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Original Article

The Impact of Cognitive Reserve on Neuroplastic Changes among Complicated Mild Traumatic Brain Injury Patients

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ABSTRACT\

Background: Cognitive reserve may influence how individuals tolerate or clinically express neurological injury, but its relationship with structural imaging patterns in complicated mild traumatic brain injury remains insufficiently characterized. Objective: To determine the association between cognitive reserve and MRI-defined structural injury patterns among adults with complicated mild traumatic brain injury. Methods: This analytical cross-sectional study included 64 patients aged 20–40 years recruited through convenience sampling from three tertiary-care hospitals in Lahore, Pakistan, between August 2024 and January 2025. Cognitive reserve was assessed using the Cognitive Reserve Assessment Scale in Health, while structural injury patterns were classified from MRI findings. Categorical variables were summarized as frequencies and percentages, and the association between cognitive-reserve category and MRI pattern was assessed using the chi-square test. Results: The mean age was 29.27 ± 6.08 years, and 34 (53.1%) participants were female. Higher cognitive reserve was identified in 34 (53.1%) participants, while 30 (46.9%) had lower reserve. Less-pronounced MRI lesions were observed in 34 (53.1%) participants and more extensive lesions in 30 (46.9%). Higher reserve corresponded completely with the less-pronounced MRI category, while lower reserve corresponded with the more extensive category (χ^2 = 64.000, df = 1, $p < 0.001$; Phi = 1.000). Conclusion: Cognitive-reserve classification was strongly associated with MRI-defined structural injury pattern, although the cross-sectional design did not establish causality or longitudinal neuroplastic change. Keywords: Cognitive Reserve; Complicated Mild Traumatic Brain Injury; Magnetic Resonance Imaging; Structural Brain Injury; Cognitive Reserve Assessment Scale in Health; Neuroplasticity.

INTRODUCTION

Traumatic brain injury (TBI) is a major cause of mortality, long-term disability, cognitive impairment, and reduced participation worldwide. It results from an external mechanical force, including falls, road traffic collisions, interpersonal violence, sports-related impacts, blasts, and rapid acceleration–deceleration movements, that disrupts normal brain structure or function. The resulting neuropathological spectrum may include focal contusions, intracranial haemorrhage, diffuse axonal injury, cerebral oedema, hypoxia, and secondary neuronal damage. Although TBI can affect individuals of all ages, children, young adults, older persons, and males exposed to occupational, transport-related, or recreational risks experience a disproportionate burden (1,2).

Traumatic brain injury is commonly classified as mild, moderate, or severe according to the Glasgow Coma Scale score, duration of loss of consciousness, duration of post-traumatic amnesia, and neuroimaging findings. Mild TBI constitutes the majority of reported cases and is generally characterized by a Glasgow Coma Scale score of 13–15, loss of consciousness lasting less than 30 minutes, and post-traumatic amnesia of less than 24 hours. Mild TBI may be further classified as uncomplicated or complicated according to the absence or presence of trauma-related intracranial abnormalities on neuroimaging. Complicated mild TBI is clinically important because patients may initially demonstrate relatively preserved consciousness while still having structural brain lesions and persistent cognitive, behavioural, or functional difficulties (2–4).

Brain injury develops through interacting primary and secondary mechanisms. Primary injury occurs at the moment of trauma and may cause direct tissue disruption, vascular injury, and axonal damage. This is followed by secondary processes that may include excitotoxicity, neuroinflammation, oxidative stress, mitochondrial dysfunction, altered cerebral perfusion, oedema, and progressive cellular injury. These mechanisms can continue after the initial event and influence the extent of neurological dysfunction and subsequent clinical outcomes (1,5,6). Recovery after TBI is heterogeneous and depends not only on the anatomical severity and location of the injury but also on age, premorbid health, educational attainment, rehabilitation exposure, psychosocial factors, and the capacity of the nervous system to adapt to injury.

