RESEARCH PAPER

Prognostic and diagnostic value of Plasma Cellular prion protein (PrPC) after moderate head trauma

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ABSTRACT

Background

Traumatic brain injury (TBI) remains a major cause of morbidity and mortality worldwide, and reliable blood-based biomarkers for early prognostic assessment are still lacking. Cellular prion protein (PrPC) is a neuronal membrane glycoprotein that may be released into the circulation following brain injury and could reflect the extent of neuronal damage.

Aim

To evaluate the temporal changes in plasma PrPC levels in first days after moderate traumatic brain injury and to assess their relationship with early neurological outcome.

Patients and Methods

This prospective observational study included 30 adult patients with isolated moderate traumatic brain injury (GCS 9–12). Plasma cellular prion protein (PrPC) levels were measured on day 1, day 2, and day 3 after admission. Neurological status was evaluated using the Glasgow Coma Scale on day 1 and day 3. Mean arterial pressure was recorded serially during the study period. Radiological assessment was performed using brain computed tomography on admission, with subsequent follow-up imaging. Correlation analysis was conducted to determine the relationship between PrPC levels and clinical parameters.

Results: Plasma PrPC levels decreased progressively through out the study period . A significant improvement in GCS was observed between day 1 and day 3 ($p < 0.001$). Plasma PrPc levels decreased progressively throughout the study period, with mean values declining from 3.97 ± 0.88 on day 1 to 3.60 ± 0.75 on day 2 and 2.70 ± 0.86 on day 3. This reduction was statistically highly significant ($P < 0.001$), suggesting a marked decline in plasma PrPc levels during follow-up

Conclusion

Plasma PrPC exhibits significant early dynamic changes after moderate TBI and correlates with neurological recovery. Serial measurement appears to be more informative than a single early value and may serve as a promising early prognostic biomarker.

Keywords

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Introduction

Traumatic brain injury (TBI) represents a major global cause of mortality and long-term disability and remains a critical public health problem across all age groups. Recent global burden analyses confirmed the persistently high incidence of TBI and its substantial socioeconomic impact despite advances in trauma systems and neurocritical care **(1, 2)**.

Early assessment of injury severity and prediction of outcome are essential for optimizing management strategies. However, current clinical tools—including the Glasgow Coma Scale (GCS) and computed tomography (CT)—have important limitations, as they may not adequately reflect the underlying molecular and cellular damage or the dynamic pathophysiological processes that occur after injury. Consequently, there has been increasing interest in identifying objective blood-based biomarkers that can improve diagnostic accuracy, enhance risk stratification, and allow early prognostic evaluation **(1)**.

Protein biomarkers have emerged as a cornerstone of modern neurotrauma research because they provide measurable indicators of neuronal and glial injury and enable real-time monitoring of disease evolution. Among the most extensively studied biomarkers are glial fibrillary acidic protein (GFAP) and ubiquitin C-terminal hydrolase-L1 (UCH-L1), which have demonstrated significant diagnostic value for detecting intracranial injury and predicting functional outcomes after TBI. Large multicenter studies, including the TRACK-TBI cohort, have shown that

early plasma levels of GFAP and UCH-L1 improve prognostic models beyond clinical variables alone **(3, 4)**.

Despite these advances, currently available biomarkers still have limitations related to sensitivity, specificity, and their ability to reflect the full spectrum of neuronal injury, and no single biomarker has yet achieved sufficient performance to be used as a stand-alone tool in routine clinical practice. This has led to ongoing research into novel biologically relevant markers that more directly reflect neuronal membrane disruption and synaptic injury **(3)**.

The cellular prion protein (PrPC) is a glycosylphosphatidylinositol-anchored membrane glycoprotein that is highly expressed in neurons and plays a key role in neuroprotection, synaptic transmission, cellular signaling, and resistance to oxidative stress. Its localization on the external surface of neuronal membranes makes it particularly vulnerable to mechanical disruption following traumatic injury. Blood–brain barrier breakdown and neuronal membrane damage can result in the release of PrPC into the systemic circulation, suggesting that circulating PrPC may serve as a direct marker of neuronal structural injury **(5, 6)**.

