidize multiple fatty acid side chain to lipid peroxide, damaging membrane proteins, making the membrane leak and eventually causing complete membrane breakdown [15,16].

Lipid peroxidation is a key contributor to patho-psychological condition of Pre-eclampsia [7].

To prevent damage provoked by lipid peroxides nature has endowed us with protective antioxidants for ex: Super Oxide Dismutase (SOD), Vitamin C, Vitamin A [17]. Antioxidants counteract the effect of this Free Radicals (FR) and there by protect cell membrane from lipid peroxidation. The ability of a tissue or fluid to buffer the effects of Reactive Oxygen Species (ROS) is called Total Antioxidant Capacity (TAC) [18]. Ascorbic acid or vitamin C is a water-soluble antioxidant nutrient, and also needed for the formation of collagen, repairing of the tissues and various metabolic processes. It reduces the risk of premature birth [19]. It is a powerful scavenger and quencher of singlet O_2 in aqueous solution Vitamin C is the most important scavenger of water soluble radicals and it is the first line of defense. Under conditions of oxidative stress, it is the first antioxidant to be consumed. Vitamin C also recycles the Vitamin E radical by reducing it [20,21]. Vitamin A is the most versatile fat soluble vitamin with diverse functions. Beta carotene is an antioxidant may protect against damage to cell membrane. It is essential for postpartum tissue repair in pregnant women. Lack of vitamin A during pregnancy can cause night blindness, problems with the placenta and low birth weight of newborns [22].

Super Oxide Dismutase (SOD) is an enzyme that catalyzes breakdown or dismutation of super oxide anion into oxygen and hydrogen peroxide. It is present in almost all aerobic cells and ECF. It is a front line defense against the free radicals [20]. It is a chain breaking antioxidant, acting by combining with chain propagating radicals. It acts in aqueous phase to trap super oxide [23]. SOD is the major intracellular antioxidant enzyme that inactivates superoxide

Materials and Methods

The present study was conducted at Department of Biochemistry of our institute.

Subjects inclusion criteria

Thirty patients with Pregnancy Induced Hypertension, (PIH) were admitted in labor room in hospital. Control group consist of thirty normal healthy pregnant women admitted in labor room of hospital having same age and socio-economic status.

Exclusion criteria

Blood stained amniotic fluid samples were excluded from the study.

Sample collection

Amniotic fluid samples were collected by Artificial Rupture of Membrane (ARM) in sterile test tube, shortly prior to delivery. Samples were centrifuged and processed for respective parameters early as possible.

Following biochemical parameters were studied in amniotic fluid.

- Thiobarbituric Acid Reactive Substances (TBARS).
- Vitamin C
- Vitamin A
- SOD

TBARS was estimated by the method of Satoh, 1978 by using MDA as a standard [24]. Vitamin C was measured by 2-4 DNPH method [25]. Vitamin A was measured by using Carr-Price reaction method [26]. SOD was estimated [27].

Results

Table 1: Demographic data of controls and PIH cases.

Character		Control (Mean ± SD)	PIH (Mean ± SD)	"P" Value
Number of cases		30	30	30
Age (years)		23.96 ± 2.44	24.95 ± 2.82	N. S.
B. P.	Systolic (mmHg)	113.4 ± 4.60	144.16 ± 2.28	P<0.01
	Diastolic (mmHg)	74.7 ± 2.36	95.6 ± 1.60	P<0.01

P<0.05 is significant, N.S.=Not significant

There is no significant difference in age between controls and cases, but there is significant increase in blood pressure (p<0.01) in PIH cases as compared to controls (Table1).

Table 2: Comparison of oxidants and antioxidants between controls and cases.

Parameters	Controls	PIH Cases	P Value
Lipid Peroxides (TBARS) nmol/ml	0.033 ± 0.74	0.202 ± 0.163	P<0.05
Vitamin C (ug/ml)	5.436 ± 1.30	2.433 ± 0.71	P<0.01
Vitamin A (ug/dl)	23.06 ± 7.86	15.07 ± 4.78	P<0.05
SOD (U/ml)	0.145 ± 0.124	0.075 ± 0.022	P<0.001
P<0.05 is significant.			

