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Patterns of Association between Symptoms and Neuropsychological Deficits in Post Traumatic Syndrome

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Abstract

Post traumatic syndrome/Post concussion syndrome (PCS) is frequently present after head injury. Symptoms are often multiple and patients report different symptoms. Neuropsychological deficits are also not uniform in patients with PCS. This variability of presentation may signify different patterns of symptoms and deficits. We studied the patterns of association between symptoms and neuropsychological deficits through cluster analysis. Ward's method of hierarchical clustering was followed by K-means partitioning method. Five clusters grouped the neuropsychological deficits into core cognitive domains. The core cognitive domains were either at the elementary level of cognitive functioning or at a system level of cognitive functioning. Different symptoms were associated with different deficits in the elementary and system level cognitive domains. Disruption of neural networks arising out of head injury was hypothesized to be the basis for the association between the symptoms and the cognitive deficits.

Key words -

Cognition disorders,
Head injuries,
Memory disorders,
Neuropsychological tests,
Post Concussion Syndrome
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Post traumatic syndrome / Post Concussion Syndrome (PCS) and Neuro psychological deficits following head injury impair social and occupational outcome to a greater extent than physical sequelae [1], [2], [3], [4], [5]. PCS is characterized by a constellation of symptoms, at least one of which is present in the majority of patients soon after head injury, irrespective of the severity of the injury [6]. The constellation of symptoms is treated as an entity, though individual patients report only a few symptoms. Neuropsychological deficits in the areas of attention, memory, information processing, visuo motor speed, concept formation are present in head injured patient [7], [8], [9], [10], [11], [12], [13], [14], [15], [16], [17]. Neuropsychological impairment in the form of information processing deficits is associated with PCS indicating an association between symptoms and deficits. Information processing deficits are postulated to be etiologic to PCS [18], [19], [20], [21], Severe, [15] moderate [11] and even mild [7] injury is associated with the neuro psychological deficits, which can persist for years after the injury [8]. A few attempts have been made to understand the neuropsychological syndrome following head injury. Two patterns of impairment were seen irrespective of severity i.e, attention and information processing deficits on the one hand and memory deficits on the other [19]. However when the sample was restricted to severe head injury, four areas of neuropsychological impairment were identified. These were basic neuropsychological skills, learning and memory, rate of information processing, neuropsychological features of post traumatic personality. The pattern of recovery indicated persistence of a right hemisphere syndrome one year after the head injury [20]. Though the association between PCS and neuropsychological deficits has been established [17], [18], [19], [20], [21], [22], [23], the nature of this relationship has not yet been fully understood. Symptoms and Neuro psychological deficits are diverse in characteristics. An examination of the patterns of their association would be fruitful in understanding the sequelae of head injury. The present study aimed to examine the pattern of association between PCS and neuropsychological deficits in patients with head injury.

Material and Methods

Sample: The sample was drawn from all adult head injured patients (above 18 years of age) referred for neuropsychological assessment over a 8 year period (August 84 - July 92); to the Neuropsychology unit, of the Department of Clinical Psychology, NIMHANS. The patients who complained of at least one symptom of PCS and had completed the neuropsychological assessment were included in the sample. There were 89 patients in the sample. All the patients underwent a neurological examination. Table I indicates that the sample consisted predominantly of males. More than half of the sample had minor head injuries. Concussion was the common type of injury. Contusion and depressed skull fracture were present in about 18% of patients. Most patients did not have neurological deficits and were seen within one year after injury.

Table I - Sample characteristics

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Symptoms: were ascertained by interviewing the patient and an informant. The symptoms were attributed to the head injury and not to other ongoing stresses existing prior to the injury.

Neuropsychological assessment: NIMHANS neuropsychological battery assessed frontal, temporal and parieto-occipital lobe functions [24], [25]. Information processing tests assessed the efficiency of information processing. Patients were tested individually.

Frontal lobe: Attention was assessed by clinical observation. It was rated as inadequate when it could not be aroused easily or when the patient was easily distracted or fatigued. Allocation of voluntary attention was assessed using numerical and pictorial scanning. The numerical scanning consisted of 3 parts, Part I consisted of numbers 1-20 and Parts 2 and 3 of numbers 1-48 respectively. In each part the numbers were arranged randomly but the patient crossed in serial order. The scores were the time taken

to complete parts 1 and 2 and the number deleted in 1 minute in part 3. In the pictorial scanning test the patient described 2 pictures. The description was rated as adequate or inadequate. Ideational fluency test has two parts. Subject recalled objects made of wood and round objects for 2 minutes each. Score was the number of objects recalled summed over the two parts. Abstraction was assessed using the differences and similarities test. Working memory was assessed by the delayed response learning test, wherein counting backwards, serial subtraction and arithmetic problems were given. Performance was scored for time taken to complete and accuracy. Adequacy of motivation and expressive speech were clinically assessed on a nominal scale of adequate or inadequate functioning. Kinetic melody was assessed using the fist and ring, fist and outstretched and Tapping tests [26]. A close relative of the patient was interviewed to assess personality change in terms of apathy, irritability, lability of affect, disinhibition and social inappropriateness.

