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Analysis of the Influence of Nifedipine + Magnesium Sulfate Therapy on the Clinical Effect of Pregnancy-Induced Hypertension, Renal Function, and Pregnancy Outcome

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Abstract: *Objective:* To investigate the effect of nifedipine + magnesium sulfate treatment on pregnancy-induced hypertension. *Methods:* From January 2020 to January 2023, 60 patients with pregnancy-induced hypertension in our hospital were randomly divided into the control group and the observation group (30 cases in each group). The control group was treated with magnesium sulfate, while the observation group was treated with nifedipine and magnesium sulfate, and the clinical efficacy of the two groups was compared. The effective rate of treatment, blood pressure indicators, renal function indicators, adverse pregnancy outcomes, and quality-of-life scores were investigated. *Results:* The effective rate of treatment and quality-of-life score in the observation group were higher than those in the control group ($P < 0.05$). On the other hand, the diastolic and systolic blood pressure, the 24 h urine creatinine and albumin, as well as the adverse pregnancy outcomes were found to be lower in the observation group as compared to the control group ($P < 0.05$). *Conclusion:* For patients with pregnancy-induced hypertension, treatment with nifedipine and magnesium sulfate can achieve significant curative and remarkable effects. While improving blood pressure, it can also improve renal function, optimize pregnancy outcomes, and improve quality of life.

Keywords: Nifedipine; Magnesium sulfate; Pregnancy-induced hypertension; Clinical effect; Renal function; Pregnancy outcome

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1. Introduction

Pregnancy-induced hypertension, commonly referred to as PIH, is a special disease during pregnancy [1]. Clinically, the patient's symptoms include edema, urinary protein, high blood pressure, *etc*. Dizziness, vertigo, headache, and other symptoms were experienced by patients in serious cases, whereas coma, convulsions, and other consequences were noted in severe cases [2]. Whilst the pathogenesis of pregnancy-induced hypertension is unknown, it is necessary to take measures such as lowering blood pressure, relieving spasms, and improving blood circulation according to the specific pathophysiological characteristics of patients and disease-inducing factors [3,4]. The commonly used antispasmodic agent clinically is magnesium sulfate, which is an antispasmodic drug that can promote the recovery of

microcirculation, but the curative effect of a single drug is limited. Nifedipine, a calcium-blocker medication, has a significant protective effect on cardiomyocytes and also the effect of dilating blood vessels. It is a commonly used antihypertensive drug in clinical practice. A combination of magnesium sulfate and nifedipine may improve renal function and clinical efficacy. The study aimed to evaluate the curative effect of nifedipine + magnesium sulfate combined treatment on patients with pregnancy-induced hypertension.

2. Materials and methods

A retrospective study was carried out between January 2020 to January 2023 at the Third Hospital of Inner Mongolia Baotou Iron and Steel Group, China. Inclusion criteria included patients being diagnosed with pregnancy-induced hypertension, with a normal mind, and able to communicate normally. Exclusion criteria included patients with mental illnesses, cancer, and those who were transferred to the hospital for treatment in the middle of the study. Sixty patients who fulfilled the inclusion criteria were recruited, informed, and signed the consent form. They were then randomly divided into two groups: the control group and the observation group.

The control group was treated with magnesium sulfate, which was applied through intravenous infusion. Patients in the control group were given 5 g of magnesium sulfate mixed with 20 mL of glucose solution over 24 h, followed by a maintenance dose of 10 g of magnesium sulfate in 500 mL glucose solution every 48 h.

The observation group was treated with a combination of nifedipine and magnesium sulfate, where magnesium sulfate was administered via intravenous infusion with doses similar to the control group, while nifedipine was taken orally at 10 mg daily. The total treatment duration was 14 days. During treatment, the patient's blood pressure was closely monitored, and magnesium toxicity was taken into account.

The observation indicators in the study included: (i) treatment efficiency, which was observed and categorized as (a) significantly effective (blood pressure below 140/90mmHg; urine protein, edema, and other symptoms disappeared), (b) effective (blood pressure between 140–150/90–100 mmHg; urine protein, edema, and other symptoms improved significantly), and (c) ineffective (did not meet the above requirements); (ii) blood pressure indicators (diastolic and systolic blood pressure); (iii) renal function indexes (24 h urine creatinine and 24 h urine albumin); (iv) adverse pregnancy outcomes; and (v) quality of life using the SF-36 scale (0–100 points), where a higher score indicated a better quality of life.

SPSS2 3.0 was used for data analysis and processing. Measurement data (mean ± standard deviation, SD) and count data (%) were tested by t and χ^2, respectively, and the difference was considered statistically significant when $P < 0.05$.

3. Results

Table 1 shows the comparison of data between the control group and the observation group, including age range, average age (mean ± SD), gestational week range, and average gestational week (mean ± SD).

