Hyperinsulinemia and Preeclampsia among Pregnant Women: A Quantitative Study

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Author's contribution

The sole author designed, analyzed, interpreted and prepared the manuscript.

Original Research Article

ABSTRACT

Hypertensive disorders in pregnancy complicate 1 in 10 pregnancies, often associated with maternal and neonatal mortality and morbidity. The study was done on 100 pregnant women's attending antenatal clinic of SreeBalaji Medical College & Hospital, Chromepet, Chennai, during the period from December 2013 - May 2015. All women who met the inclusion and exclusion criteria were taken intothe study. Fasting insulin was calculated at 18 -22weeks of gestation, and then follow up of caseswas donefor signs of development of Pregnancy-induced hypertension (PIH). In the hypertensive group, the bulk of the patients were between the ages of 25 and 30. As (P > 0.05), there is no statistical Significance in correlation between age and (PIH) in our study. Out of 100 cases studied, 64 cases (64%) had hyperinsulinemia. The remaining 36 cases (36%) had normal insulin values. The prevalence of PIH associated with hyperinsulinemia was 58% in our study. This test showed sensitivity of 87%, specificity of 82%, positive predictive value of 90%, negative predictive value 77% and diagnostic accuracy was 92%.

Keywords: Hyperinsulinemia; preeclampsia; hypertension.

1. INTRODUCTION

Pregnancy is a physiological stress associated with many complex and interrelated biochemical, physiological and anatomical alterations occurring in the body. More emphasis is laid on the biochemical changes seen in the blood during the normal pregnancy and are

exaggerated in various complications of pregnancy.Hypertensive disorders of pregnancy, one of the commonest complications of pregnancy, are a leading cause of maternal mortality. The hypertensive disorders of pregnancy are high in developing countries. Collectively these disorders complicate 5-10% of all pregnancies [1].

Hypertension in pregnancy is also responsible for fetal and infant mortality as well as 46% of infants born small for gestation [2]. Similarly it was estimated that 3-10% of infants are growth restricted. Fetal growth restriction is associated with substantiate perinatal morbidity and mortality [3,4,5].

2. MATERIALS AND METHODS

This study is a prospective study which involves a group of 100 women who attend the antenatal clinic of SreeBalaji Medical College and Hospital. The purpose of this study is to estimate fasting insulin as a predictor of pregnancy induced hyper tension.

The values are tabulated. Subsequently the subjects were followed up for regular antenatal care at 2 weekly intervals until 36 weeks & weekly thereafter until delivery and 4 weeks after delivery till 12 weeks. The primary outcome is the development of gestational hypertension.

The statistical analysis has been carried out with IBM SPSS Version - 20. Categorical variables will be analyzed with chi square test. Continuous variables are presented as Mean (SD). For normally distributed data (e,g., BMW, Age, and BP) between groups' analyses was done. For statistical significance, a two tailed probability value of less than 0.05 will be considered.

3. RESULTS

Table 1 shows, 66% in pregnancy induced hypertension group, remaining 34% are in normal group.

Table 1. Incidence of pregnancy induced hypertension

Incidence of PIH	No of cases	Percentage
Normal	34	34.0%
PIH	66	66.0%
Total	100	

Fig. 1 show the age distribution, mean age of the women in this study group with PIH is 31-37 years. Majority of women under the age group 18 -24 years in normotensive patients. Where as in hypertensive group majority of the patients were under 25 -30 years age group. As (P > 0.05), there is no statistical Significance in correlation between age and pregnancy induced hypertension.

Fig. 2 shows, hypertensive patients most of the cases were under 29 .9 BMI classification, when compared to normotensive patients. Chi square value =0.1 50, p >0.05. There is no significant difference in BMI groups and development of PIH.

Fig. 3 shows distribution according to parity, Majority of them were multigravida. There is no statistical significant difference in parity between normotensive and hypertensive patients.

Table 2. Family history

Family history	No of cases	Percentage
No	90	90.0
YES	10	10.0
Total	100	100.0

Table 2 shows, In the present study, 10% had family history of pregnancy induced hypertension, whereas remaining 90% were nil significant.