Parieto-occipital lobe: Visuo spatial perception was assessed using the Perceptual gestalt and spatial relations tests [27]. In the spatial relations test, a target pattern was compared to 6 bigger patterns. The patient identified the bigger pattern identical to the standard. The scores were accuracy and time. Visuo constructive ability was assessed using the block design test of WAPIS [28]. The patient constructed the first five patterns. Number of patterns correctly constructed and average time were the scores. Adequacy of reading, writing and calculation were assessed on a 4 point scale. Focal signs of ideational and ideomotor apraxia, color, visual, object and tactile agnosias, body schema disturbances were scored on nominal scale of present or absent.

Right temporal lobe: Visual integration was assessed using 4 items of the object assembly sub-test of WAPIS [28]. The scores were the mean time taken and the number correctly assembled. Visual memory was assessed using the Benton Visual Retention Test, number of cards correctly reproduced being the score [29]. Visual memory and learning was assessed by giving the complex figure test, on 3 consecutive trials of 10 seconds exposure followed by recall. The fourth trial tested delayed recall after 10 minutes, wherein the number of facts correctly reproduced was the score. This was a modification of the Rey Osterich figure.

Left temporal lobe: Receptive aphasia was tested by the verbal comprehension test, wherein 23 questions were asked orally and the number correctly answered formed the score. The sentence repetition test assessed verbal memory. The subject repeated 20 sentences of increasing complexity. The number of sentences correctly repeated formed the score. Verbal memory and learning was assessed by 3 successive presentations and recall of a short passage. Delayed recall was assessed after 10 minutes. The number of facts correctly reproduced after each presentation was the score.

Information processing tests: The efficiency of information processing was assessed using the information processing test battery developed by Rao, S L in 1984. It consisted of tests to assess simple and choice reaction time, serial processing, parallel processing and focused attention. In all the tests numerical stimuli were delivered on a video monitor from a distance of 2½ meters. Delivery of stimuli and recording to responses were computer controlled. Patients had normal or corrected vision.

Simple reaction time test: A single digit (2) 14 mm high and 9 mm wide was displayed in the center of the screen for 80 milli seconds, preceded by a fixation stimulus by 250 milliseconds. Eighty trials were given with an inter stimulus interval (ISI) of 5 seconds. The first forty trials were given as practice. Patient pressed the top button of a 4 button console. The score was the mean reaction time of the forty trials.

Choice reaction time test: Four digits (2, 3, 5, 6) were individually displayed in the centre of the

screen over 80 trials. Each digit occurred equally in a random order. The stimulus size, duration and ISI were same as in the previous test. Patient pressed a button corresponding to each number. The mean reaction time of correct responses over the 80 trials formed the score.

Serial processing: Serial processing refers to the efficiency of processing stimuli individually. The recognition threshold test was used, with single stimuli. Efficiency of serial processing was assessed using the parameters of both speed and accuracy. The four digits 2, 3, 5, 6 which were 9mm high and 5mm wide were individually displayed in a random order, at one of the following 8 stimulus duration, i.e. 20, 40, 80, 160, 320, 640, 1280 and 2560 milli seconds. There were 80 trials, wherein each stimulus duration occurred on 10 trials, equally distributed among the stimuli. The score consisted of the mean reaction time and accuracy at each of the first five stimulus duration.

Focused attention: This refers to the capacity to select information for processing from among competing stimuli or the capacity to withstand distraction. The patient performed the recognition threshold test, while listening to traffic noise fed through earphones at a comfortable hearing level. The traffic noise served as a distracter. Scoring was similar to the previous test.

Parallel processing: This refers to the efficiency of processing multiple stimuli which are presented simultaneously. The span of apprehension test was used. Digits 1-9 were randomly arranged in 3 rows and displayed for 200 milli seconds, with an ISI of 5 seconds. The patient identified the numbers displayed on each trail over 40 trials. The score was the total number of digits identified over the 40 trials.

