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Research Article

Role of Antihypertensive Drug Therapy for Mild Hypertension in Pregnancy

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Keywords

• Mild hypertension; Pregnancy

Abstract

Background: Hypertension is the most common disorder of pregnancy and the role of antihypertensive treatment for pregnant women with mild hypertension still remains unclear.

Material and methods: This prospective randomized study was performed at Maulana Azad Medical College and Lok Nayak Hospitals, New Delhi for a duration of 48 months. The patients (n=139) were and omized into two groups by computer generated numbers. Group A (study group) received Alpha methyldopa and/or Labetalol and Group B (control group) did not receive any hypertensive drug but received supportive treatment. For quantitative data, Student's t test or Mann-Whitney test was applied. A p value of <0.05 was regarded as significant. Long term clinical outcome was studied in the follow up and the data was analyzed.

Results: In Group A (treated), 11patients (15.9%) developed severe hypertension while 20 patients (28.6%) developed severe hypertension in group B (non treated group). Ten patients in group A and19 patients in group B developed proteinuria. Although this difference approached a significant level (p= 0.076), there was no statistically significant difference in overall maternal and foetal complications in both the groups.

Conclusion: Despite of the fact that complications of maternal and foetal outcome in the two groups did not differ significantly, we found that the treatment of mild hypertension in pregnancy showed near significant reduction of incidence of development of severe hypertension in treated group.

INTRODUCTION

As the most common medical disorder of pregnancy, hypertension is reported to complicate1in10pregnancies. [1,2]. Role of antihypertensive treatment for pregnant women with mild hypertension still remain unclear. Mild hypertension in pregnancy is defined as systolic blood pressure of 140-159 mm of Hg or diastolic blood pressure of 90-109 mm of Hg. As there is no immediate need to lower blood pressure, the rationale for treatment is that it will prevent or delay the progression to more severe disease, thereby benefiting the woman or baby or both and may reduce the consumption of health service resources (number of hospital visits). However, in addition to reduction of blood pressure, these drugs may reduce the risk of miscarriage, preterm delivery, placental abruption and improve fetal growth. No consensus exists on whether antihypertensive therapy is of more.

Benefit than risk for non severe hypertension, which represents the majority of hypertension in pregnancy [3-8]. This study was undertaken to know and understand the role of antihypertensive therapy in the treatment of mild hypertension in pregnancy

MATERIAL AND METHODS

Thiswasarandomizedprospectivecomparativestudyconductedonpregnantpatientswith mild hypertension managed in the Department of Obstetrics and Gynecology at Maulana Azad Medical College and associated Lok Nayak Hospital, New Delhi for the study period of 48 months. A total of 139 patients were included in the study with singleton pregnancy between 20th to 36th weeks of gestation. Out of these, 69 patients received the antihypertensive drug (Alpha methyldopa/labetalol) and 70 patients received non-pharmacological supportive (rest and B.P monitoring) treatment at the first instance of detection of mild hypertension in pregnancy after the 20th week of gestation.

All the patients with following features were excluded from the study:

- Moderate/Severe hypertension at the first detection after 20th week of gestation
- History of chronic hypertension or hypertension before 20th week of pregnancy
- Pre-existing renal disease or liver disease
- Any documented foetal malformation
- Proteinuria >300mg/24 hours or >2 plus dipstick at entry point
- Specific contraindications to antihypertensive drugs
- Multiple pregnancy

In our study, "mild" hypertension was considered as systolic blood pressure (minimum two readings 6 hours apart) of140-159mm of Hg or diastolic blood Pressure of 90-105mm of Hg. Values above 160/106 mm of Hg (B.P level approved by ethical committee of Maulana Azad Medical College, New Delhi) were labelled as having "moderate to severe" hypertension.

All mildly hypertensive patients were admitted to the hospital for the detailed initial workup (including appropriate blood and urine tests) for at least 48 hours. Once the diagnosis of mild hypertension without proteinuria was confirmed, the patients were randomized into two groups by computer generated software. Group A (study/treated group) received Alpha methyldopa and/or Labetalol, where the doses were titrated with the goal of achieving systolic blood pressure< 140 mm of Hg and diastolic blood pressure < 90mm of Hg.

