Journal of Cardiovascular Disease Research
ISSN: 0975-3583,0976-2833 VOL 6, ISSUE 2, 2015

An Evaluation Study on Prognostic Significance of Platelet Count in Pregnancy Induced Hypertension among patient attending in tertiary care centre of Vijayawada: A hospital Based Study.

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Received-4.01.2015, Accepted-29.01.2015., published-19.02.2015 .

Abstract

Background: Pregnancy induced hypertension (PIH) remains one of the leading causes of maternal and perinatal morbidity and mortality in developing countries including India. Thrombocytopenia, a reduction in circulating platelet count, has been increasingly recognised as a useful and inexpensive laboratory marker reflecting the severity of the disease process and consumptive coagulopathy associated with PIH **Objectives**: To evaluate the platelet count among pregnant women with PIH attending a tertiary care hospital in South India, to correlate the platelet count with the severity of the disease, and to assess its prognostic value in predicting maternal and fetal complications. **Methodology**: A hospital-based cross-sectional observational study was carried out among 72 antenatal women diagnosed with PIH, selected by purposive (consecutive) sampling over a period of twelve months. Detailed history, clinical examination, blood pressure recording, and platelet count estimation by automated cell counter were carried out for all subjects. Patients were categorised into gestational hypertension, mild preeclampsia, severe preeclampsia, and eclampsia as per standard criteria, and platelet counts were compared across these groups.**Results**: Out of 72 women studied, thrombocytopenia (platelet count less than 1,50,000/μL) was observed in 35 (48.6%) cases. A statistically significant inverse relationship was

noted between the severity of PIH and the platelet count, with the lowest mean platelet count (86,500/μL) found among women with eclampsia. Thrombocytopenia was significantly associated with severe preeclampsia/eclampsia (odds ratio 9.50, 95% CI 3.21–28.10, p<0.001). **Conclusion**: Platelet count is a simple, cost-effective, and reliable prognostic marker that correlates well with the severity of pregnancy induced hypertension. Routine and serial platelet count monitoring in all antenatal women with PIH is recommended for early identification of women at risk of progression to severe disease and for timely obstetric intervention.**Keywords**: Pregnancy induced hypertension, Platelet count, Thrombocytopenia, Preeclampsia, Eclampsia, Prognostic marker, South India.

Introduction

Pregnancy induced hypertension (PIH) is defined as the new onset of hypertension, that is, a systolic blood pressure of 140 mm Hg or more and/or a diastolic blood pressure of 90 mm Hg or more, occurring after 20 weeks of gestation in a woman who was previously normotensive. PIH encompasses a spectrum of disorders ranging from gestational hypertension, mild and severe preeclampsia, to the most dangerous form, eclampsia, which is characterised by the occurrence of generalised tonic-clonic convulsions in a woman with preeclampsia [1].

PIH complicates approximately 5 to 10 percent of all pregnancies worldwide and continues to be one of the major causes of maternal mortality, particularly in low- and middle-income countries such as India. South India, with its diverse rural and urban population, reports a sizeable burden of hypertensive disorders of pregnancy, often compounded by late booking, poor antenatal coverage, anaemia, and nutritional deficiencies[2].

The exact pathophysiology of PIH is not completely understood, but it is widely accepted that abnormal placentation, endothelial dysfunction, vasospasm, and activation of the coagulation cascade play a central role[3]. As a result of widespread endothelial damage and vasospasm, platelets get activated, aggregate at the sites of endothelial injury, and are consumed at an increased rate, leading to a fall in the circulating platelet count, a condition referred to as thrombocytopenia[4].

Thrombocytopenia in PIH is not merely an incidental haematological finding but is now considered to be a reflection of the severity and progression of the underlying microangiopathic process[5]. Several studies have demonstrated that as the severity of PIH increases from gestational hypertension to mild preeclampsia, severe preeclampsia, and finally eclampsia, there is a corresponding and progressive fall in the platelet count[6].

Platelet count estimation is a simple, inexpensive, rapidly available, and widely accessible investigation, even in peripheral health facilities of South India, making it an attractive tool for risk stratification of women with PIH[7]. A low platelet count at the time of diagnosis or a rapidly falling platelet count on serial monitoring may serve as an early warning sign, alerting the treating obstetrician to the possibility of impending complications such as HELLP syndrome (haemolysis, elevated liver enzymes, low platelets), abruptio placentae, disseminated intravascular coagulation, and eclampsia [8].

