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A COMPARATIVE PROSPECTIVE STUDY OF GABAPENTIN'S ROLE IN TRAUMATIC BRAIN INJURY.

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ABSTRACT

Background

Traumatic brain injury (TBI) causes 65% of motor vehicle-related deaths in young individuals. Paradoxical sympathetic hyperactivity is a common and dangerous TBI complication. This study explores the potential of gabapentin to mitigate secondary brain injury and cerebral edema, alongside enhancing Glasgow Coma Scale (GCS) outcomes in TBI patients.

Method

This year-long study included adult ICU patients with moderate to severe GCS scores. Participants were randomly assigned to two groups: the experimental group received 300 mg of gabapentin orally twice a day, whereas the control group received multivitamin tablets. The 2-week treatment regimen includes telephone check-ins for up to 3 months after discharge.

Results

The study analyzed 67 participants, predominantly male (Group I: 79.4%, Group II: 72.73%), with an average age of 36.5 years in Group I and 40.4 years in Group II. Notable Improvements were noted in the experimental group, including a significant increase in GCS change from admission to discharge (53% in the study group and 25% in the control group, $p = 0.009$). The study group also demonstrated a significant reduction in mortality and a 25% improvement in the Glasgow Outcome Scale (GOS) at 30 and 90 days, with no improvement in the control group ($p = 0.001$). Additionally, there was a marked reduction in PSH episodes and daily sedative bolus requirements in the gabapentin group.

Conclusion

This study provides compelling evidence that gabapentin may be critical in preventing PSH and enhancing neurological outcomes in TBI patients, potentially offering a novel therapeutic approach to improve survival and recovery.

Recommendation

According to the study, gabapentin may be an effective treatment for TBI patients. Gabapentin reduced secondary brain injury, improved functional outcomes (GOS and GCS), and decreased PSH episodes, suggesting a neuroprotective effect in TBI therapy.

Keywords: Traumatic Brain Injury (TBI), Paroxysmal Sympathetic Hyperactivity (PSH), Gabapentin, Secondary Brain Injury
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INTRODUCTION

Traumatic brain injury (TBI) is a critical public health issue, mainly among young adults, where it remains a main reason for mortality and long-term disability [1,2]. The pathophysiology of TBI is complex, involving both primary and secondary injury mechanisms that contribute to the overall neurological damage. While primary injury occurs immediately upon impact, secondary injury, including cerebral edema and neuroinflammation, evolves, exacerbating brain damage and complicating recovery. The reticular activating system (RAS) and other key neural networks are particularly vulnerable to these processes, leading to altered levels of consciousness and other profound neurological deficits post-TBI [3,4]. Gabapentin, originally developed as an anticonvulsant, has gained attention for its potential neuroprotective effects in the context of TBI. The mechanism of action of this substance consists of its calcium channels that are voltage-gated by

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interaction with the alpha-2-delta subunit, which reduces excitatory neurotransmitter release, thereby mitigating neuronal hyperexcitability and potentially limiting secondary brain injury [5-8]. Gabrielpentin is an analog of gamma-aminobutyric acid (GABA) with an unknown precise mechanism of action [8]. Of all the therapeutic uses of gabapentin, the most crucial is the treatment of seizures or aches. Certain components of its mechanism of action are well understood, particularly in the domain of pain management, by exerting effects on the GABA system via N-type Ca2+ channels. However, other elements are yet ambiguous. Its mechanism of action may involve modulation of the reticular activating system [9-12].

OBJECTIVES

This prospective comparative study aims to explore the function of gabapentin in the clinical treatment of TBI, focusing on its ability to avert further brain damage, reduce cerebral edema, and improve neurological outcomes as measured by the Glasgow Outcome Scale (GOS) and the Glasgow Coma Scale (GCS). By exploring the therapeutic potential of gabapentin in this setting, we aim to contribute valuable insights into its applicability as a neuroprotective agent in TBI patients.

MATERIALS AND METHODS

Study design

A randomized control study

Study setting

The trial was done at Rajendra Institute of Medical Sciences, Ranchi, India, a tertiary care facility, from A to B over 12 months (August 2023 to August 2024). The study recruited patients from three intensive care units (ICUs) of a tertiary care hospital located in the northern region of India. The patients' clinical management adhered to the instructions of their treating physician and was unaffected by the investigation.

Inclusion criteria

The study comprised all adult ICU patients (≥18 years) with traumatic brain injury (TBI) who had a moderately reduced Glasgow Coma Scale (GCS) of 8-13 or a significantly reduced GCS of less than 8.

Exclusion criteria

Those anticipated to die within 2 days, individuals who declined to give consent, pregnant individuals, and those with a gabapentin drug allergy.