Experimental models of blast-induced TBI have demonstrated a significant increase in plasma PrPC levels proportional to injury severity, supporting its role as a biomarker of neuronal damage. In addition, early clinical observations in patients with concussion showed that plasma PrPC concentrations were significantly elevated after injury compared with controls, highlighting its potential diagnostic value **(5, 6)**.

Another important concept in modern TBI biomarker research is the dynamic temporal profile of circulating proteins. Serial sampling has been shown to provide more accurate information about injury evolution and recovery than single time-point measurements, and current international recommendations emphasize the importance of longitudinal biomarker assessment for monitoring disease progression and improving prognostic accuracy **(1)**.

Although these findings suggest that PrPC may represent a novel and biologically relevant biomarker of neuronal injury, clinical studies evaluating its temporal changes and its relationship to neurological recovery—particularly in patients with moderate TBI—remain limited. Therefore, investigating serial plasma PrPC levels and their correlation with neurological and physiological parameters may provide additional insight into its diagnostic and prognostic utility in this important subgroup of TBI patients.

Patients and methods

Study design and ethical approval

This prospective observational study was carried out in the Intensive Care Unit of Minia University Hospital over a period of 12 months. The study protocol was reviewed and approved by the Institutional Ethics Committee of the Faculty of Medicine, Minia University. Written informed consent was obtained from the first-degree relatives of all included patients before enrollment in the study. The study was conducted in accordance with the ethical principles of the Declaration of Helsinki.

Study population

A total of 30 adult patients of both sexes with isolated moderate traumatic brain injury (TBI) were included in the present study. Moderate TBI was defined as a Glasgow Coma Scale (GCS) score ranging from 9 to 12 after initial resuscitation in the emergency department. Eligible patients were aged between 18 and 60 years.

Patients were excluded if they had associated polytrauma, a history of pre-existing neurological or neurosurgical disorders, or a GCS score less than 9 on admission. Patients who required mechanical ventilation at the time of initial assessment were not included, in order to ensure a clinically homogeneous group and to allow reliable serial neurological evaluation.

Clinical evaluation and physiological monitoring

All patients were subjected to full clinical assessment upon admission. Demographic data including age and sex were recorded. Routine monitoring of vital parameters was performed for all patients as part of standard ICU care. These parameters included heart rate, respiratory rate, body temperature, and mean arterial pressure (MAP).

Mean arterial pressure was recorded serially on day 1, day 2, and day 3 to evaluate early hemodynamic changes during the acute phase of injury. These measurements were included to explore the possible relationship between systemic physiological status and circulating levels of plasma cellular prion protein (PrPC).

Neurological status was assessed using the Glasgow Coma Scale (GCS). The initial GCS was recorded after resuscitation on admission (day 1), and a follow-up GCS assessment was performed on day 3. The change in GCS between these two time points was calculated and used as an indicator of early neurological improvement.

Radiological assessment

All patients underwent non-contrast computed tomography (CT) of the brain on admission for diagnosis and classification of intracranial lesions. The CT findings were categorized according to the type of traumatic lesion. A follow-up CT scan was

performed for all patients during the observation period, and no radiological progression was detected.

Measurement of plasma cellular prion protein (PrPC)

Venous blood samples were collected from each patient under complete aseptic conditions at three predefined time points: on admission (day 1), after 48 hours (day 2), and on day 3. The samples were processed immediately for plasma separation by centrifugation and then stored at appropriate temperatures until biochemical analysis.

Serial measurement of plasma PrPC levels was performed to evaluate its temporal pattern following moderate traumatic brain injury and to investigate its potential role as a diagnostic and prognostic biomarker.

Outcome measures

The primary outcome of the study was the temporal change in plasma PrPC levels during the first three days following traumatic brain injury.

Secondary outcome measures included early neurological improvement assessed by the change in GCS between day 1 and day 3, serial changes in mean arterial pressure, length of ICU stay, and the correlation between plasma PrPC levels and both neurological and physiological parameters.

Statistical analysis

Data were coded and analyzed using the Statistical Package for Social Sciences (SPSS) software (version 26). Quantitative data were expressed as mean ± standard deviation (SD) and range, whereas qualitative variables were presented as number and percentage.

