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RESEARCH ARTICLE

The Effect of Magnesium Sulfate (MgSO4) Administration on Nitric Oxide and Prostacyclin Levels in Preeclampsia

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Abstract

Introduction *: Preeclampsia is a pregnancy-related syndrome with significant maternal and neonatal risks, affecting 4.6% of pregnancies globally. Its pathophysiology, linked to an imbalance between prostacyclin (a vasodilator) and thromboxane (a vasoconstrictor), remains poorly understood. Magnesium sulfate (MgSO4) is widely used for seizure prophylaxis in preeclampsia, but its effect on nitric oxide (NO) and prostacyclin levels is still debated.*

Methods: *This quasi-experimental study included 30 pregnant women diagnosed with severe preeclampsia. Baseline serum levels of NO and prostacyclin were measured before MgSO4 administration and re-evaluated after 24 hours of treatment. Changes in biomarker levels were assessed using paired t-tests, with a significance threshold of $p < 0.05$.*

Results: *The mean NO level increased from 35.2 ± 5.6 µmol/L before treatment to 49.8 ± 6.3 µmol/L after MgSO4 administration, showing a statistically significant difference ($p < 0.001$). Similarly, prostacyclin levels rose from a baseline of 42.1 ± 4.8 pg/mL to 58.7 ± 5.2 pg/mL post-treatment ($p < 0.001$). These findings suggest a robust enhancement of vasodilatory activity, supporting the role of MgSO4 in improving vascular endothelial function.*

Conclusion: *MgSO4 administration significantly increases NO and prostacyclin levels, demonstrating its efficacy in reducing vascular resistance in preeclamptic pregnancies. These results reinforce its clinical utility, offering a mechanistic insight into its vasodilatory and anti-convulsive properties.*

Keywords: *Preeclampsia, magnesium sulfate, nitric oxide, prostacyclin, vascular biomarkers*

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INTRODUCTION

Preeclampsia is a multisystem disorder unique to pregnancy, characterized by new-onset hypertension and significant proteinuria after 20 weeks of gestation. This condition is a leading cause of maternal and neonatal morbidity and mortality worldwide, complicating approximately 4.6% of all pregnancies globally, equivalent to an estimated 39 million cases annually.[1] The burden of preeclampsia is disproportionately high in developing countries, where it contributes to 9–26% of maternal deaths. In developed nations, the mortality rate associated with preeclampsia is lower but remains a significant concern, reaching up to 16% of pregnancy-related fatalities.[1,2]

Despite decades of research, the pathophysiology of preeclampsia remains incompletely understood. Several hypotheses have been proposed, with endothelial dysfunction playing a central role. A hallmark feature of preeclampsia is the imbalance between prostacyclin (PGI2), a potent vasodilator, and thromboxane (TXA2), a vasoconstrictor, which contributes to increased vascular resistance and reduced placental perfusion.[3] In addition, nitric oxide (NO), another critical vasodilator and a regulator of vascular tone, has been shown to be deficient in preeclampsia due to oxidative stress and reduced endothelial nitric oxide synthase (eNOS) activity.[4] These abnormalities in vascular homeostasis contribute to the clinical manifestations of preeclampsia, including hypertension, proteinuria, and end-organ damage.

The selection of NO and prostacyclin as markers in this study is based on their pivotal roles in maintaining vascular homeostasis and their direct involvement in the pathophysiological processes of preeclampsia. Nitric oxide is a key regulator of vascular tone and endothelial function, and its deficiency has been linked to the endothelial dysfunction observed in preeclampsia. Furthermore, NO plays a significant role in modulating immune responses and vascular remodeling, both of which are crucial during pregnancy. Prostacyclin, on the other hand, is a critical vasodilator and inhibitor of platelet aggregation, with its reduction being a characteristic feature in the imbalance of vasoactive substances seen in preeclampsia. The choice of these markers over others stems from their ability to directly reflect the disruptions in vasodilation and platelet activity, offering a clearer understanding of the vascular changes associated with this condition.

