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Orienting network impairment of attention in patients with mild traumatic brain injury

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ABSTRACT

The incomplete understanding of mild traumatic brain injury (MTBI)-related cognitive impairment in the acute stage and the low cognitive needs of patients in the later stage might be the main reasons for the neglect of clinical symptoms in patients with MTBI. Patients often experience attention deficits; however, it is unclear whether these patients suffer from general deficits or selective impairment of the brain attention network. Therefore, we investigated deficits in the attention function of patients with mild brain traumatic injury. Patients (n = 50) and matched healthy controls (n = 49) completed a general neuropsychological background test and the Attention Network Test, which provided an independent assessment of the three attention networks (alerting, orienting, and executive control). We found that patients had significant deficits in the orienting network but none in the alerting and executive control networks. Furthermore, patients' cognitive task scores in attention, memory, and information processing tasks were significantly lower than the scores of the controls. Our results demonstrated that patients with MTBI had selective impairment in the orienting network and extensive cognitive impairments, including those related to general attention, memory, and information processing speed.

1. Introduction

There are approximately 50 million cases of traumatic brain injury (TBI) reported worldwide annually, and it is estimated that more than half of the population will experience one or more TBI in their lifetime [1]. Craniocerebral trauma affects brain function in many ways. Even minor trauma cases can cause long-term pain, insomnia, and cognitive decline [1]. Thus, TBI is a major public health issue.[2]

Conditions such as mild traumatic brain injury (MTBI), chronic traumatic encephalopathy, and concussion syndrome have not been given enough attention, and most patients are prescribed rest and appropriate drugs to relieve pain and sleep disorders. Patients with MTBI not only face long-term physical impairment, but also emotional, behavioral, and cognitive impairments, and most of the clinical symptoms of patients with MTBI disappear within 3–6 months [2,3]. Approximately 15% of patients have long-term post-injury symptoms [4,5]. Current studies mainly focus on exploring the neural mechanism of long-term brain injury in patients with MTBI, but few studies describe the characteristics of cognitive function changes in patients with MTBI in the short term, especially in the acute phase [6]. This is precisely mainly because post-traumatic symptoms of MTBI patients are often non-specific, and the multi-dimensional and multi-level cognitive function of MTBI patients in the acute phase needs to be explored. The possibility of long-term cognitive impairment in MTBI patients may be misestimated. Most of the clinical treatment strategies are a one-size-fits-all treatment policy, and lack of pertinence to the needs and

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[2] ANT, Attention Network Test; HAM-A, Hamilton Anxiety Rating Scale; HAM-D, Hamilton Depression Rating Scale; MoCA, Montreal Cognitive Assessment Test; MTBI, mild traumatic brain injury; RT, reaction time; TBI, traumatic brain injury; WAIS Wechsler Adult Intelligence Scale

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treatment of MTBI patients. New evidence shows that early targeted cognitive rehabilitation can effectively reduce long-term cognitive impairment in patients with MTBI. Therefore, a full understanding of the characteristics of cognitive function changes in patients with acute MTBI is the key for improving the prognosis and quality of life of MTBI.

Attention function, including visual and auditory attention, integrates many cognitive operations and is a prerequisite for other functions. Most previous studies on attention impairment in brain injury are based on the study of attention components, including the intensity of attention intensity, such as continuous attention, and selectivity, such as sustained and divided attention [7,8]. The attention control or execution function can be regarded as a policy allocation system of attention [9]. Current research has confirmed that TBI patients have different components of general attention function damage. However, the findings often focus only on one or two components, while ignoring other general attention components, such as alerting, attention allocation, and response inhibition [10], and most related studies are overly general and their results are contradictory. Some studies even show that patients with MTBI have no attention or executive function deficits [11, 12].