Of the 25 vaginal deliveries, 12 were spontaneous; one case was induced with misoprostol and12 cases with prostaglandin E2 gel. All the cases were monitored alternately by antepartum surveillance and CTG. ARM and Oxytocin acceleration was done in as per necessary with close monitoring of fetal heart rate. 3 cases required forceps extraction and 2 cases vacuum application (Table 3).

Of the total cases, 8 women had previous LSCS, repeat LSCS was done for them.In rest 62 cases and Emergency LSCS was done in view of both abnormal CTG pattern and fetal distress, failed induction.

In the present study, babies of 56.06% (37/66) with hyperinsulinemia had NICU stay which indicates adverse perinatal outcome compared to normal insulin levels and 20.58%(7 /34) of the babies born to cases with normal insulin levels had NICU stay (Table 4).
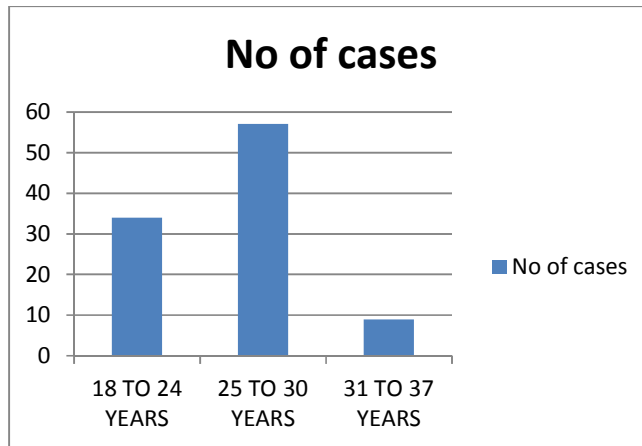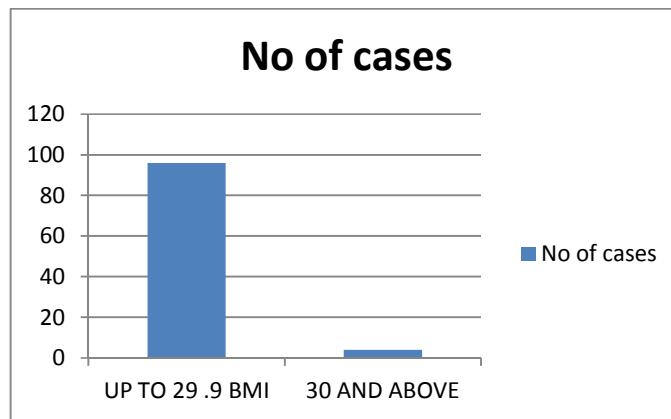
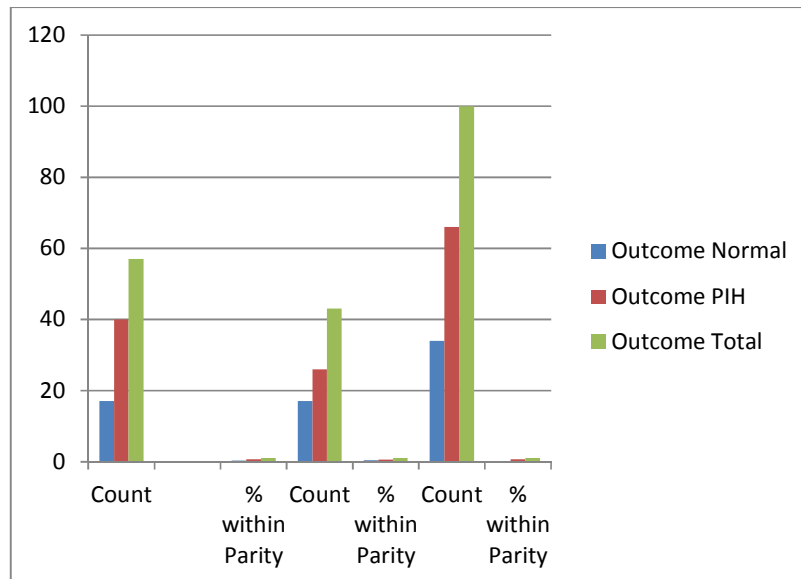**Fig. 1. Age distribution in years**

