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Review Article

Dyslipidemia in Pregnancy-Induced Hypertension

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Abstract

Pregnancy-induced hypertension (PIH) is a leading cause of maternal mortality and morbidity. Abnormal lipid profile and increased lipid peroxidation activate endothelial dysfunction causing PIH. Estimation of lipid profile in pregnancy may be helpful in predicting the development of PIH and further progression. Early diagnosis of dyslipidemia may prevent PIH thereby preventing obstetric complications. Dyslipidemia is known to be associated with essential hypertension but their role in PIH remains unclear. Early identification of at-risk women may help in taking timely preventive and curative management to prevent pregnancy related complications.

Key words*: Pregnancy-induced hypertension; Eclampsia; Dyslipidemia; Lipid profile*

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Introduction

Hypertensive disorders complicate 5–10% of all pregnancies.[1] Pregnancy-induced hypertension (PIH) includes a group of hypertensive disorders developed due to the gravid state after 20 weeks of pregnancy. Pregnancy is a physiological phenomenon accompanied by a high-energy demand with an increased oxygen requirement. This triggered aerobic environment should primarily be responsible for raised oxidative stress in pregnancy that adversely affects the mother and the fetus.[2]

The pathophysiology of PIH is still unknown. An imbalance between reactive oxygen species and antioxidants appears to be an important contributing factor.[3] Altered lipid profile and increased lipid peroxidation leading to decrease in prostacyclin (PGI_2):thromboxane A_2 (TXA_2) ratio causes the vasospastic phenomenon in kidney, uterus, placenta and brain as seen in PIH.[4] Abnormal lipid metabolism is responsible for the endothelial dysfunction,[5] and it was proposed that oxidized low-density lipoproteins (LDL) may add to endothelial dysfunction in pre-eclampsia.[6] Triglyceride (TG) rich lipoproteins may also activate endothelial dysfunction and atherothrombosis.[2]

The association of dyslipidemia with PIH has been highly suggested. Therefore estimation of lipid profile in pregnancy may be helpful in predicting the development of PIH and further progression can be monitored and managed, thereby preventing maternal and fetal complications.

Pregnancy-induced hypertension[7]

Gestational hypertension

Systolic BP ≥ 140 or diastolic BP ≥ 90 mm Hg for first time during pregnancy (after 20 weeks of gestation) without proteinuria

Preeclampsia

Systolic BP ≥ 140 or diastolic BP ≥ 90 mm Hg after 20 weeks of gestation with proteinuria ≥ 300 mg/24 hours or ≥ 1+ on dipstick testing

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Eclampsia

Women presenting with convulsion/coma along with features of preeclampsia

Lipid metabolism in preeclampsia

In pregnancy, lipolysis of TG-rich lipoproteins is reduced because of decreased lipolytic activities of the mother whereas placental VLDL receptors are up-regulated. This results in a rerouting of TG-rich lipoproteins to the fetoplacental units. In the vascularisation of the fetoplacental unit may be impaired, resulting in compensatory mechanisms that may further increase synthesis of maternal TG levels. The decreased catabolism of TG-rich lipoproteins by reduced placental uptake and decrease of lipoprotein lipolysis result in the accumulation of TG-rich remnant lipoproteins in the maternal circulation. Remnant lipoproteins may induce platelet activation and endothelial dysfunction, leading to the major clinical symptoms.[8]

The hormonal imbalance is a prime factor for the etiopathogenesis of preeclampsia. Preeclampsia is a state of hypoestrogenemia.[9] Decreased uteroplacental blood flow which is the main pathophysiological event in preeclampsia leads to impairment in the formation of dehydroepiandrosterone sulphate (DHEAS) by fetal adrenal glands. DHEAS is the important source of estrogen in pregnancy. Hypoestrogenemia also leads to decreased expression of VLDL/apo E receptors resulting in reduced transport of VLDL to fetal compartment resulting maternal hypertriglyceridemia. Further LDL taken up by the fetus for the synthesis of DHEAS is decreased due to reduced fetoplacental perfusion leading to increased LDL.[8]

