LOW DOSE ASPIRIN IN MILD PIH

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SUMMARY

Pregnancy induced hypertension is hazardous for both mother and foetus. Numerous attempts have been made for the treatment of PIH but none seems to be satisfactory. Role of low dose aspirin has come up recently in prevention of pregnancy induced hypertension. A double blind placebo controlled study was designed to evaluate the role of low dose aspirin in management of mild pregnancy induced hypertension.

The need for antihypertensive treatment was low in aspirin group (30%) as compared to placebo group (75%). Incidence of proteinuria was low in aspirin treated women with PIH (50%) as compared to placebo treated pregnant women (87.5%). Diastolic blood pressure of aspirin treated group of pregnant females decreased marginally during therapy but rose significantly at the time of delivery when aspirin was already stopped at 38-39 wks of gestation. As compared to aspirin group, the placebo group had high mean diastolic blood pressure at 38 weeks, which was statistically significant (p = 0.05).

INTRODUCTION

The hazards of hypertension in pregnancy are well established. Attempts at treatment of established preeclampsia have been numerous, but none have been satisfactory.

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The rationale for use of aspirin in hypertensive pregnancies rests upon its effects on the platelets and prostaglandins. Major effect is the correction of imbalance between thromboxane A_2 (a vasoconstrictor and promotor of platelet aggregation) and prostacyclin (a vasodilator and inhibitor of platelet aggregation).

A daily intake of low dose aspirin has been suggested by Schiff et al. (1989), Wallenburg et al. (1986), etc. for the prevention of PIH and IUGR. But literature is lacking in information regarding role of low dose aspirin in treatment of PIH. In the present study the role of low dose aspirin has been evaluated for management of mild pregnancy induced hypertension.

MATERIAL AND METHOD

A prospective, randomized, double blind placebo controlled trial of low dose aspirin for the treatment of mild pregnancy induced hypertension was carried out in the Dept of Obstetrics and Gynecology, K.G's. Medical College, Lucknow. Patients attending the antenatal clinic during 20-30 weeks of gestation were taken up for the study. 19 patients were in study group who were having mild PIH (Diastolic B.P. 90 to 110 mm of Hg and Systolic B.P. 140-160 mm of Hg). One patient was dropped from the study as she developed reaction following ingestion of coded capsule. 52 patients comprised of control group but nine were lost to follow up so ultimately 43 patients remained in the control group. They all maintained a normal B.P. through out pregnancy and were not given any drug. In all these patients, besides history taking and general examination, obstetrical examination was done.

All the patients were followed throughout pregnancy with special attention to Blood pressure, proteinuria and edema. Study group patients were given 100 mg. aspirin or starch (Placebo) by randomization, in a double blind fashion from the day of registration up to 38 weeks of pregnancy as one capsule per day. Decoding was done at the end of the study when effects of blood pressure, proteinuria and odema were noted along with fetal outcome.

RESULTS

In present study specifically blood pressure, proteinuria and oedema were noted in study group and control group patients. Results are shown in Table I and II.

Most of the cases of study group were primigravida (90%) and majority of patients developed PET between 30-34 wks gestation (55.56%).

The mean systolic and diastolic blood pressure in various groups were compared at booking at 38 wks when capsules were stopped and at the time of delivery (after stopping the capsules).

Results of our trial showed that mean systolic blood pressure (SBP) in both aspirin and placebo group were comparable at the beginning of the trial and at 38 wks of gestation. Systolic BP rose slightly after stopping of aspirin at the time of delivery. Statistically no difference in SBP existed between aspirin and placebo group. Diastolic blood pressure (DBP) showed a decrease of about 3 mm of Hg at 38 wks in aspirin group (P = NS). This DBP was significantly lower than the placebo group. Further it rose significantly on stopping aspirin at 38 weeks. (Table I)

The mean systolic and diastolic blood pressure in aspirin group at initiation of therapy was 134.2 ± 7.86 and 90.4 ± 1.26 mm of Hg respectively. At the 38th wk.