Fig. 2. BMI with outcome

Fig. 3. Parity with outcome

Table 3. Mode of delivery

Mode of delivery	No of cases (hyperinsulinemia)	No of cases· (normal insulin)	Total
Elective LSCS	4	4	8
Emergency LSCS	40	22	62
Spontaneous vaginal delivery	18	7	25
Outlet forceps application	1	2	3
Vacuum assisted	1	1	2
Total	64	36	100

Table 4. Hyperinsulinemia and Perinatal Outcome

Insulin	Healthy baby		NICU		Still birth		Total	
	No	%	No	%	No	%	No	%
Hyperinsulinemia	29	43.93	37	56.06	0	0	66	66%
Normal insulin	26	76.47	7	20.58	1	2.94	34	34%
Total	55	55	44	44	1	1	100	100.00

4. DISCUSSION

In 5 to 10% of pregnant women, hypertension develops, making it the leading cause of maternal death in our country and around the world. Establishing an accurate diagnosis of preeclampsia and proceeding with early treatments after it is established is one strategy to lessen the impact of arterial hypertension on maternal mortality.Preeclampsia is diagnosed when a pregnant woman has high blood pressure (> 140/90 mm Hg) after the 20th week of pregnancy (without a previous history of arterial hypertension) and substantial proteinuria (>300 mg in 24 hours). [6,7].

These symptoms are now thought to be a late symptom of an illness that has been present since the first trimester of pregnancy. Because of this "diagnostic delay," numerous tests have attempted to diagnose preeclampsia as soon as possible, sometimes even before the patient develops arterial hypertension.Doppler ultrasonography examination of maternal and foetal circulation, as well as uric acid, have been reported as early diagnostic tests for preeclampsia [8,9]. Hyperinsulinemia has been seen in patients with pregnancy-induced hypertension, according to the investigations. In both cross-sectional and cohort studies, pregnancy-induced hypertension has been linked to hyperinsulinemia.The majority of women with hyperinsulinemia had pregnancy-induced hypertension, according to the current study. In

our study, the prevalence of PIH associated with hyperinsulinemia was 58 %. At 12-20 weeks of pregnancy, fasting insulin was determined, and cases were followed up on for symptoms of PIH development. This test showed sensitivity of 87%, specificity of 82%, positive predictive value of 90%, negative predictive value 77% and diagnostic accuracy was 92%.

All the cases were monitored by ante partum surveillance. In the present study, 25women had vaginal deliveries. Of these, 12 had spontaneous onset of labour while others were induced. One case was induced with misoprostol and12 cases with prostaglandin E2 gel, after assessing their Bishop's score. Indication for induction was severe pre-eclampsia. During labour, ARM and Oxytocin acceleration was done when required with close monitoring of fetal heart rate.3 cases required forceps and in 2 cases vacuum was applied.

In the present study, babies of 56.06% (37/66) with hyperinsulinemia had NICU stay which indicates adverse perinatal outcome (in terms of neonatal complications which included birth asphyxia, Meconium aspiration syndrome, hyperbilirubinemia, respiratory distress syndrome, etc) compared to normal insulin levels in which 20.58% (7 /34) of the babies had NICU admission. Whereas, Gupta [10] 55.1% of cases with hyperinsulinemia had NICU admission. The prevalence of PIH associated with hyperinsulinemia was 58% in our study. This test

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showed sensitivity of 87%, specificity of 82%, positive predictive value of 90%, negative predictive value 77% and diagnostic accuracy was 92%. Early screening for preeclampsia may allow vigilant antenatal surveillance and appropriate timing of fetal delivery in order to avoid serious sequelae. Preeclampsia is frequently described as a state of insulin resistance [11]. Many features of insulin resistance like hypertension, hyperinsulinemia, glucose intolerance and lipid abnormalities are associated with this condition [12]. Insulin resistance, inflammation and atherosclerosis appear to be linked via the metabolic syndrome. Insulin resistance 1s associated with elevated levels of various proinflammatory markers which cause endothelial dysfunction and initiate the atherosclerotic cascade [13]. In several studies conducted postpartum, women with a history of preeclampsia have been shown to be more insulin resistant when compared with women with normotensive pregnancy [14-16].