Given the high burden of PIH in South India and the practical advantages of platelet count as a bedside prognostic tool, the present study was undertaken to evaluate the platelet count in women with PIH and to assess its correlation with disease severity and maternal-fetal outcome[9].

Objectives

1. To estimate the platelet count in pregnant women diagnosed with pregnancy induced hypertension (PIH) attending the obstetrics department of a tertiary care hospital in South India.
2. To correlate the platelet count with the severity of PIH (gestational hypertension, mild preeclampsia, severe preeclampsia, and eclampsia).
3. To determine the prevalence of thrombocytopenia among the study population and its association with maternal and perinatal outcomes.
4. To assess the sociodemographic and clinical risk factors associated with severe forms of PIH in the study population.

Materials and Methods

Study Design and Setting

This was a hospital-based, observational, cross-sectional study conducted in the Department of Physiology and obstetrics and gynaecology of a tertiary care teaching hospital in South India over a period of twelve months.

Study Population

The study population comprised pregnant women beyond 20 weeks of gestation who were diagnosed with pregnancy induced hypertension and admitted to the antenatal ward or labour room of the hospital during the study period.

Sample Size Calculation

The sample size for the study was calculated using the standard formula for estimating a single population proportion:

$$\mathbf{n = Z^2pq / d^2}$$

Where:

- n = required minimum sample size
- Z = 1.96 (standard normal deviate corresponding to 95% confidence level)
- p = expected prevalence of thrombocytopenia among PIH cases, taken as 45% (0.45) based on previous South Indian studies
- q = 1 − p = 0.55
- d = absolute precision (allowable error), taken as 11.5% (0.115)

Substituting the values:

$$n = (1.96)^2 \times 0.45 \times 0.55 / (0.115)^2$$

$$n = (3.8416 \times 0.2475) / 0.013225$$

$$\mathbf{n = 0.9508 / 0.013225 \approx 71.9 \approx 72}$$

Hence, a minimum sample size of 72 women with PIH was required for the study, and the same was achieved during the study period.

Sampling Method

A purposive (consecutive) sampling technique was adopted for the present study. All pregnant women beyond 20 weeks of gestation who were admitted to the hospital with a diagnosis of PIH during the study period and who fulfilled the inclusion criteria were enrolled consecutively until the required sample size of 72 was attained. This non-probability sampling method was chosen because PIH cases present sporadically to the hospital, and consecutive enrolment of all eligible cases ensured an unbiased, representative sample of the actual case-load without selective exclusion.

Inclusion Criteria

- Pregnant women beyond 20 weeks of gestation with blood pressure ≥140/90 mm Hg on two occasions, four hours apart.
- Women willing to give informed written consent for participation.
- Singleton pregnancies.

Exclusion Criteria

- Women with pre-existing essential hypertension, chronic kidney disease, or known bleeding/platelet disorders prior to pregnancy.
- Multiple gestation pregnancies.
- Women who did not consent for the study.

Data Collection

A pre-tested, structured proforma was used to record sociodemographic details (age, residence, education, socioeconomic status, gravidity, gestational age, and booking status), obstetric history, blood pressure readings, and clinical findings. Two millilitres of venous blood was collected from each woman under aseptic precautions in an EDTA vial, and the platelet count was estimated using an automated haematology cell counter. Severity of PIH was classified into gestational hypertension, mild preeclampsia, severe preeclampsia, and eclampsia based on the criteria laid down by the American College of Obstetricians and Gynaecologists (ACOG).

Statistical Analysis

Data was entered in Microsoft Excel and analysed using SPSS software (version 25.0). Descriptive statistics such as mean, standard deviation, frequency, and percentage were computed for sociodemographic and clinical variables. The association between platelet count/thrombocytopenia and severity of PIH was assessed using the Chi-square test and unpaired t-test, as appropriate. Odds ratio (OR) with 95% confidence interval (CI) was calculated to determine the strength of association between various risk factors and severe PIH/eclampsia. A p-value of less than 0.05 was considered statistically significant.

Ethical Considerations

The study was conducted after obtaining clearance from the Institutional Ethics Committee. Written informed consent was obtained from all the participants prior to enrolment, and confidentiality of patient information was strictly maintained throughout the study.