Changes in plasma PrPC levels over time were analyzed using repeated measures analysis with pairwise comparisons between different time points. Comparison between GCS values on day 1 and day 3 was performed using the paired t-test.

Variable	Studied group (n=30)
Age	
Mean ± SD	38.5 ± 17.6
Median (IQR)	37.5 (22.7 – 47.2)
Length of stay	
Mean ± SD	2.65 ± 0.47
Median (IQR)	3 (2 – 3)

Correlation between plasma PrPC levels and clinical variables, including GCS and MAP, was assessed using Friedmann test for repeated measures for the study group . A p-value less than 0.05 was considered statistically significant.

Results

The present study included 30 patients with moderate traumatic brain injury. The studied cohort showed a wide age distribution . Baseline vital parameters were within clinically acceptable ranges, and none of the patients required mechanical ventilation during the period of observation. The duration of ICU stay was short, with most patients discharged after three days, reflecting the relatively stable clinical course of this homogeneous moderate TBI group.

Radiological evaluation revealed heterogeneous intracranial lesions, with traumatic subarachnoid hemorrhage associated , cerebral contusions and Other lesions, including extradural hematoma, intracerebral hemorrhage, and skull fractures, were observed with lower and comparable frequencies. Follow-up CT brain showed no radiological progression in all studied cases, indicating early clinical and radiological stability in this cohort **.**

Serial measurement of plasma cellular prion protein (PrPC) demonstrated a progressive and statistically significant decline over the first three days following injury. Pairwise comparisons confirmed that this reduction occurred consistently between all time points, indicating a time-dependent pattern of change. The calculated mean differences further supported this finding, showing a modest reduction after the first 48 hours that became more pronounced by day 3. These results suggest that plasma PrPC levels are dynamically modulated during the early post-traumatic period and may reflect the evolving pathophysiological processes following moderate TBI **.**

A significant improvement in the Glasgow Coma Scale was observed between day 1 and day 3, indicating early neurological recovery in the studied patients. The magnitude of GCS change supports the clinical stability of the included cases and confirms the suitability of this cohort for evaluating early prognostic biomarkers **.**

Mean arterial pressure showed a gradual and statistically significant increase over the three days of follow-up. This progressive improvement reflects stabilization of systemic perfusion during the acute phase of injury. However, the relatively narrow range of MAP values suggests that all patients were maintained within a controlled hemodynamic target as part of standard ICU management **.**

Table (1): Sociodemographic data and clinical data of the studied groups

- Quantitative data were represented by mean, standard deviation and median (interquartile range), qualitative data are represented by number and percentage.

The study included 30 participants with a mean age of 38.5 ± 17.6 years (median: 37.5 years, IQR: 22.7–47.2). The median length of hospital stay was 3 days (IQR: 2–3), with a mean duration of 2.65

Variable	Studied group (n=30)
PrPc 1st day	
Mean ± SD	3.97 ± 0.88
Median (IQR)	3.9 (3.35 – 4.77)
PrPc 2nd day	
Mean ± SD	3.6 ± 0.75
Median (IQR)	3.7 (2.9 – 4.2)
PrPc 3rd day	
Mean ± SD	2.7 ± 0.86
Median (IQR)	2.8 (2.2 – 3.55)
P value	<0.001*

± 0.47 days, indicating a relatively short hospitalization period among the studied patients.

Table (2): MAP of the studied groups

- Friedmann test used for repeated measures for the study group.

****: p value < 0.05 was significant***

Variable	Studied group (n=30)
MAP 1st day	
Mean ± SD	84.6 ± 4.9
Median (IQR)	85 (80 – 88.7)
MAP 2nd day	
Mean ± SD	88.9 ± 5.5
Median (IQR)	90 (85 – 90)
MAP 3rd day	
Mean ± SD	90.3 ± 7.2
Median (IQR)	90 (85 – 97.5)
P value	0.001*

Mean arterial pressure showed a gradual increase over the three-day follow-up period, rising from 84.6 ± 4.9 mmHg on the first day to 88.9 ± 5.5mmHg on the second day and 90.3 ± 7.2 mmHg on the third day. This progressive improvement suggests better hemodynamic stabilization during hospitalization. However, the table does not report the corresponding p-value; therefore, the statistical significance of this trend cannot be determined from the available data.