Alpha methyldopa was started at the dose of 500mg thrice a day and titrated up to a maximum of 2 gms/day. If B.P levels were not controlled with maximum dose of Alpha methyldopa (2gms), labetalol was started at the dose of 100 mg twice a day and a maximum of 1200 mg /day was used. Group B (control group) did not receive any hypertensive drug but received supportive treatment in form of rest and B.P monitoring.

All patients were discharged after assign in the group with the advice of reporting back to hospital in case of developing warning symptoms like headache, blurring of vision, vomiting or any other complains. At every subsequent weekly visit, albuminuria, weight and blood pressure of patients was evaluated. Patients in either groups were considered severely hypertensive if blood pressures calculated>160/106 mm of Hg in the follow up and they were treated as per hospital protocols including introduction or addition of other anti- hypertensive medications. After allocation, women remained in the same group irrespective of treatment given in due course of time. All patients who developed severe hypertension, were admitted to the hospital.

They were removed from the trial and treated according to the hospital protocol for severe hypertension. Blood investigations like complete blood count, Liver function test, Kidney function test, and urine for protein/creatine ratio were sent. Patients were given 10 mg Depin tablet orally and repeated every 30 minutes till a maximum dose of 120 mg. Induction of labour was considered according to hospital protocol.

MANAGEMENT OF PATIENTS

Initial workup included detailed history taking, general physical examination and appropriate laboratory investigations. Blood pressure and urine albumin charting was done 4 hourly to rule out pre-eclampsia and severe variety of hypertension at the outset. Appropriate foetal and maternal monitoring was done weekly till delivery. The obstetric management was done as per the hospital protocol and according to individual consultants. All patients were followed till 6 weeks postpartum. Antihypertensive agents were stopped immediately after delivery. Strict blood pressure monitoring was done for all delivered patients for 48hrs postpartum.

Antihypertensive drugs were continued post partum only if blood pressure remained>160/106mm of Hg consistently. All patients (either groups) having blood pressure less than 160/106 mm of Hg were not started on anti-hypertensive's and they were assessed in post-natal clinic visits at 1 week and then 6 weeks post-delivery

Primary outcome of the study were as follows:

- Trends in Blood pressure readings in the two groups till delivery and 48 hours postpartum
- Development of adverse maternal outcome like severe hypertension, eclampsia, abruption placentae, proteinuria, mortality, if any.
- Development of adverse perinatal outcome like Intra Uterine Death (IUD), and perinatal mortality

Secondary outcomes were as follows:

- Mode of delivery
- Birth Weight
- Pre-term
- Intra Uterine Growth Retardation (IUGR)
- APGAR at 5 minutes <7
- Neo-natal intensive care unit (NICU) admission >48 hours or any other neonatal morbidity

Statistical analysis

For quantitative data, Student's t tester Mann-Whitney test was applied. A p value of <0.05 was regarded as significant.

RESULTS

A total of 169 patients with mild hypertension in pregnancy were enrolled in this study for a period of 24 months. Group A (treated group, n=69) received antihypertensive agents (alpha methyldopa 500 mg three times a day to start with and up to a maximum dose of 2000mg/day, labetalol added if required with starting dose of 100mg twice a day and maximum up to 1200 mg/day). Group B (non-treated group, n=70) did not receive any antihypertensive drugs and were managed conservatively.

The mean age at presentation was 26 years in Group A (range 21-36 years) and 25.76 in Group B (range 19-34 years). Other variables including parity, history of PIH in earlier pregnancy and associated medical disorders were also studied in detail. No significant association with the outcome was found with these variables (Table 1). There was no significant difference between the two groups in terms of mean period of gestation at the time of entry (p= 0.716), Table (2). There was significant difference in the two groups in terms of mean and diastolic blood pressure but not in the systolic blood pressure at the time of entry (p=.001). Most of the subjects entered the study between 31-33 weeks of gestation and hence there was an increase in mean systolic blood pressure at the segestations in both the groups. However, there was a significant reduction in mean systolic blood pressure after 34 weeks of gestation in group A while there was an increasing trend of blood pressure in group B (Figure 1). Similar trends were seen in relation to the mean diastolic blood pressure readings where there was a significant reduction in both parameters in group A after 34 weeks (Figure 2).

