ISSN Print: 2394-7500
ISSN Online: 2394-5869
Impact Factor: 5.2
IJAR 2017; 3(9): 124-127
www.allresearchjournal.com
Received: 12-07-2017
Accepted: 15-08-2017

Nisith Kumar Mohanty
Department of Nephrology, IMS and SUM Hospital, Siksha "O" Anusandhan Deemed to be University, K8, Kalinga Nagar, Bhubaneswar, Odisha, India

Ashok Kumar Panda
Department of Nephrology, IMS and SUM Hospital, Siksha "O" Anusandhan University Deemed to be, K8, Kalinga Nagar, Bhubaneswar, Odisha, India

Corresponding Author:
Ashok Kumar Panda
Department of Nephrology, IMS and SUM Hospital, Siksha "O" Anusandhan University Deemed to be, K8, Kalinga Nagar, Bhubaneswar, Odisha, India

Assessment of serum lipid and uric acid profile in Preeclamptic women in a tertiary care teaching hospital

Nisith Kumar Mohanty and Ashok Kumar Panda

Abstract
Preeclamptic is a multi-system disorder of pregnancy, which is characterised by new onset hypertension and proteinuria that develop after 20 weeks of gestation in previously normotensive women. Pre-eclampsia affects approximately 3% of all pregnancies worldwide, with onset of symptoms in the late second or third trimester, commonly after 32nd week. Our aim was to evaluate the biochemical markers which include lipid profile and serum uric acid for early diagnosis of preeclampsia. The present study consists of total 120 women subjects between the age group 20-39 years who are further subdivided into two groups. Diagnosis was confirmed as per the standard criteria. Lipid profile and uric acid levels were estimated. Total Cholesterol, Triglycerides, LDL-c, VLDL-c, and uric acid levels were raised in pre-eclampsia and statistically significant. This association may be significant in understanding the pathological processes of pre-eclampsia and may help in developing strategies for prevention and early diagnosis of pre-eclampsia.

Keywords: Preeclampsia, Lipid profile, uric acid

Introduction
Pre-eclampsia PE influences around 3% of all pregnancies overall [1], with beginning of side effects in the late second or third trimester, most usually after the 32nd week. PE is a multi-framework issue of pregnancy, which is described by new beginning hypertension and proteinuria that create after 20 weeks of growth in already normotensive women [2, 3]. The disorder has a higher occurrence among nulliparous ladies, in ladies who imagine with helped proliferation strategies, and affected by autoimmune disorders, reflecting the probable influence of an "unpracticed" or dysregulated maternal immune system in its emergence [4, 5]. Then again, women with prior metabolic, vascular or renal sickness are particularly at expanded risk for superimposed PE [6]. The mechanism of relationship of preeclampsia is not well understood. The available tools for its diagnosis are operative only when the disease sets in, and in many cases at this stage. It becomes difficult to avoid impediments. It is necessary to diagnose this condition in advance so that the future complications of mother as well as fetus might be prevented.

Besides that Preeclampsia and related disorders are known to disturb function of various organs involved in lipid and lipoprotein absorption. Several studies have shown that endothelial dysfunction is related to hyperlipidemia [7]. Altogether raised plasma centralization of Triglycerides (TG), phospholipids and total lipids and decreased high density lipoprotein – cholesterol (HDL-C) concentrations were found in women with preeclampsia in comparison to normal pregnancy [8]. Early pregnancy dyslipidemia is associated with an increased risk of PE. In pregnancy, lipolysis of TG-rich lipoproteins is reduced because of decreased lipolytic activities of the mother. In PE, the vascularization of the fetoplacental unit may be impaired, resulting in yet-undefined compensatory mechanisms that may further increase synthesis of maternal Triglyceride (TG) levels. Also, the diminished catabolism of TG-rich lipoproteins by decreased placental take-up and the putative attendant lessening of lipoprotein lipolysis brings about the aggregation of TG-rich remainder lipoproteins in the maternal course. Remainder lipoproteins may initiate platelet enactment and endothelial brokenness, consequently prompting the major clinical side effects of PE [9, 10].

