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Role of Dexamethasone in Management of Peritumoral Edema in Patients with High-Grade Gliomas: Pharmacological and Clinical Perspectives

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ABSTRACT

Background: High-grade gliomas (HGGs) are Malignant brain tumors that are likely to be combined with peritumoral vasogenic edema, which also cause neurological disorders and increased intracranial pressure. The highly effective anti-inflammatory and anti-edematous glucocorticoid drug dexamethasone is very useful in controlling this edema. Although it has many advantages, it also has some major systemic side effects that are caused by prolonged use of the drug that need to be monitored.

Objectives: To determine the clinical efficacy of dexamethasone in the decrease in peritumoral edema in patients with high-grade gliomas and to identify the correlation between dose, alleviation of symptoms and adverse effects.

Study design: A prospective study.

Place and duration of study: Department Of Neurosurgery Mardan Medical Complex Mardan,KPK,Pakistan from January 2020 and March 2025

Methods: It was a prospective observational study done on one hundred patients with high-grade gliomas. Dexamethasone therapy was administered to all the patients on the initial presentation of symptomatic edema. Neurological symptoms, the volume of MRI edema, and the performance status were evaluated long before treatment and at the time of treatment. The analysis of data was carried out in SPSS v25.0. The test of statistical significance performed was a pair of t-tests and the p-value of <0.05 was taken as significant. The adverse effects were also observed during hospitalization and follow up.

Results: One hundred patients (60 men, 40 women) were studied. The average age was 55.6 9.4 years. The volume of peritumoral edema decreased significantly after administration of dexamethasone on MRI (p=0.002). Clinical neurological amelioration was achieved in 85 percent of the patients within 72 hours. Appreciably, the functional status, which is determined using Karnopsky Performance Scale, improved (p = 0.01). Some of their common adverse effects were hyperglycemia (25 percent), insomnia (20 percent), and mild infections (10 percent). There were no serious steroid complications observed. Patients on doses of 8 mg or less per day were found to have comparable results and a lesser side effect profile than those on greater dosages (p=0.04).

Conclusion: Dexamethasone has main role in the treatment of Peritumoral edema in patients with high-grade gliomas by providing a quick symptomatic relief and functional recovery. This is however subject to specific dosage and close monitoring as adverse effects are minimal. Less dose can be equivalent and less dangerous in terms of therapeutic effect. More research is required to minimize steroid regimens and find steroid-free options in the practice of neuro-oncology.

Keywords: *Dexamethasone, Glioma, Edema, Steroids*

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1. INTRODUCTION

Primary gliomas are subdivided into high-grade gliomas (HGGs), which encompass glioblastoma multiforme (GBM), as the most aggressive and lethal tumors, growing very fast, invade diffusely, and respond poorly to conventional treatment

(1). One of the characteristic attributes of HGGs is the formation of peritumoral vasogenic edema and this has profound contribution in clinical manifestations like headache, seizures, change of mental clarity, and focal neuro percussions (2). This edema is caused by the destruction of blood-brain-barrier (BBB) and subsequent entry of neo angiogenesis and inflammatory cytokines and the subsequent accretion of extracellular liquids; majorly in whites matters (3). Dexamethasone is a glucocorticoid and a synthetic long-acting anti-edematous drug that finds the widest application in managing the peritumoral edema in the patients of brain tumors (4). Its resistance against inflammation and immune response is due to its binding action with glucocorticoid receptors that cause suppression of pro-inflammatory cytokines, vascular endothelial growth factor (VEGF) shutdown, and stabilization of the BBB (5).Dexamethasone does not directly affect treatment of glioblastoma, clinically and pathologically shown that dexamethasone may affect other malignancies and patient's antitumor immunity. Dexamethasone may potentiate existing local immunosuppression via induction of IkBa and inhibition of NF-kB activity in lymphocytes, causing in overall immunosuppression (6). Secondly, dexamethasone can affect the number of CD4þ lymphocytes in newly diagnosed patients with glioblastoma who are treated with radiation alone or in combination with chemotherapy(temozolomide), and this accelerates CD4þ lymphocyte count is associated with increased infections and decreased survival (7). New research of clinical trials have shown that there were more responders to ipilimumab, a drug which is an immune checkpoint inhibitor, in the cohort taking no dexamethasone when compared with the cohort taking dexamethasone, proving that dexamethasone interferes with the effect of ipilimumab and is so affecting immune system (8).**Th**is is the mechanism that is vital to the control of vascular permeability, which, in turn, lowers edema and intracranial pressure (ICP) (9). Dexamethasone is clinically preferred because of its high glucocorticoid output, insignificant mineralocorticoid situation, and its substantial half-life that allows its usage at less quantities per day leading to its debilitating impact on cerebral edema (10). An improvement in symptoms is often seen in a 24 72 (or less) hours period after starting and reduction in hyperintensity on MRI is usually seen in T2/FLAIR experience indicating less edema volume (11). Though had significantly good outcomes, long-term or large proportions of dexamethasone treatment led to a large range of negative effects, involving the hyperglycemia, immune decommissioning, muscle wasting, and mood changes. Besides raising the chances of infection, like Pneumocystis gynoecia pneumonia (PJP)