The most commonly used assessment methods in both research and clinical practice (i.e., Trail Making Test, Stroop, WAIS Digit Span) were developed to differentiate populations with and without gross cerebral pathology and do not reflect the contemporary cognitive neuroscience perspective of the brain/behavioral system. There are few studies from the perspective of the attention network, which regard attention function as a whole and deeply understand the changes in attention function in patients with MTBI. According to Posner et al. [13], attention is the process of concentrating psychological activity and is divided into three subnetworks: alerting, orienting, and executive control. Fan et al. [14] designed the Attention Network Test (ANT) to evaluate the different attention networks; this paradigm combines classical cue reaction time (RT) with the flanker task and tests the three attention networks quickly and effectively [14]. The ANT has been widely used in the study of children and people with schizophrenia, attention deficit hyperactivity disorder, Alzheimer's disease, depression, smoking, and cerebellar damage [15–17]. A large number of studies, using ANT and neuroimaging findings, have confirmed the coordination and synchronization of the three networks and that they have relatively independent anatomical structures. The alerting system has been associated with the frontal and parietal regions of the right hemisphere. The brain area involved in orienting function extends from the pulvinar and superior colliculus to the parietal lobe cortex and frontal eye field [18,19]. Executive control of attention is often studied through tasks that involve conflicts, such as various versions of the Stroop task, which activate midline frontal areas (anterior cingulate) and the lateral prefrontal cortex.

Lacking of sensitive and effective evaluation methods resulting in an incomplete understanding of MTBI-related cognitive impairment in the acute stage and the low cognitive needs of patients in the later stage[20] may be the main reasons for the neglect of clinical symptoms in patients with MTBI. MTBI also provides a unique opportunity to examine transient cognitive and emotional disorders and changes in corresponding neural mechanisms. As the core of cognition, the study of attention function is fundamental for us to understand the changes in cognitive function and the potential neural mechanisms in patients with MTBI. In addition, we hope that by thoroughly discussing the damage to the attention network in patients with acute MTBI and using it as a baseline, we can fully understand the changes in attention function in patients with MTBI, which is of great significance for identifying attention function damage in the early stage, and for guiding rapid rehabilitation and cognitive intervention in the later stage. Thus, in this study, we investigated the extent of cognitive deficiency in the attention networks of patients with MTBI. Further, we examined the relationship between the patient's demographic and cognitive deficits, including memory and information processing.

2. Material and methods

2.1. Patients

Patients with MTBI were defined by the diagnostic criteria of MTBI proposed by the American Congress of Rehabilitation Medicine [21] and comprised of patients who had: 1) any loss of consciousness for up to 30 min; 2) any memory loss of events up to 24 h before or after the accident; 3) any change in the mental state at the time of the accident (for example, feeling dizzy, disoriented or confused); 4) focal neurological dysfunction that may or may not has been temporary, but the severity of the injury was less severe (loss of consciousness no more than 30 min; post-traumatic amnesia no more than 24 h; and 30 min later, the Glasgow Coma Scale score did not drop below 13); and 5) no obvious abnormality found in computed tomography or/and head magnetic resonance imaging (MRI) T1 and T2 weighted images after admission. One or more of these items of traumatic brain physiological disorders could be included. A total of 54 patients with MTBI were enrolled, of which 4 patients were not included in the final statistics because of lack of cooperation, severe emotional disorders, and other reasons.

The healthy match control group comprised 49 healthy adults from the physical examination in the same period, whose age and education years were matched with those of the patients with MTBI.

The exclusion criteria were: substance abuse; alcoholism; heart, liver, lung, kidney, and other serious systemic diseases; other mental disorders; and organic encephalopathy.

All participants provided written informed consent. The present study was performed following the Declaration of Helsinki and was approved by the local ethics committee.