Neuroplasticity refers to the ability of the nervous system to modify its structural organization, functional connectivity, and patterns of neural activity in response to injury, experience, environmental demands, and rehabilitation. Adaptive neuroplasticity may support the recruitment of alternative neural pathways and the reorganization of surviving networks, whereas maladaptive changes may contribute to persistent cognitive, behavioural, or motor dysfunction. The extent and clinical relevance of neuroplastic responses vary substantially among patients and are influenced by the severity of neural damage, timing of assessment, environmental stimulation, and individual biological characteristics (7–10).

Cognitive reserve is a related but distinct construct that describes the capacity of an individual to maintain cognitive performance or cope with neuropathology by using neural networks more efficiently or recruiting compensatory cognitive strategies. Cognitive reserve is believed to develop across the lifespan through education, occupational complexity, premorbid intellectual ability, social participation, and sustained engagement in cognitively stimulating activities. It does not necessarily prevent structural brain injury; rather, it may modify the clinical expression of that injury and help explain why individuals with apparently similar neuropathology experience different levels of cognitive impairment (11–14).

Previous research involving acquired brain injury has generally shown that higher educational attainment, premorbid intelligence, occupational complexity, and social or intellectual engagement are associated with better post-injury cognitive performance. Systematic reviews have suggested that greater cognitive reserve may be associated with reduced cognitive impairment after stroke and TBI, although the strength and consistency of this relationship vary according to the reserve proxy, injury severity, outcome measure, and duration of follow-up (12,13,15). Evidence from rehabilitation and neuromodulation research also indicates that preserved cognitive and neural resources may influence responsiveness to interventions intended to support functional reorganization after brain injury (8,16,17).

Despite growing interest in cognitive reserve, several uncertainties remain. Many previous studies have combined patients with different types and severities of acquired brain injury, relied on indirect indicators such as education or estimated premorbid intelligence, or evaluated cognitive outcomes without incorporating neuroimaging findings. Research specifically examining complicated mild TBI remains limited, even though this subgroup may experience persistent symptoms despite having relatively high Glasgow Coma Scale scores. Moreover, structural lesion burden, cognitive reserve,

cognitive functioning, and neuroplasticity are conceptually related but should not be treated as interchangeable constructs. Fewer or less extensive lesions on structural magnetic resonance imaging may indicate lower injury burden or greater preservation of brain tissue, but they do not independently demonstrate longitudinal neuroplastic reorganization.

A clearer understanding of the relationship between cognitive reserve and structural neuroimaging patterns may help explain variability among patients with complicated mild TBI and support more individualized assessment and rehabilitation planning. Therefore, the present study aimed to determine the association between cognitive reserve, assessed using the Cognitive Reserve Assessment Scale in Health, and MRI-defined structural injury patterns among adults with complicated mild traumatic brain injury.

MATERIALS AND METHODS

This multicentre analytical cross-sectional study was conducted from August 2024 to January 2025 at Mayo Hospital, Lahore General Hospital, and Jinnah Hospital in Lahore, Pakistan. The study was undertaken following formal approval of the research synopsis and was designed to examine the association between cognitive reserve and MRI-defined structural injury patterns among patients with complicated mild traumatic brain injury. The reporting and interpretation of the study were aligned with the observational nature of the design; consequently, the analysis assessed associations at the recorded assessment period and was not intended to establish causality, longitudinal neuroplastic change, or the rate of clinical recovery.

The target population comprised male and female patients aged 20–40 years with non-penetrating complicated mild TBI. Complicated mild TBI was defined by a Glasgow Coma Scale score of 13–15, loss of consciousness lasting less than 30 minutes, post-traumatic amnesia lasting less than 24 hours, and the presence of trauma-related intracranial abnormalities on magnetic resonance imaging. Patients presenting within 2–8 weeks of the injury were considered eligible. Individuals were included when they met the clinical and neuroimaging criteria and voluntarily agreed to participate. Patients with a pre-existing neurological or psychiatric disorder, substance abuse within the preceding six months, uncontrolled diabetes mellitus, severe hypertension, pregnancy, or penetrating brain injury were excluded because these conditions could interfere with cognitive assessment, neurological status, or interpretation of the imaging findings.