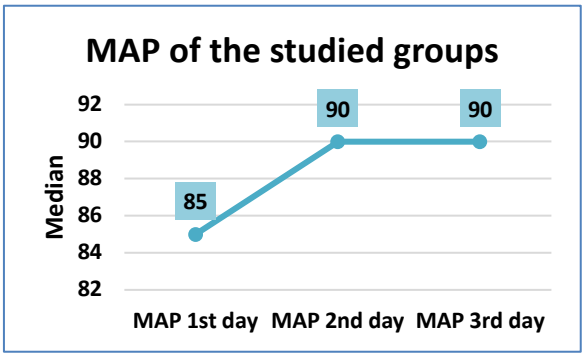


Table (3): Vital rate of the studied groups

Variable	Studied group (n=30)
HR	
Mean ± SD	85.2 ± 10.8
Median (IQR)	87 (77 – 93)
Respiratory rate	
Mean ± SD	28.6 ± 2.4
Median (IQR)	28 (27 – 30)
Temperature	
Mean ± SD	37.5 ± 0.39
Median (IQR)	37.15 (37 – 37.5)

- Quantitative data were represented by mean, standard deviation and median (interquartile range), qualitative data are represented by number and percentage.

The patients had a mean heart rate of 85.2 ± 10.8 beats/min, a mean respiratory rate of 28.6 ± 2.4 breaths/min, and a mean body temperature of 37.5 ± 0.39°C. The median values were 87 beats/min, 28 breaths/min, and 37.15°C, respectively, indicating relatively stable vital parameters within the study population.

Table (4): Plasma cellular prion protein marker (prpc) of the studied groups

- Friedmann test used for repeated measures for the study group.

****: p value < 0.05 was significant***

Plasma PrPc levels decreased progressively throughout the study period, with mean values declining from 3.97 ± 0.88 on day 1 to 3.60 ± 0.75 on day 2 and 2.70 ± 0.86 on day 3. This reduction was statistically highly significant (P < 0.001), suggesting a marked decline in plasma PrPc levels during follow-up.

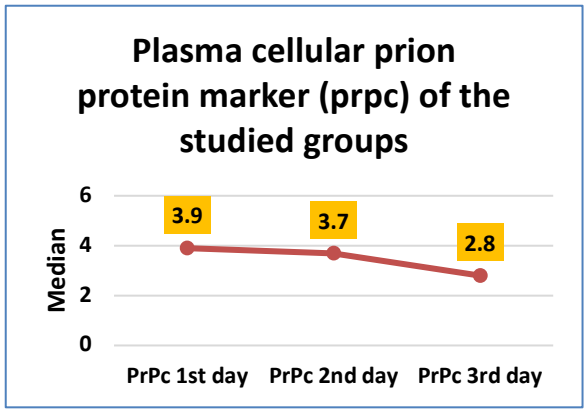
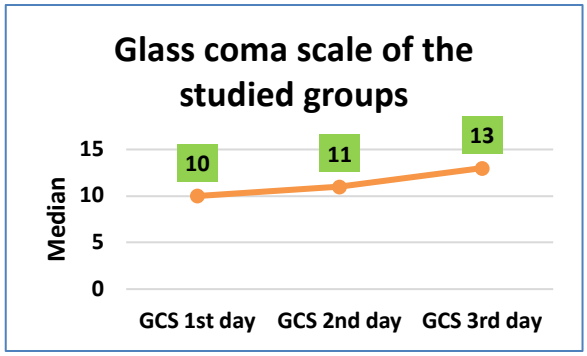


Table (5): Glass coma scale of the studied groups

- Friedmann test used for repeated measures for the study group.
****: p value < 0.05 was significant***

Variable	Studied group (n=30)
GCS 1st day	
Mean ± SD	9.8 ± 0.76
Median (IQR)	10 (9 – 10)
GCS 2nd day	
Mean ± SD	10.9 ± 1.07
Median (IQR)	11 (10 – 11)
GCS 3rd day	
Mean ± SD	12.9 ± 0.57
Median (IQR)	13 (11 – 13)
P value	<0.001*

The Glasgow Coma Scale scores improved significantly over the three days of observation. The mean GCS increased from 9.8 ± 0.76 on the first day to 10.9 ± 1.07 on the second day and 12.9 ± 0.57 on the third day. This improvement was statistically highly significant (P < 0.001), indicating a substantial enhancement in patients' neurological status during the study period.