2.2. Neuropsychological background tests

All patients completed the neuropsychological test and ANT 5–10 days after injury, with an average of (7.23 ± 1.54) days. The Montreal Cognitive Assessment Test (MoCA) [22] was used to assess general cognitive function. The digital span test (forward and backward), the Stroop color test [23], and the Trail Making Test A were used to measure attention. The auditory verbal learning test [24] was used to measure memory. Trail Making Test B, Stroop word test, Stroop interference test and Verbal fluency test were used to assess information processing and executive function. The Hamilton Anxiety Rating Scale (HAM-A) and Hamilton Depression Rating Scale (HAM-D) tests were used to evaluate anxiety and depression, respectively.

2.3. Attention Network Test (ANT)

Before the experiment, participants were familiarized with the purpose, requirements, and focus points of the test. During the experiment, the eyes of the participants were fixed on a point in the center of the screen and fingers were placed on reaction keys. The participants were required to evaluate the orientation of the target through the reaction keys. Each experiment consisted of five steps: in the first step, the fixation was shown for 400–1600 ms; in the second step, the fixation was presented for 100 ms; following a third fixation period (400 ms), the target and flankers appeared simultaneously until the participant responded with reaction keys pressing; the maximum target display duration was 2700 ms; during the fifth step, the center fixation was shown. The total time of each step was 4000 ms. The focus remained in the center of the screen throughout the experiment. The RT and accuracy of the judgement of the target arrow direction were recorded. The target item, either a left to the right arrow, appeared in the center of the scene, next to the stimulus, creating three possible conditions: the neutral condition, the consistent condition, and the inconsistent condition. The test also contained four cue conditions: no cue, central cue, double cue, and spatial cue. In the no-cue condition, participants were shown the fixation cross for only 100 ms. The central clue condition

included the clues presented in the central fixation of view. Under the condition of double clue, the cue appeared above and below the gaze to warn the participants of the upcoming stimulus array but did not provide any spatial information. In the spatial cue condition, the cue was presented above or below the central fixation point, and the target location was provided to the participants.

One session included 312 experiments, 78 for each interference state and 104 for each target, in which the number of times the target appeared above, below, left, and right from central point were equal. Different experimental conditions were presented randomly. There were 24 exercises with feedback before the actual experiment to familiarize the participants with the experiment. The total test time is approximately 17 min (comprising one 2-minute practice block and three 5-minute formal blocks), plus 3–5 min of rest between each block, for a total of approximately 30 min.

2.4. Attention network efficiency

Attention network efficiency was calculated as described previously Fan et al., [14]. RT was measured as the time between the target arrow appearance and a key press, and the following parameters were calculated:

$$\text{Alertness network} = RT_{no\ cue} - RT_{double\ cue},$$

$$\text{Orienting network} = RT_{central\ cue} - RT_{spatial\ cue},$$

$$\text{Executive control network} = RT_{incongruent} - RT_{congruent}.$$

It should be noted that the higher the alerting and orienting network efficiencies, the stronger the alert and directional efficiencies, respectively, but the higher efficiencies of the executive control, the lower the executive control function. The correct rate was defined as the number of correct trials divided by the total number of trials (312).

2.5. Statistical analyses

The statistical data were analyzed by SPSS 26.0 (IBM Corp., Armonk NY, USA). For the data that did not follow the normal distribution, we used the Mann-Whitney U test (Wilcoxon rank sum test) to analyze the data, and for the data that follow the normal distribution, we used the independent sample T-test to analyze the data. Mann–Whitney U test (Wilcoxon rank sum test) was used to evaluate the differences between the patients and the matched healthy controls. Pearson correlation coefficient and FDR correction were used to evaluate the relationship between networks efficiencies and neuropsychological background test scores. For all tests, the significance level was defined as $P < 0.05$.

