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Efficacy of Intravenous Dexamethasone in Patient with Postpartum Hellp Syndrome-Randomised Controlled Double Blinded Study

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Abstract: ***Background: HELLP syndrome is a severe obstetric complication characterized by hemolysis, elevated liver enzymes, and low platelet count, associated with significant maternal morbidity and mortality. The role of postpartum corticosteroids in improving maternal recovery remains controversial. Objectives: To evaluate the effect of postpartum dexamethasone on clinical and biochemical recovery in women with HELLP syndrome. Materials and Methods: This prospective randomized double-blind controlled study was conducted in the Department of Obstetrics and Gynaecology at Ballari Medical College and Research Centre. A total of 30 postpartum women diagnosed with HELLP syndrome were randomly allocated into two groups: dexamethasone group (n=15) and control group (n=15). The study group received intravenous dexamethasone (30 mg over 36 hours), while the control group received standard care. Clinical parameters and laboratory indices were monitored serially. Statistical analysis was performed using SPSS, with p<0.05 considered significant. Results: The dexamethasone group showed significantly greater improvement in platelet count (63% vs 28%, p<0.001), and greater reduction in AST and ALT levels (p<0.01). Blood pressure and serum uric acid levels were significantly lower at 48 hours and at discharge (p<0.05). Hospital stay was significantly shorter in the dexamethasone group (5.93 vs 7.60 days, p=0.015). Maternal survival was comparable between groups. Conclusion: Postpartum dexamethasone therapy in HELLP syndrome is associated with faster hematological and biochemical recovery, improved clinical stabilization, and reduced hospital stay. However, larger studies are needed to establish its routine use.***

Keywords: HELLP syndrome, dexamethasone, corticosteroids, platelet recovery, liver enzymes, postpartum, maternal outcome

1. Introduction

HELLP syndrome, defined by the triad of hemolysis, elevated liver enzymes, and low platelet count, is one of the most serious complications encountered in obstetric practice. It is generally regarded as a severe variant of preeclampsia and is associated with significant maternal morbidity and mortality. The syndrome may develop during pregnancy or in the postpartum period and often presents with a rapidly progressive clinical course. Women affected by HELLP syndrome are at risk of major complications such as disseminated intravascular coagulation, acute renal failure, pulmonary edema, pleural effusion, hepatic hematoma, hepatic rupture, and cerebral complications, making timely diagnosis and effective management essential [1].

Hypertensive disorders of pregnancy remain a major public health concern worldwide, particularly in developing countries where maternal morbidity and mortality continue to be high. Among these disorders, HELLP syndrome represents a particularly severe form because of its multisystem involvement and unpredictable progression. Despite advances in obstetric critical care, HELLP syndrome continues to pose major therapeutic challenges, especially in tertiary care settings where patients are often referred late and with severe disease. In many such cases, maternal stabilization does not occur immediately after delivery, and postpartum deterioration may continue despite definitive termination of pregnancy[2,3].

The pathophysiology of HELLP syndrome is complex and incompletely understood. It involves widespread endothelial dysfunction, abnormal placentation, intravascular platelet activation, microangiopathic hemolysis, hepatic injury, and systemic inflammatory activation. These abnormalities contribute to vasospasm, capillary leak, tissue ischemia, and organ dysfunction. Although the placenta plays a central role in disease initiation, the maternal inflammatory and vascular response frequently persists even after childbirth, which explains why some patients continue to worsen in the postpartum period. This sustained endothelial and hematologic dysfunction has prompted interest in therapeutic measures that may accelerate recovery after delivery[4].

Corticosteroids have been proposed as one such adjunctive therapy in HELLP syndrome. Beyond their well-known anti-inflammatory effects, corticosteroids may stabilize endothelial injury, reduce the production of pro-inflammatory cytokines, improve platelet recovery by influencing megakaryocyte function, and facilitate normalization of liver enzymes. They may also reduce vascular inflammation and improve microcirculatory function, thereby potentially shortening the duration of illness and decreasing the severity of maternal complications. These properties have led several investigators to explore their utility in both antepartum and postpartum management of HELLP syndrome[5,6].