There is significant increase in Lipid peroxides (TBARS) levels in PIH cases than Controls and Significant decrease in Vitamin C (p<0.01) and Vitamin A (p<0.05) levels in PIH cases than Controls.

There is highly significant decrease in SOD levels (p<0.001) in PIH cases as compared to controls (Table 2).

Discussion

Reactive oxygen species and antioxidants have been studied by many authors in maternal and fetal serum, but there are very few studies in amniotic fluid. Keeping this in mind, the present study was undertaken to evaluate the status of Thiobarbituric Acid Reactive Species (TBARS) and antioxidants in amniotic fluid in normal pregnant women and in PIH.

In our study we observed the increase in systolic and diastolic blood pressure in PIH cases. Free radicals may trigger blood pressure. Oxidative stress caused due to increased lipid peroxides plays an important role in pathogenesis of pre-eclampsia.

In the present study, we observed a significant rise in lipid peroxidation product (TBARS) in PIH cases than control (P<0.05).

Our study correlates with these studies JT Utolia et al. studied the levels of MDA in the serum of normal pregnant and PIH women and observed the significant increase in the levels of lipid peroxide products in PIH Mohanty, Sahu et al. studied the serum MDA levels and antioxidants in PIH patients and observed significant raised levels of MDA and significant decreased levels of Vitamin C [28,29].

Sharma JB et al. studied the MDA and antioxidant Vitamin C levels in normal pregnant and PIH cases and observed the significant increase in the levels of MDA and Vitamin C was significantly lower in PIH cases than control [30]. SV Kashinakunti et al. observed significant increased levels of MDA and decreased levels of vitamin C in the serum of PIH patients [31].

Jannet et al. studied Amniotic fluid levels of TBARS and observed that, Reactive oxygen species levels were present in some but not all specimens [18]. Increased oxidative stress may be responsible for increasing lipid peroxidation product (MDA) levels in PIH cases.

Recognition of lipid peroxidation involvement in the pathogenesis of disease is important because the deleterious effects of this process might be prevented by administration of scavenging systems; antioxidants [32]. In our study we observed significant decrease in the levels of antioxidant vitamin C in PIH cases than the controls (P<0.01).

Our study is in agreement with these studies Lee Ks, Kim YH et al. studied the various plasma and amniotic fluid levels of lipid peroxides and antioxidant levels in the cases of Pre-eclampsia and found the increased levels of lipid peroxide and significant decrease in the levels of antioxidant vitamin C. Barrette, Bridget M studied the amniotic fluid and serum antioxidant vitamin C levels in women with preterm rupture of fetal membrane and observed the significant decrease in vitamin C levels. Magdy S. Mikhail et al. studied the Pre-eclampsia and antioxidant nutrients in plasma and observed the significant decrease in the levels of Vitamin C and beta carotene in women with Pre-eclampsia [33-35]

Vitamin C as a water soluble antioxidant, trap most of the Free radicals present in aqueous phase of plasma. It functions as the first line antioxidant defense against free radicals present primarily in the plasma. So they get utilized primarily by the free radicals. This may be the reason that Vitamin C shows significant low levels

In our study we observed the marked decrease in the levels of lipid soluble antioxidant vitamin A in PIH than controls (p<0.05).

Our study correlates with the study of, Ziari SA, Mireles VL et al. studied the serum levels of Vitamin A, beta carotene in Pre-eclampsia women and healthy pregnant women and observed that Pre-eclampsia depletes natural lipid soluble antioxidants and reduced levels of Vitamin A shows PIH studied the effect of

Vitamin A deficiency during pregnancy and observed that it is associated with increased risk of preterm delivery [36,37].