3. Results

3.1. Neuropsychological background tests

Our results showed that there were no significant differences between the results of the patients with MTBI in terms of age (35.86 ± 12.08), education level (12.58 ± 2.55), MoCA (25.80 ± 4.02), HAM-D (2.26 ± 1.52), and HAM-A (2.96 ± 1.99) and the healthy controls concerning age (37.84 ± 11.09), education level (11.69 ± 3.10), MoCA (26.02 ± 1.97), HAM-D (2.53 ± 1.26), and HAM-A (3.18 ± 1.35); (all $P > 0.05$) (Table 1). However, patients performed significantly worse in memory tests, immediate recall (7.54 ± 2.10), delayed recall (7.78 ± 2.09), recognition test (7.34 ± 2.51), and information processing tests (Trail Making Test B (114.81 ± 10.90), the Stroop word test (50.61 ± 21.45), Stroop interference test (56.11 ± 27.81) and Verbal fluency test (9.54 ± 0.84)) than did the controls (immediate recall (10.51 ± 1.87), delayed recall (9.51 ± 1.79), recognition test (8.43 ± 2.01), information processing tests (Trail Making Test B (98.94 ± 15.24), Stroop word test (20.36 ± 5.45), Stroop interference test (33.22 ± 8.46) and Verbal fluency test (12.76 ± 1.75)). Moreover, as measured by the general attention test, Wechsler Adult Intelligence Scale (WAIS) Digit Span (forward) (6.16 ± 1.17), WAIS Digit Span (Backward) (4.74 ± 1.03), the Stroop color test (48.03 ± 21.32), and Trail Making Test A (65.97 ± 11.51) results were significantly lower in the patient group than the control groups' WAIS Digit Span (forward) (6.27 ± 1.29), WAIS Digit Span (backward) (5.33 ± 1.09), the Stroop color test (16.05 ± 4.45) and Trail Making Test A tests (56.10 ± 15.27) (all $P < 0.05$).

Table 1
Demographic characteristics and summary of neuropsychological test of patients and healthy controls.

	Patient group (n = 50)	HC group (n = 49)			
	Mean or Count (S. D.)	Mean or Count (S. D.)	Z / t	p	d_{cohen}
Age (years)	35.86 (12.08)	37.84 (11.09)	-0.848	0.399	-0.172
Education (years)	12.58(2.55)	11.69 (3.10)	-1.701	0.089	0.314
HAMD	2.96(1.99)	3.18(1.35)	-0.885	0.376	-0.129
HAMA	2.26(1.52)	2.53(1.26)	-0.886	0.376	-0.193
MoCA	25.80(4.02)	26.02 (1.97)	-0.527	0.598	-0.069
Attention/ concentration					
WAIS Digit Span (forward)	6.16 (1.17)	6.27(1.29)	-0.714	0.476	-0.089
WAIS Digit Span (backward)	4.74(1.03)	5.33(1.09)	-2.890	< 0.01[b]	-0.557
Stroop Color test (sec)	48.03 (21.32)	16.05 (4.45)	-7.216	< 0.01[b]	2.067
Trail Making A (sec)	65.97 (11.51)	56.10 (15.27)	-3.779	< 0.01[b]	0.731
Memory (AVLT)					
Immediate Recall	7.54(2.10)	10.51 (1.87)	-6.130	< 0.01[b]	-1.493
Delayed Recall	7.78(2.09)	9.51(1.79)	-4.414	< 0.01[b]	-0.888
Recognition	7.34(2.51)	8.43(2.01)	-2.537	0.011[a]	-0.479
Information processing and Executive function					
Trail Making B (sec)	114.81 (10.90)	98.94 (15.24)	-5.084	< 0.01[b]	-1.200
Stroop Word test (sec)	50.61 (21.45)	20.36 (5.45)	-7.034	< 0.01[b]	1.924
Stroop Interference test (sec)	56.11 (27.81)	33.22 (8.46)	-5.165	< 0.01[b]	1.109
Verbal fluency	9.54(0.84)	12.76 (1.75)	-8.304	< 0.01[b]	-2.354