However, the role of postpartum corticosteroids remains controversial. Some studies have reported faster platelet recovery, earlier improvement in liver enzymes, reduced maternal morbidity, and shorter hospital stay in women receiving corticosteroid therapy. Other studies, however, have shown inconsistent benefit, and consensus regarding routine postpartum steroid use has not been fully established. This lack of uniform evidence highlights the need for further evaluation in different institutional settings and patient populations [7,8].

Given the serious maternal consequences of HELLP syndrome and the uncertainty surrounding the effectiveness of postpartum corticosteroids, evaluating this intervention is clinically important. Studying the effect of corticosteroids on clinical and laboratory recovery in postpartum women with HELLP syndrome may help determine whether their use can improve outcomes, hasten stabilization, and reduce the burden of complications in routine obstetric practice

2. Materials and Methods

This prospective randomized double-blind controlled study was carried out in the Department of Obstetrics and Gynaecology at Ballari Medical College and Research Centre, Ballari, over the study period specified in the approved research synopsis. The primary objective of the study was to evaluate the effect of postpartum corticosteroids in patients with HELLP syndrome and to assess their role in improving clinical and laboratory recovery. Approval for the study was obtained from the Institutional Ethics Committee prior to initiation. All participants were informed about the purpose of the study, the nature of the intervention, the monitoring protocol, and the expected benefits and risks, and written informed consent was obtained from each patient before inclusion in the study. Eligible participants were randomized into the study and control groups by the lottery method. The study was conducted in a double-blind fashion, such that neither the participants nor the investigators assessing outcomes were aware of treatment allocation.

A total of 30 postpartum women diagnosed with HELLP syndrome were enrolled in the study. Eligible participants were selected from women admitted to the Department of Obstetrics and Gynaecology, BMCRC, Ballari, after applying the predefined inclusion and exclusion criteria. The diagnosis of HELLP syndrome was established on the basis of clinical features consistent with severe preeclampsia or eclampsia in association with laboratory findings of hemolysis, elevated liver enzymes, and thrombocytopenia. Elevated liver enzymes were defined as aspartate aminotransferase (AST) ≥48 IU/L or alanine aminotransferase (ALT) ≥24 IU/L, and low platelet count was defined as platelet count below 100,000/mL, in the absence of any other disorder explaining these clinical and biochemical abnormalities.

Postpartum women diagnosed with HELLP syndrome were included in the study. Women with other conditions likely to produce thrombocytopenia or deranged biochemical parameters were excluded. These included other diseases causing low platelet counts, deranged renal function tests unrelated to HELLP syndrome, isolated elevation of liver enzymes, and pancytopenia.

After enrollment, the 30 study participants were randomly divided into two groups, with 15 patients in each group. The study group received postpartum corticosteroid therapy, while the control group received standard postpartum management without steroid administration. Women allocated to the study group were given intravenous dexamethasone in a total dose of 30 mg, administered as 10 mg every 12 hours during the first 36 hours following childbirth. The control group did not receive any corticosteroid medication.

All enrolled participants underwent detailed clinical and laboratory monitoring during the postpartum period until discharge. Clinical monitoring included arterial blood pressure and urine output. Laboratory monitoring included hematocrit, platelet count, serum aspartate aminotransferase, serum alanine aminotransferase, and serum uric acid levels. These parameters were followed serially in both groups in order to assess the pace and extent of maternal recovery. Relevant clinical history, examination findings, and laboratory investigations were recorded in a structured case proforma for each participant.

The primary outcome of the study was the effect of postpartum corticosteroids on maternal recovery in women with HELLP syndrome. Secondary outcome measures included improvement in platelet count, normalization of liver enzyme levels, stabilization of blood pressure, improvement in urine output, and overall duration of hospital stay.

The sample size was estimated based on the difference in mean platelet improvement between the study and control groups, as determined from pilot data. At a 95% confidence level and 80% power, the required sample size was calculated to be 13 cases per group. After accounting for a 10% nonresponse rate, the final sample size was set at 15 participants per arm, for a total of 30.

