ORIGINAL ARTICLE

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Pilot Interventional Study Comparing Fetomaternal Outcomes of 150 mg Versus 75 mg Aspirin Starting Between 11 and 14 Weeks of Pregnancy in Patients with High Risk of Preeclampsia: A Randomized Control Trial

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Abstract

Introduction Hypertensive disorders of pregnancies complicate around 5–10% of pregnancies worldwide, and together they are a member of the deadly triad along with haemorrhage and infection that contribute to a significant amount of maternal morbidity and mortality.

Aims and Objectives To compare differences in the fetomaternal outcomes with the use of 150 mg aspirin versus 75 mg aspirin in pregnant women found to be at high risk of PE.

Methodology This was a two-armed double-blind parallel randomized control trial conducted in the Department of Obstetrics and Gynaecology, King George's Medical University, carried over a period of 1 year.

Results Preeclampsia occurred in 15 of 87 participants (17%) in the 75 mg aspirin group compared with 6 of 91 (6.5%) in the 150 mg aspirin group. There were a significantly higher incidence of PE, its severity and lesser period of gestation at delivery in the group given 75 mg dose compared to the group given 150 mg dose. There were significantly higher values of mean arterial pressure and uterine artery PI in women who developed preeclampsia compared to those who do not in both the groups. Foetal outcomes were observed in both the groups of women, and there was no statistically significant difference between them.

Conclusion This randomized trial showed that among women with singleton pregnancies who were identified by means of first-trimester screening as being at high risk of preterm preeclampsia, use of aspirin 150 mg per day started between 11 and 14 weeks till 36 weeks is a potent intervention to reduce the development of both early- and late-onset preeclampsia as compared to a dose of 75 mg per day.

Keywords Preeclampsia · Aspirin · MAP · Uterine Doppler

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Introduction

Hypertensive disorders of pregnancies including preeclampsia (PE) and eclampsia complicate around 5–10% of pregnancies worldwide, and together they are a member of the deadly triad, along with haemorrhage and infection, which contribute to a significant amount of maternal morbidity and mortality [1]. The possibility of complications is higher when the disease is severe and of early onset, requiring delivery before 37 weeks of gestation.

In low-resource settings like the Indian subcontinent, it is common to encounter patients with severe preeclampsia and its complications like eclampsia, HELLP syndrome, placental abruption, disseminated intravascular

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coagulation, intrauterine growth retardation and intrauterine death. The aetiology of preeclampsia and to an extent foetal growth restriction are attributed to abnormal placentation. A combination of maternal demographic characteristics, including medical and obstetric history, serum markers, uterine artery pulsatility index (PI) and mean arterial pressure (MAP), at 11–13 weeks of gestation has been studied and reported to identify a high proportion of pregnancies at high risk of preterm PE [2–6].

Uterine artery PI is an indirect measure of uteroplacental perfusion, and the postulated hypothesis is that high PI implies impaired placentation with the consequent increased risk of developing preeclampsia, foetal growth restriction, abruption and stillbirth [7, 8].

More than fifty trials and meta-analysis of these studies reported that the administration of low-dose aspirin in high-risk pregnancies is associated with a moderate decrease in the rate of PE and its complication [9]. More recent meta-analysis of randomized studies reported that low-dose aspirin started at or before 16 weeks was associated with a 50% reduction in the overall risk of PE and a significant reduction in preterm PE [10, 11]. Still, early screening for PE and aspirin do not find a place in national guidelines.

Moreover, majority of these studies have used relatively lower doses of the drug (75–100 mg) and very few trials have actually used 150 mg since there could be a possibility of non-responders [12, 13]. Whether aspirin in relatively higher dosage, i.e. 150 mg, is better in outcome compared to 60–80 mg is less studied and evaluated, and there is a dearth of evidence in the reported literature.