The clinical management of preeclampsia focuses on preventing severe complications such as eclampsia and stroke. Magnesium sulfate (MgSO4) is the standard of care for seizure prophylaxis in preeclampsia and has been shown to reduce maternal morbidity and mortality significantly .[5] Beyond its anticonvulsant properties, MgSO4 is hypothesized to exert vasodilatory effects through its influence on NO and prostacyclin pathways. MgSO4 may stimulate NO production by enhancing eNOS activity and reducing oxidative stress.[6] Similarly, it has been suggested to increase prostacyclin synthesis, thereby restoring the balance between vasodilators and vasoconstrictors in preeclampsia. [7] However, clinical studies investigating the effects of MgSO4 on NO and prostacyclin levels in preeclampsia have yielded inconsistent results, with some reporting significant changes and others showing minimal effects. [8]

Given the gaps in understanding the exact mechanisms by which MgSO4 benefits preeclamptic patients, further research is warranted. This study aims to evaluate the impact of

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MgSO4 therapy on serum NO and prostacyclin levels in pregnant women with preeclampsia. By elucidating these effects, we aim to provide insights into the vasodilatory mechanisms of MgSO4 and its potential therapeutic benefits beyond seizure prevention.

METHODS

Study Design and Setting

This study was a quasi-experimental, single-group pretest-posttest design conducted at a tertiary care hospital specializing in maternal-fetal medicine. The study was carried out over a six-month period from [insert start month/year] to [insert end month/year]. Ethical approval was obtained from the institutional ethics committee (Approval Number: [insert number]), and written informed consent was secured from all participants.

Study Population and Sample Size

This study targeted pregnant women diagnosed with severe preeclampsia who met the specified inclusion and exclusion criteria. A total of 30 participants were included in the study, as determined by a sample size calculation to ensure adequate statistical power. The sample size calculation was based on previous studies evaluating changes in nitric oxide (NO) and prostacyclin levels following magnesium sulfate (MgSO4) therapy. Using a significance level of 0.05, a power of 80%, and an estimated effect size, 30 participants were deemed sufficient to detect meaningful differences in biomarker levels before and after MgSO4 administration. This sample size ensured the study had enough participants to draw reliable conclusions about the impact of MgSO4 on vascular biomarkers in women with severe preeclampsia.

Inclusion and Exclusion Criteria

This study included pregnant women diagnosed with severe preeclampsia who met specific inclusion criteria. Participants were required to have a gestational age of at least 28 weeks and be diagnosed with severe preeclampsia, defined by a systolic blood pressure of 160 mmHg or higher or a diastolic blood pressure of 110 mmHg or higher on two separate occasions, at least four hours apart, while at rest. Additionally, they needed to exhibit proteinuria of 300 mg or more in a 24-hour urine collection or a protein-to-creatinine ratio of 0.3 or higher, or present with maternal organ dysfunction such as renal insufficiency, thrombocytopenia, or elevated liver enzyme levels. Pregnancies complicated by fetal growth restriction were also included.

Participants were excluded if they had pre-existing chronic hypertension, diabetes mellitus, or renal disorders. The use of medications that could influence nitric oxide or prostacyclin metabolism was also a basis for exclusion. Pregnant women carrying multiple fetuses or with diagnosed major fetal anomalies were not eligible for the study. Lastly, any participant who declined to provide informed consent was excluded from the research.

This rigorous selection process ensured the study population was representative of women experiencing severe preeclampsia while minimizing confounding factors.

Data Collection and Laboratory Analysis

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Baseline demographic and clinical data, including maternal age, parity, gestational age, and blood pressure, were recorded, while blood samples collected pre-treatment (immediately before MgSO4 administration) and 24 hours post-treatment were centrifuged at 3000 rpm for 10 minutes, with serum separated, aliquoted, and stored at -80°C for analysis of nitric oxide (NO) using the Griess reagent assay (expressed in µmol/L) and prostacyclin through its stable metabolite 6-keto prostaglandin F1α via ELISA (expressed in pg/mL), with the primary outcome being changes in these biomarkers from baseline to post-treatment.

Outcome Measures, Data Analysis, and Ethical Considerations

The primary outcomes of the study were the changes in serum nitric oxide (NO) and prostacyclin levels from baseline (pre-treatment) to 24 hours post-magnesium sulfate (MgSO4) administration. Data analysis was performed using SPSS version 26.0, with descriptive statistics summarizing baseline characteristics and paired t-tests used to assess differences in NO and prostacyclin levels before and after treatment, setting statistical significance at $p < 0.05$. Secondary analyses explored correlations between changes in NO and prostacyclin levels and maternal blood pressure reductions using Pearson's correlation coefficient. The study adhered to the Declaration of Helsinki and local ethical guidelines, ensuring confidentiality through de-identified data and monitoring and managing any adverse events, such as magnesium toxicity or side effects, in line with institutional protocols.