Abbreviations: SD, standard deviation; HAMA, Hamilton Anxiety Rating Scale; HAMD, Hamilton Depression Rating Scale; MoCA, Montreal Cognitive Assessment Test; WAIS, Wechsler Adult Intelligence Scale; AVLT, Auditory Verbal Learning Test.
[a] compared to HC group ($p < 0.05$); [b] compared to HC group ($p < 0.01$)

3.2. Attention network efficiency

We found no significant differences in alerting and executive control network efficiencies between patients with MTBI and controls (Table 3). However, there was a significant decrease in the patients' orienting network efficiency (20.14 ± 21.68) when compared to the controls (49.06 ± 23.93) ($t = -6.304$, $P < 0.01$, Cohen $d = -1.267$). In addition, there was a significant difference in the mean RT and accuracy between the patient and the control group ($Z = 1.970$, $P = 0.049$, Cohen $d = 0.456$; $Z = -2.456$, $P = 0.014$, Cohen $d = -0.447$). Fig. 1.

Table 2 and Fig. 2 summarized RT data pooled from correct trials as a function of cue and flanker conditions. The ratio was used to examine specific effects that are not affected by overall RT differences. For each participant, the RT for each subnetwork was divided by the total time taken by the participants. The network ratio scores of the patient group

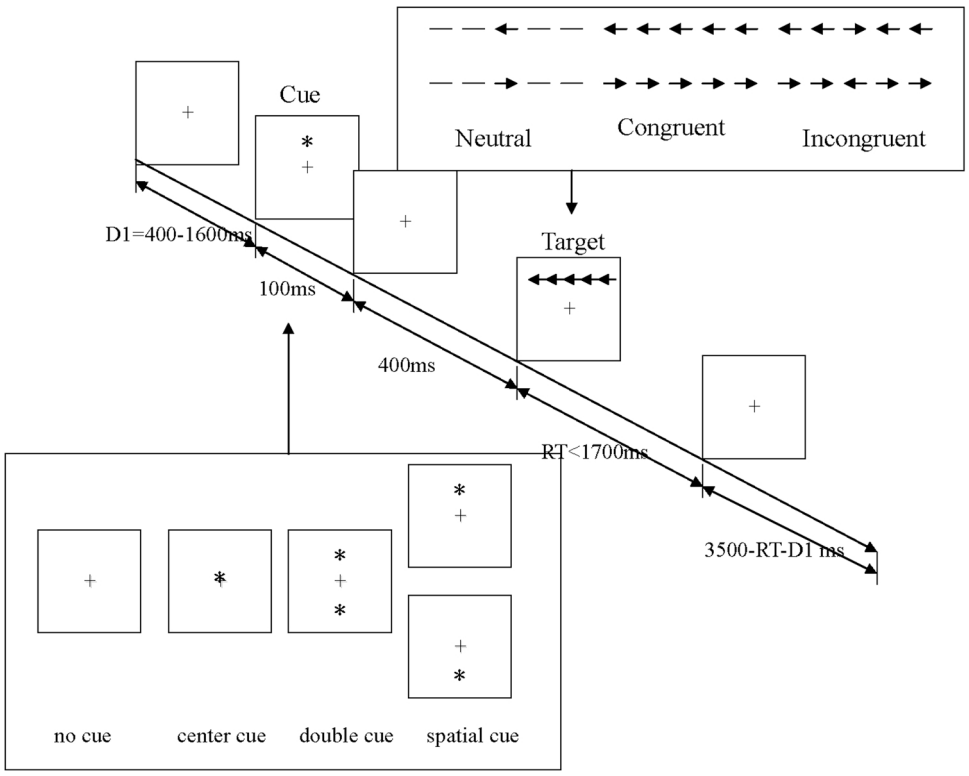

Fig. 1. Schematic of the attention network test (ANT). (a) The four cue conditions. (b) The six stimuli used in the present experiment. (c) An example of the procedure.

Table 2
Each trial mean RTs (msec) and standard deviations of patients.