First-trimester use of aspirin is not associated with the increased risk of foetal abnormalities and there is no evidence of any increase in maternal bleeding form any site or placental abruption [14]. Also, there is no association between low-dose aspirin during the third trimester and antenatal closure of the ductus arteriosus, intraventricular haemorrhage or neonatal bleeding [10].

There is a paucity of data for the Indian population regarding their response to different dosages of aspirin. There is growing concern that the dose of aspirin 75 mg per day used commonly in clinical settings has no benefit in majority of pregnant women, and to our knowledge, there are limited trials which have actually compared dosage of 150 mg with 75 mg.

The present study is planned to compare differences in the fetomaternal outcomes with the use of 150 mg aspirin compared to another group given 75 mg aspirin which is the usual recommended dose in majority of standard guidelines. Primary outcome was the development of preeclampsia before 37 weeks. Secondary outcomes were comparison of severity of PE, foetal growth restriction, neonatal morbidity and mortality.

Materials and Methods

The trial was a two-armed double-blind parallel randomized control trial conducted in the Department of Obstetrics and Gynaecology, King George's Medical University. The pregnant women attending the outpatient department between 11 and 14 weeks of gestational age (at crown-rump length of 45–84 mm) and meeting the inclusion criteria were enrolled in the study after a written informed consent. The study was carried out over a period of 1 year.

Inclusion criteria were women with high risk of preeclampsia (diagnosed by FMF Eclampsia calculator which involved history, body mass index, medical disorders, mean arterial pressure and uterine artery PI). Exclusion criteria were active peptic ulcer disease, bleeding disorder, chronic kidney disease, thrombocytopenia (platelet count < 1.5 lac), history of vaginal bleeding and foetus diagnosed with malformations/anomaly/aneuploidy.

All women were asked for previous obstetric history, pre-pregnancy weight and past history of preeclampsia. Weight and height were recorded. Mean arterial pressure was calculated for each arm in sitting position. Transvaginal Ultrasound (Toshiba Xario) was done, and crown-rump length and bilateral uterine artery PI were recorded for all enrolled women between 11 and 14 weeks. After enrolment, these women were randomized according to computer-generated random number table into two groups: one receiving 150 mg aspirin and the other 75 mg aspirin per day at bed time. Allocation concealment was done by sequentially numbered opaque sealed envelopes. The principal investigator was blinded for allocation sequence. Pharmacist distributed the sealed drug packets to the women of both groups without disclosing the content and group to the patient. Health care providers were unaware of the intervention drug. Outcome analysis was done after decoding the allocation sequence. Every patient was instructed to take one capsule at bed time starting from the time of enrolment and continued until 36 weeks. Patients were followed till delivery and outcome was recorded. The trial was registered in CTRI no: CTRI/2018/01/011155, and ethical clearance was obtained from the Institutional Ethical Committee of King George's Medical University (reg no ECR/262/Inst/UP/2013): 83rdECM IIB-IMR-F/P2.

