

1 Study of Feto-Maternal Outcome in Pregnancy Induced 2 Hypertension

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6

7 **Abstract**

8 Introduction: Pregnancy induced hypertension is one of the most common causes of both
9 maternal and fetal morbidity and mortality. This study aims to determine the feto-maternal
10 outcome and correlation with severity of PIH. Material and methods: 250 cases of PIH were
11 studied and divided according to severity. The maternal and fetal outcome parameters were
12 documented and analysed using statistical methods. Results: More the severity of PIH, more
13 are the chances of maternal and fetal complications. Earlier onset of PIH was also seen more
14 in severe cases as were the number of inductions. Conclusion: The clinical course of PIH is
15 progressive and is characterised by continuous deterioration that is ultimately stopped only by
16 delivery. Early detection and appropriate management of the pregnancy may improve the
17 outcome for both the mother and the fetus.

18

19 **Index terms**— pregnancy induced hypertension, maternal outcome, fetal outcome.

20 **1 Introduction**

21 Pregnancy induced hypertension is one of the most common causes of both maternal and fetal morbidity and
22 mortality. It is a pregnancy specific syndrome that can virtually affect every organ system. It is a challenge to
23 be addressed and overcome if there is to be any significant improvement in maternal and perinatal health.

24 Although the cause of PIH still remains unknown, evidence for its manifestation begins early in pregnancy.
25 Covert pathophysiological changes occur that gain momentum across gestation and eventually become clinically
26 apparent. Unless delivery supervenes, these changes ultimately result in multi -organ involvement with a clinical
27 spectrum ranging from barely noticeable to one of cataclysmic deterioration.

28 Eclampsia, disseminated intravascular coagulopathy, acute renal failure, HELLP syndrome, intracerebral
29 haemorrhage, antepartum haemorrhage and even maternal death can occur. Long term complications like
30 persistent hypertension and cardiovascular morbidity are known risks for the mothers suffering from PIH.

31 Fetal complications like intra -uterine growth retardation, sudden intra -uterine fetal death, still births, preterm
32 and low birth weight babies, increased need for NICU care, increased neonatal morbidity and mortality are
33 prevalent.

34 An attempt has been made in the present study to identify the factors affecting feto -maternal outcome in cases
35 of pregnancy induced hypertension so as to be able to identify them at the earliest and offer a better outcome to
36 both mother and baby.

37 **2 II.**

38 **3 Materials and Methods**

39 This was a prospective study carried out over a period of 1 year from 1 st Jan 2009 till 31 st Dec 2009 at
40 Grant medical college and Sir J. J. Group of hospitals after clearance from ethical committee. 250 patients of
41 pregnancy induced hypertension were studied. They were divided into mild, moderate and severe PIH. The cases

8 DISCUSSION

42 with systolic blood pressure greater than 130 mmHg, diastolic blood pressure greater than 90 mmHg on two
43 measurements taken 6 hours apart, in association with proteinuria more than 300 mg in 24 hours urine were
44 included in the mild preeclampsia group. The cases were accepted as mild preeclampsia if the the diastolic blood
45 pressure was less than 100 mmHg and as moderate preeclampsia if the diastolic blood pressure was 110 mmHg.
46 Severe cases were defined if the following criteria were present: Systolic blood pressure ? 160 mm Hg, Diastolic
47 blood pressure ? 110 mm Hg and Proteinuria 3+ or more.

48 A prestructured proforma was filled and parameters of maternal and fetal outcome were tabulated. Statistical
49 tests like the Chi square test and calculation of Spearman's rho were applied. A p value < 0.001 was accepted
50 as significant. The results obtained were compared with other studies from textbooks and journals. 149 (60%)
51 of the patients were primigravidae, 55 (22%) were of second gravida, 27 (11%) were G3 and 19 (7%) were
52 grand multigravidae. 32 i.e. 68% of the patients with severe PIH were primigravidae and 5 (10.7%) were grand
53 multigravidae. 6 (12.8%) and 4 (8.5%) were of the second and third gravida respectively.

54 4 III.