Table 1. Data comparison of the two groups

Group	Number of cases (n)	Age range (years)	Average age (years)	Gestational weeks (weeks)	Average gestational week (weeks)
Control group	30	20–36	27.45 ± 3.26	28–38	36.59 ± 2.50
Observation group	30	20–37	27.51 ± 3.35	28–39	36.48 ± 2.71
χ^2/t (%)		–	0.062	–	0.185
P		–	0.485	–	0.625

The treatment effective rate between the control group and the observation group after the treatment is shown in **Table 2**. Whilst the control group had more effective cases (83.33%) and a few ineffective cases (16.67%), the observation group showed that all cases were effective (100%), where the effective cases were slightly more than the significantly effective cases (53.33% versus 46.67%). Hence, the rate of treatment effectiveness found in the observation group was significantly higher than that observed in the control group ($P = 0.020$).

Table 2. The treatment effective rate of the two groups

Group	Number of cases (n)	Significantly effective	Effective	Ineffective	Total effective rate
Control group	30	10 (33.33)	15 (50.00)	5 (16.67)	25 (83.33)
Observation group	30	14 (46.67)	16 (53.33)	0 (0.00)	30 (100.00)
χ^2	-	1.111	0.067	5.455	5.455
P	-	0.292	0.796	0.020	0.020

Data are given in n (%).

The blood pressure indicators of the two groups before and after the treatment are shown in **Table 3**. Patients in both groups had lower diastolic and systolic blood pressure after treatment. However, the observation group appeared to have a lower blood pressure than those of the control group (diastolic blood pressure 81.48 ± 2.36 versus 85.26 ± 8.20, $P = 0.000$; systolic blood pressure 130.15 ± 2.11 versus 135.26 ± 8.56, $P = 0.000$).

Table 3. Blood pressure indicators of the two groups

Group	Number of cases (n)	Diastolic blood pressure (mmHg)		Systolic blood pressure (mmHg)	
		Before treatment	After treatment	Before treatment	After treatment
Control group	30	104.15 ± 6.26	85.26 ± 8.20	154.28 ± 9.25	135.26 ± 8.56
Observation group	30	104.48 ± 6.35	81.48 ± 2.36	154.19 ± 9.18	130.15 ± 2.11
t	-	0.261	9.265	0.185	10.265
P	-	0.845	0.000	0.478	0.000

Data are given in mean ± SD.

Table 4 showed the renal function indexes of the two groups before and after the treatment. Both groups showed a decrease in 24 h urine creatinine and albumin after the treatment, where the amounts were lower in the observation group as compared to the control group (creatinine 7.45 ± 0.26 versus 9.26 ± 1.52, $P = 0.000$; albumin 125.26 ± 4.08 versus 140.26 ± 11.50, $P = 0.000$).

Table 4. Renal function indexes of the two groups

Group	Number of cases (n)	24 h urine creatinine (mmol/L)		24 h urine albumin (mg)	
		Before treatment	After treatment	Before treatment	After treatment
Control group	30	10.45 ± 2.65	9.26 ± 1.52	180.56 ± 50.26	140.26 ± 11.50
Observation group	30	10.51 ± 2.71	7.45 ± 0.26	181.01 ± 50.41	125.26 ± 4.08
t	-	0.084	5.948	0.162	9.584
P	-	0.695	0.000	0.487	0.000

Data are given in mean ± SD.

The adverse pregnancy outcomes of both groups after the treatment are shown in **Table 5**, where there were lesser adverse pregnancy outcomes observed in the observation group as compared to the control group (total incidence of 3.33% versus 23.33%, $P = 0.023$).

Table 5. Adverse pregnancy outcomes of the two groups

Group	Number of cases (n)	Neonatal asphyxia	Respiratory distress	Premature rupture of membrane	Fetal macrosomia	Total incidence
Control group	30	2 (6.67)	2 (6.67)	2 (6.67)	1 (3.33)	7 (23.33)
Observation group	30	1 (3.33)	0 (0.00)	0 (0.00)	0 (0.00)	1 (3.33)
χ^2	-	0.351	2.069	2.069	1.017	5.192
P	-	0.554	0.150	0.150	0.313	0.023

Data are given in n (%).

The quality-of-life score between the control group and the observation group is shown in **Table 6**. The quality of life of the observation group in terms of vitality, physiological functions, emotional functions, and social functions appeared to be higher than that of the control group ($P = 0.000$).