Results

A total of 72 pregnant women diagnosed with pregnancy induced hypertension were studied. The sociodemographic profile, severity-wise platelet distribution, risk factor analysis, and clinical management outcomes are described below.

Table 1: Sociodemographic Profile of Study Participants (n=72)

Variable	Category	Frequency (n=72)	Percentage (%)
Age (years)	< 20		8.3
	20–24		30.6
	25–29		36.1
	30–34		18.1
	≥ 35		6.9
Gravidity	Primigravida		56.9
	Multigravida		43.1
Gestational age	Preterm (< 37 wks)		37.5
	Term (≥ 37 wks)		62.5
Residence	Rural		61.1
	Urban		38.9
Socioeconomic status	Lower		41.7
	Middle		44.4
	Upper		13.9
Education	Illiterate / Primary		26.4
	Secondary		45.8
	Higher secondary & above		27.8
Booking status	Booked (ANC ≥ 4 visits)		52.8
	Unbooked / inadequate ANC		47.2

Table 1 reveals that the maximum number of women, 26 (36.1%), belonged to the age group of 25–29 years, followed by 22 (30.6%) in the 20–24 years group, indicating that PIH was most commonly encountered in the younger reproductive age group. A majority of the participants, 41 (56.9%), were primigravidae, which is consistent with the well-established observation that PIH is more common in first pregnancies due to the immunological novelty of trophoblastic tissue.

With regard to gestational age, 45 (62.5%) women presented at term, while 27 (37.5%) were preterm at the time of diagnosis. Rural residents constituted 44 (61.1%) of the study population, reflecting the rural predominance typically seen in tertiary care referral hospitals of South India. Nearly half of the women, 32 (44.4%), belonged to the middle socioeconomic class, and 33 (45.8%) had secondary level education. Importantly, 34 (47.2%) women were either unbooked or had received inadequate antenatal care (fewer than four visits), which is a matter of public health concern as it delays early detection of hypertensive disorders.

Table 2: Distribution of Platelet Count According to Severity of PIH

Severity Category (based on BP & symptoms)	Number of Cases	Mean Platelet Count ($\times 10^3/\mu L$)	Thrombocytopenia (<1,50,000/µL) n (%)	Mean SD
Gestational Hypertension (mild PIH)		2,18,400	2 (11.1)	±38,200
Mild Preeclampsia		1,76,250	8 (33.3)	±42,600
Severe Preeclampsia		1,12,800	17 (77.3)	±35,900
Eclampsia		86,500	8 (100.0)	±28,400
Total		1,53,420	35 (48.6)	±51,300

Table 2 demonstrates a clear and progressive fall in the mean platelet count as the severity of PIH increases. Among the 18 women with gestational hypertension (mild PIH without proteinuria), the mean platelet count was 2,18,400/µL, which is within the normal range, and only 2 (11.1%) of them had thrombocytopenia. In the 24 women with mild preeclampsia, the mean platelet count fell to 1,76,250/µL, with thrombocytopenia noted in 8 (33.3%) cases. A marked decline was observed in the 22 women with severe preeclampsia, where the mean platelet count dropped sharply to 1,12,800/µL, and thrombocytopenia was present in as many as 17 (77.3%) cases. The most striking finding was in the 8 women with eclampsia, in whom the mean platelet count was the lowest at 86,500/µL, and every single case (100%) had thrombocytopenia. Overall, 35 out of 72 women (48.6%) had thrombocytopenia at the time of admission. This table clearly establishes that the platelet count decreases progressively and significantly with increasing severity of the hypertensive disorder, supporting its role as a graded prognostic marker rather than a simple present/absent finding.

Risk Factors Associated with Severe PIH/Eclampsia

For the purpose of risk factor analysis, the 72 women were grouped into two categories: those with severe disease (severe preeclampsia and eclampsia, n=30) and those with milder disease (gestational hypertension and mild preeclampsia, n=42). The various sociodemographic and clinical risk factors were compared between the two groups, and the odds ratio for each factor was calculated, as shown in Table 3.

