

Effective factors on linguistic disorder during acute phase following traumatic brain injury in adults

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ABSTRACT

Traumatic brain injury (TBI) has been known to be the leading cause of breakdown and long-term disability in people under 45 years of age. This study highlights the effective factors on post-traumatic (PT) linguistic disorder and relations between linguistic and cognitive function after trauma in adults with acute TBI. A cross-sectional design was employed to study 60 post-TBI hospitalized adults aged 18–65 years. Post-traumatic (PT) linguistic disorder and cognitive deficit after TBI were respectively diagnosed using the Persian Aphasia Test (PAT) and Persian version of Mini-Mental State Examination (MMSE) at discharge. Primary post-resuscitation consciousness level was determined using the Glasgow Coma Scale (GCS). Paraclinical data was obtained by CT scan technique. Multiple logistic regression analysis illustrated that brain injury severity was the first powerful significant predictor of PT linguistic disorder after TBI and frontotemporal lesion was the second. It was also revealed that cognitive function score was significantly correlated with score of each language skill except repetition. Subsequences of TBI are more commonly language dysfunctions that demand cognitive flexibility. Moderate, severe and frontotemporal lesion can increase the risk of processing deficit in linguistic macrostructure production and comprehension. The dissociation risk of cortical and subcortical pathways related to cognitive-linguistic processing due to intracranial lesions can augment possibility of lexical-semantic processing deficit in acute phase which probably contributes to later cognitive-communication disorder.

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1. Introduction

Traumatic brain injury is the major factor of death and chronic disability in people younger than 45 years old in industrialized countries (Bruns & Hauser, 2003) and is the leading cause of death in Iran in 15–45 year-old individuals following road traffic accident (Yousefzadeh et al., 2007). Results of epidemiologic studies in a Trauma Centre in north of Iran disclosed that more than one half of admitted traumatic patients suffer head injury (Hemmati et al., 2009). Of all injuries, TBI particularly affects different aspects of health (Fork et al., 2005; McCathy, Dikmen, Langlois, Selassie, & Horner, 2006). Brain injury survivors demonstrate disabilities in psychosocial, emotional and especially cognitive-linguistic domains, which have negative effects on communicative activities and social-interactive skills (Herry, Phillips, & Crawford, 2006). The cognitive deficit including attention, memory, visual spatial

perception, information processing and abstract reasoning are willingly identified as common sequela of brain injury and as onset of the disorganized language, which is a characteristic of this population (Chapey, 2008, chap. 19). More recently, greater emphasis has been put on disruption of the frontal lobe executive function including initiation, goal setting, planning, and self monitoring, as factors in communication problems (McDonald, Flashman, & Saykin, 2002). The most commonly recognized linguistic deficits after head injury occur in visual naming, verbal fluency, and following complex command (Whelan & Bruce, 2006). It was indeed beheld that after TBI, there is a dysfunction in maintenance of hippocampal Long-Term Potentiation which is supposed to be the physiologic basis for memory formation and consolidation (Sanders, Sick, Perez-Pinzon, Dietrich, & Green, 2000). Empirical models in TBI showed that hippocampus is sensitive to acute apoptotic event (Dietrich, Alonso, & Helley, 1994; Kotapka et al., 1991). It was known that head injury disrupts working memory (WM) function (McDowell, Whyte, & D'Esposito, 1997). WM is also consequential for a wide diversity of cognitive skills, such as mental schemes representation, planning, problem solving and active

