

## **DELUSIONAL DISORDERS AFTER HEAD INJURY**

S. SABHESAN<sup>1</sup>  
M. NATARAJAN<sup>2</sup>

### **SUMMARY**

Delusional disorders have been fundamental to the behaviour problems seen in patients during the early recovery phase of head injury. One hundred and twenty three patients admitted in the Trauma Ward were followed up and the nature and types of the delusions were studied. Their emergence in relation to cognitive recovery and the significance of other factors, such as pre-traumatic personality, alcohol abuse, severity of injury etc., in the genesis of such delusions are presented.

Delusions, as morbid judgements, are marked by the extraordinary conviction, subjective certainty, imperviousness to other experiences and the impossibility of their contents (Jaspers 1963.) Jaspers sums up the three different points of views about the genesis of delusions as follows: 'the first denies that there is any experience at all of delusion proper, all delusions are understandable and secondary; the second believes that lack of critical capacity due to poor intelligence allows delusions to emerge from any kind of experience; the third requires the singular phenomenon of delusional experience'. Delusions as sequelae of head injury form a fascinating area for the study of neurobiological basis of behaviour.

From the management point of view delusions underlie most of the behavioural problems of patients with head injury in the early stages, their contents deciding the subjective distress and the nature of behaviour. Cummings (1985) quotes that majority of families of patients with organic delusions consider the symptom as a major management problem. Relatives of our own patients take it as a definite sign of

loss of mentation and irretrievability of the brain damage (Raja Sakthivel et al. 1985).

Delusions as late sequelae of head injury have been described by many authors. (Fahy et al. 1967; Achte et al. 1969; Merskey and Woodforde 1972). Few authors have described them in the early phases of recovery (Bell 1982; Levin et al. 1982, Bond 1985). The present study is concerned with the phenomenology of delusions following head injury and the clinical factors associated with the emergence of these delusions.

### **Material and Methods**

The study was conducted in the Trauma Ward of the Dept. of Neurosurgery, in Govt. Rajaji Hospital, Madurai between September 1984 and April 1985. A total of the 123 patients with predominant injury to the head were seen from the day of admission to the day of discharge, by the psychiatrist along with the neurosurgery team. During the stay in the hospital the clinical features were assessed by the psychiatrist and information about ward behaviour was obtained from the nursing personnel. Five of these patients

1. Investigator in Psychiatry  
2. Chief Investigator

1. I.C.M.R. Project on Adjustmental problems of Head Injured Patients  
Dept. of Neurosurgery, Govt. Rajaji Hospital, Madurai.

absconded from the wards and the one was discharged at request. The duration of hospital stay in the rest of the patients ranged between 12 and 38 days. The availability of beds was a constraining factor, often leading to premature discharge of patients. They were followed-up as out-patients at weekly intervals to begin with, and seen at least once in three months for one year. During the follow-up details of behavioural problems were obtained from a close relative.

Severity of the head injury was made out by the length of the Post Traumatic Amnesia (PTA), because it reflects both the severity of injury and the kind of trauma the brain receives (Carlsson et al. 1968). PTA is defined as the time taken by the patient to regain his continuous memory (Jennet 1977). Alcohol abuse was defined according to DSM III. Delusions were defined according to Jasper's criteria. There were seventeen patients in whom such delusions were made out through clinical interviews. Rest of the head injured patients seen during this period formed the control group. Clinical data such as age, nature of injury, alcohol abuse and early seizures, were compared with this control group. Since all patients in the deluded group had suffered acceleration-deceleration injuries, a similar group of sixteen consecutive patients with acceleration-deceleration injuries were taken as controls for comparison of PTA.

Chi-Square tests for the discrete variables and 't' test for the continuous variables were the statistical methods used for the comparisons.

### Results

The deluded patients constituted 13.82% of total population and 22.67% of those with closed head injuries. Comparison of the clinical data with those of con-

trol is given in Table 1. The comparison indicates that severity of the injury, its type and history of alcohol abuse are significantly associated with delusional disorders in head injured patients.

Table 1  
Comparison of Clinical variables of deluded patients and the controls

	Deluded	Controls	Statistical Inference
<b>Age 18-35</b>	11	68	
36-50	3	29	$\chi^2 - .632$
51 plus	3	0	Not Sig.
<b>Type of Injury</b>			
Open	0	48	
Closed	17	58	-
<b>Alcohol Abuse</b>			
Present	10@	26	$\chi^2 - 6.749$
Absent	7	80	Sig. at 1%
<b>Early Seizure</b>			
Present	3	4	$\chi^2 - 2.329$
Absent	14	102	Not Sig.
<b>PTA (N-16)</b> in both groups			
Mean	16.688@@	3.938	$t - 3.177$
S.D.	16.491	2.999	Sig at 1%

@ Includes two patients who had stopped alcohol abuse 3 to 4 months prior to injury.