55 5 Results

56 6 There

57 155 i.e. 62% of the patients had > 3 ANC visits, 72 i.e. 28% had between 1 and 3 visits while 23 i.e. 9.2% were
58 unregistered.

59 There is a significant negative correlation between the number of ANC visits and PIH severity when analysed
60 statistically (Spearman's rho= -0.311, p<0.001).

61 Out of 250 cases, 81.2% had BMI in the normal range, 4.8% were underweight and 14% patients were
62 overweight. No correlation was seen between BMI & severity of PIH (Spearman's rho= -0.046, p=0.468).

63 When the position of placenta was studied, lateral placenta was seen in 44.6% cases of severe PIH whereas
64 mild and moderate cases had lateral placenta. Thus we can see that in cases of severe PIH, the incidence of
65 lateral position of placenta was significantly higher (Chi-square = 16.874, p<0.001). In severe cases of PIH, there
66 were CCU admissions in 6.4% cases, imminent eclampsia in 27.8% cases and abruptio placentae, DIC, acute
67 renal failure in 2.1% cases. Maternal mortality was seen in 4.3% cases. There is a significant positive correlation
68 between occurrence of maternal complications & severity of PIH (spearman's rho= 0.532, p<0.001) i.e. more the
69 severity of PIH, more are the chances of complications.

70 175 i.e. 70 % cases delivered spontaneously and 75 i.e. 30% needed induction.

71 When correlated with severity of PIH, 42 (89.3%) of severe PIH cases required induction, 26 (50%) of cases
72 of moderate and 7 (4.6%) cases of mild PIH needed induction. There is a significant positive correlation between
73 induction of labour and severity of PIH (spearman's rho = 0.729, p<0.001) i.e. Severe PIH cases needed to be
74 induced. Out of all the cases, 153 i.e. 61.2% cases were delivered vaginally and 97 i.e.

75 7 38.8% required LSCS, the most common indication being fetal 76 distress.

77 Table no 2 shows the fetal outcome. There was a significant negative correlation of severity of PIH with birth
78 weights (Spearman's rho = -0.323, p<0.001). Thus cases of severe PIH had babies with lower birth weights. In
79 our study, total 69 babies needed NICU admissions i.e. 27.6%. The most common reason for admission was
80 preterm with low birth weight (52% The gestational age of onset of PIH was compared in the 3 groups. In 22
81 (8.8%) cases, the onset was at < 28 wks, in 33 (13.2%) between 28 -32 wks, in 82 (32.8%) between 32 -36 wks
82 and in 113 (45.2%) the onset was beyond 36 wks gestation.

83 In cases of severe PIH, the onset < 28 wks was seen in 31.9% cases whereas in mild PIH it was in 1.3% cases.
84 There was a significant negative correlation between gestational of onset of PIH and severity of PIH (spearman's
85 rho=-0.467, p<0.001), thus showing that severe PIH cases have an earlier onset. Pregnancy induced hypertension
86 is a pregnancy-specific multi system disorder affecting both the mother and the baby.

87 In our study, total 250 cases were classified as per severity of PIH. 151 (60%) patients had mild PIH. The rest
88 were almost equally distributed as moderate or severe cases -52 cases (21%) with moderate PIH and 47 cases
89 (19%) with severe PIH. 20 patients i.e. 43% cases with severe PIH were in the extremes of age groups.

90 8 Discussion

91 Eskenazi B, Fenster L et al in a multivariate analysis of risk factors of PIH in 1991 found that women that either
92 spectrum of age were more susceptible to PIH. (1) Similar findings were also seen in a study by C. J. Lee et al
93 in a study for risk factors of PIH in the Asian population in 2000. (2) PIH often affects young and nulliparous
94 women and this was shown in our study as well as other studies done by Eskenazi B, Fenster L and Sidney S (1)
95 and Campbell DM et al (3) .