It is suggested that pregnancy interceded changes in serum uric acid are essentially the result of modified renal dealing with. Expanded serum uric acid in women with preeclampsia has been reliably depicted for over 80 years. The expansion in serum uric acid has been ascribed to diminished renal urate clearance because of renal dysfunction [11]. Taking these factors in to consideration, the present study includes assessment of lipid profile and uric acid levels in preeclampsia.

Materials and Methods

The present study was carried out at Department of nephrology, IMS and Sum hospital, Siksha 'O' Anusandhan University, Bhubaneswar, Odisha, India, during the period from January 2016 to June 2017. The study protocol was approved by the Ethics committee of IMS and Sum hospital. The present study consists of total 120 women included between the age group 20-39 years who are further again divided into two groups. One group having normotensive pregnant women (n= 50) as controls. Another group B, Consists of pregnant women with preeclampsia (n= 70) as real study cases. Written consent was taken indivisually from all women in this study. Patient height was measured, maintaining an accuracy of 0.5cm.Weight was measured, up to adjacent 100gm. Prepregnancy body mass index was calculated as weight in kilograms/height in square meters. According to WHO norm, normal BMI ranges from 18.5 to 24.9 kg/m2. BMI between 25-29.9 kg/m2 is overweight, Similarly a BMI > 30 kg/m2 is considered obese. Blood pressure was measured by sphygmomanometer in right arm in left lateral position after 10 minutes of rest. Preeclampsia was diagnosed as blood pressure >140/90 mmHg on 2 separate occasions 4 hours apart in association with proteinuria (>0.3gm in 24 hours or at least 1+ on dipstick examination). An overnight fasting blood sample were collected under all aseptic precautions 5-10 ml of blood was collected and analysed for all the standard parameter in biochemical analysis.

All values in this observational study were expressed as mean ± sd. Here we used student t-test and Pearson's correlation coefficient to get the statistical significance. P-value <0.05 was taken to be consideration as statistically significant.

Observation and output

The study was conducted on 120 nulliparous pregnant women in their third trimester between the ages twenty to thirty nine years. Seventy patients were of pre-eclampsia in this study group and 50 normotensive non-pre-eclamptic women in control group. Pre-eclampsia was diagnosed on the basis of history, clinical examination, blood pressure findings and presence of proteinuria. Table-1 shows the Demographic & Clinical characteristics of the Cases & Controls group. Table-2 shows the Comparison of biochemical parameters between the two groups where TG, VLDL, LDL, cholesterol, serum uric acid level were significantly higher in preeclamptic women ($p< 0.001$), while serum HDL was significantly low in preeclamptic women ($p< 0.001$).

Table 1: Demographic & Clinical characteristics of the Cases & Controls group

Parameters	Controls (n=50) (mean ± sd)	Cases (n=70) (mean ± sd)
Age (Yrs.)	22.06 ± 10.16	29.06 ± 10.16
Weight (kg)	44.06 ± 10.16	47.8 ± 7.7
Height (cm)	76.03 ± 6.90	77.85 ± 6.88
BMI (Kg/m^2)	27.0 ± 2.1	29.4 ±2.8
Systolic BP	114.76 ±0.43	152 ±0.47
Diastolic BP	68 ±0.82	104 ±0.26

Statistically Significant ($P< 0.05$)

Table 2: Comparison of biochemical parameters between the two groups

Parameters	Controls (n=50) (mean ± sd)	Cases (n=70) (mean ± sd)
Total Cholesterol (mg/dl)	176.4 ±12.07	218.34 ± 24.88
Triglycerides (mg/ dl)	129.84 ±11.32	187.4 ± 29.2
LDL-c(mg/ dl)	96.17 ±14.75	137.10 ±13.75
HDL-c(mg/ dl)	41.5 ± 7.9	29.6 ±4.80
VLDL-c(mg/ dl)	25.79 ±2.41	31.4 ±4.57
Uric acid (mg/ dl)	5.4 ±1.03	7.3 ± 0.6