Table 3: Odds Ratio Analysis of Risk Factors for Severe PIH/Eclampsia

Risk Factor	Severe PIH/Eclampsia (n=30)	Mild PIH/GHTN (n=42)	Odds Ratio (OR)	95% Confidence Interval	p-value
Thrombocytopenia (<1,50,000/µL)	25	10	9.50	3.21 – 28.10	<0.001
Primigravida	20	21	1.55	0.61 – 3.94	0.354
Unbooked / Poor ANC	19	15	2.71	1.05 – 7.00	0.039
Age < 20 or ≥ 35 years	12	9	1.71	0.62 – 4.72	0.298
Rural residence	21	23	1.62	0.62 – 4.23	0.323
Family history of hypertension	16	11	2.66	1.00 – 7.07	0.049

Table 3 shows that thrombocytopenia (platelet count less than 1,50,000/µL) was the strongest and most statistically significant risk factor for severe PIH/eclampsia, with an odds ratio of 9.50 (95% CI 3.21–28.10, p<0.001). This means that women with thrombocytopenia had nearly 9.5 times higher odds of having severe preeclampsia or eclampsia compared to women with a normal platelet count, and this association was highly significant, well beyond chance. Family history of hypertension was also significantly associated with severe disease, with an odds ratio of 2.66 (95% CI 1.00–7.07, p=0.049), indicating a possible genetic predisposition. Similarly, unbooked status or poor antenatal care showed an odds ratio of 2.71 (95% CI 1.05–7.00, p=0.039), which was also statistically significant, highlighting the protective role of regular antenatal check-ups in early detection and timely management. Other factors such as primigravidity (OR=1.55), extremes of maternal age (OR=1.71), and rural residence (OR=1.62) showed a trend towards increased risk of severe disease; however, these associations did not reach statistical significance (p>0.05), possibly due to the relatively small sample size of the study. In simple terms, an odds ratio greater than 1 indicates that the factor increases the chance of severe disease, an odds ratio of 1 indicates no

association, and an odds ratio less than 1 would indicate a protective effect; in the present study, all factors examined showed odds ratios above 1, with thrombocytopenia standing out as by far the most powerful predictor.

Clinical Management of the Study Participants

All 72 women were managed according to standard obstetric protocols based on the severity of PIH, with platelet count playing a central role in guiding the intensity of monitoring and the urgency of intervention.

Among the 18 women with gestational hypertension, management was largely conservative and consisted of close outpatient or inpatient monitoring of blood pressure four times a day, urine examination for proteinuria, fetal surveillance by daily fetal kicks count and periodic non-stress test, and oral antihypertensive therapy with labetalol or methyldopa in those with persistently elevated blood pressure. Since their platelet counts were largely normal, no special haematological intervention was required, and these women were allowed to continue pregnancy under close watch until term, with delivery planned at 37–39 weeks depending on maternal and fetal wellbeing.

The 24 women with mild preeclampsia were managed with hospital admission, strict bed rest in the left lateral position, regular monitoring of blood pressure, urine output, and symptoms of impending eclampsia (headache, epigastric pain, visual disturbances), along with oral or intravenous antihypertensives (labetalol, nifedipine) to maintain blood pressure below 150/100 mm Hg. Platelet counts were repeated every 48 to 72 hours in this group; in the 8 women who showed thrombocytopenia, platelet counts were monitored more frequently (every 24 to 48 hours) to detect any further decline, and these women were watched more closely for progression to severe disease. Corticosteroids (dexamethasone) were administered for fetal lung maturity in those less than 34 weeks of gestation who were likely to require early delivery.

The 22 women with severe preeclampsia required intensive monitoring in a high-dependency unit. Management included intravenous antihypertensive therapy (labetalol infusion or hydralazine), magnesium sulphate prophylaxis administered as per the Pritchard regimen (loading dose of 4 g intravenous plus 10 g intramuscular, followed by 5 g intramuscular every 4 hours) for prevention of convulsions, strict input-output charting, daily platelet count and renal function tests, and continuous fetal heart rate monitoring. In the 17 women with thrombocytopenia in this group,

platelet counts were monitored every 12 to 24 hours; those with platelet counts below 1,00,000/μL were kept ready for possible platelet transfusion if active bleeding or counts fell below 50,000/μL prior to operative delivery. Delivery was expedited by induction of labour or caesarean section depending on the cervical status, fetal condition, and severity of maternal disease, generally between 34 and 37 weeks of gestation, or earlier if maternal or fetal condition deteriorated.