Table 6. The quality-of-life score of the two groups

Group	Number of cases (n)	Vitality (points)		Physiological functions (points)	
		Before treatment	After treatment	Before treatment	After treatment
Control group	30	52.48 ± 8.15	60.36 ± 7.15	51.39 ± 8.15	61.59 ± 8.36
Observation group	30	52.36 ± 8.20	72.95 ± 2.15	51.48 ± 8.20	75.20 ± 2.19
t	–	0.265	8.595	0.018	9.265
P	–	0.889	0.000	0.487	0.000
Group	**Number of cases (n)**	**Emotional functions (points)**		**Social function (points)**	
		Before treatment	**After treatment**	**Before treatment**	**After treatment**
Control group	30	53.48 ± 8.41	62.59 ± 6.26	51.39 ± 8.15	63.68 ± 7.22
Observation group	30	53.20 ± 8.19	72.51 ± 2.15	51.84 ± 8.20	73.61 ± 2.26
t	–	0.084	9.481	0.018	7.985
P	–	0.862	0.000	0.869	0.000

Data are given in mean ± SD.

4. Discussion

During pregnancy, due to the increase of progesterone and luteinizing hormone in the body over time, the blood coagulation function and fibrinolytic activity in the body of pregnant women will change accordingly. However, if there is a disorder of the fibrinolytic and coagulation system, it is likely to cause pregnancy-induced hypertension, which is also clinically known as gestational hypertension [5,6]. When the disease develops to a certain stage, it may lead to spasms of small blood vessels throughout the patient's body, as well as certain damage to the vascular endothelium, which will aggravate the coagulation dysfunction, eventually leading to a hypercoagulable state and form a thrombus, which may endanger the health of the patients and fetuses [7,8]. Pregnancy-induced hypertension is the most common complication during pregnancy, and its occurrence is related to hemodynamic abnormalities caused by systemic small vessel spasms. The onset of pregnancy-induced hypertension is insidious, and the condition is critical. Some patients may manifest signs of organ failure or coma, which threatens the lives of women and fetuses [9].

In recent years, the incidence of pregnancy-induced hypertension has been increasing year by year, and the current annual incidence has reached 9% [10]. Pregnancy-induced hypertension not only poses a great threat to the health of mothers and infants but also causes maternal and perinatal deaths. Clinically, the patients' manifestations are mostly persistently elevated blood pressure, proteinuria, and edema. Some women with very mild symptoms suffer from mild dizziness and elevated blood pressure, and they do not feel any other discomfort at all [11,12]. However, when the situation becomes more serious, the patients will have various symptoms, such as dizziness, nausea, headache, vomiting, persistent right upper quadrant pain, *etc*., along with a significant increase in blood pressure, a more serious situation of edema, and a large amount of proteinuria. Some patients also experience convulsions and coma. According to some surveys, the risk of "postpartum hemorrhage" is very high in the third trimester, and it will cause great harm to fetuses [13]. Therefore, adequate attention should be given to the prevention and treatment of the disease.

Currently, in the process of treating pregnancy-induced hypertension, doctors will conduct all-around observation of the mother's and baby's body, provide oxygen support promptly, and supply protein and calories on time. Patients with severe edema will require salt-intake control. The current commonly used antihypertensive drug in clinical practice is magnesium sulfate, which has the effect of dilating blood vessels and improving microcirculation. Magnesium ions in the drug can expand blood vessels and regulate blood pressure by inhibiting the contraction of vascular smooth muscle (VSM) and also reducing the resistance in blood vessels and spasm of small blood vessels [14]. Nifedipine is a calcium ion channel antagonist, which can inhibit the calcium ion from activating the calcium ion pump, thereby maintaining the calcium ion concentration in the blood [15]. In addition, nifedipine can expand blood vessels throughout the body to a certain extent, reduce peripheral blood pressure, and increase blood flow in uterine arteries. The combination of magnesium sulfate and nifedipine has a certain synergistic effect that can alleviate the stress damage on VSM cells and is beneficial to improving hemodynamic disorders. In addition, the use of nifedipine will increase the blood perfusion of the kidneys during treatment, thereby preventing oxidative stress damage of the glomerular filtration membrane caused by abnormal blood perfusion of the kidneys. The occurrence of pregnancy-induced hypertension is also closely related to oxidative stress. The changes in oxidative metabolism in the body likely lead to intensified oxidative stress in the body. Nifedipine can remove free radicals in the body and regulate the metabolism, thereby improving the antioxidant capacity in the body and significantly improving the renal function of patients. In this paper, the observation group obtained significant therapeutic effects after combining the above two drugs. Compared with the control group, which was given magnesium sulfate, the observation group had a higher effective rate, lower blood pressure indicators, better renal function indicators, and lower adverse pregnancy outcomes. Higher quality of life scores suggested that the combined use of nifedipine and magnesium sulfate is of significant value.

5. Conclusion

In summary, nifedipine and magnesium sulfate can be combined in the treatment of pregnancy-induced hypertension. The combination has a significant effect, can improve blood pressure indicators and renal function, and can well control pregnancy outcomes and reduce adverse events. After treatment, the quality of life of patients will be greatly improved.

Disclosure statement

The authors declare no conflict of interest.

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