96 Antenatal care is one of the most important determinants of early detection of PIH. Regular visits will help
97 identify such cases at the earliest and enable prompt intervention, thus improving the pregnancy outcome. In the
98 present study, of all 250 cases, 155 i.e. 62% had more than 3 ANC visits, 72 i.e. 28% cases had between 1-3 visits,

99 while 23 i.e. 9.2% were unregistered that is they had not received any antenatal care. There was a significant
100 negative correlation found in this study between number of ANC visits and PIH severity indicating that patients
101 with fewer ANC visits had more severe PIH. Bandar Abbas et al in their study showed that women of PIH with
102 IUGR babies had less than three antenatal visits during pregnancy. (4) There was no correlation found between
103 BMI and severity of PIH in this study. However other studies like Lisa et al (5) and Dorothea Mostello et al (6)
104 have shown the increased incidence of PIH with higher BMI. Ahmet Ursavas reported obesity as an independent
105 risk factor for PIH and preeclampsia in 2008. (7) In our study, in cases of severe PIH the incidence of lateral
106 placenta was significantly higher. This result is in accordance with the study of Kofinas et al (8) who state that
107 of their preeclamptic women, 74% had unilateral placental location and a 2.8 fold risk of preeclampsia.

108 In our study, the total maternal complications seen were 45 i.e. in 18 % of the cases. 3 (1.2%) patients
109 were admitted in the critical care unit, 18 (7.2%) had imminent eclampsia, 14 (5.6%) suffered from eclampsia, 4
110 (1.6%) had abruptio placentae and disseminated intravascular coagulopathy and acute renal failure was seen in
111 1 (0.4%) case. In cases of severe PIH in particular, there were CCU admissions in 6.4% cases, imminent eclampsia
112 in 27.8% cases and abruptio placentae, DIC, acute renal failure in 2.1% cases and mortality was seen in 4.3%
113 cases.

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117 Maternal mortality was seen in 3 (1.2%) cases. One such was of a second gravida with full term pregnancy
118 with severe PIH and Intrauterine Fetal Demise. Patient had only 2 ANC visits. She had been brought to the
119 hospital in DIC and was immediately admitted in the CCU. However despite blood product transfusion she went
120 into Acute Renal Failure and could not be resuscitated. The second case was of a primigravida with 32 weeks
121 pregnancy who presented with eclamptic convulsions and fresh still birth. In the third case, the patient had
122 presented with severe PIH with term pregnancy with eclampsia. Emergency LSCS had been done which was
123 uneventful. Patient was in the ward as the baby was in the NICU for preterm status. On day 18 post delivery,
124 there was a sudden rise in her blood pressure which had previously come to normal post delivery. She suffered
125 from a Cerebrovascular accident and died despite immediate CCU transfer and resuscitation.

126 In the present study, there was a significant positive correlation between occurrence of maternal complications
127 & severity of PIH (spearman's rho= 0.532, p<0.001) i.e. more the severity of PIH, more are the chances of
128 complications. These were similar to results obtained by Yucesoy et al (9) and Yadav et al (10) . In cases of
129 PIH, due to uteroplacental insufficiency, there are increased chances of intra -uterine growth restriction. Also in
130 severe cases needing early induction, preterm births are common. Thus the babies are of lower birth weights.

131 In the present study, 113 i.e. 45.2% babies had birth weight > 2.5 kg, 65 (26%) between 2 -2.5 kg, 29 (11.6%)
132 between 1.5 -2 kg, 28 (11.2%) between 1 -1.5 g and 15 (6%) with < 1 kg. There was a significant negative
133 correlation of severity of PIH with birth weights (Spearman's rho = -0.323, p<0.001). Thus cases of severe PIH
134 had babies with lower birth weights. Ye RW et al (11) in their study in 2010 showed the incidence rates of low birth
135 weights in mild, moderate, and severe subgroups as 2.5% 4.9% and 11.9% respectively. The rates increased with
136 the severity of PIH. in another study by Buchbinder et al, they have shown that in women who have gestational
137 hypertension or preeclampsia, increased rates of preterm delivery and delivery of smallfor-gestational-age infants
138 are present only in those with severe disorder. (12) In our study, a significant positive correlation was seen
139 between the NICU admissions and severity of the cases, i.e. severe PIH cases had more chances of the baby
140 getting admitted in NICU which has also been studied by Ray et al (13) .