Table I

Distribution of mean systolic and diastolic blood pressure at booking, 38 wks and At the time of delivery in various groups

Blood Pressure	Aspirin Gr. (n = 10)		Placebo Gr. (n = 8)		Control Gr. (n = 43)	
Mean ± S.D.	Systolic	Diastolic	Systolic	Dystolic	Systolic	Diastolic
At Booking	134.2 ± 7.86	90.4 ± 1.26	136.25 ± 7.44	96.25 ± 5.6	114.5 ± 5.26	76.5 ± 7.9
At 38 weeks	132.2 ± 6.63	87.2 ± 4.34	135.4 ± 7.72	95.1 ± 4.74	112 ± 8.37	75.2 ± 8.67
At Delivery	138.8 ± 8.39	94.4 ± 7.82	134.0 ± 8.21	94.6 ± 4.28	115.9 ± 3.4	77.9 ± 3.6

when capsules were stopped, the mean systolic and diastolic blood pressure were 132.2 ± 6.63 and 87.2 ± 4.34 mm of Hg which shows marginal fall in systolic as well of diastolic blood pressure value.

At delivery however the systolic and diastolic blood pressure raised to 138.8 ± 8.39 and 94.4 ± 7.82 mm of Hg. The diastolic blood pressure showed a significant rise from 38 wks of gestation after stopping aspirin.

In the placebo group the mean systolic and diastolic blood pressure at booking, at 38 wks and at the time of delivery were 136.25 ± 7.44, 96.25 ± 5.6 and 135.4 ± 7.72 and 95.1 ± 4.74 and 134.0 ± 8.21 and 94.6 ± 4.28 mm. of Hg respectively. There was no fall in systolic or diastolic B.P.

As compared to aspirin group the placebo group had high mean systolic and diastolic B.P. at 38 wks.

Of the 10 patients of aspirin group one developed severe PIH with traces of protein in urine at 39 wks, after 7 days of stoppage of aspirin and had to undergo LSCS for loss of fetal movement.

Over all five patients (50%) in aspirin group and 7 patients (87.5%) in placebo group had protenuria.

Seven out of eight patients required antihypertensive drugs in placebo group while in aspirin group only three cases required antihypertensive treatment. Out of these three cases, two cases required it when aspirin was stopped at 38 wks.

DISCUSSION

Since aspirin has an antiplatelet action, its use in PIH was renewed.

Table II

Distribution of patients developing severe PIH/Proteinuria or requiring antihypertensive drugs in aspirin and placebo groups

	Aspirin (n = 10)		Placebo (n = 8)	
	No.	%	No.	%
1. No. of women who developed moderate to severe PIH	1	10	0	0
2. Period of Gestation for development moderate to severe PIH.	39 Wk.		-	
3. No. of women who developed proteinuria	5	50	7	87.5
4. No. of women requiring antihypertensive drugs	3	30	6	75

Encouraging results have been obtained in women habitually taking aspirin (Crandon and Isherwood 1979), women at high risk of development of PET (Beaufils 1985), with recurrent IUGR etc. (Wallenberg and Rotmans, 1987). But studies using aspirin as treatment of PIH however are limited.

Schiff et al. (1990) in their study found no effect of low dose aspirin in treatment of mild PIH. Toppozada et al. (1991) has shown that in severe pree-clampsia acetyl salicylic acid treated patients showed a significant reduction of systolic and diastolic blood pressure, and decrease in proteinuria as compared to conventional treatment. Similar are the results in our study. Aspirin treated patients on an average maintained lower systolic and diastolic blood pressure, which tend to get increased once therapy was stopped.

Majority of placebo group of patients (6 out of 8) required either sedatives alone at night or methyl dopa orally for control of blood pressure. While only 3 (30%) of aspirin treated patients required antihypertensives, that too, 2 of them needing it once the aspirin was stopped.

This clearly showed the reduced need of antihypertensive medicines in patients taking aspirin for PIH (Table II). Protenuria was found to occur less frequently in aspirin group (50%) as compared to placebo group (87.5%). Similar observations have been made by Toppozada et al (1991) in aspirin treated severe PIH. McParland et al (1990). Wallenberg & Rotmans (1986), Schiff et al (1989) have all shown reduction of protenuria and pre-eclampsia in aspirin treated patients.

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