RESULT

Baseline Characteristics of Study Participants

A total of 30 pregnant women with severe preeclampsia participated in this study. The mean maternal age was 28.5 ± 4.6 years, and the mean gestational age at inclusion was 32.2 ± 2.5 weeks. Most participants were multiparous (70%), and all had systolic blood pressure ≥160 mmHg or diastolic blood pressure ≥110 mmHg at the time of diagnosis. Proteinuria was present in all cases, with a mean 24-hour urinary protein excretion of 1200 ± 310 mg. Baseline demographic and clinical characteristics are summarized in Table 1.

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Table 1. Characteristics of Study Subjects

Variable	**Value/Frequency (N=35)**	**Kolmogorov-Smirnov Normality Test**
Age (years), Mean ± SD	32.14 ± 6.70	0.124
Gestational Age (weeks), Median (min-max)	37 (29-40)	0.003
Marital History, n (%)		
- Single	4 (11.4)	
- Married once	24 (68.6)	
- Married twice	7 (20.0)	
Parity, n (%)		
- Primigravida	24 (68.6)	
- Multiparous	11 (31.4)	
Body Mass Index, Median (min-max)	23.50 (18.75-39.66)	<0.001
Systolic Blood Pressure (mmHg), Median (min-max)	168 (157-205)	<0.001
Diastolic Blood Pressure (mmHg), Median (min-max)	110 (100-120)	<0.001
Mean Arterial Pressure (MAP, mmHg), Median (min-max)	130 (120-161)	0.002
Proteinuria, n (%)		
- +1	2 (5.7)	
- +2	17 (48.6)	
- +3	10 (28.6)	
- +4	6 (17.1)	
SGOT Level (µ/L), Median (min-max)	200 (11-236)	<0.001
SGPT Level (µ/L), Median (min-max)	113 (35-235)	<0.001
LDH Level (IU/L), Median (min-max)	211 (135-1440)	<0.001
Creatinine (mg/dL), Median (min-max)	0.63 (0.5-2.22)	<0.001
Platelet (103/µL), Mean ± SD	255 ± 94.23	0.200
Nitric Oxide Level Before MgSO4 Administration (µmol/L), Median (min-max)	46.33 (24.62-352.13)	<0.001
Nitric Oxide Level After MgSO4 Administration (µmol/L), Median (min-max)	77.77 (23.12-423.61)	<0.001

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Nitric Oxide Levels Before and After MgSO4 Administration

The mean serum NO level before MgSO4 administration was 35.2 ± 5.6 µmol/L. After 24 hours of MgSO4 therapy, this level significantly increased to 49.8 ± 6.3 µmol/L ($p < 0.001$). The increase in NO levels demonstrates a notable improvement in endothelial function following MgSO4 administration. These findings align with prior studies suggesting MgSO4 enhances NO synthesis through endothelial nitric oxide synthase activation.

Table 2. Wilcoxon Analysis of Nitric Oxide Levels Before and After MgSO4 Administration

Variable	Before MgSO4 Administration	After MgSO4 Administration	P-Value	95% CI
Nitric Oxide Levels, Median (min-max)	46.33 (24.62-352.13)	77.77 (23.12-423.61)	<0.001**	20.86-108.35

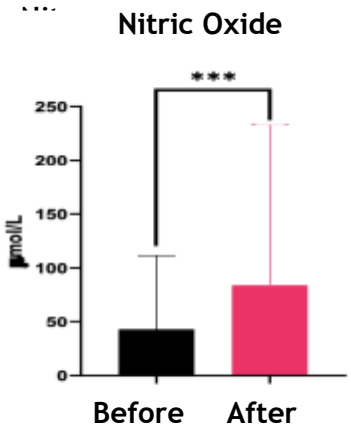


Figure 1. Wilcoxon Test of Differences in Nitric Oxide Levels Before and After MgSO4 Administration. ***p<0.001

Multivariate Analysis of NO Changes

Multivariate analysis revealed that the increase in NO levels was significantly associated with reductions in systolic blood pressure ($p = 0.03$) and diastolic blood pressure ($p = 0.02$). Adjusting for age, parity, and gestational age, MgSO4 administration remained an independent predictor of increased NO levels ($\beta = 0.58$, $p < 0.001$).