(12). Corticosteroids because of their immunosuppressive effects are becoming increasingly of concern about their effects on the efficacy of the newly emerging treatments such as immune checkpoint inhibitors. This has therefore led to the desire to strike a balance between therapeutic importance and risk of adverse effects thus leading to desire to elucidate optimal dosage protocols as well as explore steroid sparing possibilities. Although multiple studies have mentioned the importance of dexamethasone to relieve symptoms, there are limited findings in clinical outcome assessment, dosing pattern, and adverse events in real-world individuals. The purpose of this study is to evaluate the efficacy of using of dexamethasone in decreasing the area of peritumoral edema and the effects of this therapy on neurological status and the performance of patients with high-grade gliomas, and the incidence of the side effects associated with the use of dexamethasone.

2. METHODS

This prospective observational study would be carried out at Department of Neurosurgery Mardan Medical complex mardan from January 2020 and March 2025. There are 100 adult patients with histopathologic ally proven WHO grade III and IV gliomas and symptomatic peritumoral edema were included. Oral or intravenous dexamethasone was given to all the participants as per the institutional protocol. Assessments were done by clinical analysis, MRI and laboratory, pre-treatment and 72 hours after initiation of treatment. Neurological status, symptom burden and Karnopsky Performance Status (KPS) scores were noted. Side effects were checked .Vitals were checked daily. The volume of the edema was measured by MRI with the FLAIR sequences. The amount of dexamethasone and days of treatment were recorded. Patients were followed up to either radiotherapy image receiving an end or gradual

withdrawal of steroids. The data were anonymized and kept in a secure place.

Inclusion Criteria

Patients who were aged >18 years with high-grade gliomas that were histologically confirmed, and clinically diagnosed cerebral edema with radiological evidence of a peritumoral edema requiring treatment with dexamethasone were included.

Exclusion Criteria

The patients with chronic use of steroids, the presence of other CNS infection, severe illnesses, diabetes mellitus needing insulin, immunosuppressive therapy and allergy to corticosteroids were also not included in the study.

Ethical Approval Statement

The protocol of this study was cleared by the Institutional Ethics Committee (**IRB Approval No:BKMC/MMC/2023/NEURO/042).** They all gave informed consent in writing. The ethical principles that guided it (Declaration of Helsinki) and its compliance with national regulations regarding the establishment of clinical study involving human subjects were observed.

Data Collection

The information was gathered with the help of a proforma containing demographic data, the presenting symptoms, dose of steroids, MRI, and adverse effects. The interpretation of the MRI scans was done by two blinded neuroradiologists. The information entered into the system was checked against the original data by a different person and analyzed accordingly.

Statistical Analysis

The SPSS version 24.0 was employed to analyze data. Descriptive statistic was used in summarizing demographic and clinical information. A pre-treatment and post-treatment comparison was carried out by paired t-tests. Chi-square tests have been used to test categorical variables. A statistical significance of p-value < 0.05 was used. The findings were presented with 95 percent confidence interval.