Results

A total of 1090 women with singleton pregnancies were screened for preeclampsia, and 200 (18.0%) were found to be at high risk. However, 10 women (5%) were not fulfilling
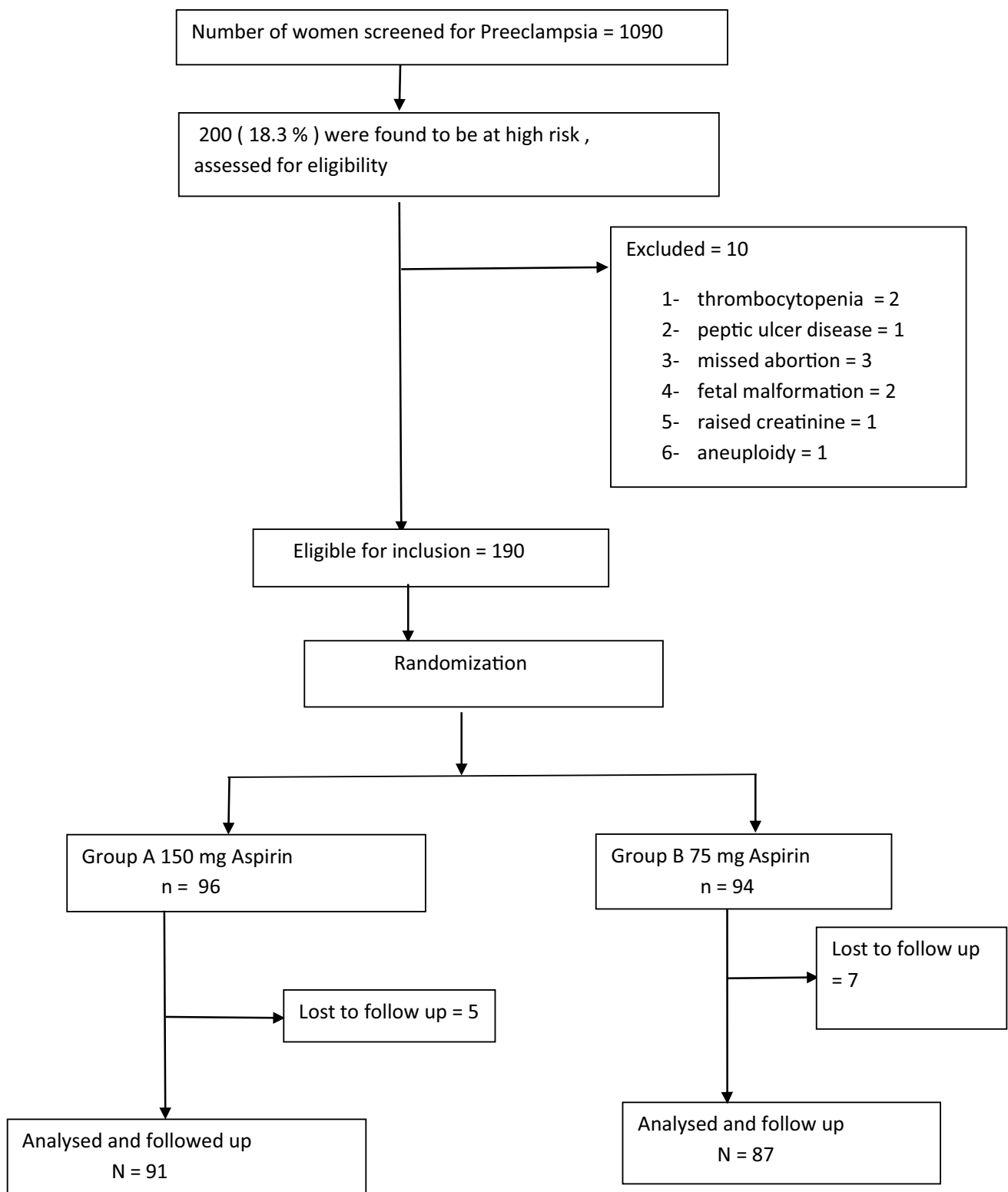
Fig. 1 Flow chart of randomization

the inclusion criteria and were excluded (Fig. 1). After randomization of 190 women who participated in the trial, 12 were lost to follow-up. There were 91 women in group A (150 mg aspirin) and 87 women in group B (75 mg aspirin). After enrolment, five women in group A and seven women in group B refused to participate in the study.

Data were analysed as mean standard deviation (SD), frequency and percentage whenever appropriate. Comparison of the different variables between the study groups was done using t test (for independent samples in case of continuous data) and χ^2-test. For smaller values, Fisher's exact test was used.

There were no significant differences between the aspirin group and the placebo group with regard to the characteristics of the participants at baseline (Table 1).

Preeclampsia occurred in 15 of 87 participants (17%) in the 75 mg aspirin group compared with 6 of 91 (6.5%) in the 150 mg aspirin group. There were a significant difference in the incidence of PE, its severity and period of gestation at delivery in the two groups as shown in Table 2. There was no significant difference in the mode of delivery in both the groups ($p = 0.638$).