The elevated triglycerides result in increased atherogenic small dense LDL and reduced HDL levels in gestational hypertension. Hypothesized mechanisms for the dyslipidemia and preeclampsia association have been described in literatures. Investigators have noted that elevated plasma lipid and lipoprotein may induce endothelial dysfunction secondary to oxidative stress. They noted that dyslipidemia may impair trophoblast invasion thus contributing to a cascade of pathophysiologic events that lead to the development of preeclampsia.[10,11]

Risk factors - NICE and PRECOG Guidelines

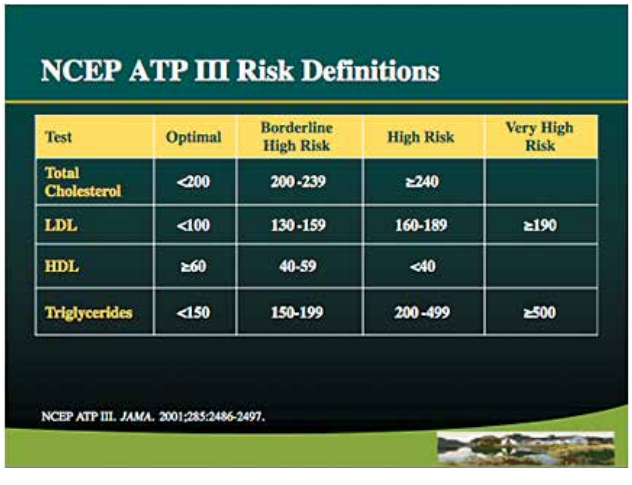
NICE guidelines 2008

Previous preeclampsia
Multiple pregnancy
Pre existing vascular disease such as hypertension or pre existing renal disease
Nulliparity
Pregnancy interval of more than 10 years
Age ≥ 40 years
Body mass index ≥ 30
Family history of preeclampsia

Preeclampsia community guidelines (PRECOG)

Previous preeclampsia
Multiple pregnancy
Underlying medical conditions
Pre-existing hypertension/ renal disease/ diabetes
Presence of anti phospholipid antibodies
Or any two of the following:
Nulliparity
Pregnancy interval of more than 10 years
Age ≥ 40 years
Body mass index ≥ 35
Family history of preeclampsia
Booking diastolic pressure ≥ 80 and ≤ 90 mmHg

ATP III classification of lipid profiles



Comparison of lipid profiles of PIH subjects of different studies

Authors	TC (mg/dL)	HDL-C (mg/dL)	LDL-C (mg/dL)	TG (mg/dL)
Saxena et al[2] (n=70)	226.2±43.9	41.6±9.0	127.8±39.5	283.4±73.9
Singh et al[12] (n=25)	218.40±40.29	45.38±22.74	136±46.2	210±57.3
Vani et al[9] (n=50)	227±31	51±5	132±34	219±58
Enquobahrie et al[13] (n=57)	198.11±3.72	64.14±1.43	107.54±2.95	137.81±6.69
Shivanagappa et al[14] (n=70)	259.9±76.6	43.3±14.7	154.68±47.9	275.38±123.24
De et al[15] (n=50)	236.3±35.63	45.9±8	135.4±23.36	275.6±38.93
Belo et al[16] (n=51)	268.2±94.7	54.2±14.5	140.3±60.9	238.8±85.6
Evruke et al[17] (n=84)	237.7±58.4	50.2±15.3	137.1±42	251.9±108.6
Sahu et al[18] (n=30)	293.3±15.7	50±2.7	196.7±15.3	233.57±34.6

Conclusion

Normal human pregnancy results in pronounced physiologic hyperlipidemia involving gestational rise in blood triglycerides and cholesterol. Women with preeclampsia show additional alteration in lipids reflecting a disordered lipid and lipoprotein metabolism. Predominantly low HDL and high triglyceride concentration may promote vascular dysfunction and oxidative stress seen in hypertensive disorders in pregnancy. Future studies are required to evaluate causative factors for altered lipid profile in preeclampsia.