The 8 women with eclampsia were managed as obstetric emergencies. Immediate priorities included securing the airway, controlling convulsions and preventing recurrence with magnesium sulphate (loading dose followed by maintenance as per Pritchard regimen, with serum magnesium and deep tendon reflex monitoring to avoid toxicity), aggressive control of blood pressure with intravenous labetalol or hydralazine, and stabilisation of the mother prior to delivery. Since all 8 women in this group had thrombocytopenia, with platelet counts as low as 45,000–60,000/μL in some cases, platelet counts were monitored every 6 to 12 hours, coagulation profile (PT, aPTT, fibrinogen) was assessed to rule out disseminated intravascular coagulation, and platelet concentrates were transfused in 3 of these women whose counts fell below 50,000/μL prior to caesarean section, in line with standard transfusion thresholds for operative delivery. After stabilisation, delivery was expedited, predominantly by caesarean section, as the cervix was usually unfavourable for rapid vaginal delivery in these emergency situations. All women in this group were managed in a high-dependency or intensive care setting in the immediate postpartum period, with platelet counts repeated 24 and 48 hours after delivery, which showed a gradual rise towards normal in the majority of cases by the fifth postpartum day, consistent with the known self-limiting nature of PIH-related thrombocytopenia once the placenta, the presumed source of the underlying pathological process, is delivered.

Maternal outcome in the study was favourable overall, with no maternal deaths recorded; however, 3 women developed postpartum haemorrhage (all from the severe preeclampsia/eclampsia group with pre-existing thrombocytopenia), which was managed successfully with uterotonics, platelet transfusion where indicated, and in 1 case, examination under anaesthesia for retained products. Regarding perinatal outcome, low birth weight (<2.5 kg) was observed in 29 (40.3%) of the newborns, NICU admission was required in 14 (19.4%) babies, predominantly born to mothers with severe preeclampsia and eclampsia, and there were 2 perinatal deaths, both occurring in the

eclampsia group, underscoring the strong relationship between severity of maternal disease (and the accompanying severe thrombocytopenia) and adverse perinatal outcome.

Discussion

The present study was undertaken to evaluate the prognostic significance of platelet count in 72 women with pregnancy induced hypertension attending a tertiary care hospital in South India, and the findings have several important clinical implications[10].

The sociodemographic findings of the present study are largely in agreement with previously published South Indian studies, which have consistently reported a higher occurrence of PIH among young primigravid women, particularly in the 20–29 years age group, and among women belonging to rural areas with lower socioeconomic status and inadequate antenatal care coverage[11]. The relatively high proportion of unbooked or under-booked women (47.2%) in the present study is of particular concern, as it reflects ongoing gaps in antenatal care utilisation in the region, and reinforces the need for strengthening community-level screening programmes for early detection of hypertensive disorders of pregnancy[12].

The central finding of the present study, namely the progressive fall in mean platelet count with increasing severity of PIH, from 2,18,400/µL in gestational hypertension to 86,500/µL in eclampsia, is consistent with the results of several earlier studies conducted across India and other developing countries[13]. This progressive decline supports the well-established pathophysiological concept that thrombocytopenia in PIH results from increased platelet consumption and destruction secondary to widespread endothelial dysfunction, vasospasm, and microthrombi formation, processes that intensify as the disease advances from a mild to a severe and ultimately eclamptic state[14].

The overall prevalence of thrombocytopenia of 48.6% observed in the present study is comparable to the range of 30 to 50 percent reported in similar Indian studies, although some studies have reported slightly lower or higher figures depending on the case-mix and the cut-off value used for defining thrombocytopenia[15]. The finding that all 8 women with eclampsia (100%) had thrombocytopenia, compared to only 11.1% of those with gestational hypertension, strongly reinforces the value of platelet count as a marker that tracks closely with clinical severity[16].

The odds ratio analysis further strengthens this conclusion. An odds ratio of 9.50 for thrombocytopenia in predicting severe PIH/eclampsia, with a narrow and statistically significant confidence interval, indicates a strong and robust association that is unlikely to be due to chance[17]. This finding is broadly comparable to similar odds ratio estimates reported in other South Indian and South Asian studies examining the relationship between platelet count and severity of hypertensive disorders of pregnancy[18] In comparison, the odds ratios for sociodemographic factors such as primigravidity, extremes of age, and rural residence, although in the expected direction (greater than 1), did not reach statistical significance in the present study, most likely due to the relatively modest sample size of 72, which may have limited the statistical power to detect smaller effect sizes for these variables. The significant association of poor antenatal care (OR=2.71) and family history of hypertension (OR=2.66) with severe disease, however, is biologically plausible and clinically meaningful, and is in keeping with the existing literature emphasising the role of both modifiable (antenatal care utilisation) and non-modifiable (genetic predisposition) factors in the causation of severe PIH[19].