Among various high-risk factors seen in women as shown in Table 3, on univariate analysis there was no significant difference in medical diseases or prior history of PE. There were significantly higher values of mean arterial pressure

and uterine artery PI in women who developed preeclampsia compared to those who did not, in both the groups.

Foetal outcomes were observed in both the groups of women, and there was no statistically significant difference between them as shown in Table 4.

Discussion

In this trial, we identified women at high risk of preeclampsia by combined screening which included maternal factors, uterine artery pulsatility index and mean arterial pressure; however, placental growth factor and pregnancy-associated plasma protein A were not incorporated due to financial constraints in a low-resource setting. Studies have shown that the detection rate of preeclampsia at less than 37 weeks by maternal characteristics is 33% while it increases to 72% if uterine artery PI and mean arterial pressure are added to it [15]. Further addition of serum biomarkers improves the detection rate to 77% [16].

Table 1 Characteristics of included women in the trial

	150 mg aspirin n = 91	75 mg aspirin group n = 87	p value
Gestational age (median) (crown-rump length mm)	62.2	63.3	0.458
Age (mean ± SD)	28.1 ± 4.9	27.7 ± 5.2	0.554
Body mass index (mean ± SD)	24.5 ± 3.7	25 ± 4.2	0.478
Method of conception			
(a) Spontaneous	86 (94.5%)	80 (92%)	0.446
(b) OI	3 (3.2%)	(2.3%)	
(c) IVF	2 (2.2%)	5 (5.7%)	
Obstetric history			
(a) Nullipara	67 (73.6%)	60 (68.9%)	0.762
(b) Multipara with PE	7 (7.69%)	(9.2%)	
(c) Multipara without PE	17 (18.68%)	19 (21.89%)	

Table 2 Comparison of the development of PE, complication and mean period of gestation in both the groups

Outcomes	150 mg aspirin n = 91	75 mg aspirin n = 87	p value
Development of preeclampsia	6 (6.5%)	15 (17.2%)	0.046
Early-onset preeclampsia	1 (1%)	5 (5.7%)	0.114
Severe PE	2 (2.1%)	9 (10.3%)	0.051
Placental abruption	2 (2.1%)	4 (4.5%)	0.637
Severe PE	2 (2.1%)	9 (10.3%)	0.051
(a) Thrombocytopenia	0	1 (1.1%)	
(b) HELLP	1 (1%)	2 (2.2%)	
(c) Pulmonary oedema	0	2 (2.2%)	
(d) BP > 160/110 mm Hg	1 (1%)	4 (4.5%)	
Mean POG at the time of delivery	37.3	36.6	0.007

Table 3 Maternal high-risk factors in women who developed PE

	150 mg aspirin n=91			75 mg aspirin n=87			Overall	
							p	Unadjusted OR (95% CI)
Hypothyroidism	8 (8.7%)			7 (8%)			0.858	0.908 (0.315–2.620)
APLA	1 (1%)			3(3.4%)			0.316	3.214 (0.328–31.508)
Autoimmune diseases	1 (1%)			1 (1.1%)			0.974	1.047 (0.064–16.996
GDM	5 (5.4%)			7 (8%)			0.500	1.505 (0.459–4.934)
Type 2 diabetes	1 (1%)			3 (3.4%)			0.316	3.214 (0.328–31.508)
Chronic hypertension	2(2.1%)			5(5.7%)			0.617	1.589 (0.259–9.749)
Mean arterial pressure	102.5±5.24	85.11±7.52	<0.001	99.87±8.36	86.9±7.51	<0.001	0.138	
Uterine artery PI	2.84±0.32	2.23±0.47	0.002	2.66±0.48	2.31±0.50	0.015	0.166	
History of early-onset preeclampsia in previous pregnancy	2 (2.19%)	3 (3.4%)	0.819	0.143 (CI 0.003–5.951)				
History of late-onset PE in previous pregnancy	5 (5.5%)	4 (4.6%)	0.764	0.667 (CI 0.047–9.47864)				