	Warning Type			
congruency	No-cue	Double-cue	Center-cue	Spatial-cue
congruent	691.78 (104.62)	649.12 (100.48)	664.82 (106.58)	646.06 (99.96)
incongruent	779.20 (86.14)	752.84 (79.45)	753.90 (86.39)	738.43 (93.330)
neural	643.40 (107.40)	595.32 (108.74)	622.66 (112.12)	597.34 (114.63)

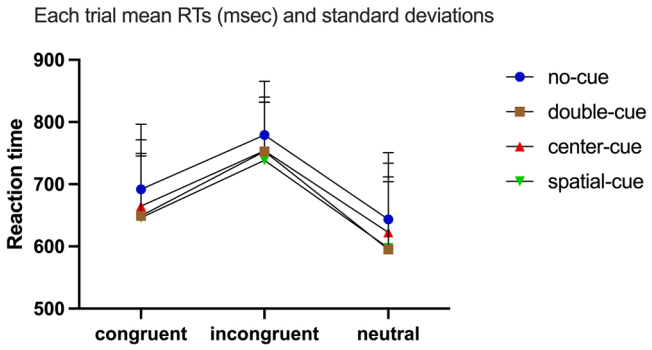

Fig. 2. Results of mean RT from correct trials in the patient group as a function of cue and flanker conditions.

and the control group were shown in Fig. 3. There was also a significant decrease in the patients' orienting network ratio (0.03 ± 0.03) when compared to that of the controls (0.08 ± 0.04) ($Z = -5.536$, $P < 0.01$, Cohen d =−1.416).Table 3.

3.3. Correlation analysis

A correlation analysis revealed that there was no correlation between the three attention networks and the results of neuropsychological background test in patients with MTBI (all $P > 0.05$).

4. Discussion

In this study, we investigated cognitive deficits in patients with MTBI in the acute phase and found extensive cognitive impairment in general attention, memory, and information processing speed, similar to results in other published studies [25,26]. We used the ANT paradigm to further evaluate the attention network and found a selective impairment of the orienting network in patients with MTBI while alerting and executive networks were comparable to controls. In addition, there was no correlation between the three attention networks and the neuropsychological background test.

Orienting network mediate the ability to prioritize sensory input by selecting a channel or location and allow for solving complex problems and high-level cognitive tasks. The orienting network rely on the joint coordination of the dorsal frontoparietal network and ventral frontoparietal network [27,28] and are regulated by top-down and bottom-up signals [29]. TBI in the area of the ventral network is often accompanied by functional impairment of the dorsal network. Damage to the orienting network may manifest in several closely related mechanisms. First is the neuro-metabolism (glutamate release and ion flux, energy crisis, cytoskeletal damage, axonal dysfunction, altered neurotransmission, and cell death) [30], which in acute TBI is closely related to cognitive function decline. Current research suggests that both single and multiple MTBI can cause similar pathophysiological changes in the brain both in the acute and chronic phases. In addition, patients with MTBI often experience attention and distraction issues and repeated processes of cognitive engagement and disengagement can easily consume cognitive reserves, resulting in mental fatigue [31,32]. This can account for the