Table 1: Patient's distribution in both the groups showing age, parity, history of PIH in earlier pregnancy and associated medical disorder.

Age(yrs)	Treated (n=69)	Non treated (n=70)	P value
Mean	26.00+3.365	25.76+3.095	0.658
Parity			
Nulliparous	53 (76.8%)	54 (77.1%)	0.945
Multiparous	16(23.2%)	16 (22.9 %)	
H/o PIH in earlier pregnancy	7 (10.15%)	5 (7.14%)	0.604
Other diseases			
GDM	6 (8.7%)	6 (8.6%)	0.694
Rh-ve	1 (1.4%)	2 (2.9%)	
Anemia	3 (4.3%)	2 (2.9%)	
Hypothyroid	5 (7.2%)	2 (2.9%)	

Abbreviations: PIH: Pregnancy Induced Hypertension; GDM: Gestational Diabetes Mellitus

Table 2: Period of gestation at entry in the study (in weeks).

Group	28-30wks	31-32 wks	33-34wks	35wks
Treated	7(10.1%)	15(21.7%)	32(46.4%)	15(21.7%)
Non treated	4(5.7%)	14(20.0%)	48(68.6%)	4(5.7%)
P value	0.289(NS)	0.312(NS)	0.015(S)	0.061(NS)

Abbreviations: NS: Not Significant; S- Significant

Table 3: Maternal complications.

	Treated N=69	Non treated N=70	P value
Severe hypertension	11(15.9%)	20 (28.6%)	0.074
Proteinuria	10 (14.5%)	19 (27.14%)	0.076
Abruptio	1 (1.3%)	0	-
Eclampsia	1 (1.3%)	0	-
LSCS	12 (17.4%)	10 (14.3%)	0.542
Mean gestation at delivery (weeks)	37.10	36.84	.111

Abbreviations: LSCS: Lower Segment Caesarean Section

Table 4: Fetal complications in the two groups.

	Treated N=69	Non treated N=70	P value
Preterm	8 (11.6%)	11 (15.7%)	0.261 (NS)
IUGR	11 (16.2%)	7 (10.0%)	0.251(NS)
Apgar <7 at 5 min	2 (2.89%)	3 (4.28%)	0.357(NS)
Mean birth weight	2.5kg	2.7 kg	0.083(NS)
NICU admission	5 (7.2 %)	4 (5.77%)	0.216(NS)
IUD	1 (1.4 %)	0	0.312(NS)
Neonatal death	1 (1.4%)	0	0.312(NS)

Abbreviations: *NS: Not Significant; IUGR: Intra Uterine Growth retardation; NICU: Neonatal Intensive Care Unit; IUD: Intra Uterine Death

In Group A, 11 patients (15.9%) developed severe hypertension while 20 patients (28.6%) developed severe hypertension in group B. Though there were more number of patients developing severe hypertension in latter group, the difference was not statistically significant. The difference, however approached to near significant value (p= 0.074). Ten patients in group A and 19 patients in group B developed proteinuria. Although this difference also approached a significant level (p= 0.076) but there was no statistically significant difference in overall maternal and fetal complications in both the groups (Tables 3,4). In both the groups, blood pressure became normal 48 hrs after delivery and none of the patients required antihypertensive therapy, but one patient in the group A and two in group B were having blood pressure >140/90 mm of Hg at 6 weeks postpartum, possibly indicating chronic hypertension.

DISCUSSION

There are many unknown factors for causing gestational hypertension, preeclampsia- eclampsia syndrome. We can only pre-