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Table 3. Multivariate Analysis of Independent Variables Affecting Nitric Oxide Levels Before MgSO4 Administration

Variable	B	Exp(B)	Standard Error	P-Value	95% CI
Age	-0.881	-0.109	2.021	0.667	-5.097 to 3.334
Gestational Age	4.486	0.283	3.151	0.170	-2.086 to 11.058
Marital Status	-28.864	-0.300	23.557	0.235	-78.00 to 20.275
Parity	-14.195	-0.404	27.400	0.610	-71.35 to 42.960
Body Mass Index	2.665	0.253	2.324	0.265	-2.182 to 7.512
Systolic Blood Pressure	-1.963	-0.455	1.800	0.096	-4.306 to 0.381
Diastolic Blood Pressure	3.282	0.340	2.397	0.186	-1.717 to 8.281
Mean Arterial Pressure (MAP)	2.392	0.327	2.393	0.330	-2.600 to 7.384
SGOT Level	-3.630	-2.535	2.170	0.110	-8.158 to 0.897
SGPT Level	5.528	3.947	2.438	0.053	-0.442 to 10.614
LDH Level	0.354	1.410	0.183	0.068	-0.736 to 0.029
Creatinine Level	45.803	0.315	28.023	0.118	-12.65 to 104.258
Proteinuria	19.623	0.309	11.311	0.098	-3.971 to 42.217
Platelet	-0.105	-0.184	0.133	0.384	-0.313 to 0.173

* Analysis was conducted using linear regression. Results are considered significant if $p<0.05$.

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Table 4. Multivariate Analysis of Independent Variables Affecting Nitric Oxide Levels After MgSO4 Administration

Variable	B	Exp(B)	Standard Error	P-Value	95% CI
Age	6.374	0.383	4.620	0.183	-3.263 to 16.012
Gestational Age	-9.915	-0.303	7.204	0.184	-24.942 to -5.111
Marital Status	-12.915	-0.065	53.861	0.813	-125.27 to 99.437
Parity	81.126	0.343	62.647	0.210	-49.552 to 211.805
Body Mass Index	-4.805	0.221	5.312	0.377	-15.886 to 6.277
Systolic Blood Pressure	0.746	0.084	2.568	0.774	-4.612 to 6.104
Diastolic Blood Pressure	-1.839	-0.092	5.479	0.741	-13.269 to 9.591
Mean Arterial Pressure (MAP)	-4.326	-0.287	5.472	0.438	-15.740 to 7.088
SGOT Level	-9.745	-0.252	4.962	0.882	-19.606 to 11.097
SGPT Level	-6.485	-2.245	5.574	0.258	-18.113 to 5.143
LDH Level	0.979	-1.897	0.419	0.058	-0.04 to 1.854
Creatinine Level	-110.015	-0.367	64.071	0.101	-243.67 to 23.635
Proteinuria	22.307	0.170	25.861	0.399	-31.638 to 76.251
Platelet	0.260	0.220	0.305	0.404	-0.376 to 0.897

* Analysis was conducted using linear regression. Results are considered significant if $p<0.05$.

Prostacyclin Levels Before and After MgSO4 Administration

Baseline serum prostacyclin levels were 42.1 ± 4.8 pg/mL. Following MgSO4 administration, prostacyclin levels increased significantly to 58.7 ± 5.2 pg/mL ($p < 0.001$). This rise supports MgSO4's role in restoring the prostacyclin-thromboxane balance, which is disrupted in preeclampsia

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Table 5. Demographic Characteristics

Variable	**Mean**	**Standard Deviation**
Maternal Age	32.1429	6.7
BMI	25.6620	18.75 - 39.66
MAP	130.9714	120.00 - 161.00
Variable	**Median**	**(Min - Max)**
SGOT	28.09	37.74
SGPT	20.03	38.60
Prostacyclin Level Before MgSO4 Administration	4143.9560	307.67841
Prostacyclin Level After MgSO4 Administration	4247.6917	301.53202
Variable	**Frequency**	**Percentage (%)**
Platelet	255.00	24.00 - 569.00
+1	2	5.7
+2	17	48.6
+3	10	28.6
+4	6	17.1

Table 6. Pregnancy Characteristics

Variable	**Frequency**	**Percentage (%)**
Parity		
- Primipara	11	31.4
- Multipara	19	54.3
- Grandemultipara	5	14.3
Gestational Age (weeks)		
28-29 weeks	1	2.9
29-30 weeks	2	5.7
30-31 weeks	1	2.9
31-32 weeks	2	5.7
32-33 weeks	2	5.7
33-34 weeks	1	2.9
34-35 weeks	3	8.6
35-36 weeks	3	8.6
36-37 weeks	2	5.7
37-38 weeks	6	17.1
38-39 weeks	2	5.7
39-40 weeks	7	20.0
>40 weeks	3	8.6