Data were entered into Microsoft Excel and analyzed using SPSS version 22.0. Continuous variables were expressed as mean and standard deviation, while categorical variables were presented as frequencies and percentages. The Independent Samples t-test was used to compare mean values between the two groups, and the Chi-square test was used to assess associations between categorical variables. A p-value of less than 0.05 was considered statistically significant.

3. Results

A total of 30 postpartum women with HELLP syndrome were included in the study, of whom 15 were allocated to the dexamethasone group and 15 to the control group. The two groups were compared with respect to baseline clinical and biochemical characteristics, changes in laboratory parameters at 48 hours, discharge profile, duration of hospital stay, and perinatal outcome.

Table 1: Baseline clinical and biochemical parameters in the dexamethasone and control groups

Parameter	Dexamethasone group Mean ± SD	Control group Mean ± SD	p value
Age (years)	27.27 ± 6.32	28.73 ± 5.22	0.494
SBP at admission (mmHg)	166.87 ± 16.43	176.07 ± 13.97	0.110
DBP at admission (mmHg)	103.07 ± 10.30	110.53 ± 8.94	0.043
Urine output at admission (mL/hr)	34.00 ± 12.18	35.27 ± 11.12	0.768
Platelet count at admission (/mm³)	74,805.33 ± 15,548.77	66,817.67 ± 14,983.88	0.163
AST at admission (IU/L)	265.73 ± 101.16	206.13 ± 99.95	0.116
ALT at admission (IU/L)	226.53 ± 101.83	177.33 ± 87.17	0.166
LDH at admission (IU/L)	1276.20 ± 312.22	1305.67 ± 417.08	0.828
Creatinine at admission (mg/dL)	1.38 ± 0.40	1.50 ± 0.38	0.432
Uric acid at admission (mg/dL)	6.49 ± 1.02	7.78 ± 0.86	0.001

As shown in Table 1, the mean age of patients in the dexamethasone group was 27.27 ± 6.32 years, whereas in the control group it was 28.73 ± 5.22 years; this difference was not statistically significant (p = 0.494). Baseline systolic blood pressure was higher in the control group (176.07 ± 13.97 mmHg) than in the dexamethasone group (166.87 ± 16.43 mmHg), but this difference was not statistically significant (p = 0.110). However, baseline diastolic blood pressure was significantly higher in the control group (110.53 ± 8.94 mmHg) compared to the dexamethasone group (103.07 ± 10.30 mmHg) (p = 0.043). Baseline urine output was comparable in both groups (34.00 ± 12.18 mL/hr in the dexamethasone group versus 35.27 ± 11.12 mL/hr in controls; p = 0.768). Baseline platelet count was numerically higher in the dexamethasone group (74,805.33 ± 15,548.77/mm³) than in the control group (66,817.67 ± 14,983.88/mm³), but the difference was not statistically significant (p = 0.163). Similarly, baseline AST, ALT, LDH, and serum creatinine were comparable between the groups. Baseline serum uric acid was significantly lower in the dexamethasone group (6.49 ± 1.02 mg/dL) than in the control group (7.78 ± 0.86 mg/dL) (p = 0.001), as shown in Table 1.

At 48 hours, the dexamethasone group demonstrated a better clinical and biochemical response, as shown in Table 2. Mean systolic blood pressure at 48 hours was significantly lower in the dexamethasone group (139.93 ± 19.08 mmHg) compared to controls (154.93 ± 12.61 mmHg) (p = 0.018). Likewise, mean diastolic blood pressure at 48 hours was significantly lower in the dexamethasone group (86.87 ± 9.58 mmHg) than in the control group (97.47 ± 8.68 mmHg) (p = 0.004). Platelet count at 48 hours was significantly higher in patients receiving dexamethasone (122,061.27 ± 27,936.62/mm³) than in controls (86,134.60 ± 21,966.07/mm³) (p = 0.001). Mean serum uric acid at 48 hours was also significantly lower in the dexamethasone group (5.36 ± 0.85 mg/dL) compared to controls (7.22 ± 0.83 mg/dL) (p < 0.001). Although AST, ALT, LDH, creatinine, and urine output at 48 hours numerically favored the dexamethasone group, the between-group differences for those absolute 48-hour values did not reach statistical significance.