Participants were recruited through non-probability convenience sampling from the participating tertiary-care hospitals. Eligibility was established from the available clinical history, Glasgow Coma Scale findings, documented duration of loss of consciousness and post-traumatic amnesia, and magnetic resonance imaging evidence. Written informed consent was obtained before enrolment and cognitive assessment. Each participant was assigned a study code, and identifying information was kept separate from the analytical data to preserve confidentiality.

A total sample of 64 participants was included. The sample size was calculated using Epitools at a 5% level of significance, as specified in the approved study protocol. All enrolled participants provided the cognitive-reserve and MRI information required for the reported analyses and were included in the final dataset.

Cognitive reserve was assessed using the Cognitive Reserve Assessment Scale in Health (CRASH). The instrument was administered to each participant after enrolment and generated a total cognitive-reserve score together with general, affective, and non-affective domain scores. In accordance with the scoring approach used in the study, participants were categorized as having lower or higher cognitive reserve on the basis of the total CRASH score. The domain scores were also classified according to the cut-off values applied in the original data collection and analysis. To avoid ambiguity at category boundaries, the

categories were treated as mutually exclusive during data coding. The total CRASH score was the principal cognitive-reserve variable used for examining its relationship with the MRI classification.

Structural brain findings were evaluated from the magnetic resonance imaging examination documented approximately one week after the injury. The MRI findings were categorized according to the extent of the reported structural abnormalities. The first category comprised less-pronounced lesions, fewer microbleeds, and greater preservation of brain volume, whereas the second category comprised more extensive lesions, a greater microbleed burden, and signs of mild cerebral atrophy. These categories were treated as indicators of MRI-defined structural injury burden rather than direct measurements of neuroplasticity or functional recovery. No inference regarding the formation of new neural pathways or longitudinal brain reorganization was made solely from the structural MRI classification.

The primary exposure variable was cognitive-reserve category based on the total CRASH score, and the principal outcome variable was the dichotomized MRI-defined structural injury pattern. General, affective, and non-affective CRASH domain classifications were summarized as secondary descriptive variables. Participant age and sex were also recorded for descriptive characterization of the sample. The temporal sequence of assessment was documented by distinguishing the MRI examination obtained approximately one week after injury from the cognitive-reserve assessment undertaken when participants presented within the specified 2–8-week recruitment period.

Completed data-collection forms were reviewed for completeness and internal consistency before coding. Data were entered into a computerized database and checked to reduce transcription and classification errors. Particular attention was given to ensuring that each participant was assigned to only one category for the total CRASH score, each CRASH domain, and the MRI-defined structural injury pattern. All 64 participants had complete data for the principal variables and were retained in the analysis.

Data were analysed using IBM SPSS Statistics version 26.0. Continuous variables were summarized using the mean and standard deviation, while categorical variables were presented as frequencies and percentages. The distribution of total cognitive reserve, CRASH domains, sex, and MRI-defined structural injury patterns was initially examined descriptively. The chi-square test of independence was used to evaluate the association between cognitive-reserve category and MRI-defined structural injury pattern. The analysis was based on the complete 2×2 contingency table, and the expected cell frequencies were assessed when determining the suitability of the chi-square test. Statistical significance was established at $p < 0.05$, and values generated by the software as 0.000 were interpreted and reported as $p < 0.001$.

The study was conducted in accordance with accepted ethical principles for research involving human participants. Participation was voluntary, written informed consent was obtained before data collection, and participants retained the right to withdraw without affecting their clinical care. Confidentiality and privacy were maintained throughout data collection, data entry, analysis, and reporting. The study procedures were undertaken following formal institutional approval of the research synopsis and in accordance with the principles of the Declaration of Helsinki.