@@ Excludes one patient who developed post-traumatic Korsakoff syndrome.

Lateralization of the neurological damage in closed head injury patients is often inconclusive. Focal seizures indicate the site of damage. Among our patients out of the total, seven with early seizures, two with open injuries (due to assaults) and two with left sided seizures, did not develop delusions. Two with right sided seizures and one with generalized seizures developed delusions. Lateralization based on soft tissue contusion and fracture sites could point out six patients with right sided injury, seven with left sided damage and four with indefinite/extensive site of impact. Those with right sided damage include three patients with affective

syndromes and one with schizophreniform psychosis. Thus, though numerically small and methodologically not very definitive, clinical evidence of lateralization points to the predominance of left sided injury in these patients.

The psychiatric syndromes which could be assigned to these patients were made according to DSM III criteria. There were five patients with (Post-traumatic) delirium, three with organic affective syndrome, four with organic delusional syndromes and four with mixed organic syndromes. The last patient may best be diagnosed as suffering from schizophreniform psychosis. Among the five patients with delirium three were alcohol abusers.

In our study, we have classified the delusions on the basis of chronological evolution during the phase of recovery (Table 2). Taking recovery from PTA as a

Table 2  
Delusional contents and their relation to PTA period

Types of delusions	During PTA	Immediately after PTA	Days/ Weeks after PTA
<b>Delusions of persecution</b>			
Simple elementary type	5(3)		
Complex type	6(2)	2(1)	1
<b>Delusions of infidelity</b>	3(1)		1
<b>Affect-laden delusions</b>			
Grandiose type	2(1)		
Depressive type			1
<b>Delusions of reference</b>	1		
Total	17	2	3

Figures in parenthesis indicate the number of alcohol abusers.

Total number of delusions exceeds the number of patients because of the co-existence of more than one type of delusion.

significant event in the cognitive restoration, they have been classified as those occurring during the PTA period, those during and immediately after recovery and those occurring after restoration of continuous working memory. The nature and psychopathology of these delusions with typical case-histories are given below:

#### *Delusions prior to the end of PTA:*

Delusions of persecution are predominant in this group. Five cases had simple, elementary, transient delusions as part of the post-traumatic delirium. Visual hallucinations were often present specially at night. They were associated with only minimal cognitive restoration. The complex type of delusions occurred a little later in the recovery process, with the patients being conscious and showing normal orientation. The hallucinations were usually auditory and the delusions were more elaborate and permanent. The contents were congruent with the hallucinations, if present.

*Case 1.* Mr. S. aged 22 years had sustained a road traffic accident. From 6th day onwards the patient was frightened about an impending assault. He heard a feminine voice which threatened him about assault and torture in the hands of his relatives. The voice told him frequently that even if he tried to escape, he would be found out and tortured. The hallucinations disappeared early and the delusions had disappeared about 18 days after injury. His PTA was about 22 days.

Towards a better level of cognitive restoration, delusions with affective colouring emerged. The manic changes in our patients with their grandiose delusions resembled in form and content those of primary mania.

*Case 2.* Mr. M. aged 42 years was hit by a lorry while cycling. Following a brief period of delirium, he exhibited elation of mood, volubility, irritability and grandiose delusions. He believed that he was the local ruler. He used to order that doctors should enter the ward only with his permission. They should examine him first and with his permission others could be seen later. He was restless and walked out of the ward. Two weeks later he was traced in a town about 80 km from the hospital. He had been beaten by the local residents because he was ordering that buses should ply only with his permission. He was demanding that the hoteliers should bring food to his place. The manic episode lasted for a total of four weeks and his PTA was about 45 days.

One patient developed delusions of reference. One common feature about all these delusions is that, as is expected, none of them remember any part of them once they recover from their PTA.

*Those immediately following recovery.*

These delusions emerge at a time when there is growing realization of the details of injury. Delusions of persecution weave around the event of head injury, the amnesic period and the behaviour of others during and after the injury.

*Case 3.* Mr. C. 23 years, fell down from a moped and a stranger who admitted him had deposited the valuables with the police out-post. Prior to the injury, he had quarrelled with his tenant over the rent arrears. Recovering after a PTA of 5 days, he denied the fall and attributed the injuries to the assault by the person who was an agent of the tenant. His rivals only wanted to teach him a lesson and were not interested in the jewels. He reasoned out that if he had really suffered such a severe injury (as doctors said), his moped would

not have escaped with just a few scratches.

*Delusions after recovery of continuous memory.*

Certain delusions arise after the cognitive processes have fully recovered and often form a part of the functional psychosis. The course and the symptoms are similar to functional psychosis and the role of head injury is probably in triggering the episode.