Table 4 Comparison of foetal outcome in both the groups

	150 mg aspirin n=91	75 mg aspirin n=87	p
Preeclampsia			
Still birth	1 (1%)	3 (3.4%)	0.359
Neonatal death	1(1%)	2 (2.3%)	0.264
NICU admission	2 (2.1%)	6 (6.9%)	0.249
Foetal growth restriction	2 (2.1%)	8 (9.2%)	0.088
Baby weight at birth	2.38±0.44	2.03±0.58	0.148
No preeclampsia			
Still birth	0	1 (1.1%)	0.488
Foetal growth restriction	7 (7.7%)	6 (6.9%)	0.838
Baby weight at birth	2.91±0.57	2.83±0.55	0.374
NICU admission	6 (6.6%)	7 (8%)	0.932
Neonatal mortality	1 (1%)	2 (2.3%)	0.264
Total FGR	9 (9.9%)	14 (16%)	0.312

Bujold [10] did a meta-analysis of 34 RCTs in which they compared low-dose aspirin given at ≤ 16 weeks versus placebo and found a higher incidence of preeclampsia (9.3 vs. 21.3%), severe PE (0.7 vs. 15%), FGR (7 vs. 16%), gestational HTN (16.7 vs. 29.7%) and preterm birth (3.5 vs. 16.9%) in the control group. However, studies done later questioned the efficacy of 75 mg aspirin compared to a higher dose. Stephanie Roberge [17] did a meta-analysis of 45 randomized controlled trials, comparing the effect of daily aspirin in various doses or placebo during pregnancy, and reported that prevention of preeclampsia and foetal growth restriction using aspirin in early pregnancy is associated with a dose–response effect (50–150 mg) with higher dosage having a better effect on prevention of preeclampsia ($p < .001$), severe preeclampsia ($p = .008$) and foetal growth restriction ($p < .001$), while low-dose aspirin initiated at more than 16 weeks' gestation has a modest or no impact on the risk of preeclampsia, severe preeclampsia and foetal growth restriction. The entire focus is now shifted on early identification of women at risk of preeclampsia and starting low-dose aspirin preferably before 16 weeks.

Evidence also suggests that preterm PE can be predicted in early pregnancy and prevented with a minimum dose of aspirin initiated also in early pregnancy [15, 18]. The ASPRE trial [19] which compared 150 mg aspirin with placebo has also shown a potential benefit of 150 mg aspirin in prevention of preeclampsia, but they suggested that prevention is restricted to preterm preeclampsia, while we realized that in our population, there is reduction of late preeclampsia as well.

The incidence of women found to be at high risk of preeclampsia in our setting was 18%, and 10.5% of them developed PE. The incidence of PE is high and implies that the pregnant women in this part of the world are more prone to develop preeclampsia. The incidence of preeclampsia has been reported differently in different studies; it is certainly affected by the demography of the population studied. Studies have reported incidence to vary from 1% [20], 21% [11] to as high as 62% [21].

In the group of women who were given 150 mg aspirin, preeclampsia occurred in 6.5% versus 17.2% in women given 75 mg aspirin. There was significant group difference between the development of severe PE (10.3 vs. 2.1%) and mean period of gestation at the time of delivery ($p = 0.007$) as shown in table 2. Both the groups required one or two antihypertensives (labetalol 600–800 mg and nifedipine 10–20 mg) for treatment of hypertension. Magnesium sulphate prophylaxis was given to all women who developed severe PE. Difference in occurrence of placental abruption was not statistically significant in both the groups, and none of the women turned into eclampsia in both the groups.