RESULTS

A total of 64 participants with complicated mild traumatic brain injury were included in the analysis. The mean age was 29.27 ± 6.08 years, with a 95% confidence interval of 27.75–30.79 years. Females accounted for 34 (53.1%) participants, while 30 (46.9%) were males. Complete cognitive-reserve and MRI classification data were available for all participants.

The distribution of the Cognitive Reserve Assessment Scale in Health scores is presented in Table 2. Based on the total CRASH score, 34 (53.1%) participants were classified as having higher cognitive

reserve and 30 (46.9%) as having lower cognitive reserve. The general domain was classified as high in 36 (56.3%) participants and low in 28 (43.8%). The affective domain was high in 34 (53.1%) and low in 30 (46.9%). All 64 participants were classified within the high non-affective category, resulting in no variability in this categorical domain.

Table 1. Demographic Characteristics of the Participants

Characteristic	Value
Total participants	64
Age, years, mean ± SD	29.27 ± 6.08
Mean age, 95% CI	27.75–30.79
Female, n (%)	34 (53.1)
Male, n (%)	30 (46.9)

Abbreviations: CI, confidence interval; SD, standard deviation.

Table 2. Distribution of Total and Domain-Specific CRASH Classifications

CRASH measure	Classification	n (%)	95% CI
Total cognitive-reserve score	Lower cognitive reserve	30 (46.9)	35.2–58.9
	Higher cognitive reserve	34 (53.1)	41.1–64.8
General domain	Low	28 (43.8)	32.3–55.9
	High	36 (56.3)	44.1–67.7
Affective domain	Low	30 (46.9)	35.2–58.9
	High	34 (53.1)	41.1–64.8
Non-affective domain	High	64 (100.0)	94.3–100.0

Abbreviations: CI, confidence interval; CRASH, Cognitive Reserve Assessment Scale in Health. Confidence intervals for proportions were calculated using the Wilson method. Categories were treated as mutually exclusive according to the scoring classification applied in the study.

MRI evaluation identified less-pronounced structural lesions with fewer microbleeds and greater preservation of brain volume in 34 (53.1%) participants. More extensive lesions, a greater microbleed burden, and signs of mild cerebral atrophy were identified in 30 (46.9%) participants.

Table 3. Distribution of MRI-Defined Structural Injury Patterns

MRI-defined structural injury pattern	n (%)	95% CI
Less-pronounced lesions with fewer microbleeds and greater preservation of brain volume	34 (53.1)	41.1–64.8
More extensive lesions with greater microbleed burden and signs of mild cerebral atrophy	30 (46.9)	35.2–58.9
Total	64 (100.0)	—

Abbreviations: CI, confidence interval; MRI, magnetic resonance imaging.

The relationship between total cognitive-reserve category and MRI-defined structural injury pattern is shown in Table 4. All 34 participants classified as having higher cognitive reserve had the less-pronounced MRI pattern, whereas all 30 participants classified as having lower cognitive reserve had the more extensive MRI pattern. No participant with higher cognitive reserve was classified in the more extensive MRI category, and no participant with lower cognitive reserve was classified in the less-pronounced MRI category.

Table 4. Association Between Cognitive-Reserve Category and MRI-Defined Structural Injury Pattern

Cognitive-reserve category	Less-pronounced MRI pattern, n (%)	More extensive MRI pattern, n (%)	Total, n (%)
Lower cognitive reserve	0 (0.0)	30 (100.0)	30 (100.0)
Higher cognitive reserve	34 (100.0)	0 (0.0)	34 (100.0)
Total	34 (53.1)	30 (46.9)	64 (100.0)

Pearson $\chi^2 = 64.000$; df = 1; $p < 0.001$; Phi coefficient = 1.000. Percentages in the MRI columns are calculated within each cognitive-reserve category. The conventional odds ratio was not estimable as a finite value because both discordant cells contained zero observations.

The chi-square test demonstrated a statistically significant association between total cognitive-reserve category and MRI-defined structural injury pattern ($\chi^2 = 64.000$, df = 1, $p < 0.001$). The Phi coefficient of

1.000 indicated complete separation between the two classifications in the analysed sample. Because the 2 × 2 table contained two zero cells, a finite conventional odds ratio and corresponding Wald confidence interval could not be calculated. The observed association should therefore be interpreted as a complete categorical correspondence within this sample rather than as evidence that cognitive reserve caused differences in structural brain injury.