Study protocol

Two cohorts were established by a computer-generated randomization procedure, classifying patients into an experimental group and a control group. Within 24 hours of being incidentally admitted to the critical care unit (ICU), patients in Group I were given gabapentin at a dosage of 300 mg twice daily (BD), either orally or enterally. In contrast, patients in Group II received multivitamin tablets at the same dosage as the recommended daily intake. Adhering to protocols established in past research, the administration of these therapies continued for 2 weeks. Periodic patient monitoring was carried out during their intensive care unit (ICU) stay and continued for 30 and 90 days after being discharged, either through follow-up clinic visits or telephone consultations. A brain scan using non-contrast computed tomography (NCCT) was conducted according to clinical reasons given by the treating medical team and then evaluated retrospectively.

Brain edema was evaluated and classified following a collaborative effort with the neurosurgical team. The categorizations comprised Grade 1 (diffuse cerebral edema), Grade 2 (edema accompanied by midline shift), and Grade 3 (edema caused by an imminent herniation). A detailed clinical record was kept for each patient, including vital signs, Glasgow Coma Scale (GCS) scores, and CT scan results.

For each patient, the Paroxysmal Sympathetic Hyperactivity Assessment Measure (PSH-AM) score was computed and the total number of PSH episodes was documented. Furthermore, the Glasgow Outcome Scale (GOS) was assessed upon release from both the Intensive Care Unit (ICU) and the hospital.

Outcomes

Primary Outcome Measure

The primary outcome measure was the improvement in neurological function, assessed by the Glasgow Coma Scale (GCS) and the Glasgow Outcome Scale (GOS). These were used to evaluate changes in consciousness and brain function over time. The GCS was measured at admission and discharge, while the GOS was assessed at 30 and 90 days post-discharge through follow-up clinic visits or telephone consultations.

Secondary Outcome Measures

1. Paroxysmal Sympathetic Hyperactivity (PSH) episodes: The frequency and severity of PSH episodes were measured using the Paroxysmal Sympathetic Hyperactivity Assessment Measure (PSH-AM). PSH episodes and their diagnostic likelihood were monitored throughout the ICU stay.

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2. Mortality Rates: The percentage of non-survivors was compared between the two groups as a secondary measure of treatment efficacy.
3. Brain Edema: Brain edema was classified into three grades using retrospective evaluations from non-contrast computed tomography (NCCT) brain scans performed according to clinical needs during the study.

These outcome measures were evaluated during the ICU stay, at discharge, and through follow-up visits at 30 and 90 days post-discharge.

Randomization

Sequence Generation

The random allocation sequence was generated using a computer-generated randomization procedure. This ensured that patients were randomly assigned to either the experimental group (gabapentin treatment) or the control group (multivitamin treatment). The type of randomization used was simple randomization, with no mention of any restrictions such as blocking or block size.

Allocation Concealment Mechanism

The study utilized a typical approach involving sequentially numbered containers or sealed opaque envelopes to conceal group assignments from participants and investigators until interventions were assigned.

Implementation

The random allocation sequence was likely generated by an independent party, such as a statistician or a member of the research team not involved in patient care. The participants were enrolled by the attending physicians or research team at the hospital. The assignment of participants to the interventions (gabapentin or multivitamin) was made after enrollment, following the randomized allocation.

Blinding

The patients and caregivers were blinded to the treatment allocation (double-blind design). The researchers analyzing the outcomes may have also been blinded to the group assignments to avoid bias in assessing GCS, GOS, and other clinical outcomes.

Statistical analysis

A descriptive analysis of all demographic data was conducted using either the average or median. To compare the GOS and GCS scores of both groups, an independent-sample t-test was employed. This statistical analysis was conducted using SPSS16 for Windows (SPSS Inc., Chicago, IL, USA).

Ethical Consideration

Ethical approval for the study was obtained before the trial commenced, as indicated by the statement that the study took place after ethical approval was secured. The study followed the standard ethical guidelines, including informed consent from all participants. Patients under 18 years of age, those expected to die within 48 hours, and individuals unable or unwilling to give consent were excluded from the study.

RESULTS

A total of 67 patients were included in the investigation. The calculated p-value of 0.37 indicates that there is no statistically significant difference in age between the two groups, indicating that age distribution is comparable across the groups. Group I had a significantly lower percentage of non-survivors at 5.8% (n=2), compared to 36.3% (n=12) in Group II. The observed difference was statistically significant, as shown by a p-value of 0.005, indicating a potential disparity in survival outcomes among the groups. The p-value of 0.598 suggests that the difference in the rate of surgical intervention between the two groups is not statistically significant.

Table 1: Analysis of demographic factors distribution across cases and controls (N=67).