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listening (Jonides, 1995). WM is a primary and critical component of all dimensions of cognition. Accordingly, its impairment can hugely disturb daily life activities such as communicative function in this population (Bales, Wagner, Kline, & Dixon, 2009). Although some studies were carried out to investigate the association between locus of lesion and type of aphasia following a non-traumatic injury (Halon, Lux, & Dromerick, 1999; Saygin, Wilson, Dronkers, & Bates, 2004), in the case of PT linguistic disorder thus far, the role of lesion types and other factors for substance TBI severity, plurality and locus of lesion, and neurosurgical intervention remain largely unexplored. Some authors reported that brain-injured patients often had anomia as in Wernicke's aphasia. Separate from these types of aphasic syndromes, other investigators pointed to the high frequency of transcortical sensory aphasia following TBI (Heilman, Safran, & Geschwind, 1971; Levin, Grossman, & Kelly, 1976; Vukovic, 1998). Clinical studies demonstrated that language impairment proceeding TBI had a cognitive-communication nature (Angeleri et al., 2008; Bhatnagar, 2002, chap. 19). Moreover, it is clear that executive dysfunctions after TBI can imperil several aspects of cognition, such as verbal memory (Tremont, Halpert, Javorsky, & Stern, 2000) and visual memory (Ricker, Hillary, & Deluca, 2001). The data provided by Martin and Ayala (2004) suggested that deficit in short-term verbal memory function was correlated with a deficit in word processing. Several earlier studies highlighted a correlation between language skill and problem solving in TBI patients (Baldo et al., 2005; Hjelmquist, 1989). This study focuses on PT linguistic disorder and examination of language profile in adult TBI victims. Apparently, the determination of critical factors on PT linguistic disorder are of high importance for proper diagnosis and early intervention. In order to do so, we characterized PT linguistic disorder among subjects and determined some effective factors on it such as locus and type of lesion and TBI severity. We also scrutinized the relationship between tested general cognitive and linguistic functions (verbal fluency, word discrimination, word repetition, word production, story comprehension, semantic acceptability, and semantic category).

2. Materials methods

2.1. Participants

We designed a cross-sectional study to determine the effective factors on linguistic disorder occurring after TBI. 60 TBI adults (55 males, 5 females; mean age: 32.5 ± 13.1) admitted in Poursina trauma center participated in current project. All subjects were monolingual, Farsi-speaking and right handed. Samples were separated to groups with and without PT linguistic disorder regarding their linguistic profile in PAT. Inclusion criteria were conscious survivors at the time of testing, a favorable outcome according to Glasgow outcome scale (GOS), abnormal findings on neuroimaging during the initial hospitalization, an age range of 18–65 years at the time of injury, having literacy at least 4 years of formal education, and existing post traumatic amnesia (PTA) of 20 min or more. Exclusion criteria for participants included documented history of psychiatric illness, pre-existing speech and language disorders, drug abuse, and TBI, neurological conditions such as brain tumor, stroke, dementia and Parkinson. Blind, deaf and mental retard patients were also deleted. Demographic, clinical and paraclinical data of patients including gender, age when injured, education level, ICU and hospitalization duration, etiologic factor, TBI severity, presence of cognitive deficit after TBI, neurosurgical intervention, side of hemisphere lesion, site and type of lesion were recorded in a questionnaire.