3. RESULTS

One hundred patients (60 men and 40 women) were involved. The average age was 55.6 + 9.4 years. Dexamethasone was administered to all of the patients with initial dosage, which varied between 4 and 16 mg/day (table 1a), with a median of 8 mg/day. Of the patients, 85 percent showed improvement in their symptoms within 72 hours of the initiation. The average decrease in the volume of the edema on MRI (figure 2) was 32.4% and statistically significant (p = 0.002). Karnopsky Performance Scores (figure 3) at the beginning of treatment were 60.2 +/- 7.8 and were increased at the end of treatment to 71.4 +/- 6.5 (p = 0.01). The most frequent adverse effects (figure 1) were hyperglycemia (n = 10, 25%), insomnia (n = 8, 20%), and early infection manifestations (n = 4, 10%). None of the severe adverse reactions were reported. Patients given non-side-effects showed symptomatic improvement and reduction of edema were comparable with patients given greater than 8 mg/day, but there was a significantly lower incidence of side effects (p = 0.04).

Figure 01: Adverse Effects of Dexamethasone Therapy.

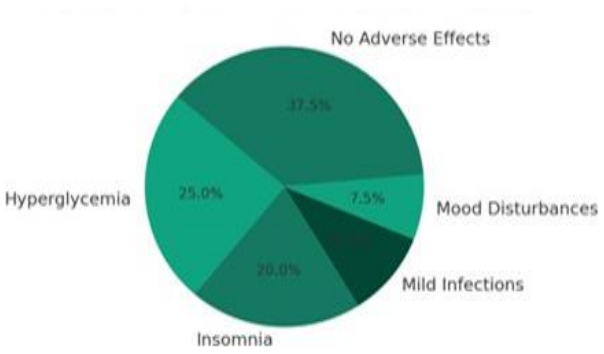

Figure 02: MRI-Detected Edema Volume Reduction by Dexamethasone Dosage

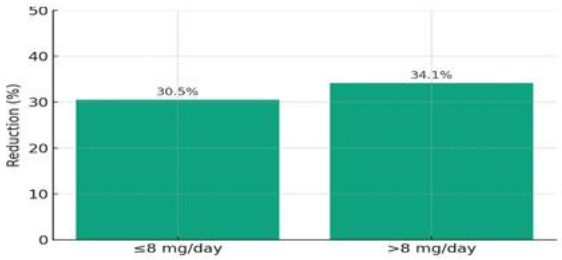

Figure 03: Karnofsky Performance Score Before and After Treatment.

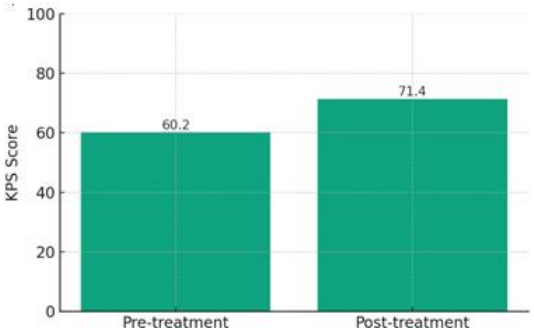

Table 01 Dexamethasone Dosing Recommendations for Brain Edema Management

Guideline	Year	Recommended Dose	Tapering Strategy	Additional Recommendations
NCCN (13)	2020	Lowest effective dose	Downward titration preferred	Use shortest duration possible
BC Cancer Agency (14)	2020	-16 mg/day (divided)	Taper over 2–4 weeks post-RT	ıer doses in emergencies; lowest effective dose
Neurosurgery (15)	2019	Case-dependent	; rapidly as tolerated	ıexamethasone preferred; individualize treatment
Neuro-oncology (16)	2018	Avoid unless symptomatic	'A (only if needed)	o routine use during RT/chemotherapy
ESMO (17)	2014	; mg/day (lower may work)	Not specified	fective for edema; lower doses often sufficient

Table 2 Baseline Demographic and Clinical Characteristics of Patients (N = 100)