The general and affective CRASH domains also showed broadly similar marginal distributions to the total cognitive-reserve classification. However, inferential associations between these domains and the MRI pattern were not reported because the required domain-specific cross-tabulated counts were unavailable. The non-affective category could not support comparative analysis because all participants were classified at the same level.

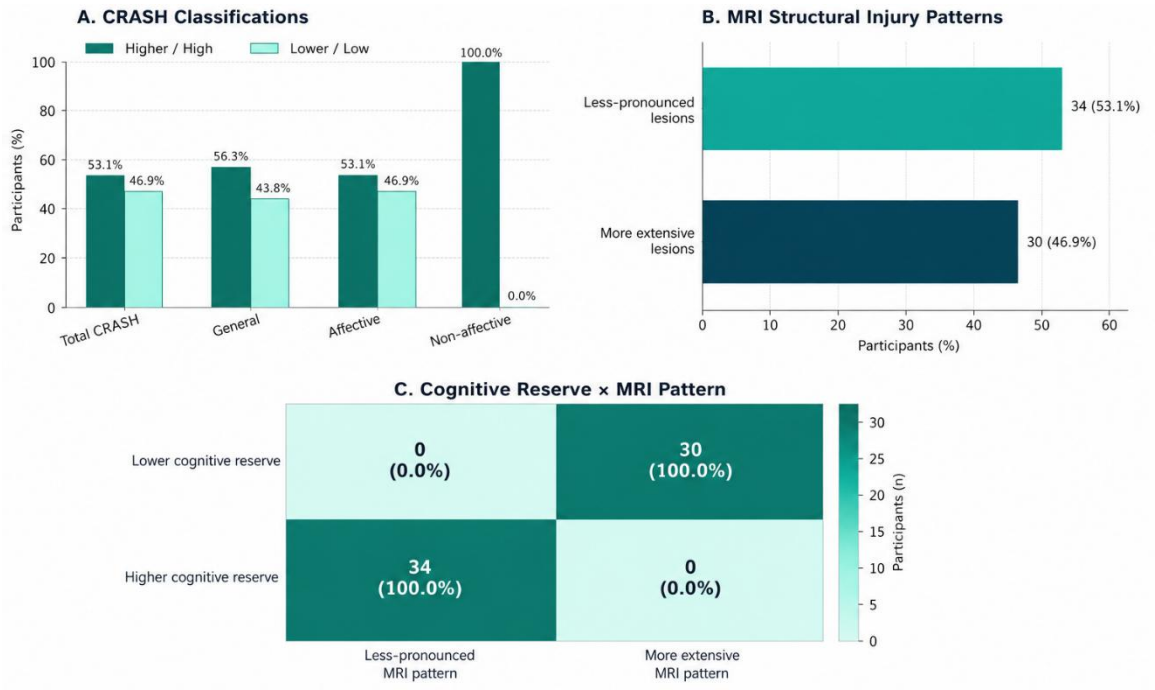

Figure 1 Cognitive reserve classifications and MRI-defined structural injury patterns among patients with complicated mild traumatic brain injury. Panel A presents the distribution of total and domain-specific Cognitive Reserve Assessment Scale in Health classifications. Higher total cognitive reserve was observed in 34 participants (53.1%), while 30 (46.9%) had lower cognitive reserve. High general and affective domain classifications were recorded in 36 (56.3%) and 34 (53.1%) participants, respectively, whereas all 64 participants (100.0%) were classified within the high non-affective category. Panel B shows that 34 participants (53.1%) had less-pronounced MRI lesions with fewer microbleeds and greater preservation of brain volume, while 30 (46.9%) had more extensive lesions, greater microbleed burden, and signs of mild cerebral atrophy. Panel C demonstrates complete categorical separation: all participants with higher cognitive reserve had the less-pronounced MRI pattern, whereas all participants with lower cognitive reserve had the more extensive MRI pattern. Panel D summarizes the strength of this association ($\chi^2 = 64.000$, df = 1, $p < 0.001$; Phi = 1.000). This relationship represents a cross-sectional association and does not establish causality or longitudinal neuroplastic change. CRASH, Cognitive Reserve Assessment Scale in Health; MRI, magnetic resonance imaging.