References

1. Cunningham FG, Leveno KJ, Bloom SL, Hauth JC, Rouge DJ, Spong CY. Pregnancy Hypertension. In: Williams obstetrics. 23rd edition. New York: McGraw-Hill; 2010. P.706–756.
2. Saxena S, Thimmaraju KV, Srivastava PC, Mallick AK, Das B, Sinha N et al. Role of dyslipidaemia and lipid peroxidation in pregnancy induced hypertension. J Clin Sci Res 2015; 4: 205–212.
3. Boutet M, Roland L, Thomas N, Bilodeau JF. Specific systemic antioxidant response to preeclampsia in late pregnancy: the study of intracellular glutathione peroxidases in maternal and fetal blood. Am J Obstet Gynecol 2009; 200: 530 e1–7.
4. Robson SC. Hypertension and renal disease in pregnancy. In: Edmonds DK, editor. Dewhurt's textbook of obstetrics and gynaecology for postgraduates. 6th edn. New York: Blackwell Science Ltd. 1999.p.167–169.
5. Zeiher AM, Drexler H, Wollschlager H, Just H. Modulation of coronary vasomotor tone in humans. Progressive endothelial dysfunction with different early stages of atherosclerosis. Circulation 1991; 83: 391–401.
6. Sattar N, Bendomir A, Berry C, Shepherd J, Greer IA, Packard CJ. Lipoprotein subfraction concentrations in pre-eclampsia: Pathogenic parallels to atherosclerosis. Obstet Gynecol 1997; 89: 403.
7. Dutta DC. Brothers Medical Publishers; 2013.p.219.
8. Vani I, Gayathri A, Nagamani T, Sunieeta H. Lipid profile parameters in normal and preeclampsia complicating pregnancies- A prospective observational study. The Ame J Sci & Med Res 2015; 1(1): 61–66.
9. Leeman L, Fontaine P. hypertensive disorders of pregnancy, American Family Physician 2008; 78(1): 93–100.
10. Sachdeva PD, Patel BG, Bhatt MV. A study of incidence and management of pregnancy induced hypertension in central Gujarat, India. International Journal of Universal Pharmacy and Life Sciences 2011; 1(3): 61–70.
11. WHO-Global Database on Body mass index.
12. Singh A, Khambra P, Rani KU, Mandal AK.

Assessment of Serum βhCG, lipid profile and uric acid levels in early second trimester as predictors of pregnancy induced hypertension. Annals of Pathology and Laboratory Medicine 2016; 3(3): 157–161.

13. Enquobahrie DA, Williams MA, Butler CL, Frederick IO, Miller RS, Luthy DA. Maternal plasma lipid concentrations in early pregnancy and risk of preeclampsia. The American Journal of Hypertension 2004; 17: 574–581.
14. Shivanagappa M, Singh S, Sujatha MS, Mahadevaih M. Comparative study of lipid profile in normotensive and hypertensive pregnant women. International Journal of Scientific Study 2015; 7(3): 222–225.
15. De J, Mukhopadhay AK, Saha PK. Study of serum lipid profile in pregnancy induced hypertension. Indian Journal of Clinical Biochemistry 2006; 21(2): 165–168.
16. Belo L, Caslake M, Gaffney D, Santo-Silva A, pereira-Leite L, Quintaniha A et al. Changes in LDL size and HDL concentration in normal and preeclamptic pregnancies. Atherosclerosis 2002; 162: 425–432.
17. Evruke IC, Demir SC, Urunsak IF, Ozgunen FT, Kadayifci O. Comparison of lipid profiles in normal and hypertensive pregnant women. Ann Saudi Med 2004; 24: 382–385.
18. Sahu S, Abraham R, Vedavalli R, Daniel M. Study of lipid profile, lipid peroxidation and vitamin E in pregnancy induced hypertension. Indian J Physiol Pharmacol 2009; 53: 365–369.