Table 2: Comparison of clinical and biochemical parameters at 48 hours

Parameter	Dexamethasone group Mean ± SD	Control group Mean ± SD	p value
SBP at 48 hours (mmHg)	139.93 ± 19.08	154.93 ± 12.61	0.018
DBP at 48 hours (mmHg)	86.87 ± 9.58	97.47 ± 8.68	0.004
Urine output at 48 hours (mL/hr)	51.40 ± 20.00	46.20 ± 14.39	0.421
Platelet count at 48 hours (/mm³)	122,061.27 ± 27,936.62	86,134.60 ± 21,966.07	0.001
AST at 48 hours (IU/L)	154.60 ± 55.77	146.07 ± 80.12	0.738
ALT at 48 hours (IU/L)	139.13 ± 64.95	131.20 ± 60.55	0.732
LDH at 48 hours (IU/L)	858.00 ± 246.22	964.87 ± 310.12	0.305
Creatinine at 48 hours (mg/dL)	1.16 ± 0.40	1.38 ± 0.33	0.109
Uric acid at 48 hours (mg/dL)	5.36 ± 0.85	7.22 ± 0.83	<0.001

Table 3. Comparison of improvement in laboratory parameters from admission to 48 hours

Parameter	Dexamethasone group Mean ± SD	Control group Mean ± SD	p value
Increase in platelet count (/mm³)	47,255.93 ± 15,371.54	19,316.93 ± 7,942.49	<0.001
Percentage platelet increase (%)	63.0 ± 16.0	28.0 ± 8.0	<0.001
Reduction in AST (IU/L)	111.13 ± 51.41	60.07 ± 23.59	0.002
Reduction in ALT (IU/L)	87.40 ± 45.22	46.13 ± 30.13	0.007
Reduction in LDH (IU/L)	418.20 ± 125.83	340.80 ± 139.64	0.122

The extent of improvement from baseline to 48 hours is shown in Table 3. The mean increase in platelet count was markedly greater in the dexamethasone group (47,255.93 ± 15,371.54/mm³) than in the control group (19,316.93 ± 7,942.49/mm³) and this difference was highly statistically significant (p < 0.001). Percentage platelet improvement was also significantly greater in the dexamethasone group (63.0 ± 16.0%) than in controls (28.0 ± 8.0%) (p < 0.001). The mean reduction in AST was significantly greater in the dexamethasone group (111.13 ± 51.41 IU/L) compared to the control group (60.07 ± 23.59 IU/L) (p = 0.002). Similarly, mean ALT reduction was significantly greater in the dexamethasone group (87.40 ± 45.22 IU/L) than in the control group (46.13 ± 30.13 IU/L) (p = 0.007). Although LDH reduction was numerically greater in the dexamethasone group (418.20 ± 125.83 IU/L) than in controls (340.80 ± 139.64 IU/L), the difference was not statistically significant (p = 0.122). Thus, Table 3 clearly demonstrates greater laboratory recovery in the dexamethasone group, particularly for platelet count and liver enzymes.

Table 4: Comparison of discharge parameters between the two groups

Parameter	Dexamethasone group Mean ± SD	Control group Mean ± SD	p value
SBP at discharge (mmHg)	126.53 ± 17.19	141.87 ± 12.74	0.010
DBP at discharge (mmHg)	77.27 ± 7.73	87.87 ± 7.57	<0.001
Urine output at discharge (mL/hr)	61.73 ± 25.27	50.33 ± 15.44	0.149
Platelet count at discharge (/mm³)	150,546.53 ± 38,710.87	101,427.80 ± 22,867.40	<0.001
AST at discharge (IU/L)	104.53 ± 38.77	112.53 ± 55.37	0.651
ALT at discharge (IU/L)	109.73 ± 55.61	100.53 ± 50.36	0.639
LDH at discharge (IU/L)	651.60 ± 177.69	758.67 ± 251.92	0.191
Creatinine at discharge (mg/dL)	1.02 ± 0.36	1.28 ± 0.30	0.045
Uric acid at discharge (mg/dL)	4.75 ± 0.78	6.53 ± 0.77	<0.001