DISCUSSION

In this study of patients with moderate traumatic brain injury (TBI), plasma cellular prion protein (PrPC) demonstrated a clear time-dependent decline over the first three days after admission, accompanied by early neurological improvement as reflected by increasing GCS . Although the follow-up CT remained stable in all cases, PrPC still showed significant temporal change, suggesting that PrPC may reflect evolving biological processes after injury even in clinically and radiologically stable moderate TBI. In addition, PrPC measured on day 3 correlated with contemporaneous neurological status and with the magnitude of neurological improvement , supporting the concept that later PrPC levels—and dynamic change—may provide prognostic information beyond a single early measurement.

our cohort showed higher early PrPC values on day 1 (mean) followed by a significant reduction by day 3 (mean ≈2.75 ng/mL). This pattern is biologically plausible given that PrPC is a membrane-anchored protein enriched in the CNS and may be released into blood after neuronal membrane disruption and blood–brain barrier dysfunction. Mechanistically, circulating soluble PrPC can arise through regulated proteolytic shedding of membrane PrPC—particularly via ADAM10—besides passive release from injured tissue, which could help explain dynamic changes during early recovery **(7, 8)**.

When compared to previous work, the magnitude of our day-1 PrPC levels is notable. In hemorrhagic stroke (ICH), Wu et al. reported median plasma PrPC 4.20 ng/mL in patients vs 2.02 ng/mL in controls (P<0.001), indicating that acute brain injury states can produce circulating PrPC values in the same range as our early post-TBI measurements **(6)**.

Human concussion data also support post-injury elevation. In the sport-related concussion pilot study by Pham et al., the mean post-concussion PrPC was 2.96 ng/mL (±0.37 SEM, n=6) versus 1.59 ng/mL (±0.07 SEM, n=76) in athlete baseline samples, and 1.70 ng/mL (±0.07 SEM, n=103) in combined baseline controls (P<0.0001). our day-3 PrPC mean (~2.75 ng/mL) approaches the "post-concussion" range described there, while our day-1 mean (~4.03 ng/mL) is closer to levels reported in more severe acute brain injury cohorts such as ICH **(5)**.

Preclinical evidence aligns with injury-associated elevation as well. In a rat model of primary blast-induced TBI, Pham et al. found mean plasma PrPC 3.97 ng/mL (±0.13 SE) vs 2.46 ng/mL (±0.14 SE) in controls (p<0.0001) at 24 hours post-insult, supporting the idea that TBI can raise plasma PrPC into the 3–4 ng/mL range **(9)**.

Taken together, prior animal and human studies mainly emphasize post-injury elevation of PrPC. our

study adds an important complementary observation: in a clinically stable moderate TBI cohort, PrPC appears to decline significantly over the first 72 hours (Table 2). This "early high → subsequent decline" may represent resolution of acute release/shedding and/or stabilization of BBB integrity and neuronal membrane stress during early recovery.

In our correlation analysis, PrPC on day 3 showed a significant negative association with GCS on day 3 and with the degree of neurological improvement .This directionality is consistent with evidence from ICH: Wu et al. reported a strong inverse correlation between plasma PrPC and GCS ($r = -0.645$, $P < 0.001$) and also demonstrated prognostic performance for 90-day outcome **(6)**.

Similarly, in aneurysmal subarachnoid hemorrhage, Yao et al. (Clinical Chimica Acta) found that plasma PrPC concentrations were significantly higher in patients than controls and were "tightly correlated" with severity scales including GCS, with independent prediction of 90-day poor outcome **(10)**.