2.2. Instruments and procedure

Severity of TBI was rated on the basis of the primary post-resuscitation GCS (Rengachary & Ellenbogen, 2005, chap. 19). In existing study, severe, moderate and mild TBI were respectively determined by score of 8 or less, 9–12 and 13–15 on the GCS. Functional outcome was estimated by GOS (Rengachary & Ellenbogen, 2005, chap. 19) at discharge and categories 4 and 5 were considered as favorable outcome. Type and location of brain lesions were obtained by CTscan at maximum of 24 h after head trauma. A neuroradiologist reviewed all of the scans independent of the cognitive and linguistic data. The findings were entered on a code sheet that specified the side, anatomic location including involved lobe or area and Pathology including penomosephalus, contusion, concussion, intracranial hemorrhage (ICH), subarachnoid hemorrhage (SAH), subdural hematoma (SDH), epidural hematoma (EDH) of each focal abnormal intensity. In each patient, among multiple lesions, the most intense and wide site was considered as analysable lesion site. A speech and language pathologist screened patients with PT linguistic disorder using PAT. Linguistic functions were measured by verbal fluency, visual naming and story auditory comprehension, descriptive speech, phrase or sentence repetition, and word discrimination tasks. *Verbal fluency* task involves asking patient to generate as many words as possible in one minute beginning with a specific phoneme such as /s/, /p/ called phonological fluency as well as pertaining to a specific category such as animals, fruits, flowers i.e. semantic fluency. This subtest can also be utilized in assessing executive function (Herry et al., 2006). *Descriptive speech* task is used to measure narrative discourse skill. It elicits a narrative discourse using four picture sequence cards that are shown to the patient when asking to generate a complete story based on the actions shown. The examiner recorded descriptive speech and then transcripts it. Velocity and efficiency of narrative discourse production is measured according to guidance of test (Nilipour, 1993). Semantic categories and semantic acceptability subtests of Persian version of Bilingual Aphasia Test (Paradis, 1989) evaluate convergent thinking, divergent thinking, and judicative thought functions (Chapey, 2008, chap. 19) were also employed to assess linguistic functions. The score of every task was transformed to the percentage. Patients' actual cognitive status was appraised using MMSE; the instrument able to screen abnormal mental status systematically. It evaluates several domains of cognitive function: orientation, registration, attention and calculation, recall, language, and visual construction. The maximum score is 30. Cognitive impairment is defined by score of 23 or lower, albeit according to patient's grade of education (Folstein, Folstein, & McHugh, 1975). This scale was standard in 101 Farsi-Speaking dementia patients with literacy at least 4 years of formal education and its reliability was estimated using Chronbach's alpha 0.78. Specificity and sensitivity of scale were accounted 80% and 90% in cut-off point 23 respectively (Foroughan, Jafari, Shirinbayan, Ghaemmagham Farahani, & Rahgozar, 2008). Total linguistic and cognitive examinations were carried out at discharge; length of time of post injury at which participants were tested synchronically with length of hospitalization was averagely 1 week.

2.3. Statistical analyses

By using SPSS version 16.0, we used Fisher Exact test for univariate analysis of the association between PT linguistic disorder and all qualitative variables in our study. Non-parametric Mann–Whitney *U* test and parametric *t*-test were respectively administered to examine the significant difference of quantitative variables with abnormal and normal distribution between post-traumatic aphasic and non-aphasic groups. For determining predictors of linguistic disorder after TBI in multivariate analysis; we applied multiple

logistic regression by Back Ward method. The normality of variables distribution was monitored by Kolmogorov–Smirnov test where abnormal distribution of variables was obtained. Then, Spearman correlation coefficient was undertaken to examine correlation between general cognitive and linguistic skills scores. Otherwise, Pearson's correlation coefficient was used. Hypothesis tests were two-tailed and the significance level in all tests was considered 0.05.

3. Results

The general and clinical characteristics of patients are given in Table 1. According to Fig. 1, most patients with linguistic disorder had poor performance in some linguistic tasks such as verbal fluency (34.35%), story auditory comprehension (38.46%), semantic acceptability (58.74%), semantic category (55.67%) and confronting naming (65.94%). Despite of producing semantically impoverished descriptive speech (43.28%), their speech velocity was near to standard (81.28%) and repetition skill approximately remained intact (81.28%).

Results of univariate analysis to examine the association of PT linguistic disorder with individual and disease-related characteristics indicated that there was a significant association between

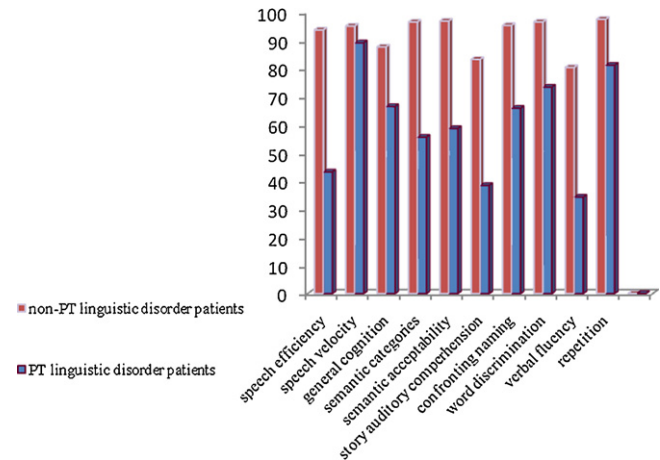


Fig. 1. description of linguistic and general cognition functions in groups with and without PT linguistic disorder.