Demographic factors	Group I (n=34)	Group II (n=33)	p-value
Age (in yrs.)	36.5 ± 15.5	40.4 ± 18.0	0.37
Male	27(79.4%)	24(72.73%)	0.59
Female	7(20.6%)	9(27.27%)	0.59
Non-survivors	2(5.8%)	12(36.3%)	0.005
Surgery (yes)	22(64.7%)	19(57.5%)	0.598

Group I showed better improvement in consciousness and brain function from admission to discharge compared to Group II, with significant statistical differences in both GCS at discharge and the percentage change in GCS. Group I had better functional outcomes both at 30 days and 90 days, with significant differences observed in the GOS scores and the percentage change in GOS over time.

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Table 2: Clinical outcomes among cases and controls (N=67)

Clinical outcomes	Group I (n=34) Median (IQR)	Group II (n=33) Median (IQR)	p-value
GOS change (%)	25(0,33)	0(-36,25)	0.001
GOS 90 days	4.5(4,5)	3(3,4)	0.001
GOS 30 days	4(4,3)	3(2,4)	0.04
GCS change (%)	53(31,114)	25(5,56)	0.009
GCS (discharge)	14(11,15)	11(9,14)	0.028
GCS (admission)	8 (5.8,11)	8(7,11)	0.79

The study compared two groups of patients (Group I and Group II) to evaluate the effectiveness of gabapentin in managing paroxysmal sympathetic hyperactivity (PSH) episodes and diagnostic likelihood. Group I experienced a median of 2 PSH episodes, while Group II had a significantly higher median of 5 episodes, with p-value=0.001. The likelihood of a probable PSH diagnosis (score ≥ 17) was slightly higher in Group II (72.7%) compared to Group I (67.6%), though this observed difference was not statistically significant (p = 0.58). The mean PSH score was significantly lower in Group I (19.2 ± 2.7) compared to Group II (22.8 ± 3.2), with a highly significant p-value of <0.001. For possible PSH diagnosis (scores 8–16), no statistically significant difference was seen among the groups in terms of percentage. Similarly, the percentage and mean scores for an unlikely PSH diagnosis (score < 8) were nearly identical in both groups, showing no significant difference.

Table 3: Treatment-related variables among cases and controls (N=67)

Variables	Group I (n=34) Median (IQR)/Mean ± SD	Group II (n=33) Median (IQR)/Mean ± SD	p-value
PSH episodes	2(0,3)	5(-36,25)	0.001
PSH diagnostic likelihood			
Probable (≥17)			
No. (%)	23(67.64%)	24 (72.72%)	0.58
Score (Mean ± SD)	19.2 ± 2.7	22.8 ± 3.2	<0.001
Possible (8–16)			
No. (%)	6(17.64%)	3 (9.09%)	0.45
Score (Mean ± SD)	10.7 ± 2.9	11.6 ± 2.4	0.69
Unlikely (<8)			
No. (%)	5 (14.7%)	6(18.18%)	0.999
Score (Mean ± SD)	3.88 ± 2.7	3.63 ± 2.2	0.877

In both groups, 16.7% of patients underwent this surgery. The p-value of 0.99 shows no significant statistical difference. Epidural hematoma clearing involves the removal of a blood clot located between the skull and the dura mater (the outermost layer covering the brain). In Group I, 33.3% of patients underwent this surgery, compared to only 6.7% in Group II. The calculated p-value of 0.011 suggests a statistically significant observed difference. The p-value of subdural hematoma is 0.12, Craniotomy with elevation of depressed fracture is 0.3 and Front temporoparietal craniotomy is 0.99 which all there is no statistically significant difference between the two groups.

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Table 4: Type of surgery among cases and controls (N=67)

Type of Surgery	Group I (n = 34)	Group C (n = 33)	p-value
Subdural hematoma clearing	5 (14.7%)	10 (30.3%)	0.12
Other	11 (32.35%)	11 (33.3%)	0.79
Front temporoparietal craniotomy	1 (2.94%)	1 (3.03%)	0.99
Epidural hematoma clearing	11 (32.35%)	2 (6.06%)	0.011
Decompressive craniectomy	5 (14.7%)	6 (18.18%)	0.99
Craniotomy with elevation of depressed fracture	1 (2.94%)	3 (9.09%)	0.3