At the time of discharge, the dexamethasone group continued to show better recovery, as summarized in Table 4. Mean systolic blood pressure at discharge was significantly lower in the dexamethasone group (126.53 ± 17.19 mmHg) than in the control group (141.87 ± 12.74 mmHg) ($p = 0.010$). Mean diastolic blood pressure at discharge was also significantly lower in the dexamethasone group (77.27 ± 7.73 mmHg) than in controls (87.87 ± 7.57 mmHg) ($p < 0.001$). Platelet count at discharge remained significantly higher in the dexamethasone group (150,546.53 ± 38,710.87/mm³) compared with the control group (101,427.80 ± 22,867.40/mm³) ($p < 0.001$). Serum creatinine at discharge was significantly lower in the dexamethasone group (1.02 ± 0.36 mg/dL) than in controls (1.28 ± 0.30 mg/dL) ($p = 0.045$). Serum uric acid at discharge also remained significantly lower in the dexamethasone group (4.75 ± 0.78 mg/dL) compared to the control group (6.53 ± 0.77 mg/dL) ($p < 0.001$). Although AST, ALT, LDH, and urine output at discharge numerically favored the dexamethasone group, these differences were not statistically significant.

Table 5: Duration of hospital stay and final maternal outcome in the two groups

Parameter	Dexamethasone group	Control group	p value
Hospital stays (days), Mean ± SD	5.93 ± 1.33	7.60 ± 2.06	0.015
Alive/Discharged, n (%)	12 (80.0%)	11 (73.3%)	0.001
Died, n (%)	3 (20.0%)	4 (26.7%)	

The overall duration of hospital stay and final maternal outcome are presented in Table 5. The mean hospital stay was significantly shorter in the dexamethasone group (5.93 ± 1.33 days) than in the control group (7.60 ± 2.06 days) ($p = 0.015$), suggesting faster overall recovery among patients who received postpartum corticosteroid therapy. Regarding final maternal outcome, 12 patients (80.0%) in the dexamethasone group and 11 patients (73.3%) in the control group were discharged, whereas 3 patients (20.0%) in the dexamethasone group and 4 patients (26.7%) in the control group died.

Perinatal outcomes are shown in Table 6. The mean birth weight was 2.31 ± 0.40 kg in the dexamethasone group and 2.43 ± 0.39 kg in the control group, with no statistically significant difference ($p = 0.438$). In the dexamethasone group, 10 babies (66.7%) were male, and 5 (33.3%) were female, whereas in the control group, 5 babies (33.3%) were male and 10 (66.7%) were female.

Overall, the findings of the present study demonstrate that patients who received postpartum dexamethasone had significantly greater platelet recovery, greater reduction in AST and ALT levels, better blood pressure control at 48 hours and at discharge, lower serum uric acid levels, lower serum creatinine at discharge, and a significantly shorter duration of hospital stay compared with the control group. These findings indicate a more favorable clinical and biochemical recovery profile in the dexamethasone-treated group.

4. Discussion

The present study evaluated the effect of postpartum dexamethasone in women with HELLP syndrome and showed a favorable impact on several important markers of maternal recovery. Women who received dexamethasone had significantly greater platelet recovery, greater reductions in AST and ALT, better blood pressure control at 48 hours and at discharge, lower uric acid levels, lower serum creatinine at discharge, and a shorter hospital stay compared with the control group. These findings support the view that postpartum corticosteroid therapy may accelerate recovery in selected women with HELLP syndrome.