PT linguistic disorder and the followings; lesion type ($P < 0.01$), lesion sites ($P < 0.02$), brain injury severity ($P < 0.001$), surgical intervention after TBI ($P < 0.002$), and post-traumatic cognitive

Table 1
Demographic, clinical and paraclinical data of patients.

Variables N (%)	Aphasic patients (N = 39)	Non aphasic patients (N = 21)	P-Value	All patients (N = 60)
Age at injury (years) M ± SD	33.7 ± 14.02	30.38 ± 11.2	NS ^a	32.5 ± 31.1
Grade of education (years) M ± SD	8.35 ± 2.76	8.25 ± 2.3	NS	8.41 ± 2.6
ICU duration (days) M ± SD	2.0 ± 2.76	0.06 ± 0.25	<0.001	1.06 ± 2.2
Hospitalization (days) M ± SD	10.64 ± 4.49	6.44 ± 3.22	<0.001	8.61 ± 4.4
Gender, n (%)			NS	
Male	36 (92.3)	19 (90.5)		55 (91.7)
Female	3 (7.7)	2 (9.5)		5 (8.3)
Etiologic factor, n (%)			NS	
Accident	34 (87.3)	13 (63)		47 (78.3)
Falling	4 (10.2)	5 (24)		9 (15)
Other	1 (2.5)	3 (13)		4 (6.7)
TBI severity, n (%)			<0.001	
Mild	6 (15.4)	19 (90.5)		25 (41.7)
Moderate	22 (56.4)	2 (9.5)		24 (40)
Severe	11 (28.2)	0 (0)		11 (18.3)
Cognitive deficit, n (%)			<0.005	
Present	17 (43.6)	1 (4.8)		18 (30)
Absence	22 (56.4)	20 (95.2)		42 (70)
Neurosurgical intervention, n (%)			<0.002	
Present	21 (53.8)	3 (14.3)		24 (40)
Absence	18 (46.2)	18 (85.7)		36 (60)
Hemisphere lesion n (%)			NS ^a	
Left	25 (64.1)	14 (66.7)		39 (65)
Right	8 (20.1)	5 (23.8)		13 (21.7)
Bilateral	6 (15.8)	2 (9.5)		8 (13.3)
Site of lesion n (%)			<0.02	
Frontal	6 (15.3)	5 (33.8)		11 (18.3)
Temporal	5 (12.8)	5 (33.8)		10 (16.6)
Fronto-temporal	13 (33.3)	1 (4.8)		14 (23.3)
Fronto-parietal	1 (2.6)	0 (0)		1 (2.6)
Tempo-parietal	5 (12.8)	7 (33.3)		12 (20)
Parietal	1 (2.6)	2 (9.5)		3 (5)
Fronto-tempo-parietal	8 (20.5)	1 (4.8)		9 (15)
Type of lesion n (%)			<0.01	
Penomosephalus	0 (0)	4 (19)		4 (6.7)
Subarachnoid	4 (10.3)	3 (14.3)		7 (11.5)
Subdural	8 (20.5)	5 (23.8)		13 (21.6)
Epidural	3 (7.7)	5 (23.8)		8 (13.5)
Intracerebral	9 (23.1)	2 (9.5)		11 (18.4)
Contusion	3 (7.7)	1 (4.8)		4 (6.7)
Contusion and hemorrhage	12 (30.7)	1 (4.8)		13 (21.6)

^a Not significant.