These cross-condition findings support a broader interpretation: circulating PrPC may function as a general marker of acute CNS injury burden and may become more clinically informative when measured after the initial stabilization period. In our data, the absence of a significant association between day-1 PrPC and admission GCS could reflect heterogeneity in injury timing before sampling, early physiologic confounding, or the fact that immediate clinical scores may be dominated by transient factors (e.g., agitation, pain, prehospital events), while later measurements better align with evolving neuronal injury biology.

MAP increased significantly over time (Table 4), likely reflecting early systemic stabilization and optimized ICU management rather than direct injury progression. Importantly, all follow-up CT scans were unchanged , which strengthens the argument that the observed PrPC dynamics are not simply a byproduct of radiological deterioration but may capture subtler biological changes (inflammation, oxidative stress, synaptic/membrane turnover) that are not necessarily visible on short-interval CT. Mechanistically, PrPC is implicated in neuroprotective signaling and oxidative stress responses, providing biological plausibility for dynamic regulation during recovery **(11, 12)**.

Current biomarker frameworks emphasize that no single marker perfectly captures the heterogeneous biology of TBI, and that temporal kinetics are crucial for clinical utility. Large efforts in TBI biomarkers have highlighted the value of time-sensitive markers (e.g., GFAP and UCH-L1) for diagnosis and risk stratification, and underscore the importance of specifying sampling windows and interpreting biomarkers in relation to time after injury **(13, 14)**.

In that context, our serial PrPC sampling over three days is a methodological strength because it addresses kinetics rather than relying on a single admission value. our findings suggest that PrPC may have potential value as (1) a dynamic marker in the acute phase, and (2) a late-acute prognostic correlate (day-3 association with neurological status and improvement; , warranting evaluation in larger cohorts with longer outcome follow-up (e.g., 30–90 days).

Clinically, our data support the hypothesis that PrPC is responsive to early post-traumatic changes and that later measurements may better reflect neurological trajectory . Future work should aim to (i) standardize sampling relative to injury time, (ii) include a matched control group, (iii) extend follow-up to functional outcomes (e.g., GOS-E at 3 months), and (iv) evaluate PrPC alongside established biomarkers (GFAP/UCH-L1) to test whether PrPC adds incremental prognostic value in multivariable models **(6, 13)**.

Limitations

This study has limitations that should be considered when interpreting the results. First, the sample size was relatively small and derived from a single center, which may limit generalizability. Second, follow-up was limited to three days and did not include longer-term functional outcomes, Finally, details that can influence biomarker interpretation—such as exact time from injury to first blood sampling and assay kit characteristics—should be explicitly standardized and reported in future studies.

CONCLUSION

Plasma cellular prion protein (PrPC) demonstrated a significant time-dependent decline in first 3 days after moderate traumatic brain injury, and this dynamic change was accompanied by early neurological improvement as reflected by GCS scores. Despite the absence of radiological progression on follow-up CT, PrPC showed a distinct temporal pattern, suggesting that it reflects ongoing biological processes not detected by routine imaging. Moreover, PrPC levels on day 3 and their rate of change were significantly associated with neurological recovery, indicating its potential value as an early prognostic biomarker. Serial measurement of PrPC appears to be more informative than a single early determination.

RECOMMENDATIONS

- Larger multicenter studies are recommended to validate the prognostic role of PrPC.
- Future research should include long-term functional outcome assessment.

- Combined evaluation of PrPC with established TBI biomarkers may improve prognostic accuracy.
- Standardization of the timing of blood sampling is needed to better define its kinetic profile.
- Further studies across different TBI severities are required to explore its role in risk stratification.

Author contribution

- **Heba Allah Abd El-Kareem Ali Mohamed:** Conceptualization, study design, patient recruitment, data collection, laboratory work, statistical analysis, and drafting of the manuscript.
- **Amany Khairy Abu El-Hussein:** Study supervision, methodology development, critical revision of the manuscript for important intellectual content.
- **Khaled Ahmed Abdo:** Methodology, data interpretation, and manuscript revision.
- **Hany Kamal:** Clinical supervision, patient management, and final approval of the manuscript.
- **Asmaa Khalf Allah Kamel**: clinical supervision, lab invetigation and manuscript revision
- All authors read and approved the final version of the manuscript.

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Conflict of interest
The authors declare that there is no conflict of interest regarding the publication of this paper.

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