DISCUSSION

The present study identified a strong cross-sectional association between cognitive-reserve classification and MRI-defined structural injury pattern among adults with complicated mild traumatic brain injury. Slightly more than half of the participants were classified as having higher cognitive reserve, and an equivalent proportion demonstrated the less-pronounced MRI injury pattern. Within the reported cross-tabulation, all participants with higher cognitive reserve were categorized as having less-pronounced lesions, whereas all participants with lower cognitive reserve were categorized as having more extensive lesions. The resulting chi-square statistic and Phi coefficient indicated complete categorical separation within the analysed sample. This finding should nevertheless be interpreted as an association rather than evidence that cognitive reserve prevented structural injury, caused tissue preservation, or produced neuroplastic recovery.

The observed relationship was broadly consistent with the cognitive-reserve framework, which proposes that education, occupational complexity, premorbid intellectual ability, and sustained cognitive or social engagement may influence the ability to maintain cognitive functioning in the presence of neurological pathology. Cognitive reserve is generally considered a functional construct reflecting efficient network utilization and compensatory cognitive processing rather than a direct measure of preserved brain anatomy. Consequently, individuals with higher reserve may demonstrate fewer clinical manifestations for a given degree of pathology, although cognitive reserve does not necessarily reduce the initial mechanical injury caused by trauma (11–14).

Previous studies involving traumatic and other acquired brain injuries have generally reported that higher premorbid intelligence, educational attainment, occupational complexity, socioeconomic resources, and social engagement were associated with better cognitive outcomes. A systematic review of acquired brain injury found that greater cognitive reserve was associated with less cognitive impairment and more favourable cognitive performance, although the included studies varied in design, population, reserve indicator, and outcome measurement (12). A systematic review focused specifically on TBI similarly reported a generally positive relationship between cognitive-reserve proxies and post-injury cognitive performance, while emphasizing substantial heterogeneity in injury severity, assessment timing, and methodological quality (13). These findings support the potential relevance of cognitive reserve in explaining interindividual variability following brain injury but do not establish that reserve determines structural lesion burden.

Steward et al. reported that cognitive reserve was associated with cognitive functioning after TBI, although injury severity remained an important determinant of recovery trajectory (14). This distinction is relevant to the present findings because the MRI categories may primarily reflect variation in the severity and anatomical extent of the original injury. Participants classified as having less-pronounced lesions may have sustained less severe trauma independently of their cognitive-reserve status. Without adjustment for injury mechanism, lesion location, Glasgow Coma Scale score, duration of post-traumatic amnesia, rehabilitation exposure, and time since injury, it was not possible to determine whether cognitive reserve independently accounted for the observed MRI distribution.

The complete correspondence between the cognitive-reserve and MRI categories was substantially stronger than would ordinarily be expected in a heterogeneous clinical population. A Phi coefficient of 1.000 represented the maximum possible association between two binary variables. Although such a relationship may occur, it also raises the possibility that the variables were classified using overlapping information, that one category was derived from the other, or that a coding or cross-tabulation error occurred. The original participant-level data and SPSS output should therefore be verified to confirm that cognitive-reserve classification and MRI classification were independently coded and that the reported 2 × 2 cell frequencies were correct.

The absence of variability in the non-affective CRASH category also requires consideration. All participants were classified at the high level, preventing the assessment of any relationship between this domain and the MRI findings. This pattern may reflect a genuine characteristic of the sample, limited discriminatory capacity of the selected threshold, restricted score distribution, or a coding issue. Reporting the continuous CRASH total and domain scores, together with their means, standard deviations, medians, ranges, and internal-consistency estimates, would provide a more informative evaluation than categorical scores alone. Analysing the total score continuously could also reduce information loss associated with dichotomization.