deficit ($P < 0.005$). In group with PT linguistic disorder contusion accompanied with hemorrhage (30.7%) and ICH (23.1%), fronto-temporal lesion (33.3%) and frontotempo-parietal lesion (20.5%), moderate injury (56.4%) and severe injury (28.2%), are respectively more common lesion types, lesion sites and injury severity category rather than group without PT linguistic disorder. About 53% of patients with PT linguistic disorder underwent neurosurgery. While percentage of neurosurgery intervention in patients without PT linguistic disorder was 14.3%. Post-traumatic cognitive deficit in group with PT linguistic disorder was 56.4% compared with group without PT linguistic disorder which was 4.8%. No significant association was witnessed between PT linguistic disorder and age ($P < 0.305$), gender ($P < 0.999$), education status ($P < 0.781$), and damaged hemisphere ($P < 0.141$). There was a significant difference regarding the length of stay in ICU ward between the two groups, more dedicating to patients with PT linguistic disorder (mean $\pm$ SD: 2 ± 2.76 Mann–Whitney U test, $Z = -3.89$, $P < 0.001$). In other words, survivors with PT linguistic disorder had more length of stay in ICU ward. They had also a duration of hospitalization significantly more than without PT linguistic ones (mean $\pm$ SD: 10.64 ± 4.49 , $t = 4.12$, $df = 58$, $P < 0.001$); the more the length of hospitalization, the more the possibility of PT linguistic disorder would be. More details are represented in Table 1. According to Table 2, participants who indicated contusion co-occurring with SDH or ICH (OR = 8.21; 95%CI = 1.25–11.51) and fronto-temporal lesion (OR = 10.00; 95%CI = 1.20–16.02) in their neuroimaging findings were more likely at risk of the PT linguistic disorder compared to those with other intracranial lesions. Similarly, subjects who had moderate and severe brain injury ($4 < \text{GCS score} < 12$) at primary post resuscitation (OR = 15.27; 95%CI = 3.73–20.82), undergoing neurosurgery (OR = 4.45; 95%CI = 1.88–7.12), with cognitive deficit after TBI (OR = 7.00; 95%CI = 1.77–9.27) were also significantly more likely at risk of the PT linguistic disorder. We entered all above

Table 2

The results from Fisher Exact analysis for effective variables on PT linguistic.

Variables	P-Value	OR	CI95%
Brain injury severity	0.001	15.27	3.73–20.82
Neurosurgical intervention	0.002	4.45	1.81–7.12
Cognitive deficit	0.005	7.00	1.71–9.27
Hemisphere lesion	NS ^a	0.89	0.29–2.73
Subarachnoid hemorrhage	NS	0.36	0.08–1.546
Subdural hemorrhage	NS	0.43	0.12–1.48
Epidural hemorrhage	NS	0.50	0.09–2.37
Intracerebral hemorrhage	NS	3.70	0.74–7.18
Contusion	NS	0.15	0.01–1.62
Contusion and SDH or ICH	0.022	8.21	1.2–11.51
Frontal lesion	NS	0.70	0.19–2.55
Temporal lesion	NS	0.47	0.11–1.86
Fronto-temporal lesion	0.013	10.00	1.2–16.02
Fronto-parietal lesion	NS	1.55	1.28–1.78
Tempo-parietal lesion	NS	0.29	0.08–1.08
Parietal lesion	NS	0.25	0.02–2.93
Fronto-tempo-parietal lesion	NS	1.37	0.50–4.38

^a Not significant.

significant variables (P -value less than 0.1) in the multiple logistic regression (Table 3). In the final modeling processing step, fronto-temporal lesion and brain injury severity variables remained in the final model, so we can consider them as predictors of PT linguistic disorder. Brain injury severity and Fronto-temporal lesion are the first powerful significant predictor (OR = 10.91; 95%CI = 1.09–19.81) and the second strong significant predictor of PT linguistic disorder successively (OR = 14.77; 95%CI = 2.21–23.09). It was also found that transcortical sensory aphasia (33.3%), Anomia (23.7%), and Wernicke's aphasia (17.9%) were respectively more prevalent than other types in language profile of patients with PT linguistic disorder. Most aphasia type in moderate injury category belonged to fluent aphasia which was characterized as transcortical

Table 3

Results from multiple logistic regression analysis for PT linguistic disorder predictor factors.