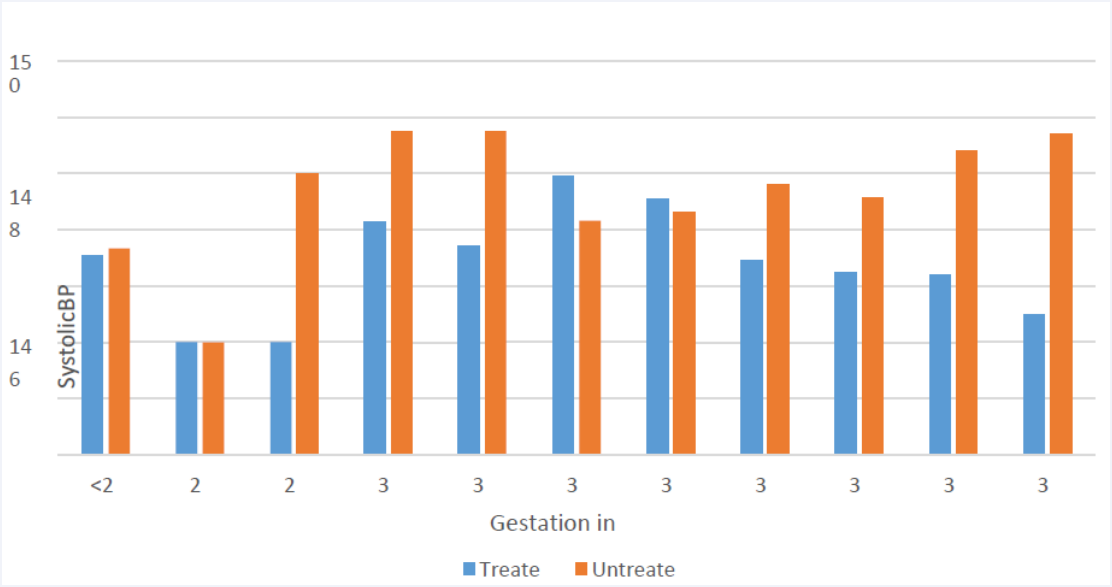

Figure 1 Trends of Mean SBP at each week of gestation in two groups.

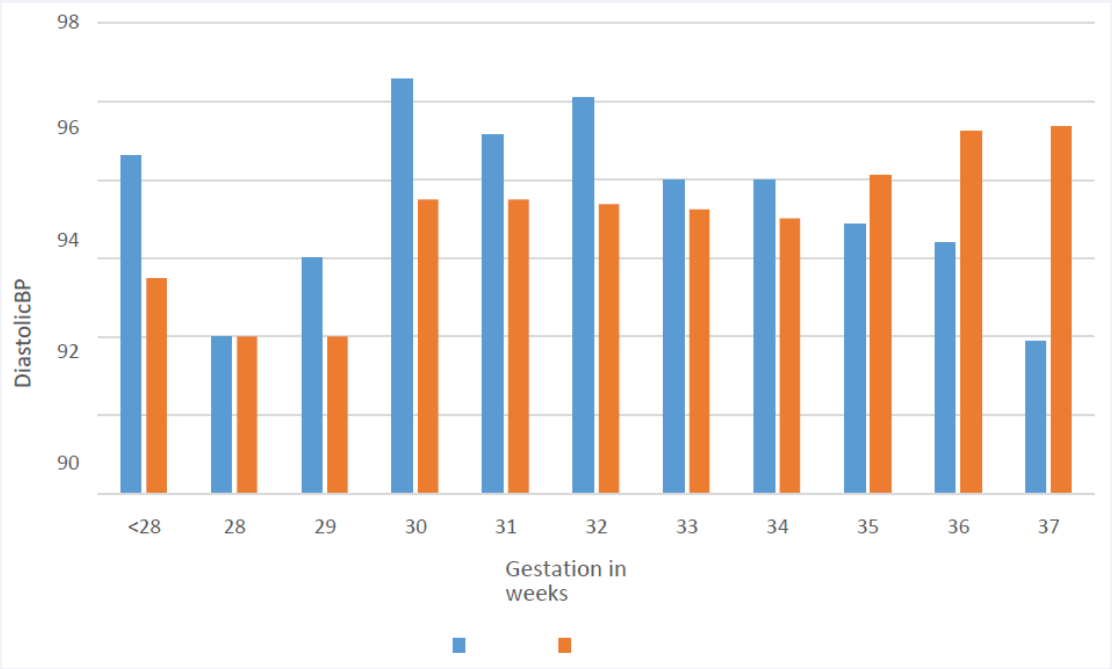

Figure 2 Trends of Mean DBP at each week of gestation in two groups.