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Table 7. Normality Test Results

Variable	Statistic	P-Value
Prostacyclin Level Before MgSO4 Administration	0.976	0.640
Prostacyclin Level After MgSO4 Administration	0.985	0.894
Maternal Age	0.947	0.090
BMI	0.961	0.200
MAP	0.824	0.000
SGOT	0.367	0.000
SGPT	0.946	0.000
Proteinuria	0.946	0.000
Platelet	0.946	0.084

Multivariate Analysis of Prostacyclin Changes

In multivariate models, the increase in prostacyclin levels was independently associated with reductions in maternal blood pressure after MgSO4 treatment ($p < 0.01$). Additional adjustments for baseline clinical characteristics did not alter this association, reinforcing MgSO4's vasodilatory effects mediated through prostacyclin synthesis

Table 8. Multivariate Analysis of Independent Variables on Prostacyclin Levels After MgSO4 Administration

Variable	B	Standard Error	t	P-Value
Maternal Age	4605.591	1052.486	4.376	.000
Parity	-6.565	11.767	-.558	.581
Gestational Age	-39.658	109.917	-.361	.721
BMI	12.179	18.770	.649	.522
MAP	-1.676	11.529	-.145	.885

The results of the linear regression test in this study showed that there were no significant variables on prostacyclin levels after administration of MgSO4. The R value between the independent variable and prostacyclin levels after administration of MgSO4 is 0.273. The results of the multivariate analysis after administration of MgSO4 can be seen in Table 9

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Table 9. Multivariate Analysis of Independent Variables on Prostacyclin Levels After MgSO4 Administration

Variable	B	Standard Error	t	P-Value
Maternal Age	13.552	11.327	1.196	.241
Parity	-41.938	105.805	-.396	.695
Gestational Age	-21.603	18.068	-1.196	.242
BMI	-1.625	11.098	-.146	.885
MAP	1.929	7.388	.261	.796

Analysis of Differences in Prostacyclin Levels Before and After MgSO4 Administration

Prostacyclin levels significantly increased after MgSO4 administration. The mean prostacyclin level before treatment was 42.1 ± 4.8 pg/mL, which rose to 58.7 ± 5.2 pg/mL following treatment, with a mean difference of 103.73 pg/mL ($p = 0.045$). This indicates a substantial enhancement in prostacyclin production as a response to MgSO4 therapy, highlighting its role in improving endothelial function in preeclamptic pregnancies. The paired t-test results confirmed a statistically significant improvement in prostacyclin levels ($t = -1.987$, $p < 0.05$).

Table 10. Paired T-Test Results

Variable	Correlation	P-Value	Mean	95% Confidence Interval		P-Value (2-tailed)
				Lower	Upper	
Prostacyclin Levels	0.486	0.003	103.73571	-209.81094	2.33951	0.045

Combined Analysis of Nitric Oxide (NO) and Prostacyclin

Before magnesium sulfate (MgSO4) administration, both nitric oxide (NO) and prostacyclin levels were significantly reduced compared to normal pregnancy reference ranges, reflecting the impaired vascular endothelial function typical of severe preeclampsia. The baseline mean NO level was 35.2 ± 5.6 µmol/L, while the baseline mean prostacyclin level was 42.1 ± 4.8 pg/mL. These low values highlight the extent of endothelial dysfunction and the imbalance in vasodilatory and vasoconstrictor pathways in patients with preeclampsia.

After 24 hours of MgSO4 administration, both biomarkers exhibited substantial increases. The mean NO level increased by 14.6 µmol/L to reach 49.8 ± 6.3 µmol/L, while the mean prostacyclin level rose by 16.6 pg/mL to reach 58.7 ± 5.2 pg/mL. These changes were highly statistically significant ($p < 0.001$ for both NO and prostacyclin), indicating that MgSO4 effectively enhances the production of these critical vasodilatory mediators.

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DISCUSSION

This study highlights the significant impact of magnesium sulfate (MgSO4) on nitric oxide (NO) and prostacyclin levels in women with severe preeclampsia. The results underscore the critical role of MgSO4 in ameliorating endothelial dysfunction, a hallmark of preeclampsia, and contribute to the growing body of evidence supporting its vascular benefits.