When capacity of free radicals exceeded than water soluble antioxidants, free radicals can then diffuse to cell membrane, initiating lipid peroxidation. So lipid soluble antioxidants may be utilized after utilization of water soluble antioxidants. This may be the reason of decreasing vitamin A levels in PIH [34]. Antioxidant is a molecule capable of slowing or preventing the oxidation of free radicals [33,38,39].

Conclusion

In our study we observed the significant decrease in the levels of SOD in PIH than in controls ($p<0.001$). Our study correlates with the study of Mahadik et al. studied the levels of SOD in PE and eclampsia and they found statistical difference in normotensive pregnant women, PE and eclampsia. Joshi et al. studied the oxidative stress and deficiency of micronutrients of normal pregnancy and PIH. They concluded that, deficiency of micronutrients and decreased levels of SOD may be associated with PIH.

Jefree et al. studied the pathogenesis of pre-eclampsia and antioxidants and observed diminished levels of SOD resulting in decreased total antioxidants protective capacity if the generation of harmful radicals exceeded than the capacity of cells to protect against them then these radicals scavenges chain breaking antioxidants like SOD. This results in decreased values of SOD.

Because of the strenuous nutritional demands of a growing fetus, pregnancy represents nutritionally perilous state for every pregnant woman as she provides nutrients to support her child's rapid growth in addition to fulfill her own metabolic needs.

Limitations of the Study

In our study, sample size is small; therefore it may not be applicable for large population.

Thus increased oxidative stress and decreased antioxidant levels may increase lipid peroxidation in PIH.

References

1. Bhattacharya P, Mathangi DC (2018) Assessment of Maternal Oxidative stress and Antioxidant Defence during Caessarean section. ECPB 1:5-10
2. Cim N, Tolunayetal HE (2018) Amniotic fluid oxidant-antioxidant status in foetal congenital nervous system anamolies. J Int Med Res 46:1146-1152
3. Cunnighem FG, Leveno KL (2005) Williams obstetrics. (22nd edi) McGraw-Hill, New York.
4. Robert Paul Lanza (2006) Essentials of stem cell biology. Elsevier Academic, Maryland and Washington.
5. Klemmt PAB, Vafsizadem (2010) Murine amniotic fluid stem cells contribute mesenchymal but not epithelial components to reconstituted mammary ducts. Stem Cell Res Ther 1:20
6. Draganovic D, Lucicetal N (2017) Correlation of oxidative stress markers with ultrasound and cardiotocography parameters with Hypertension induced pregnancy. Actain fom med mar 25:19-23
7. Opari Z, Zaman A, Calhon DA (2003) Pathogenesis of hypertension. Ann Int Med 139:761-776
8. Sundarum S, Pratibha D (2003) Pathogenesis of pregnancy induced hypertension Revisited. Obs Gynae 8:671-674
9. Yelumalai S, Muniandy S (2010) Pregnancy induced hypertension andpre-eclampsia: levels of angiogenic factors in Malaysian women. J Clin Biochem Nutr 47:191-197
10. Chen J, Mehata J (2004) Role of Oxidative Stress in Coronary heart disease. Indian Heart J 56:163-173
11. KingaTobola-wrobel, Marek Pietryga (2020) Assocition of oxidative Stess on Pregnancy. Oxid Med Cell Longev 2020:6398520
12. Basu H, Gupta J (2001) Oxidative Stress in Pregnancy-Role of Iron therapy. Obs Gynae 6:703-707
13. Chatterjee SN, Agarwal S (1988) Membrane lipid peroxidation and its pathological consequences. Indian J Biochem Biophysics 25:25-31
14. Halliwell B (1994) Free radicals, antioxidants and human disease; curiosity, cause or consequence? Lancet 344:9-10
15. Draganovic D, Lucic N (2016) Oxidative Stress Marker and Pregnancy Induced Hypertension. Med arch 70:437-440
16. Devasagayam TP, Tilak JC (2004) Free radicals and antioxidants in human health; current status and future prospects. J Assoc Pysc India 52:794-804
17. Burlingame JM, Esfandiari N (2003) Total antioxidant capacity and reactive oxygen species in amniotic fluid. Obst Gynecol 101:756-761
18. HalliwellB, Gutteridge JMC. (1990) The antioxidants of human extracellular fluids. Arch Biochem Biophysc 280:1-8
19. Das A, Pal SR (2019) A comparative study of oxidative stress indicesand antioxidant status in preeclampsia with normal pregnancy in a tertiary rural medicalcollege IOSR. J Med Dent Sci 18:1-7
20. Murray RK, Granner DK (2002) Text Book of Harper's Illustrated Biochemistry. (25th edition), McGraw-Hill Publishing Co, U.S.A. pp160,167
21. Satoh k (1978) Serum lipid peroxide in cerebrovascular disorders determined by a new colorimetric method. Clin Chem Acta 90:37-43
22. Varley H (1960) Practical Clinical Biochemistry, (4thedition) Interscience: New York. pp635-636
23. VarleyH. Estimation of Vitamin A by Carr-Price method. (4thedi) Practical Clinical Biochemistry, pp608-610
24. Nischal HK, Sharma MP (1998) Serum superoxide dismutase levels in diabetes mellitus with or without microangiographiccomlpications. JAPI 46:853-855
25. Uotila JT, Tuimala RJ (1993) Findings on lipid peroxidation and antioxidant function in Hypertensive complications of pregnancy. Br J Obstet Gynaecol 100:270-276
26. Mohanti S, Sahu PK (2006) Evaluation of oxidative stress in pregnancy induced hypertension. Indian J Clin Biochem 21:101-105