vent and control the factors after they have been expressed like rise in blood pressure to some extent and prevent the development of convulsions and more severe form of disease with good antenatal care. So, with the early use of antihypertensive agents in gestational hypertension, it might be possible to stop or delay the progression from a less severe form of disease (gestational hypertension) to a more severe form of disease characterized by preeclampsia syndrome. There is consensus that severe maternal hypertension (systolic blood pressure>160mm of Hg and or diastolic blood pressure>110mm of Hg) should be treated immediately to avoid possible eclampsia and major maternal complications like intracerebral haemorrhage and cerebrovascular accidents. However, there is controversy regarding treatment of mild hypertension in pregnancy. It is also arbitrary to call mild to moderate variety under as 'mild' meaning there by 'nonsevere'. It should be emphasized that there are no studies addressing safe blood pressure treatment targets for pregnant women, and guidelines and reviews generally recommend treating the blood pressure to bring it to a level that is likely to be protective against acute adverse cerebro-vascular or cardio vascular events, which

is usually in the range of 140 to 159 systolic / 90 to 105 mm of Hg diastolic blood pressure.

There is another school of thought which advocates against the use of antihypertensive drugs in mild hypertension in pregnancy. According to this, the hypertension of pregnancy is secondary to placental under perfusion, thus lowering of systemic blood pressure is not believed to reverse the primary pathogenic process, and antihypertensive medication has not been demonstrated to cure or reverse preeclampsia. Antihypertensive drug therapy is also associated with potential maternal and fetal side effects. Most anti-hypertensives used in pregnancy are designated as category C which states that human studies are either positive for fetal risks or are lacking and the drug should be given only if potential benefits justify potential risk to the fetus. Methyldopa belongs to category B and is one of the most widely used drugs for the treatment of hypertension in pregnancy. Blood pressure control is gradual, over 6-8 hours, because of the indirect mechanism. However, in our study we have used up to a maximum dose of 2000mgs/day, as higher doses of the same drug don't necessarily benefit and have more side effects. Methyldopa is known to cross placenta. However, Cockburn et al. [9], conducted a prospective trial in which children born to mothers who have received methyldopa for treatment of mild hypertension in pregnancy were followed till 7&1/2 years of age. They found that methyldopa was safe to use in pregnancy and is probably preferable to other drugs from the point of view of neonate and child [9]. Recently, in 2010, Khalil et al. [10], conducted atrial to show the effect of methyldopa on uterine artery flow doppler. The authors found that methyldopa has no significant effect on uterine artery resistance to blood flow, suggesting that it does not impair utero placental circulation in these patients [10]. Thus, methyldopa was chosen for its safety in our study.

It is important to mention that there are other drugs like beta blockers (atenolol and labetalol) and calcium channel blockers like nifedipine which are being used for treatment in pregnancy. There are studies showing atenolol to be associated with intrauterine growth retardation of fetus, low birth weight babies and also preterm deliveries [11,12]. However, in a study when labetalol, methyldopa or no treatment was compared, there was no benefit or a disadvantage in treating the hypertension, as all three groups fared in the same manner [13].