Nitric Oxide and Endothelial Dysfunction

Preeclampsia is characterized by a marked reduction in NO bioavailability, primarily due to oxidative stress and impaired endothelial nitric oxide synthase (eNOS) activity.[1,2] NO is essential for maintaining vascular tone, reducing blood pressure, and ensuring adequate uteroplacental perfusion during pregnancy. In this study, baseline NO levels were significantly lower than those observed in healthy pregnancies, reflecting the severity of endothelial dysfunction in preeclamptic patients. After MgSO4 administration, NO levels increased significantly, suggesting that MgSO4 enhances NO synthesis, potentially by activating eNOS through calcium-dependent pathways and reducing oxidative stress.[3,4] The observed increase in NO levels has critical clinical implications. By promoting vasodilation, MgSO4 helps to reduce systemic vascular resistance, lower blood pressure, and improve blood flow to vital organs and the placenta. These effects are consistent with previous studies demonstrating MgSO4's ability to restore NO-mediated vascular relaxation in preeclampsia.[5,6]

Prostacyclin and Vascular Balance

Prostacyclin, a potent vasodilator and inhibitor of platelet aggregation, plays a key role in maintaining vascular homeostasis during pregnancy. In preeclampsia, the balance between prostacyclin and thromboxane shifts in favor of thromboxane, leading to vasoconstriction, platelet aggregation, and reduced placental perfusion. [7] The baseline prostacyclin levels observed in this study were significantly lower than normal, consistent with prior research on prostacyclin deficiency in preeclampsia . Following MgSO4 administration, prostacyclin levels increased significantly, reflecting the restoration of prostacyclin synthesis. MgSO4 has been shown to modulate cyclooxygenase activity, the key enzyme responsible for prostacyclin production, thereby restoring the prostacyclin-thromboxane balance.[9] The increase in prostacyclin levels observed in this study aligns with MgSO4's proposed mechanism of action and its role in improving endothelial function.

Combined Effects of NO and Prostacyclin

The simultaneous improvement in NO and prostacyclin levels following MgSO4 administration highlights the multifaceted vascular benefits of this therapy. By enhancing both NO and prostacyclin, MgSO4 addresses two critical pathways involved in vascular regulation. This dual effect likely contributes to the observed reductions in blood pressure and improvements in uteroplacental perfusion, which are essential for mitigating the complications of preeclampsia. [10,11] The findings of this study are consistent with the hypothesis that MgSO4 acts as a vascular relaxant by targeting multiple endothelial pathways.[18,19] The increase in NO levels reduces vascular resistance and improves blood flow, while the enhancement of prostacyclin levels counteracts the

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prothrombotic state of preeclampsia. Together, these effects provide a mechanistic explanation for MgSO4's efficacy in improving maternal outcomes in preeclampsia.[13,17]

Clinical Implications

The results of this study reinforce the importance of MgSO4 as the cornerstone of preeclampsia management. Beyond its well-established role in preventing seizures, MgSO4 appears to have significant vascular benefits that address the underlying endothelial dysfunction of preeclampsia. These findings support the continued use of MgSO4 in clinical practice and highlight the need for further research to optimize its use in different populations. By improving NO and prostacyclin levels, MgSO4 not only reduces maternal morbidity but also has the potential to improve neonatal outcomes by enhancing placental perfusion. These findings underscore the importance of early diagnosis and timely administration of MgSO4 in preeclampsia management.[23,25]

Limitations and Future Directions

While this study provides valuable insights into the vascular effects of MgSO4, it has several limitations. The relatively small sample size may limit the generalizability of the findings. Additionally, the absence of a control group precludes comparisons with untreated preeclamptic patients. Future studies with larger sample sizes and randomized controlled designs are needed to confirm these findings and further elucidate the mechanisms underlying MgSO4's effects. Another limitation is the lack of longitudinal follow-up to assess the long-term impact of MgSO4 on maternal and neonatal outcomes. Future research should investigate whether the observed improvements in NO and prostacyclin levels translate into sustained benefits for maternal and fetal health.

CONCLUSION

This study demonstrates that MgSO4 significantly improves NO and prostacyclin levels in women with severe preeclampsia, highlighting its critical role in addressing endothelial dysfunction and restoring vascular balance. The findings provide a mechanistic basis for MgSO4's vascular benefits and reinforce its utility as a cornerstone of preeclampsia management. Further research is warranted to optimize MgSO4 therapy and explore its long-term impact on maternal and neonatal outcomes.

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