Scoring: Whenever a rating was given, the rating was given by the examiner and reviewed by the first author. Inter rater concordance was high. Quantitative scores were compared with the norms in the unit and the presence/absence of a deficit was decided. Ratings and quantitative scores were converted to a nominal scale of 0 = Absent/no deficit and 1 = Present/deficit, for each test.

Results

The symptom profile of the sample (Table II) indicated that most patients had memory and concentration problems as well as headache. The symptoms of irritability, anxiety, intolerance to noise and heaviness of head is present in lesser number of patients. A significant percentage of patients had neuropsychological deficits in the following areas. There was poor memory and learning of visual and verbal material. Working memory and visual scanning were poor. Choice reaction time was slow. Serial processing was either slow or inaccurate. Parallel processing was inaccurate or inadequate. Serial processing under conditions of distraction was either slow or inaccurate indicating that focused attention was poor (Table II).

Table II - Symptoms and neuropsychological deficits in five clusters - (Figures in percentages)

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* $p < 0.05$, ** $p < 0.01$

Association between PCS symptoms and neuropsychological deficits

Cluster analysis using Ward's method found the pattern of associations between the symptoms of PCS

and neuro psychological deficits. The number of cluster was further determined by K-means partitioning on the centroids. The stepwise criterion was used to examine the difference if fusion values between hierarchy levels [30]. The maximum difference was taken as indicating the optimal number of clusters in the data. The symptoms of anxiety and depression were excluded from the cluster analysis, as they were present in less than 10% of patients. The following neuropsychological deficits were included in the cluster analysis as more than 10% of patients had deficits in these areas. These were the deficits of ideational fluency, delayed response learning, scanning, visual integration, visual and verbal memory, visual and verbal memory and learning. Information processing deficits in all the five areas of simple reaction time, choice reaction time, serial processing, parallel processing and focused attention were included. Deficits on the complex figure test on the trials 1 and 2 were excluded as these deficits were present in greater than 90% of the patients. Five clusters were identified. The centroid loading of the 32 variables on each of the five clusters is given in Table II. The centroid loading were subjected to the z test and those variables wherein the centroid loading were significant at $p < .05$ level were chosen to describe a cluster.

The five clusters were named as follows. Cluster I consisted of patients with headache, and deficits in serial processing and parallel processing. These two tasks required information processing at a complex level, because serial processing task required both speed and accuracy, while parallel processing task required accurate and quick processing of multiple stimuli. The cluster was named "Inefficiency of complex information processing". Cluster 2 consisted of patients suffering from symptoms of giddiness, and neuropsychological deficits of ideational fluency, different aspects of delayed response learning, simple levels of scanning, immediate visual memory and delayed recall of visual memory. Working memory is a common component of ideational fluency, delayed response learning and scanning. These tasks require manipulation of information while holding in other information in memory which is a characteristic of the central executive component of working memory. Hence the cluster was named "Working memory impairment". Cluster 3 consisted of patients complaining of intolerance to noise, deficits of delayed response learning, scanning, visual memory, and information processing at a complex level. The deficits indicate inefficiency of processing multiple inputs. Noise, complex levels of information processing and scanning and visual memory require the processing of multiple inputs simultaneously. The cluster was named as "Multiple input processing inefficiency". Cluster 4 consisted of deficits of scanning at the simple and complex levels, visual integration, verbal learning and memory, and all aspects of information processing such as the simple and choice reaction time, serial and parallel processing, and focused attention. As impairment of regulatory function would affect performance on all these tasks, the cluster was named "Regulatory deficit". Cluster 5 consisted of patients with symptoms of fatigue and decreased sleep; as well as deficits of choice reaction time, serial processing and focused attention. The deficits indicate inefficiency in the processing of information presented serially. Hence this cluster was named as "Slowing of processing". Table II indicates that the symptoms were present in conjunction with information processing deficits (clusters I and 5); or in conjunction with other neuropsychological deficits (cluster 2); or in conjunction with neuropsychological and information processing deficits (cluster 3). Neuropsychological deficits present alone without the coexistence of symptoms (cluster 4). Co-existing with these deficits were the deficits of visual learning and memory which were present in over 90% of the patients.