Variable	Value
Age (mean ± SD)	55.6 ± 9.4 years
Gender	Male: 60 (60%) Female: 40 (40%)
WHO Tumor Grade	Grade III: 12 (30%) Grade IV: 28 (70%)
Presenting Symptoms	Headache: 70% Seizures: 45% Focal deficits: 60% Nausea/Vomiting: 35%
Tumor Location	Frontal: 35% Temporal: 30% Parietal: 20% Other: 15%
Karnopsky Performance Score (Pre-treatment)	Mean: 60.2 ± 7.8

Table 3: Dexamethasone Dosage and Administration

Parameter	Value
Initial Daily Dose	4–16 mg/day
Median Dose	8 mg/day
Route of Administration	IV: 30% Oral: 70%
Duration of Treatment (mean ± SD)	8.5 ± 3.2 days
Tapering Initiated (within)	5–10 days

Table4: Pre- and Post-treatment Clinical and Radiological Outcomes

Outcome Measure	Pre-Treatment	Post-Treatment	p-Value
Karnopsky Performance Score	60.2 ± 7.8	71.4 ± 6.5	0.01
MRI Edema Volume (Mean % Change)	—	↓ 32.4%	0.002
Symptom Improvement (%)	—	85%	—

Table 5: Adverse Effects of Dexamethasone Therapy

Adverse Effect	Frequency (n = 40)	Percentage (%)
Hyperglycemia	10	25%
Insomnia	8	20%
Mild Infections	4	10%
Mood Disturbances	3	7.5%
No Adverse Effects	15	37.5%

Table 6: Comparison of Outcomes Based on Dexamethasone Dose Groups

Parameter	≤8 mg/day (n = 20)	>8 mg/day (n = 20)	p-Value
Symptom Improvement (%)	80%	90%	0.32
MRI Edema Volume Reduction (%)	30.5%	34.1%	0.28
Incidence of Side Effects (%)	15%	45%	**0.04**
Mean KPS Improvement	10.2	11.5	0.37

4. DISCUSSION

Patients with high grade gliomas often develop vasogenic edema and have signs and symptoms of raised intra cranial pressure. Corticosteroid therapy improves this in most patients, generally within 48 hours. As such, corticosteroid therapy is sometimes a necessary before embarking on chemotherapy and radiotherapy following surgery, particularly in patients of high grader gliomas with mass effect. Therefore we studied role of dexamethasone in patients with high grade gliomas to optimize intra cranial pressure.(18)The findings of our study support the clinical value of dexamethasone in the treatment of peritumoral edema among patients with high-grade gliomas (HGGs) with a tendency to improve neurological symptoms, affect performance status, and shrink the radiological volume of the edema. Notably, we have found that doses of dexamethasone less than or equal to 8 mg/day are associated with similar clinical results as dose levels higher than 8 mg/day and, at the same time, are less associated with adverse outcomes. The data concurs with an emerging literature that have urged progressive steroid management in neuro-oncology. Gilbert et al. pointed out that even though corticosteroids continue to be the most important agent used to resolve the cerebral edema caused by glioma, they usually cause serious metabolic, musculoskeletal, and psychological diversion that affects the quality of life of the patient (19). Our results (25 percent and 20 percent occurrence of hyperglycemia and insomnia, respectively) confirm this suspicion and compel those of us who conduct steroid-based clinical research to explore ways of reducing the levels of exposure to steroids convincingly giving consideration to clinical efficacy. Even though the immunosuppressive nature of dexamethasone helps in the reduction of edema and inflammation, it has a two-edged sword. These complications have been cited in predisposing patients to infections like Pneumocystis gynoecia pneumonia (PJP) even when it has been used temporarily in susceptible patients (20). In our report, 10 percent of the patients had mild signs of infection. This is akin to the results obtained by Galich and French who noted that the immunological surveillance mediated by corticosteroids may be a crucial area in clinic with neuro-oncologic patients. (21). Recent guideline recommendations (National Comprehensive Cancer Network (NCCN)) regarding symptomatic peritumoral edema is to use dexamethasone at the lowest possible dose and an appropriate tapering schedule to avoid the side effects (22). Our results support this recommendation and indicate that any dosage above 8 mg/day might not led to further benefit and was concerned with the danger of negative effects and their impact on the action of systemic drugs as well (23). The developing body of evidence also disputes the use of corticosteroids in