All parameters Statistically Significant at ($P< 0.05$)

Discussion

Definition of PE has changed with due course of time. The underlying universal arrangement and meaning of the hypertensive issue of pregnancy assembled by Davey *et al*., characterized it to have a diastolic circulatory strain of 90 mm Hg on two events, or 110 mm Hg on a solitary event [13]. In PE the glomerular boundary is unquestionably changed and there is an expanded discharge of protein including egg whites. At the point when complete protein discharged (TPE) surpasses 1 g/24 hours or 1+ by dipstick, cylindrical protein reabsorption will be soaked and singular proteins discharge rates will be identified with their molecular loads [14]. We chose nullipara women for the present investigation as nulliparity isn't just have high danger of PE yet is the most widely recognized maternal hazard factor which can undoubtedly be surveyed just by the history. Duckitt *et al*., deduced in their investigation that nulliparity isn't just the most widely recognized maternal hazard factor yet it has been appeared to practically significantly increase the danger of PE [15].

In this study, the results revealed that mean maternal age, weight & height of preeclamptic women (group-B) was statistically significant than normotensive pregnant healthy women (group-A), ($p< 0.05$). The mean systolic and diastolic blood pressure in pre-eclamptic women (group-B) was significantly higher than normotensive pregnant women (group-A) (Table-1). Women with preeclampsia had significantly higher BMI compared with controls ($p< 0.05$) (Table-1) which is similar to finding of Sharami *et al*. [18]. Probable, mechanism of increased BMI in preeclampsia is increased insulin resistance and a state of inflammation associated with obesity [19]. Insulin resistance leads to lipolysis, leading to increased flux of fatty acids to liver promoting synthesis of TG20. Also maternal obesity is

independently associated with development of placental endothelial dysfunction and ultimately preeclampsia [19].

The mean levels of TG, VLDL, LDL and cholesterol were altogether higher in preeclamptic women than in normotensive controls ($p< 0.001$). Likewise there was a critical abatement in HDL in study bunch when contrasted with control ($p< 0.001$) (Table-2). Hypertriglyceridemia in preeclampsia is likewise ascribed to insulin opposition because of corpulence. During early pregnancy, anabolic stage empowers lipogenesis and fat stockpiling in anticipation of fast fetal development in late pregnancy. Along these lines there is physiologic hyperlipidemia with gestational ascent in triglyceride and cholesterol as high as a few times in third trimester [21]. Danger of preeclampsia was multiple times higher in ladies with raised TG [22]. Estrogen incites biosynthesis of endogenous triglyceride by invigorating hepatic lipase [23]. There is diminished action of lipoprotein lipase which is answerable for diminished catabolism at fat tissue level, Thus in preeclampsia, there is hypertriglyceridemia while placental VLDL receptors are up controlled. This resultant in rerouting of TG rich lipoproteins to feto-placental unit. Anyway in preeclampsia the vascularization of fetoplacental unit might be impeded, coming about in yet-indistinct compensatory systems that may additionally build combination of maternal TG levels [24]. As of now talked about, weight and insulin obstruction likewise advances union of TG.