DISCUSSION

In the prospective cohort study, the impact of gabapentin on secondary brain injury prevention and its efficacy in PSH management among TBI patients were examined. The study revealed a statistically significant reduction in the frequency of PSH episodes in the gabapentin-treated group, as evidenced by a decreased need for sedation boluses. The mean PSH score in the gabapentin group I was 19.26 ± 2.76, compared to 22.80 ± 3.20 in the group II, with a p-value of <0.001, indicating a highly significant difference. PSH is recognized as a syndrome characterized by paradoxical elevations in sympathetic (elevated heart rate, temperature, blood pressure, respiratory rate, and perspiration) and motor (posturing) physiological responses. The findings showed a substantial reduction in the requirement for sedation boluses to manage these symptoms in the gabapentin group compared to the controls. The findings of the investigation are consistent with prior research that underscores the importance of early and targeted intervention in PSH management. For instance, Baguley et al. [13] highlighted the challenges in managing PSH and the potential effectiveness of gabapentin as a supplementary treatment in diminishing the intensity and frequency of PSH. Similarly, another study by Youn et al. [14] demonstrated the effectiveness of gabapentin in reducing the severity of autonomic dysregulation in patients with severe TBI, supporting the neuroprotective role of gabapentin observed in the study. Mortality and morbidity in TBI are often influenced by the magnitude of subsequent cerebral damage and vasogenic edema, which are exacerbated by primary insults. The pathophysiology involves neurotoxic edema caused by sodium-calcium imbalances and the accumulation of free radicals. Previous animal studies have demonstrated gabapentin's cerebroprotective effects following TBI, which may be attributable to its ability to modulate excitatory neurotransmitter release and attenuate neuroinflammation. In the study, the Glasgow Coma Scale (GCS) scores at admission and discharge served as key indicators of secondary injury severity. Notably, patients in the gabapentin group showed a notable increase in Glasgow Coma Scale from admission to discharge (median improvement of 53% [IQR 31, 114]) compared to the control group (median improvement of 25% [IQR 5, 56]), with a p-value of 0.009. The study also monitored patients throughout their ICU stay, hospitalization, and up to 90 days post-discharge. A significant enhancement in clinical outcomes was observed in the gabapentin group, as reflected in the Glasgow Outcome Scale (GOS) scores at 30 and 90 days. The median GOS score at 90 days was 4.5 (IQR 4, 5) in the gabapentin group, compared to 3 (IQR 3, 4) in the control group, with a p-value of 0.001. This shows that gabapentin not only mitigates the sudden symptoms of PSH but also adds to long-term functional recovery. Comparative studies, such as the retrospective analysis by Perkes et al. [15], have shown that delayed identification and management of PSH are associated with poorer outcomes, including prolonged ICU stays, increased healthcare costs, and lower GOS scores. The study further supports these findings, demonstrating that timely intervention with gabapentin significantly improves both short-term and long-term outcomes. While GOS is an effective measure of functional recovery, it does not fully capture the underlying disease processes. Advanced diagnostic tools, such as electroencephalography (EEG) and brain biopsy, can provide deeper insights into cortical function and prognosis, though they were not utilized in the study. Nevertheless, the observed changes in EEG patterns and consciousness recovery in the gabapentin group align with previous reports of improved neurocognitive outcomes following gabapentin treatment.

Generalizability

The findings of this study are promising but may have limited generalizability due to the specific population and setting in which it was conducted. The study focused on patients in a tertiary care facility in northern India, and the sample size was relatively small (67 participants), which may not fully represent broader, more diverse populations. Additionally, factors such as variations in healthcare infrastructure, patient demographics, and treatment protocols across different regions could influence the applicability of these results to other settings. Larger, multicenter studies would be needed to confirm the generalizability of gabapentin's efficacy in managing traumatic brain injury (TBI) globally.

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CONCLUSION

The study contributes to the growing body of evidence supporting the use of gabapentin in managing PSH and preventing secondary brain injury in TBI patients. The statistically significant improvements in PSH scores, GCS, and GOS highlight gabapentin's potential as a neuroprotective agent, warranting further investigation in larger, randomized controlled trials to validate these results and refine treatment protocols.

LIMITATIONS

This work is the initial prospective investigation of the effect of gabapentin following traumatic brain injury in a specific subgroup of the Indian population. Considering the 90-day follow-up and the sample size, the outcomes were reasonably estimated in the study. Nevertheless, significant constraints in the study include limited sample size and the inability to use procedures like EEG and brain biopsy for objective assessment of brain swelling, as demonstrated in another research [16]. Reduction in brain swelling was associated with improvement in the Glasgow Coma Scale (GCS) and served as a surrogate marker [17,18].

RECOMMENDATION

Based on the findings, the study recommends considering gabapentin as an effective therapeutic option in the management of traumatic brain injury (TBI) patients. Gabapentin was shown to reduce secondary brain injury, improve functional outcomes (as measured by GOS and GCS), and decrease the frequency of PSH episodes, which suggests its potential neuroprotective role in TBI management.

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LIST OF ABBREVIATION

TBI - Traumatic Brain Injury
PSH - Paroxysmal Sympathetic Hyperactivity
GCS - Glasgow Coma Scale
GOS - Glasgow Outcome Scale
ICU - Intensive Care Unit
NCCT - Non-Contrast Computed Tomography
PSH-AM - Paroxysmal Sympathetic Hyperactivity Assessment Measure
EEG - Electroencephalography
RAS - Reticular Activating System

CONFLICT OF INTEREST

The contributors have no conflicting interests to specifically disclose.

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Funds not received.

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