Variables	<i>b</i>	SE	df	P-Value	OR	CI 95%
Step 3						
Fronto-temporal lesion	2.39	1.17	1	0.042	10.91	1.09–19.81
Brain injury severity	3.25	0.85	1	0.001	14.77	2.21–23.09

Table 4

Distribution of aphasia types in PT linguistic disorder patients according to severity of TBI.

	Aphasic patients			
	Aphasia type <i>N</i> (%)	Severe TBI patients (<i>N</i> = 11)	Moderate TBI patients (<i>N</i> = 22)	Mild TBI patients (<i>N</i> = 6)
Wernicke's aphasia	7 (17.9)	3 (27.2)	4 (18)	0 (0)
Anomic aphasia	9 (23.7)	2 (18.1)	3 (13.6)	4 (66.6)
Transcortical sensory aphasia	13 (33.3)	3 (27.2)	9 (40)	1 (16.6)
Global aphasia	4 (10.3)	2 (18.1)	2 (9.1)	0 (0)
Broca's aphasia	2 (5.1)	0 (0)	1 (4.5)	1 (16.6)
Motor transcortical aphasia	1 (2.6)	0 (0)	1 (4.5)	0 (0)
Mixed transcortical aphasia	2 (5.1)	1 (9.1)	1 (4.5)	0 (0)
Conduction aphasia	1 (2.6)	0 (0)	1 (4.5)	0 (0)

Table 5The results of the correlation analysis between linguistic functions and general cognitive function.^a

	Repetition	Verbal fluency	Word discrimination	Confronting naming	Semantic categories	Semantic acceptability	General cognition
Repetition							
Verbal fluency	0.234						
Word discrimination	0.662 [*]	0.415 [*]					
Confronting naming	0.471 [*]	0.644 [*]	0.563 [*]				
Semantic categories	0.565 [*]	0.574 [*]	0.754 [*]	0.582 [*]			
Semantic acceptability	0.652 [*]	0.527 [*]	0.632 [*]	0.436 [*]	0.674 [*]		
General cognition	0.558 [*]	0.621 [*]	0.771 [*]	0.537 [*]	0.780 [*]	0.542 [*]	
Story auditory comprehension	0.458 [*]	0.535 [*]	0.632 [*]	0.365 [*]	0.781 [*]	0.658 [*]	0.694 [*]

^a Spearman non-parametric test.^{*} $P < 0.01$.

sensory aphasia in most cases (40%). Anomic was the most aphasia type in mild injury category, nearly 66% (Table 4). The correlation analysis of the investigated linguistic skills during acute phase in patients with PT linguistic disorder directed us to a significant inter-correlation of all these skills except repetition, lacking any correlation with verbal fluency skill. It was also elucidated that cognitive function score was significantly correlated with the score of each individual linguistic function ($P < 0.01$). The results of the correlation analysis of investigated skills in all brain-damaged victims are illustrated in Table 5.