VLDL transports TG in fringe blood in this way hypertriglyceridemia additionally prompts expanded serum levels of VLDL [19]. As of now talked about, insulin obstruction causes lipolysis, prompting expanded transition of unsaturated fats to liver advancing union of VLDL [20]. Expanded LDL levels are because of raised estrogen and progesterone levels in preeclampsia. It has been indicated that LDL (extraordinarily oxidized LDL) increments blood vessel affectability to pressor specialists and represses endothelium dependant vasodilatation. This endothelial brokenness, prompts glomerular sores and hence proteinuria, which additionally gives a sign of its seriousness. Low HDL in preeclampsia is because of insulin opposition [26]. As indicated by Pirzado *et al*. [27], there is an immediate relationship between's fat tissue lipoprotein lipase movement and plasma HDL. This is answerable for low degrees of HDL. HDL conveys abundance, possibly hurtful cholesterol from fringe tissues to liver, where it very well may be discharged. Also, it is engaged with initiating lipoprotein which discharges unsaturated fats that can be oxidized by β-oxidation pathway to give vitality. Low degrees of HDL may reduce these capacities.

Hypercholesterolemia promotes formation of free radicals. Thus several studies have linked atherogenic lipid profile as a potential contributor to increased risk of preeclampsia [19]. Thus, dyslipidemia mediated endothelial dysfunction & placentally derived endothelial disturbing factors like lipid peroxides could possibly contribute in pathogenesis of pregnancy induced hypertension. Thus, estimation of lipid profile may have a predictive role in assessing extent of endothelial damage and may help by preventing or foreseeing complications in pre-eclampsia [21]. Serum uric acid level were significantly higher in preeclamptic women ($p< 0.001$). This is consistent with previous studies [28, 29]. Excessive cellular activity is associated with placental ischemia also leads to overproduction of uric acid which serves as a marker of the disease. Uric acid levels have been consistently reported to be elevated in preeclampsia. Hyperuricemia may predate proteinuria by several weeks [28, 29]. Previous studies also indicate that measurement of serum uric acid may be a better indicator of fetal prognosis as compared to blood pressure in preeclampsia [30]. Monitoring of serum uric acid level in those with preeclampsia will help to predict those that will develop eclampsia [31].

Conclusion

These discoveries propose that total Cholesterol, Triglycerides, LDL-c, VLDL-c, and uric corrosive levels were brought up in pre-eclampsia and factually noteworthy, whereas HDL-c levels were brought up in these patients however measurably no significant, it very well may be inferred that there exists a relationship in lipid profile and uric corrosive with preeclampsia in this way dyslipidemia and raised uric acid levels are the highlights of pre-eclampsia in nullipara pregnant patiets in their third trimester. This relationship might be critical in understanding the obsessive procedures of preeclampsia and may help in creating systems for counteractive action and early conclusion of preeclampsia.

Reference

1. World Health Organization (WHO) World health report: Make every mother and child count. Geneva: WHO, 2005, 63. [Google Scholar]
2. Redman CW, Sargent IL. Latest advances in understanding preeclampsia. Science. 2005; 308:1592-94. [PubMed] [Google Scholar]
3. Sibai B, Dekker G, Kupferminc M. PE. Lancet. 2005; 365:785-99. [PubMed] [Google Scholar]
4. Saito S, Shiozaki A, Nakashima A, Sakai M, Sasaki Y. The role of the immune system in preeclampsia. Mol Aspects Med. 2007; 28:192-209. [PubMed] [Google Scholar]
5. Sargent IL, Borzychowski AM, Redman CW. Immunoregulation in normal Pregnancy and PE: an overview. Reprod Biomed Online. 2006; 13:680-86. [PubMed] [Google Scholar]
6. Catov JM, Ness RB, Kip KE, Olsen J. Risk of early or severe preeclampsia related to pre-existing conditions. Int. J Epidemiol. 2007; 36:412-19. [PubMed] [Google Scholar]
7. Robert JM, Redman CWG. Preeclampsia: more than pregnancy induced hypertension. Lancet. 1993; 41:1447-51.
8. Wockhardt Hospitals Blog, Causes, Symptoms and Complications of Preeclampsia: Is your Pregnancy at Risk, Thursday, 2009.
9. Lyall F, Ian AG. The vascular endothelium in normal pregnancy and pre-eclampsia. Reviews of Reproduction 1996; 1:107-16.
10. Robert JM, Lain KY. Recent insight into the pathogenesis of preeclampsia. Placenta 2002; 23:359-72.
11. Gary Cummingham F, Norman FG, Kenneth *et al*. Hypertensive disorders in pregnancy, Williams Obstetrics, 22 Edition, Me. Graw Hill, 2005, 761-764.
12. Tietz Kidney function tests. Tietz textbook of clinical chemistry and molecular diagnostics. 4th edition. Elsevier, 807.
13. Davey DA, Macgillivary I. The classification and definition of the hypertensive disorders of pregnancy.