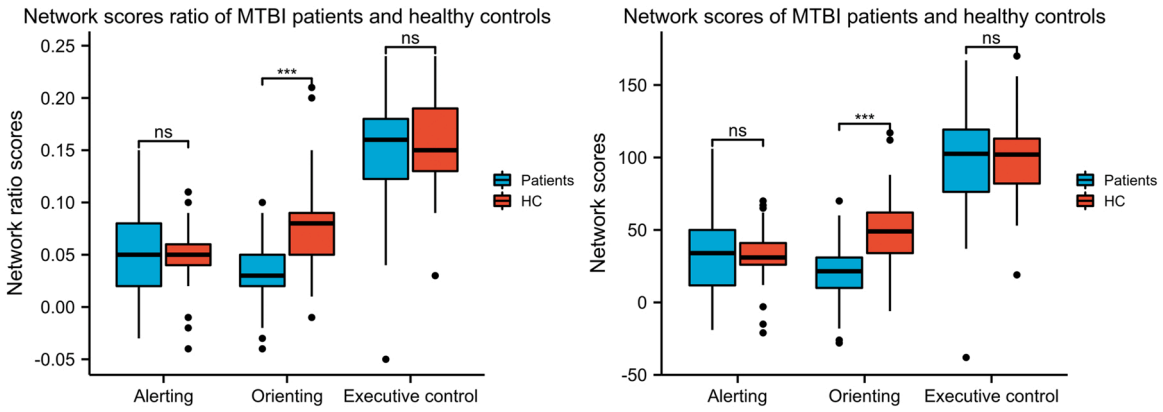

Fig. 3. Network scores and network ratio scores of patients with MTBI and healthy controls [*** indicates $p < 0.01$]. The quartiles (Q3, Q1) are respectively the upper and lower parts of the box, the maximum and minimum observed values of the interval [Q1–1.5*, IQR; Q3 + 1.5* IQR] are respectively the upper and lower parts of the line, and the horizontal lines in the box represent the position of the median. When an observation exceeds the upper and lower ends of the line, it is considered an outlier and marked with a black dot (outliers were included in statistical analysis). IQR, interquartile range; = Q3-Q1.

Table 3
Attention performance of patients and healthy controls.

Performance	Patients group (n = 50)	HC group (n = 49)			
	Mean (S.D.)	Mean (S.D.)	*Z* / *t*	p	d_{cohen}
Alerting	33.16(26.55)	32.63(17.31)	-0.112	0.911	0.024
Ratio	0.05(0.04)	0.05(0.03)	0.070	0.944	< 0.01[b]
Orienting	20.14(21.68)	49.06(23.93)	-6.304	< 0.01[b]	-1.267
Ratio	0.03(0.03)	0.08(0.04)	-5.536	< 0.01[b]	-1.416
Executive	100.12(35.88)	100.96 (28.08)	-0.056	0.955	-0.026
Ratio	0.15(0.05)	0.16(0.04)	-0.714	0.475	-0.221
Mean RT	669.82(76.22)	637.57 (64.53)	-1.970	0.049[a]	0.456
Accuracy (%)	96.90(3.84)	98.20(1.44)	-2.456	0.014[a]	-0.447

The scores of three groups in the table are the derived scores. HC, healthy control; RT, reaction time; SD, standard deviation.
[a] compared to HC group ($p < 0.05$); [b] compared to HC group ($p < 0.01$);

low performance of patients with MTBI in general background tests, but it does not explain the lack of selective impairment of the orienting network selective in patients with MTBI in this study.

Another reason may be the microstructure changes that may occur even in the absence of cell death. Although this study lacks the support of imaging research, it is not difficult to review the literature and find axonal injury after TBI is common and it could be responsible for cognitive impairment associated with MTBI. Blumbergs et al. [33] early confirmed that TBI had an axonal injury, especially in the fornix. Viano et al. [34]also showed that the fornix is obviously susceptible to the stress-strain effect of concussion, and it is a common TBI injury area in moderate to severe TBI. Damage to the integrity of the corticospinal tract has also been indicated [35]. In most situations, the cumulative effect of widespread white matter tract damage across many tracts is likely to be a key factor affecting brain network function, rather than focal damage in a particular location [36]. Similar changes have been found in the white matter of patients with MTBI [37,38]. Cohen et al. [39] showed the global decline of the neuronal marker N-acetylaspartate (NAA), as well as gray (GM) and white matter (WM) atrophy after mild TBI. In addition, the speed of processing is dependent on the integrity of white matter pathways maintaining their optimal inter-connectedness. The decrease in the information processing speed of patients with MTBI in the acute phase in this study also indicated that the integrity of the white matter conduction tract was impaired.