4. Discussion

As a matter of fact, linguistic processing deficit after TBI reflected to executive dysfunction (Davis & Coelho, 2004), WM and declarative memory deficits (Youse & Coello, 2005). Later impairment is a remarkable deficit in TBI that involved prefrontal cortex, limbic system, hippocampus, and mediodorsal and ventroanterior thalamic nuclei functions. These regions control cortical selective engagements for conscious representation of successive linguistic schemas (Cohen & Banich, 2003). Executive function aspects of cognitive process play a critical role in comprehension and production of a message in linguistic macrostructures (Chapey, 2008, chap. 19; Coelho, Grela, Corso, Gamble, & Feinn, 2005). Due to the fact that declarative memory acts in lexical-semantic information encoding and retrieval (Bourgeois & Hickey, 2009), is believed to be the cause of our patients' poor performance in auditory comprehension, descriptive speech, verbal fluency, semantic acceptability, and semantic category tasks. In existing project, linguistic profile of most patients belonged to fluent aphasia in the traditional sense. Namely, semantic paraphasia, low lexical richness, poor speech efficiency, and dissociated and confused statements were observed in their descriptive speech. Their comprehension skill was more impaired, however, the speech rate was approximately normal, and repetition was appeared intact. A research demonstrated that these patients had massively faced difficulty in linking information between sentences and formulating semantically in the narrative discourse. Despite of these dysfunctions, some components of their discourse including speech fluency and syntactic structure appeared intact (Dennis & Barnes, 2000). Less impaired repetition skill in this population results in higher frequency of transcortical sensory aphasia and anomia in our study where repetition skill is perfect. There was also a significant correlation between the cognitive function assessed by MMSE and all investigated linguistic functions in this study. Based on literature, cognitive deficit is predominantly noted in people with auditory comprehension deficit such as those with Wernickes' aphasia (Helm-Estabrooks, 2002). Along with present study, Vukovic et al. discovered that after trauma, auditory comprehension, naming, and verbal fluency dysfunctions were significantly correlated with some cognitive dysfunctions including short and long-term verbal memory, short and long-term visual memory, and reasoning deficits after brain injury in the acute phase (Vukovic, Vucsanovik, & Vukovic, 2008). Since verbal fluency test is used as a known instrument for measuring executive function of the frontal region, it can be more implicated in terms of executive dysfunctions (Spreen & Risser, 2003). Results of fMRI studies during implementing verbal fluency test in healthy individuals showed the activity of bilateral posterior-lateral prefrontal, cingulate gyrus, inferior frontal gyri, and fronto-temporal cortex (Chua et al., 2005). The role of damaged ventrolateral prefrontal cortex in weak performance of TBI patients in variety of tasks, including picture naming and WM was also reported (Thothathiri, Schwartz, & Thompson-Schill, 2010). These reports imply importance of alleged brain regions in executive function components. Herein, frontal lobe was injured alone

in fewer patients with PT linguistic disorder rather than when accompanying with other lobes. Moreover, verbal fluency was defected in these patients. Laterality of lesion was not a significant factor in PT linguistic disorder. Thus, our study ingenerated PT linguistic disorder occurred in either left or right hemisphere lesions. It is recalled that executive function is managed in bilateral prefrontal lobe, therefore, it is predicted that PT linguistic disorder is appeared following either of hemisphere damages. Some studies have suggested a significantly positive correlation between verbal fluency score and three scale scores of verbal intelligence, receptive lexicon and narrative discourse in TBI children (Harvey, Song, Ewing-Cobbos, Chapman, & Mendelssohn, 2001; Mitrushina, Boon, & D'Elia, 2000). Previous researches distinguished the influence of compromised linguistic skill on problem solving and rational reasoning disabilities in brain injury patients (Baldo, Bunge, Wilson, & Dronkers, 2010; Baldo et al., 2005). The association between rational reasoning disability and vital damaged brain areas for language processing such as left middle and superior temporal gyri and left inferior parietal lobule was determined by Baldo et al. (2010). Acute auditory comprehension disorder and poor descriptive speech performance were the other findings of this study. The patients had problems in evoking necessary propositional phrases to convey and comprehend the narrative events, keeping the gist of the narrative speech, sequencing the occurrences of story, summarizing and inferring the central concept. Such deficits are demonstrated as semantic-pragmatic disorder which is probably contributes to later communicative and social disorders in patients with severe and sustained TBI (Davis & Coelho, 2004). Consequently, PT linguistic disorder is mostly related to complex linguistic operations that demand cognitive flexibility in their execution (Bear, Connors, & Paradis, 2007, chap. 20). Results of our study presented widespread and multi-focal brain injuries specifically contusions accompanied ICH and SDH in fronto-temporal region being significantly more prevalent in patients with PT linguistic disorder. Such deteriorations were preferably related to lexical-semantic process impairment. Another study without cognitive-linguistic examination detected contusion and intraparenchymal hemorrhage as the most common lesions following moderate and severe brain trauma (Yousefzadeh, Dehnadi, Ebrahimi, Safaie, & Mohammadi, 2010). Therefore, it is expected that patients with moderate and severe injuries may face high PT linguistic disorder risk. This risk in mild injury was less than moderate and severe injury. High percentage of PT linguistic disorder in moderate injury may be due to great moderate samples included in this survey. Total severe injury brought about PT linguistic disorder. Creation of a sequence of neurochemical events post trauma which in turn leads to secondary short and long-term neuronal and cellular impairments accompanied by cognitive, behavioral and emotional alterations and occurs more seriously in severe brain injury (Margulies, 2002). Findings of this study also illustrate different linguistic outcomes in various severity levels which can justify more linguistic disorder in more injury severity, poor linguistic comprehension and semantically impoverished speech can arise from dissociation of synapse in each or both brain cortical and subcortical pathways of language processing (Sidoros et al., 2009). In subsequence of brain trauma, some changes in central nervous system may also influence on linguistic and cognitive-communication processing. These changes occur on a neurophysiological basis (Stern et al., 2003) and include apoptosis, reduction in the size and number of neurons in some brain regions, decrease in activities of brain regions which are distant from lesion site, diminished efficiency in synaptic contacts and neurochemical changes such as a change in the concentration of some neurotransmitters like Dopamine. Brain imaging studies demonstrated that subsequently, brain trauma brings about disruptions in brain neuroanatomic structures especially in