Am J Obstet Gynecol. 1988; 158:892-98. [PubMed] [Google Scholar]
14. Davison JM. The kidney in pregnancy. Martinus Nijhoff, Boston; Renal function during normal pregnancy and the effect of renal disease and PE, 1986, 65-80. In: Andreucci VE (Ed) [Google Scholar]
15. Duckitt K, Harrington D. Risk factors for PE at antenatal booking: systematic review of controlled studies. BMJ. 2005; 330:565.
16. Sharami SH, Tangestani A, Faraji R, Zahiri Z, Azam A. Role of dyslipidemia in pre-eclamptic overweight pregnant women. Iran J Reprod Med. 2012; 10:105-112.
17. Ephraim R, Doe PA, Amoah S, Antoh EO. Lipid profile and high maternal body mass index is associated with preeclampsia: A case-control study of the Cape Coast Metropolis. Ann Med Health Sci. Res. 2014; 4:746-750.
18. Pradnya Phalak, Mona Tilak. Study of lipid profile in pre-eclampsia. Indian Journal of Basic & Applied Medical Research. 2012; 5(2):405-409.
19. Ray JG, Vermeulen MJ, Schull MJ, Redelmeier DA. Cardiovascular health after maternal placental syndromes (CHAMPS): Population-based retrospective cohort study. Lancet. 2005; 366:1797-1803.
20. Swapan Das, Debasish Char, Sanjay Sarkar, Prakash Das, Tushar Kanti Saha, Sucheta Biswas. Comparison of lipid profiles in normal pregnancy and in pre-Eclampsia: A case control study. IOSR Journal of Dental and Medical Sciences. 2013; 11(4):53-55.
21. Karl W, Birgit W, Michael MH *et al*. Triglyceride rich lipoproteins are associated with hypertension in preeclampsia. The Journal of Clinical Endocrinology & Metabolism. 2003; 88(3):1162-1166.
22. Cekmen MB. Erhagci AB, Balat A, Duman C, Maral H, Ergen K *et al*. Plasma lipid and lipoprotein concentrations in pregnancy induced hypertension. Clin. Biochem. 2003; 36(7):575-578.
23. Pirzado ZA, Sangi SA, Malik R. High density lipoprotein cholesterol metabolism and its role in ischemic heart disease. Pak J Med Res. 1999; 38:38-41.
24. Hawkins TL, Roberts JM, Mangos GJ, Davis GK, Roberts LM, Brown MA. Plasma uric acid remains a marker of poor outcome in hypertensive pregnancy: A retrospective cohort study. BJOG. 2012; 119:484-492.
25. Wu Y, Xiong X, Fraser WD, Luo ZC. Association of uric acid with progression to preeclampsia and development of adverse conditions in gestational hypertensive pregnancies. Am J Hypertens. 2012; 25:711-717.
26. Varma TR. Serum uric acid levels as an index of fetal prognosis in pregnancies complicated by pre-existing hypertension and pre-eclampsia of pregnancy. International Journal of Gynecology& Obstetrics. 1982; 20(5):401-8.
27. Sahijwani D, Desai A, Oza H *et al*. Serum Uric acid as prognostic marker of pregnancy induced hypertension. Journal of South Asian federation of Obstetrics & Gynecology. 2012; 4(3):130-3.