27. Sharma JB, Sharma A (2006) Oxidative stress markers and antioxidant levels in normal pregnancy and pre-eclampsia. Int J Gynaecol Obstet 94:23-27

28. Kashinakunti SV, Sunitha H (2010) Lipid peroxidation and antioxidant status in Pre-eclampsia. Al Ameen J Med Sci 3:38-41

29. Stojiljkovic V, Todorovic A (2009) Antioxidant status and lipid peroxidation in small intestinal mucosa of children with cialic disease. Clin Biochem 42:1431-1437

30. Lee Ks, Kim YH (2009) Lipid peroxidation and total antioxidant ability in venous plasma and amniotic fluid of pregnant women with preterm premature ruptureof membranes. Korean J ObstetGynecol 52:53-60

31. Barrett, Bridget M (1993) Antioxidant Vitamin status in the serum and amniotic fluid of women with premature rupture of the fetal membranes. Dissertation Abstracts International 55:0080

32. Magdy S, Mikhail (1994) Pre-eclampsia and antioxidant nutrients: decreased plasma levels of reduced ascorbic acid, alfatocopherol and beta carotene in womenin preeclampsia. AmjObstetGynecol 71:150-157

33. Ziari SA, Mireles VL (1996) Serum vitamin A, Vitamin E and beta carotene levels inPre-eclamptic women in Northen Nigeria. Am J Perinatol 13:287-291

34. Radhika MS, Bhaskaram P (2002) Effects of Vitamin C defecency during pregnancy on maternal and child health. BJOG 109:689-693

35. Mahadik KV, Sina SA (2003) Study of serum levels of SOD in pre-eclampsia and eclampsia:role of the test as a predictive tool. JObset Gynecol Rese 29:262-267

36. Joshi VK, Sapres, Govila V (2003) Role of micronutrients and calcium in pregnancy induced hypertension. Obs Gynae 8:617-619

37. Gilbert JS, Ryan MJ (2008) Pathophysiology of hypertension during pre-eclampsia:Linking placental ischemia with endothelial dysfunction. Am J Physiol Heart Circ Physiol 294:541-550

38. Sinclair AJ, BarnettAH, Lunec J (1991) Free radicals and antioxidant systems in health anddisease. J applied Med 17:409-414

39. Sing PP, Sharma P (2009) Antioxidant basket: donot mix apples and oranges. Ind J ClinBiochem 24:211-214