frontal cortex, prefrontal cortex, fronto-temporal, hippocampus, fronto-limbic and mesocorticolimbic and Nigrostriatal dopaminergic pathways (Baldo & Kelley, 2007; Bales et al., 2009; Tashlykov, Katz, Gazit, & Zohar, 2007). Post-trauma change in concentration of Dopamine disturbs cognitive functions (Ridley, Cummings, Leow-Dyke, & Baker, 2006). Clinical and experimental models reported axonal degeneration striatum (Ding, Yao, Lai, & McAllister, 2001), decrease in PFC glucose metabolism (Fontaine, Azouvi, Remy, & Bussel, 1999) and frontal lobe activity during memory tasks (Ricker et al., 2001) affected by TBI. Reasons of these events are not still entirely vivid (Falo, Reeves, & Phillips, 2008). It seems that these events are related to PT linguistic disorder in communication context. We postulated surgical intervention of damaged mature brain related to PT linguistic disorder and guessed it most probably due to resection of intact brain tissues concomitant with damaged ones. Perhaps the trifle surface difference between intact and damaged tissues is the reason of this phenomenon. Resection of intact brain tissues particularly in crucial neural pathways dealing with linguistic and executive function leads to reduction and deterioration of neurons and synapses involving in language processing. Wong, Wong, Mok, Wong, and Poon (2010) reported a remained cognitive dysfunction post endovascular surgery in patients with aneurismal subarachnoid hemorrhage. Craniectomy after brain injury was determined as a significant factor for poor functional outcome in another study (Kuo et al., 2011). More clarifications certainly necessitate further investigations. Naturally, the more injured the brain, the more stay in hospital and ICU ward would be. Due to the reason that PT linguistic disorder was correlated with brain injury severity, the significant difference of ICU duration and hospitalization between post-traumatic aphasic and non-aphasic patients can be referred to the difference of severity of TBI between two groups.

5. Conclusion

Finally, post-traumatic cognitive deficit causes problem in process of complex linguistic schemes for message comprehension and production of linguistic macrostructures. Undoubtedly, it is obvious that clarifying the role of lesion site, type and neurosurgical intervention on linguistic outcome after TBI requires further and precise investigations. Moreover, the small sample size is considered as a limitation in present study, and it is proposed that a similar research be done with a larger sample size. With respect to the effect of neurosurgical intervention on emersion of aphasia, it is proposed that an extremely accurate neurophysiologic survey be done in addition to neuropsycholinguistic and neuropsychological scopes among operative and non-operative TBI patients with controlled brain injury severity. Additionally, in order to determine the appropriate rehabilitation and medical therapeutic guidelines to remedy PT linguistic disorder, its spontaneous recovery procedure and involved factors should be surveyed.

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