Attention function depends on the cooperation of a wide range of distributed networks, and TBI often selectively damages the higher cognitive functions that require coordination of multiple brain networks [40]. Functional connectivity magnetic resonance imaging data greatly expands our knowledge of the TBI effect on cognitive function and corresponding compensation mechanisms [36]. The integrity and strength of neuronal connections play an important role in behavior research. Ham et al. [41] found abnormal functional connections in the attention network between the anterior island and dorsal cingulate cortex. The decrease in long-distance connections, such as the superior and inferior longitudinal bundles, inferior frontal occipital bundles, cingulate bundles, and fornix was also found in patients with MTBI, consistent with Diffusion Tensor Imaging studies [40,42]. Comparison of MRI for tasks and resting state shows that there are specific extensive structural abnormalities or abnormal connections in the frontoparietal network in patients with TBI [43]. Synchronization of the ventral and dorsal systems can significantly improve the sensitivity of the visual attention system and allow for a faster response to visual stimuli [44]. A wide range of abnormal connections, coupled with the composition of a wide range of orienting network brain regions, give us reasons to believe that orienting networks are also affected. The orienting network impairment in patients with MTBI found in this study may be closely related to neurometabolic disorders, related white matter damage, and changes in the mode of cooperation within or between neural networks.

Although this study found a decline in the speed of executive control and information processing in the general background test, it was not found significant deficit in the alerting and executive control networks in the MTBI group. Previous studies have shown that valid orienting facilitates, and invalid orienting inhibits, conflict processing. There is some brain overlap between orienting and executive control network, including the frontal eye field and the areas near/along the intraparietal sulcus. This gives us reason to believe that orienting network damage will also cause damage to the executive control network. First, due to the difference of the attention network measurement tools from the specific executive function tests, such as the Tower of London, the Wisconsin card task, and the Six Elements Test, which cannot fully represent executive function, there may be different results in the future combined with electrophysiological research. Second, because this study strictly excludes patients with focal brain injury, the patients do not see obvious structure damage as is the case with moderate and severe TBI, still have complete conflict monitoring function, and can compensate for errors to complete the test in time. Finally, coupled with the relatively long stimulus onset asynchrony [45] in the ANT of this study, patients with MTBI made greater efforts to maintain a considerable level of task

performance. This can also reflect the concealment of cognitive impairment in patients with MTBI, especially in the acute stage, and the importance of a comprehensive evaluation.

This study has several limitations. First, because this study examined acute injury, a follow-up is necessary. Second, it would be worthwhile to include patients with non-brain injuries as an additional control group. Although we also used scales, such as HAMA and HAMD to eliminate the effects of anxiety and emotional disorders on cognitive tests, they cannot be avoided completely. Finally, neuroimaging will be necessary to confirm the results of this study since this was purely an observational study. ANT is mainly the study of selective visual attention; whether patients with MTBI also have selective attention network impairment such as auditory channels is a desirable direction for future research.

With the incomplete understanding of mild traumatic brain injury (MTBI)-related cognitive impairment in the acute stage and the low cognitive needs of patients in the later stage, clinicians find it difficult to identify cognitive decline in patients with MTBI. Our study is a step towards understanding the attention deficit disorder in patients with MTBI. Our results showed that patients with MTBI had extensive cognitive impairment in general attention, memory, and information processing speed, confirming there was selective impairment in the orienting network but no significant impairment in the alerting and executive network.

Data Availability

Data will be made available on request.

Acknowledgements

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Declarations of Interest

The authors declare no conflict of interest.

Author contributions

HWC and XGC conceived and designed the study. YYW and YWZ supervised the data, collection initiated the study and drafted the manuscript. XYZ was involved in data collection. KW supervised the whole work and revised the manuscript. All authors read and approved the final manuscript.

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