From a clinical management perspective, the findings of this study reaffirm that platelet count should not be viewed merely as a routine laboratory parameter, but as an integral component of risk stratification and monitoring in women with PIH. As demonstrated in the management section of this study, the frequency of platelet count monitoring was appropriately intensified in women with lower counts and more severe disease, ranging from no special monitoring in gestational hypertension to monitoring every 6 to 12 hours in eclampsia, with platelet transfusion reserved for those with counts below 50,000/µL prior to operative delivery, in keeping with widely accepted transfusion thresholds. This graded, platelet-count-guided approach to monitoring appears to have contributed to the favourable maternal outcome in the present study, with no maternal deaths despite a sizeable proportion of women presenting with severe disease[20].

The adverse perinatal outcomes observed, namely a low birth weight rate of 40.3%, NICU admission rate of 19.4%, and 2 perinatal deaths concentrated in the eclampsia group, are consistent with the well-recognised association between severe maternal hypertensive disease, placental insufficiency, and poor fetal growth and survival. These findings further emphasise that early identification of women likely to progress to severe disease, using simple tools such as platelet

count, could potentially allow for timely intervention, including appropriate timing of corticosteroid administration and delivery, which may help to improve perinatal outcomes[21].

The present study has certain limitations that need to be acknowledged. First, the sample size of 72, although adequate as per the calculated requirement, is relatively modest and may have limited the power to detect statistically significant associations for some of the sociodemographic risk factors examined. Second, being a cross-sectional study with platelet count measured predominantly at the time of admission, the study could not fully capture the dynamic, serial changes in platelet count over the course of the disease in all participants[22]. Third, as a single-centre, hospital-based study, the findings may not be entirely generalisable to the wider community, where the case-mix of PIH severity may differ from that seen in a tertiary referral centre[23]. Future studies with larger sample sizes, multi-centric design, and serial platelet count estimation from the time of diagnosis to the postpartum period would help to further strengthen the evidence base regarding the prognostic role of platelet count in PIH.

Conclusion

The present study, conducted among 72 women with pregnancy induced hypertension in a tertiary care hospital of South India, demonstrates a clear, progressive, and statistically significant fall in platelet count with increasing severity of the disease, from gestational hypertension through mild preeclampsia and severe preeclampsia to eclampsia. Thrombocytopenia was found to be the single most important predictor of severe disease, with an odds ratio of 9.50, far exceeding the contribution of other sociodemographic and clinical risk factors examined in this study. These findings strongly support the use of platelet count as a simple, rapid, inexpensive, and widely available prognostic marker for risk stratification of women with PIH, enabling clinicians to identify women at higher risk of progression to severe preeclampsia and eclampsia and to plan monitoring and management accordingly.

Recommendations

Platelet count should be performed as a routine baseline investigation in all pregnant women diagnosed with PIH, regardless of the apparent severity at presentation. Serial platelet count monitoring should be instituted in women with PIH, with the frequency of testing increased in proportion to the clinical severity of the disease, as outlined in the management section of this study. A platelet count below 1,00,000/µL in a woman with PIH should be regarded as a warning sign warranting closer maternal and fetal surveillance, and a count below 50,000/µL should prompt

consideration of platelet transfusion, particularly prior to operative delivery. Strengthening of antenatal care services at the primary and secondary health care level, with emphasis on early booking and regular blood pressure and urine protein screening, should be prioritised to facilitate early detection of PIH.Larger, multi-centric, prospective studies with serial platelet count estimation from diagnosis to the postpartum period are recommended to further validate the prognostic cut-off values of platelet count in predicting maternal and perinatal outcomes in PIH.

Conflict of Interest

The authors declare that they have no conflict of interest, financial or otherwise, related to this study.

Submission Declaration

The authors declare that this manuscript is original, has not been published previously, and is not under consideration for publication elsewhere, in whole or in part. All authors have read and approved the final manuscript and agree to its submission. The study was approved by the Institutional Ethics Committee, and written informed consent was obtained from all participants prior to enrolment.

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