EFFECT OF ASPIRIN ON PLATELET COUNT AND ADHESIVENESS IN PREGNANCY INDUCED HYPERTENSION

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SUMMARY

Low dose aspirin (100 mg) was given in patients with history of eclampsia/ pre-eclampsia in previous pregnancy and women with PIH in present pregnancy. Platelet count was done before starting and after stopping therapy and was compared with PIH patients on anti-hypertensive therapy. In PIH group (30 cases) on anti-hypertensive therapy the mean platelet count was 1.92 lac/mm^3 which was on lower side. While PIH group on aspirin therapy had mean platelet count 2.1 lac/mm^3 (before therapy) and 2.11 lac/mm^3 after therapy. In PIH group on anti-hypertensive therapy platelet adhesiveness was 31.9% which was on higher side (P<0.01). In PIH group on aspirin therapy platelet adhesiveness was 21.15% before therapy and 17.92% after therapy which showed a significant decrease in platelet adhesiveness in patients on aspirin therapy (P<0.001).

INTRODUCTION

The term hypertensive disorder of pregnancy includes a heterogenous collection of most disease. In most countries pregnancy induced hypertension (PIH) appears to be the single largest cause of maternal death (Dekker & Sibai Baha, 1993).

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Pregnancy induced hypertension (PIH) occurs in 10% of all pregancies out of which 5% develop eclampsia (Ratnam et al, 1992). In PIH there is imbalance between thromboxane and prostacyclin which causes generalised vaso-constriction and platelet aggregation. This reduces the foeto-maternal blood flow. Aspirin restores the balance of prostacyclin and thromboxane and improves utero-placental circulation

(McParland and Pearce, 1991).

MATERIAL AND METHODS

The study was conducted on 90 patients in the Department of Obstetrics and Gynaecology and Neonatology, J.N.M.C., A.M.U., Aligarh from Jan. 93 to April 94.

Causes were selected after taking a detailed history. Control group (30 cases) included healthy normotensive pregnant females without any overt medical illness, and study group (60 cases) included patients with pregnancy induced hypertension (PIH) which were subgrouped as follows:

a) Patients of PIH antihypertensive therapy in third trimester of pregnancy (30 cases).

b) Patients receiving Low Dose Aspirin (30 cases) which were subgrouped as:

(i) PIH with history of Eclampsia or Pre-eclampsia in previous pregnancy and PIH in present pregnancy (19 cases)

(ii) IUGR on basis of clinical suspicion (4 cases)

(iii) Premature labour pains in present pregnancy (7 cases).

Coagulation studies were done in both the groups and included Bleeding Time (Duke's method), Platelet count (Plasma Dilution Method) and Platelet Adhesiveness.

$$\left[\frac{\text{Initial platelet count - Final platelet count}}{\text{Initial platelet count}}\right] \times 100 = \%\ \text{of platelet adhesiveness}$$

(Dacie & Lewis, 1984).

Blood sample in control group and patients on anti-hypertensive therapy was collected in 3rd trimester, whereas in patients in LDA it was collected before starting the therapy and after stopping therapy.

Coagulation study in both control and study group included Bleeding time, Platelet count and platelet adhesiveness.

Dose and duration of aspirin therapy: 100 mg of aspirin was started from 14-16 weeks of gestation and was stopped 2 weeks before E.D.D.

RESULTS

The mean platelet adhesiveness in normal pregnancy was 13.37% and was significantly higher ($P<0.01$) in patients with PIH (31.9%). In PIH group on LDA the platelet adhesiveness before therapy was 21.15% which was found to be higher ($P<0.01$) than after stopping the aspirin dose (17.92%).

DISCUSSION

In PIH patients the mean platelet count was 1.92 lac/mm^3 which was reduced as compared to the control group (2.35 lac/mm^3). Kelton et al (1985) have also found mild thrombocytopenia (platelet count 1-1.1 lac/mm^3) in pre-eclampsia. Redman et al (1978) and Lindheimer et al (1981) found that the platelet count generally decreased in females with pre-eclampsia compared with corresponding pregnant control women.

In pre-eclamptic women there is an increase in platelet bound and circulatory platelet bindable immunoglobulin (Samules et al, 1987) suggesting an alteration in the platelet surface. Pre-eclamptic patients have evidence of both an in-vivo and in-vitro platelet function defect.

Burrows et al (1987) have reported the platelet associated IgG in pre-eclamptic females even in the abscence of thrombocytopenia.

In aspirin treated patients pl. count before and after therapy did not show any sig-

Table 1
Platelet count and Platelet adhesiveness in control and study group

	No. of Cases	Pl. count (lacs/mm³)	Pl. adhesiveness (%)	P. value
1. Control	30	2.35 ± 0.56	13.37 ± *2.46	*<0.01
2. PIH	30	1.92 ± 0.43	31.90 ± *11.20	
3. Low dose aspirin				
a. PIH	19			
- before therapy		2.10 ± 0.40	21.15 ± 7.7+	+<0.001
- after therapy		2.11 ± 0.50	17.92 ± 7.6+	
b. IUGR + PLP	11			
- before therapy		2.10 ± 0.40	18.44 ± 7.8**	**NS
- after therapy		1.98 ± 0.30	16.91 ± 7.7**	

The mean platelet count in normal pregnancy was 2.35 lac/mm³ while there was mild thrombocytopenia (1.92 lac/mm³) in patients of PIH group. In patients on aspirin therapy the mean platelet count before and after treatment did not show any variation.

nificant change.

Platelet adhesiveness In normal pregnancy platelet adhesiveness was 13.37%, while in PIH it was 31.9% (P<0.01). This increased platelet aggregation is because of increased thromboxane level and decreased prostacyclin level in PIH. In pre-eclampsia placenta produces over seven times more thromboxane as prostacyclin (Walsh, 1985).

Aspirin inhibits platelet adhesion to collagen under condition of stasis of low flow, but not under normal condition that is at physiological rates of shear and haemotocrit levels (McParland et al, 1991).

It has been suggested that low dose aspirin restores the thromboxane and prostacyclin balance and thus be of use in the prevention and possible treatment of pre-eclampsia (McParland et al, 1991).

Aspirin inhibits synthesis of PG, by acetylation of cyclo-oxygenase enzyme (Roth and Majerus, 1975). According to Burch et al 1978, aspirin inhibits pl. aggregation by irreversible acetylation of cyclo-oxygenase enzyme.There is a differential inhibitory effect of aspirin on cyclo-oxygenase enzym in two tissues i.e. platelet and the endothelium (Moncado & Vane 1979).

According to them low dose aspirin preferentially inhibits platelet thrombox-

ane synthesis leaving endothelial prostacyclin synthesis, relatively intact. This selective mode of action appears to be related to both dosage and kinetics of aspirin. Dosage as low as 160 mg inhibited platelet cyclo-oxygenase activity by more than 80% and large doses have little additional effect. This goes in harmony with our study which shows that platelet adhesiveness is remarkably decreased in patients on aspirin. This decrease is statistically significant ($P<0.001$) in patients before starting aspirin and after the aspirin therapy.

CONCLUSION

Aspirin decreases platelet adhesiveness in PIH patients but has no effect on platelet count.

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