The distinction between structural injury and neuroplasticity is central to the interpretation of these results. The MRI categories used in this study described lesion extent, microbleed burden, brain-volume preservation, and signs of mild atrophy. These findings represented structural injury patterns and did not directly measure adaptive neural reorganization. Neuroplasticity is more appropriately evaluated through repeated structural imaging, functional MRI, diffusion tensor imaging, electrophysiological

measures, or longitudinal changes in cognitive and functional outcomes. Therefore, the present study could not determine whether participants developed new neural pathways, recruited alternative networks, or demonstrated greater longitudinal brain reorganization.

The findings nevertheless had potential clinical relevance. Cognitive-reserve assessment may contribute to a broader understanding of premorbid and contextual factors that influence how patients present after complicated mild TBI. When considered alongside injury severity, clinical examination, neuroimaging, cognitive testing, and psychosocial information, reserve-related measures may support individualized prognosis and rehabilitation planning. However, the present results did not establish that increasing cognitive reserve after injury would reduce existing structural lesions or directly improve recovery. Evidence from cognitive-training and neuromodulation research suggests that rehabilitation may promote adaptive responses after acquired brain injury, but treatment effects require evaluation through prospective interventional designs (8,16,17).

The multicentre recruitment of patients from three tertiary-care hospitals and the focus on complicated mild TBI were strengths of the study. This subgroup is clinically important because patients may demonstrate intracranial abnormalities despite relatively preserved levels of consciousness. The use of a structured cognitive-reserve instrument and the inclusion of MRI findings also permitted simultaneous consideration of reserve classification and structural injury pattern.

Several limitations reduced the certainty and generalizability of the findings. The cross-sectional design prevented assessment of temporal sequence, within-person change, neuroplastic development, or recovery trajectory. Convenience sampling may have introduced selection bias, and the relatively small sample was restricted to adults aged 20–40 years from tertiary hospitals in Lahore. The analysis did not adjust for education, occupation, socioeconomic status, premorbid intelligence, injury mechanism, Glasgow Coma Scale score, lesion location, time since injury, rehabilitation exposure, or other potential confounders. The cognitive-reserve and MRI variables were dichotomized, and the psychometric basis of the applied thresholds was not fully documented. The MRI classification was broad and lacked reported details regarding imaging sequences, lesion quantification, reader blinding, and inter-rater reliability. The MRI and CRASH assessments were also undertaken at different post-injury periods. Finally, the complete separation between the principal variables and the absence of variation in the non-affective domain require verification against the original dataset.

Future studies should use prospective longitudinal designs with larger and more diverse samples. Cognitive reserve should be evaluated using validated continuous measures and established premorbid proxies, while structural injury should be quantified using standardized imaging criteria. Repeated cognitive, functional, structural, and functional-neuroimaging assessments would help distinguish initial injury burden from subsequent neuroplastic adaptation. Multivariable analyses should account for injury severity, lesion characteristics, education, occupation, socioeconomic conditions, rehabilitation exposure, and other clinically relevant confounders. Interventional research may subsequently determine whether cognitive training, enriched rehabilitation, or combined cognitive and physical approaches modify functional recovery across different levels of cognitive reserve.

CONCLUSION

Higher cognitive-reserve classification was strongly associated with a less-pronounced MRI-defined structural injury pattern among adults with complicated mild traumatic brain injury, while lower cognitive reserve corresponded with the more extensive injury category in the analysed sample. These findings suggested a relationship between reserve-related characteristics and structural injury patterns but did not establish causation, protection against traumatic lesions, longitudinal neuroplastic change, or improved recovery. Confirmation through verified participant-level data, standardized imaging measures, continuous cognitive-reserve assessment, adjustment for injury severity and other confounders, and prospective longitudinal research is required.

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