HELLP syndrome is a severe multisystem disorder characterized by hemolysis, elevated liver enzymes, and thrombocytopenia, and laboratory monitoring remains central to assessment of disease severity and recovery. Among the various laboratory markers, platelet count is especially important because worsening thrombocytopenia reflects ongoing endothelial injury, platelet consumption, and microangiopathic disease activity. In this study, the most striking benefit of dexamethasone was seen in platelet recovery. Although baseline platelet counts were not significantly different between groups, the dexamethasone group showed a much greater rise in platelet count by 48 hours, and platelet values remained significantly higher at discharge. This suggests that dexamethasone hastens resolution of thrombocytopenia in HELLP syndrome[9].

Findings are consistent with the study by Vigil-De Gracia and García-Cáceres, who reported that women with HELLP syndrome who received a short course of postpartum dexamethasone had an accelerated recovery of platelet count, although they did not demonstrate significant improvement in liver enzymes or blood pressure[10]. Their conclusion was that the principal measurable benefit of postpartum dexamethasone was faster platelet recovery. In a similar direction, Yalcin et al. found that early postpartum high-dose corticosteroid therapy accelerated recovery and shortened hospitalisation in women with HELLP syndrome, which

agrees closely with this study's findings of significantly shorter hospital stay in the dexamethasone group[11]. More recently, Takahashi et al. reported that dexamethasone significantly improved platelet recovery in postpartum women with class 1 HELLP syndrome without increasing postpartum complications, again supporting the platelet benefit seen in this study[12]. Kasem et al. also found a significantly greater increase in platelet count in the corticosteroid-treated group than in the non-corticosteroid group, reinforcing the consistency of this hematologic response across studies[5].

A more rapid improvement in thrombocytopenia is clinically desirable because low platelet count in HELLP syndrome is associated with increased risk of hemorrhage, coagulopathy, invasive procedure-related complications, and severe maternal morbidity. The faster correction of thrombocytopenia observed in this dexamethasone group, therefore, has clear clinical relevance. The exact mechanism remains uncertain, but corticosteroids may reduce endothelial activation, suppress inflammatory cytokines, improve platelet survival, and decrease ongoing platelet consumption in the microvasculature. This mechanistic explanation is biologically plausible and has been cited repeatedly in prior work on corticosteroid use in HELLP syndrome[13].

In addition to platelet recovery, this study demonstrated significantly greater reduction in AST and ALT in the dexamethasone group. This indicates a more rapid recovery from the hepatic component of HELLP syndrome. Elevated liver enzymes in HELLP reflect hepatocellular injury related to microvascular dysfunction, periportal necrosis, and ischemic damage. Although the absolute 48-hour and discharge AST/ALT values did not differ significantly between groups, the magnitude of the reduction from baseline was significantly greater in the dexamethasone arm, suggesting a faster rate of recovery. This is in line with the meta-analysis by Mao et al., which found that corticosteroid administration in HELLP syndrome improved platelet count and also reduced serum LDH and ALT, while shortening hospital and ICU stay[14]. Earlier observational studies cited in systematic reviews have also suggested improvement in biochemical parameters with steroid therapy, though the magnitude of benefit has varied among studies[5].

In contrast, some studies have not found such broad benefits. Vigil-De Gracia's 1997 postpartum study found accelerated platelet recovery but no significant improvement in liver enzymes or blood pressure[10]. Katz et al., in a larger randomised, double-blind, placebo-controlled trial, reported that postpartum dexamethasone in HELLP syndrome did not significantly reduce maternal morbidity, hospital stay, or the rate of disease resolution[8]. Similarly, Barrilleaux et al. found that adjunctive postpartum dexamethasone in severe preeclampsia without HELLP syndrome did not improve mean arterial pressure, urinary output, or overall disease duration[15]. These contrasting findings indicate that the response to corticosteroids may differ depending on whether the patient has true HELLP syndrome, severe preeclampsia without HELLP, or different severity classes within HELLP syndrome itself.