5. CONCLUSION

The high frequency of preeclampsia in women with diabetes underscores the need for more study into the pathogenesis, prognostic markers, therapy, and long-term health consequences of preeclampsia in this population. From this study, we can conclude that pregnant women with increased insulin values are likely develop pregnancy induced hypertension. The occurrence of preeclampsia is significantly linked to increased insulin resistance. Insulin resistance can thus be utilized as a biomarker for preeclampsia diagnosis.

DISCLAIMER

The products used for this research are commonly and predominantly use products in our area of research and country. There is absolutely no conflict of interest between the authors and producers of the products because we do not intend to use these products as an avenue for any litigation but for the advancement of knowledge. Also, the research was not funded by the producing company rather it was funded by personal efforts of the authors.

CONSENT AND ETHICAL APPROVAL

Informed consent was obtained from all study participants and as per international standard or university standard written ethical approval has been collected and preserved by the author(s).

ICH /GCP guidelines were followed.

COMPETING INTERESTS

Author has declared that no competing interests exist.

REFERENCES

1. Andrea G, Witlin, siblings BM. Hypertension in pregnancy: current concepts of preeclampsia. Ann Rev Med. 1997;48:115-127
2. Montan S, Sjoberg 0-0, Svenningsen N. Hypertension in pregnancy-fetal and infant outcome. Clin Exp Hypertens. 1987;B62:337-348.
3. Paz I, Gale R, Laor A, Danon YL, Stevenson DK, Seidmans D, The cognitive outcome of full term small for gestational age infants at late adolescence. Obstet Gynecol. 1995;85 :452.
4. Piper JM, Xenakin CMJ, McFarland M, Elliot BD, Barkers MD, Langer D: Do growth retarded premature infants have different rates of perinatal morbidity and mortality than appropriately grown premature infants? Obstet Gynecol 1996;87:169.
5. Minior VK, Divon MY: Fetal growth restriction at term: Myth or reality? Obstet Gynecol. 1998; 92:57.
6. Ekholm E. Hemodynamic measures in prediction of pre- eclampsia. Acta Obstet Gynecol Scand 1997;76:101- 103.
7. Grunewald C. Biochemical prediction of pre- eclampsia ..Acta Obstet Gynecol Scand 1997;(Suppl 164);76:104- 107.
8. Jose L. Bartha, Raquel Romero-Carmona, Rafael Torrejon-Cardoso, Rafael Cormoino-Delgado, Insulin, insulin-like growth factor-I and insulin resistance in women with pregnancy-induced hypertensio n, Am J Obstet Gynecol. 2002;187:735-402
9. Ellen W. Seely, Caren G. Solomon, Insulin resistance and its potential role in pregnancy induced hypertension, J Clin Endocrinol Metab. 2003;88:2392-2398.
10. Gupta DS. Recent advances m obstetrics and gynecology, Istedn.,vol. 8, New Delhi, Jaypee Brothers medical Publishers pvt. Ltd.1994:14-31.
11. Julie Nigro, Narin Osman, Anthony M. Dart, Peter J. Little, Insulin resistance and atherosclerosis, Endocrine Reviews. 2006;27:242- 259.

12. Kim E. Innes, Jeffrey H. Wimsatt, Pregancy induced hypertension and insulin resistance: evidence for a connection, Acta Obstet Gynecol Scand 1999;78:263- 284.

13. Nisell H, Erikssen C, Persson B, Carlstron K. 1999. Is carbohydrate metabolism altered among women who have undergone a preeclamptic pregnancy? GynecolObstet Invest 48:241-246.

14. Bauman WA, Maeimen M, Langero, Solomon A, B. An association between hyper insulinemia and hypertension during 3rd trimester of pregnancy. Am J Obstet Gnynaecol. 1998;159(2):446-50.

15. Solomon CG, Caroll JS, Okumura K, Graves SW, Seely EW. Higher cloesterol and insulin levels are associated with increased risk for pregnancy induced 8hypertension. Am J Hypertens. 1999;12:276-82.

16. Kaaja R, Tikkanten MJ, Vinikka L, Ylikorkala O. Serum lipoproteins insulin and urinary prostanoid metabolites normal and hypertensive pregnant women. Obstet Gynecol 1995; 85:353-56.

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