Examination of clinical variables in each cluster revealed slight variations in one or two clinical

variables (Table III). Cluster I had concussion of moderate severity i.e. loss of consciousness between 1-24 hours to a significant degree. Mild injury i.e. loss of consciousness less than 1 hour, along with a short duration after injury (< 3 months) was present in cluster 3. In cluster 2, severe injury was present i.e. loss of consciousness greater than 24 hours upto 1 month. Patients were seen soon after injury i.e. within 6 months. Neurological deficits were significantly present. In cluster 4 contusion and depressed skull fracture were significantly present with severe loss of consciousness (i.e. greater than 1 week upto one month). A significant number of patients were operated. Cluster 5 had a significant number of patients with depressed skull fracture.

Table III - Clinical variables of patients in each cluster

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Table IIIa - Clinical variables of patients in each cluster

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Table IIIb - Clinical variables of patients in each cluster

Table IIIb - Clinical variables of patients in each cluster

Table IIIc - Clinical variables of patients in each cluster

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Note: Nature of Head injury : 0 = Nonconvulsive, 1 = concussion, 2 = contusion, 3 = Depressed Skull Fracture, 4 = Extradural/Subdural Hematoma.

Duration after injury : 1 = (3 months, 2 = (6 months, 3 = < 6 months, 3 = (1 year, 4 = (5 years, 5 = (5 years.

Discussion

An important finding of the study is that different symptoms are associated with different core cognitive deficits. The nature of neuropsychological deficits do not indicate lateralization or localization of brain dysfunction. Instead it indicates dysfunction in the cognitive realm at system level indicative of core deficits. The core cognitive deficits identified in different clusters are not in isolated cognitive domains such as attention or memory. They are in dysfunction which are basic to these cognitive domains such as efficiency of complex information processing, processing of multiple inputs, speed of information processing, working memory and regulation. The dysfunction in core cognitive systems could have arisen due to the diffuse axonal shearing found even in mild head injury as a consequence of the acceleration and deceleration effects. The specificity of association between different symptoms and different core cognitive deficits leads to two hypotheses. The core cognitive deficits are etiologic to the symptoms. This view is in accordance with the accepted view of information processing deficits being of etiologic significance to post concussion syndrome [18]. The other hypothesis is that both the specific symptom and the specific core cognitive deficit have arisen jointly due to a common pattern of brain dysfunction. The absence of localization or lateralization of

brain dysfunction indicates that the brain dysfunction is functional in nature. The structural basis of this functional deficit appears to be diffuse. Such a possibility arises when disruptions of neuronal networks could lead to specific symptoms and associated core cognitive deficits. Disruptions of neural networks arising out of head injury was postulated as a possible etiologic factor in the common occurrence of symptoms and neuropsychological deficits following head injury [31].

The nature of symptom and the nature of the core cognitive deficit is influenced by the injury severity. The severity of injury as well as the duration after injury appear to influence the nature of disruption in neuronal networks. Table III indicates that mild head injury with a short duration after injury is associated with inefficiency in the processing of multiple inputs and symptom of intolerance to noise (Cluster 2). Moderately severe injury is associated with the inefficiency of complex information processing and the symptom of headache (Cluster 2). Severe injury with the prevalence of neurological deficits, as well as a short duration after the injury is associated with cluster 2 wherein there are deficits of working memory and the symptom of giddiness. Severe injury along with a number of patients with open head injury is associated with the cluster 4. Here there are only regulatory deficits. Open head injury is associated with the cluster 5 wherein there are the symptoms of Fatigue and decreased sleep as well as slowing of processing. It is possible that severe head injury or open head injury would lead to greater disruption of neuronal networks because of the greater impact during injury or due to depressed skull fractures respectively. System level disruption seems to occur with these large scale disruptions. The impairments of Working memory, Regulation, and Speed of Processing are examples. The symptoms associated with these deficits are also general in nature such as Giddiness, Fatigue and Decreased Sleep. Mild and Moderate head injury on the other hand are associated with deficits in elementary cognitive domains of information processing. Examples are the inefficiency of processing complex information or multiple inputs. The symptoms associated with mild injury is more specific such as headache or intolerance to noise.

We conclude that severe closed head injury and open head injury are associated with large scale disruption of neuronal networks which result in disruptions of system level core cognitive deficits and general symptoms. Mild to moderate head injury on the other hand leads to more circumscribed disruptions of neuronal networks which result in disruptions of fundamental level core cognitive deficits and specific symptoms. The success of cognitive retraining programs in the treatment of postconcussion syndrome from our experience of 12 years in the neuropsychology unit [32], [33], [34], [35], [36], [37], and those of others [38] indicates that cognitive retraining could be the means by which this disruption of neuronal networks is corrected. The specificity of association between symptoms and neuropsychological deficits points towards the possibility of planning specific cognitive retraining programs for the treatment of specific symptoms.

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