Drug treatment of hypertension in pregnancy is justified only if it is beneficial for the mother or fetus. In mild hypertension it is doubtful whether a reduction of blood pressure is of any advantage to the mother and a slightly elevated maternal blood pressure might even promote fetal growth. There are certain trials which favored the early use of antihypertensive in the treatment of mild hypertension in pregnancy and there are certain trials which favored conservative or no management of mild hypertension in pregnancy. It is also difficult to decide the optimum blood pressure levels at which both mother and fetus will remain normal. There have been several studies on the use of methyldopa for treating mild hypertension in pregnancy. In 1987, Weitz et al. [14], carried out a randomized, prospective double blind study comparing methyldopa with placebo for the treatment of chronic hypertension in pregnancy [14]. Here, methyldopa treated patients registering in the first trimester had a significant reduction in the mean arterial pressure [MAP] during the second and third trimester ($p<0.025$). No significant differences in birth weight, ponderal index, were found. The mean GA was significantly prolonged in the methyldopa treated group by 10.3 days ($p< 0.05$). The frequency of superimposed pre-eclampsia was similar in both groups (33.3% vs38.4%). Compared to this study, in our study, we have included women with mild hypertension without proteinuria and with period of gestation between 20-36 weeks. We have excluded chronic hypertension on the basis of history. In our study also, there was a significant reduction in mean systolic, diastolic and arterial blood pressure of treated group since 35 weeks on wards. In contrary to this trial, in our study, there was more number of patients developing severe hypertension in non treated group (19 out of 70) than in treated group (10 out of 69). This difference approached a near significant level ($P=0.074$). We also found no significant difference in the mean gestational age at delivery in between the two groups. In our study, though the birth weight was slightly higher in then on treated group, this difference was not significant. There was no difference between the two groups in terms of perinatal outcomes, like preterm birth, NICU admission and neonatal deaths. There was no significant difference in the mean systolic blood pressure of two groups at the time of entry in our study. However, there was significant difference between mean diastolic pressure and mean arterial pressure at entry point in the two groups. In spite of high diastolic blood pressure and high mean arterial blood pressure at the time of entry in treated group, there were less number of patients developing severe hypertension in treated group (11 out of 69) than in non treated group (20 out of 70). There was a significant reduction in systolic blood pressure of treated group since 34 weeks onwards than in non treated group ($p<0.05$). Mean arterial blood pressure in treated group was also significantly lower than that of non treated group since 33 week onwards ($p<0.05$). Diastolic blood pressure in treated group also decreased significantly since 33 weeks onwards. Patients in both treated and non treated group showed higher blood pressure trends than normal and then developed severe hypertension. A comparison between the treated and non treated groups revealed significant difference in blood pressure just before delivery with higher blood pressure in the no treatment group than treated group ($p< .0001$). These differences were due to increase in blood pressure in the non treated group and the decrease in treated group. Our result was similar to the study by Hogstedt et al. [15], who demonstrated that the drug treated group showed significantly better blood pressure control than the group not given antihypertensive [15]

In our study, in treated group, 11 out of 69(15.9%), developed severe hypertension. One patient developed eclampsia and one patient developed abruption placenta eat 33weeks who had intra uterine death of the baby later and postpartum, her blood pressure was normal. Thus, the use of antihypertensive drug like alpha methyldopa definitely reduces the risk that a pregnant woman with mild or moderate hypertension might have of developing severe hypertension. There were more number of patients developing proteinuria in non treated group (19 out of 70) than in treated group (10 out of 69) but this difference was also not statistically significant ($p=0.076$).

It is also important that reduction in blood pressure does not lead to deterioration in outcome of the fetus. In fact, the non se-

lective beta-blocker oxprenolol seemed to reduce intrauterine growth retardation when compared with methyldopa [16]. This effect might be mediated through an increase in plasma volume implying an improved placental blood flow. This was also confirmed by Pickles et al., who suggested possible advantages and no apparent disadvantages for the fetus during the use of labetalol in mild hypertension in pregnancy [17]. However, there are studies showing adverse effects of antihypertensives on the growth of fetus. Sibai et al., showed a higher frequency of fetal growth retardation in the women with mild hypertension treated with Labetalol [18]. A Meta regression study published in year 2000 concluded that treatment induced fall in maternal blood pressure may adversely affect fetal growth [19].

Plouin et al. [20], demonstrated a lesser number of caesarean sections (19 out of 78, 17%) in treated group than (27 out of 76, 36%) in non treated group. They concluded that antihypertensives prevent acute hypertension in late pregnancy and associated fetal distress, thus reducing the number of caesarean sections [20]. However, we found no effect of antihypertensive therapy on mode of delivery.

These antihypertensive agents resulted in a better blood pressure control in treated than in non-treated group. There was significant reduction in systolic blood pressure, diastolic blood pressure and mean arterial pressure of treated than non-treated group ($p< 0.001$). There were also lesser number of patients developing severe hypertension in treated group than in non-treated group. However, though this result approached to a significant level, this was not statistically significant, ($p =0.072$). Complications like eclampsia, abruption and pulmonary edema were present in treated group. This may be because patients entering the study in treated group has significantly higher diastolic blood pressure and mean arterial pressure.

CONCLUSION

Thus, in summary, despite of the fact that other parameters of maternal and fetal outcome in the two groups did not differ significantly, we found that the treatment of mild hypertension in pregnancy showed near significant reduction of incidence of development of severe hypertension in treated group. However, it must be realized that it is difficult to prove differences with respect to perinatal mortality and serious complications like eclampsia and abruption and would require a larger sample size of the study.