In a study done by Plasencia et al. [2], they reported that uterine artery screening PI at $11 + 0$ to $13 + 6$ weeks and

the change in uterine artery PI between $11 + 0$ to $13 + 6$ and $21 + 0$ to $24 + 6$ weeks of gestation provided significant independent contribution to the prediction of preeclampsia with the detection rates of early- and late-onset preeclampsia being 90.9 and 31.0%. In our study, we found a higher level of uterine artery PI in women who developed PE. Almost all women had PI value of more than 2.5 irrespective of exact period of gestation between 11 and 14 weeks.

Mean arterial pressure of women who develop preeclampsia was higher than those who did not. In a study by Poon [22], they evaluated the performance of screening for preeclampsia (PE) by maternal medical history and mean arterial pressure (MAP) at 11 weeks to 13 weeks 6 days. The detection rate of PE by log multiple of the median MAP and maternal variables was 62.5% for a false-positive rate of 10%. The mean arterial pressure was affected by ethnic origin, body mass index and personal history of PE. Literature has shown that the predictive strength of mean arterial pressure is moderate [4]. In our study also, we realized that the baseline mean arterial pressure was higher in women who developed PE compared to those who did not.

The incidence of foetal growth restriction among women who developed PE was lower in women given 150 mg aspirin, although the difference was not statistically significant [19]. Other foetal outcomes did not show any significant difference in this trial as shown in Table 4.

A study was carried out by Ebrashy et al. [21] on 139 women found to be at high risk of preeclampsia through uterine artery Doppler at 11–14 weeks, and 75 mg aspirin was started in one arm versus placebo in other. They found that in the aspirin group, the incidence of preeclampsia was 35%, severe PE 7.7% and foetal growth restriction 18.9%, while in this trial among the 91 women who were given 150 mg, the incidence of PE was 6.5%, severe PE 2.1% and early-onset PE 1%. The incidence in the control group of the study by Ebrashy et al. showed a significantly higher incidence of PE, severe PE and foetal growth restriction. It indicates that aspirin is effective in a dose of 75 mg also; however, the efficacy increases with a doubling of dose.

There were no reported side effects noted in the form of bleeding or peptic ulcer in both the groups. Similar to ASPRE trial, our trial showed that the incidence of preeclampsia in the 150 mg aspirin group was lower than that in 75 mg group; however, we also realized that in our study population, 150 mg aspirin also helps to lessen PE developing between 34 and 37 weeks.

The main limitation of the study is the small sample size. The study if carried out at a larger scale could lead to better inference and impact. Also, incorporation of biochemical markers in the form of PAPPA and PLGF would have helped to more precisely predict the women having higher risk of preeclampsia. The biggest strength is the direct comparison of the most commonly used dosage with the recommended dose of the drug. In the light of the trial finding, the benefit applicable in Indian scenario is a consistent use of 150 mg aspirin, a tested dose which is effectively preventing PE without any identifiable harm.

Conclusion

The incidence of both preeclampsia and eclampsia is high in a developing country like India. We encounter maternal deaths due to hypertensive disorders; hence, potential benefit by early screening and intervention is clear and laudable. This randomized trial showed that among women with singleton pregnancies who were identified by means of first-trimester screening as being at high risk of preterm preeclampsia, use of aspirin 150 mg per day started between 11 and 14 weeks till 36 weeks is a potent intervention to reduce the development of both early- and late-onset preeclampsia as compared to a dose of 75 mg per day.

Also, the trial showed that in resource-constrained settings where availability of biomarkers for determination of women at high risk of preeclampsia is very much restricted, screening by history and mean arterial pressure can be of great help.

Compliance with Ethical Standards

Conflict of interest There is no conflict of interest among the authors. There is no financial relationship with any organization.

Ethical Statement All procedures followed were in accordance with the ethical standards of the responsible Institutional Committee on human experimentation and with the Helsinki Declaration of 1975, as revised in 2008 (5).

Informed consent Informed consent was obtained from all patients for being included in the study.

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