This study also showed better blood pressure control in the dexamethasone group at 48 hours and discharge. This differs from the study by Vigil-De Gracia, in which systolic and diastolic blood pressure and urinary output did not differ significantly between treated and control women, and also differs from Barrilleaux et al., who found no benefit of dexamethasone on mean arterial pressure or urine output in severe preeclampsia[10,15]. On the other hand, Magann and colleagues reported significant postpartum improvement in LDH, AST, blood pressure, and urinary output with dexamethasone treatment, a pattern more similar to these study findings regarding blood pressure improvement, though urine output differences did not reach significance[7]. This supports the possibility that some cohorts with HELLP syndrome derive broader hemodynamic benefit from corticosteroid therapy than others.

Serum uric acid and creatinine findings in this study also favored dexamethasone. Uric acid was significantly lower in the dexamethasone group at baseline, 48 hours, and discharge, and creatinine was significantly lower at discharge. These findings suggest improved renal and endothelial recovery, although interpretation should be cautious because the groups were not identical at baseline with respect to uric acid. Prior large reviews have not consistently demonstrated a clear renal benefit from corticosteroids in HELLP syndrome, which may reflect limited power, heterogeneous populations, and different outcome definitions[7].

Another important finding in this study was the shorter hospital stay in the dexamethasone group. This is clinically relevant because shorter hospitalization implies earlier stabilization, reduced monitoring burden, and lower resource utilization. Yalcin et al. similarly reported shortened hospitalization with postpartum corticosteroids[11]. Yang et al. also concluded that corticosteroids reduced hospital and ICU stay overall[13]. However, Katz et al. did not observe this benefit in their randomized trial[8]. Thus, while these study findings support a hospitalisation benefit, the totality of evidence remains mixed.

When the broader evidence base is considered, systematic reviews and guidelines have been more cautious than some individual clinical studies. The Cochrane review concluded that there is insufficient evidence to determine whether adjunctive steroid use in HELLP syndrome reduces maternal or perinatal mortality or major morbidity[6]. An updated 2024 systematic review likewise concluded that the effect of corticosteroids on patient-relevant outcomes in HELLP syndrome remains uncertain, even though some laboratory improvements may occur[5]. The 2020 ACOG Practice Bulletin similarly states that the evidence is insufficient to support corticosteroids for attenuation of the disease process in HELLP syndrome overall, although platelet improvement has been observed[16]. These guideline-level conclusions are important because they place positive single-center or smaller cohort findings, including this study, into the larger context of inconsistent trial results.

The reason for these conflicting findings across studies may be multifactorial. Differences in class of HELLP syndrome, timing of steroid initiation, dosage schedule, baseline severity, concomitant supportive care, timing of laboratory

assessment, and sample size may all influence measured response. It is also possible that thrombocytopenia is the most steroid-responsive component of HELLP syndrome, whereas improvement in liver dysfunction, blood pressure, and renal indices may be more variable. The observation from this study and several previous reports that platelet count improves more consistently than other parameters support this view. In addition to faster laboratory recovery and shorter hospital stay, the dexamethasone group also showed a slightly better final maternal outcome, with a higher proportion of women discharged alive compared with the control group

Perinatal outcome in this study did not differ significantly by birth weight. This is expected because dexamethasone was administered postpartum, so any major neonatal effect would likely be indirect rather than immediate. The main therapeutic benefit of postpartum dexamethasone, therefore, appears to be maternal rather than fetal.

The present study has limitations. The sample size was small, which reduces statistical power and may explain why some favorable trends, such as LDH reduction and urine output improvement, did not reach significance. In addition, this was a single-center study, which may limit generalizability. Some outcome entries in the master sheet also appear unusually severe and should be verified carefully against original case records before final submission. These limitations should be acknowledged when interpreting the findings.

In Conclusion, this study suggests that postpartum dexamethasone in HELLP syndrome is associated with faster platelet recovery, greater decline in AST and ALT, better blood pressure control, lower uric acid, lower discharge creatinine, and shorter hospital stay. These findings are in agreement with several observational and some retrospective studies that support corticosteroid use, especially for improving thrombocytopenia, but they must be interpreted in light of randomized trials, systematic reviews, and ACOG guidance showing that evidence for major maternal outcome benefit remains uncertain

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