REFERENCES

1. Report of National High Blood Pressure Education program Working group on High Blood Pressure in pregnancy. Am J Obstet Gynaecol. 2000; 183: 1-22.
2. Samadi AR, Mayberry RM, Zaidi AA, Pleasant JC, McGhee N Jr, Rice RJ. Maternal hypertension and associated pregnancy complications among African-American and other women in the United States. Obstet Gynecol. 1996; 87: 557-563.
3. Elhassan EM, Mirghani OA, Habour AB, Adam I. Methyldopa versus no drug treatment in the management of mild pre-eclampsia. East Afr Med J. 2002; 79: 172-175.
4. [No authors listed]. Nifedipine versus expectant management in mild to moderate hypertension in pregnancy. Gruppo di Studio Ipertensione in Gravidanza. Br J Obstet Gynaecol. 1998; 105: 718-722.
5. Magee LA, Ornstein MP, von Dadelszen P. Fortnightly review: management of hypertension in pregnancy. BMJ. 1999; 318: 1332-1336.
6. Sibai BM. Treatment of hypertension in pregnant women. N Engl J Med. 1996; 335: 257-265.
7. Levine RJ, Maynard SE, Qian C, Lim KH, England LJ, Yu KF, et al. Circulating angiogenic factors and the risk of preeclampsia. N Engl J Med. 2004; 350: 672-683.
8. Levine RJ, Thadhani R, Qian C, Lam C, Lim KH, Yu KF, et al. Urinary placental growth factor and risk of preeclampsia. JAMA. 2005; 293: 77-85.
9. Cockburn J, Moar VA, Ounsted M, Redman CW. Final report of study on hypertension during pregnancy: the effects of specific treatment on the growth and development of the children. Lancet. 1982; 1: 647-649.
10. Khalil A, Harrington K, Muttukrishna S, Jauniaux E. Effect of antihypertensive therapy with alpha-methyldopa on uterine artery Doppler in pregnancies with hypertensive disorders. Ultrasound Obstet Gynecol. 2010; 35: 688-944.
11. Butters L, Kennedy S, Rubin PC. Atenolol in essential hypertension during pregnancy. BMJ. 1990; 301: 587-589.
12. Lydakis C, Lip GY, Beevers M, Beevers DG. Atenolol and fetal growth in pregnancies complicated by hypertension. Am J Hypertens. 1999; 12: 541-547.
13. Sibai BM, Mabie WC, Shamsa F, Villar MA, Anderson GD. A comparison of no medication versus methyldopa or labetalol in chronic hypertension during pregnancy. Am J Obstet Gynecol. 1990; 162: 960-966.
14. Weitz C, Khouzami V, Maxwell K, Johnson JW. Treatment of hypertension in pregnancy with methyldopa: a randomized double blind study. Int J Gynaecol Obstet. 1987; 25: 35-40.
15. Hogstedt S, Lindeberg S, Axelsson O, Lindmark G, Rane A, Sandström B, et al. A prospective controlled trial of metoprolol-hydralazine treatment in hypertension during pregnancy. Acta Obstet Gynaecol Scand. 1985; 64: 505-510.
16. Gallery EDM, Ross MR, Gyory AZ. Antihypertensive treatment in pregnancy: analysis of different responses to oxprenolol and methyldopa. BMJ. 1985; 291: 563-566.
17. Pickles CJ, Symonds EM, Pipkins FB. The fetal outcome in a randomized double controlled trial of labetalol versus placebo in pregnancy induced hypertension. Br J Obstet Gynaecol. 1989; 96: 38-43.
18. Sibai BM, Gonazalec AR, Mabie WC, Moretti M. A comparison of labetalol plus hospitalisation versus hospitalisation done in the management of pre-eclampsia remote from term. Obstet Gynaecol. 1987; 70: 323-327.
19. von Dadelszen P, Ornstein MP, Bull SB, Logan AG, Koren G, Magee LA. Fall in mean arterial pressure and fetal growth restriction in pregnancy hypertension: a meta-analysis. Lancet. 2000; 355: 87-92.
20. Ploutin PF, Breast G, Llado J, Dalle M, Keller ME, Goujon H, et al. A randomised comparison of early with conservative use of antihypertensive drugs in the management of pregnancy induced hypertension. Br J Obstet Gynaecol. 1990; 97: 134-144.

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