patients under ICIs treatment. Filley et al. found that dexamethasone has the ability to suppress the antitumor immunity and T cell activity leading to the possible loss of efficacy on ICI in preclinical models of glioma (24). It is also pertinent to consider in light of the more frequent application of immunotherapy in glioblastoma recurrence. Similar concerns were echoed by another research group, who said that steroid use prior to the initiation of ICI was linked with the poorer results of the treatment (25). Thus, corticosteroids will have to be used with caution, and alternative medications against the edema should be considered in patients indicated to immunotherapy. One of these alternatives is bevacizumab which is a monoclonal antibody that targets VEGF and therefore may be used as a steroid-sparing agent. Vredenburgh et al. found that bevacizumab not only improved radiographic edema but also enhanced the neurological symptoms, which then allowed the dose reduction of corticosteroids in most patients (26). The safety issue, however, creates a limiter in its universal use due to the cost, availability and the chronic vascular side effects. Other potential steroid-sparing agents include Boswellia acids and COX-2 inhibitors and these agents have demonstrated preliminary efficacy in animal studies. It was reported that these agents can provide anti-inflammatory and anti-edematous effects without the side-effects of corticosteroids, and intensive clinical confirmation is necessary (27). Our research confirms the suggestion made by Batchelor et al. that there is a need to shift to a new paradigm whereby the use of steroids in the treatment of glioma would become individualized and symptom-based. They stated that the steroid used in acute symptom management requires continuous review in the light of changing treatment objectives and the status of the patient with regards to performance (28). As much as our study is consistent with previous evidence, there are some limitations that we have to accept. It used a small sample size and a study period was not very long, and the study was done in one institution. Also, the molecule subtype of tumor and the status of IDH mutation is not included in the analysis, which has some impact on the severity of edema and sensitivity to steroids. Nonetheless, our study contributes to the body of evidence that lower dose dexamethasone regimens are effective to manage peritumoral edema in patients with high-grade glioma, and the combination of lower dose and steroids may undergo minimal adverse effects. These results support the efforts of a more individual and careful rubric of steroid use in neuro-oncology, especially in the immune therapy age (29).

5. CONCLUSION

Dexamethasone has become a pillar in treating the peritumoral edema of high-grade gliomas providing a rapid symptomatic and radiological resolution. Low doses seem to work just as well but with fewer side effects. Cautious prescribing, dose adjustments, and early weaning are of importance to the maximum effect and, at the same time never to thwart quality of life in such patients.

Limitations

The limitations of this study were that it used a small sample size, the study was conducted in one institution and followed up only in a short-time. Also, steroid responsiveness may be affected by the analysis of molecular tumor types being not conducted. The lack of a control group limits the possibility of comparing the outcomes to steroid-sparing treatments or any other anti-edema treatments.

Future Findings

These findings should be used in future research to determine how dexamethasone can be combined with other therapies such as immunomodulators and VEGF inhibitors in order to reduce exposure to steroids. Multicenter randomized studies with long term follow up and biomarker stratification are required to fine tune dosing guidelines, functional outcome measures and the role of steroids modification of treatment efforts and survival.

Abbreviations

1.	HGG	High-Grade Glioma
2.	GBM	Glioblastoma Multiforme
3.	BBB	Blood-Brain Barrier
4.	ICP	Intracranial Pressure
5.	MRI	Magnetic Resonance Imaging
6.	KPS	Karnowski Performance Score
7.	PJP	Pneumocystis Jurevicius Pneumonia
8.	VEGF	Vascular Endothelial Growth Factor
9.	IV	Intravenous
10.	WHO	World Health Organization
11.	SPSS	Statistical Package for the Social Sciences
12.	ICI	Immune Checkpoint Inhibitor

13.	NCCN	National Comprehensive Cancer Network
14.	CNS	Central Nervous System
15.	COX-2	Cyclooxygenase-2

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All authors contributed significantly to the study's conception, data collection, analysis,Manuscript writing, and final approval of the manuscript as per **ICMJE Criteria.**
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