141 Sudden vasospasm, chronic utero-placental and feto-placental insufficiency and complications like abruptio
142 placentae put the babies of PIH mothers at higher risk of perinatal mortality. In the present study, the fetal
143 wastage like abortion, still births and neonatal deaths were studied and were seen more in severe cases of PIH.
144 In studies by Yadav et al (10) and Yucesoy et al (9) , perinatal mortality rate was found to be higher in severe
145 cases of PIH.

146 In the present study, cases with earlier onset of PIH had a more severe course of the disease and increased
147 maternal and fetal morbidity as also shown by study conducted by Ingrid PM et al (14) .

148 Termination of pregnancy is the only cure for PIH. In milder cases if the fetus is premature, conservative
149 management can be employed to reduce the risk of neonatal death or serious morbidity due to prematurity. In
150 such cases assessment of fetal well being and placental function are done along with strict toxæmia monitoring
151 of the mother. If the PIH does not improve or it worsens then the pregnancy has to be terminated irrespective
152 of the gestational age to avoid maternal complications and morbidity. In our study, in 144 i.e. 95.4% cases of
153 mild PIH the patients were admitted and spontaneous labour was awaited and 7 i.e. 4.65 needed to be induced.
154 In severe cases however, 42 i.e. 89.3% cases needed to be induced. There was a significant positive correlation
155 between induction of labour and severity of PIH (spearman's rho = 0.729, p<0.001) i.e. severe cases of PIH had
156 to be terminated resulting in preterm and low-birth weight babies. Similar results were seen in a study conducted
157 by Bailey et al (15) and Ye RW et al (11) where cases of severe PIH had to be induced at an earlier gestational
158 age as compared to the mild cases.

159 V.

11 CONCLUSION

11 Conclusion

160 The clinical course of PIH is progressive and is characterised by continuous deterioration that is ultimately
161 stopped only by delivery. Emphasis should be on early registration and regular ANC visits so as to detect cases of
162 pregnancy induced hypertension as early as possible in turn preventing severity and its associated complications.
163 The fetal well being should be monitored with non stress tests, modified biophysical profile, serial USG with
164 amniotic fluid estimation, Doppler studies so as to detect fetal compromise. Maternal parameters of blood
165 pressure, proteinuria, serum uric acid levels as well as premonitory signs and symptoms should be monitored so
166 as to decide a timely intervention for best feto -maternal outcome.

No

COMPLICATIONS	1 : Maternal Complications		
	MILD PIH	MODERATE PIH	SEVERE PIH
CCU ADMISSION	0	0	3 (6.4%)
IMMINENT ECCLAMPSIA	0	5 (9.6%)	13 (27.8%)
ECLAMPSIA	0	3 (5.8%)	11 (23.4%)
ABRUPTIO PLACENTAE	1 (0.7%)	3 (5.8%)	1 (2.1%)
CEREBROVASCULAR ACCIDENT	0	0	1 (2.1%)
DIC	0	0	1 (2.1%)
ACUTE RENAL FAILURE	0	0	1 (2.1%)
MORTALITY	0	1 (1.9%)	2 (4.3%)
TOTAL	151	52	47

Figure 1: Table No

Age Of On-set	Maternal Complications	Induction Of Labour	Preterm Delivery	De-livery	Lscs
< 28 wks	28	9 (40%)	11	18 (81.8%)	20
-32 wks		(33.3%)		(60.6%)	(87.9%)
					5 (22.7%)
					13
					(39.4%)
32 -36 wks	>36wks	18 (21.9%)	22	(26.8%)	44 (53.7%)
	TOTAL	7 (6.2%)	15	(13.3%)	0 93 Fetal (40.2%)
		45 Sga	75	Nicu	Wastage
				Admission	46 (40.7%)
					97
< 28 wks	28 -	14 (63.6%)	8	(36.4%)	18 16 (72.7%)
32 wks	32 -36	19 (57.6%)		(54.5%)	20 7 (21.2%)
wks	>36wks	32 (39%)		(24.4%)	23 8 (9.8%) 6
	TOTAL	26 (23%)		(20%)	69 (5.3%) 37
			91		113 250

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[Note: Table No 5 : Gestational Age Of Onset Of Pih And Fetal Outcome IV.]

Figure 2:

11 CONCLUSION

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