*Case 4.* Mr. T. aged 38 years, had 4 weeks of PTA and two weeks later, the relatives were disturbed to note his frequent crying spells, insomnia and fatigue. He had two wives and began to suspect both of them. His jealousy was further aggravated by his lack of libido. In spite of their reassurances, he looked for 'possible stains' in their sarees and used to follow them whenever they went out of the house. But he blamed himself for their behaviour and justified their behaviour. Amidst the depressive self-blaming, the delusional conviction was obvious.

*Case 5.* Mr. M., aged 45 years was hit in a clash. He recovered consciousness within an hour and on the second day began to show psychotic changes. Initially only negativistic, he subsequently developed stupor. After a week's treatment with phenothiazines, he was selectively communicative. He described the auditory hallucinations as voices of his enemies conversing among themselves. The voices were also commanding, threatening or advising him at times. He was deluded that his property had been looted and that his persecutors had infiltrated even the hospital staff to somehow do away with him. Referential ideas, suspicion of other patients and running away marked his clinical picture.

Premorbid factors were also considered. In general no particular type of

personality was found among these patients. Traits of paranoid personality or of cyclothymic personality (in patients with secondary affective disorders) were not evident in their personal histories. One patient with organic affective illness had suffered a prolonged grief following the death of his only daughter. Case 5 had a family history of schizophrenia. Delusions in some patients incorporated certain pre-traumatic life events. Otherwise the only psychological morbidity prevalent among our patients was alcohol abuse. Among eight patients in whom the metabolic sequelae of alcohol abuse could be considered, the aetiological role was possible (if at all) in only one patient. The relatively higher number of alcoholics among those with simple delusions associates its significance atleast in these types. Among other types, with the available small sample size a mere chance association could not be ruled out.

Management of these patients necessitated use of major tranquillizers during the hospital stay. All the patients in first group were started on phenothiazines and/or butyrophenones and the maximum dosage ranged upto 300 mgs/day of chlorpromazine or 15 mgs/day of haloperidol in divided doses. The duration of the treatment ranged between 2 weeks to 7 weeks, with complete disappearance of the delusions at the time of recovery from PTA. During the one year follow-up, none of them had any recurrences of the symptoms. In the second group, both patients continued to harbour the delusions during the follow-up, though they were not unduly suspicious or otherwise psychologically disturbed. Only one patient was maintained on trifluoperazine (ranging from 15 to 30 mgs/day during the course) for about 10 weeks. Later the family was not keen about continuing the drugs. It is difficult to say if the treatment

had any desired affect on the outcome of delusions. The third group was treated along the lines of functional psychoses. Case 4 was treated with antidepressants for about 18 weeks when the treatment was terminated following recovery. Case 5 was treated with chlorpromazine (300 to 500 mgs/day) for about eight months. He discontinued the medicines on his own but during follow up was not found to be disturbed.

### Discussion

The fairly high incidence of delusions after head injury indicates the magnitude of the problem. Running out of the ward by five patients also indicates the degree of management problem posed by these patients.

The psychopathology behind the delusions varies at different times. Those in the early phase arise out of 'cognitive turbulence' when the discriminatory faculty is non-functional and is swayed by the plethora of disturbances in perception, memory, orientation etc. The confused gestalt leads to perception of hostile surroundings and the unstable cognition underlies the impermanence of the delusions. After some improvement, the critical faculty is not differentiated. Thus, the hallucinatory contents determine the theme and content of the delusions. With still further recovery, the delusions occur by distortion of thinking by the pervading affective changes. With better restitution the delusions acquire a greater degree of permanence. Though the cognitive systems have recovered enough to weave an elaborate system of thinking, the memory has not still recovered. The patients do not remember the delusions, after recovery from PTA.

The delusions which occurred around the time of recovery from PTA incorpo-

rated previous life events into the delusional system. Whitty and Zangwill (1977) refer to emergence of systematized paramesias during the post-traumatic confabulatory states. Quoting a case of Russel they explain that such involvement of real life events might lead to false accusations and even legal difficulties. Though it was apparently a logically deducible belief, the contents were inherently unlikely and they were not amenable to reason or contradictory experiences. Whereas in the previous delusions the root cause was a disturbance or non-existence of the critical faculty, in the second type, the critical faculty was put in the service of the delusion and there was a falsification of the past.

In the third type, the delusions appear to be independent of the event of the injury. A constitutional susceptibility due to inherited vulnerability and/or a possible precipitation by the injury could have resulted in the present psychotic episode. The evidence for the same was present, at least in Case 5. In general further the psychoses are from the reintegration of consciousness, more independent they appear to be from the effects of head injury and the delusions take the typology seen in the functional psychoses.

The significance of a few clinical variables of the injury in relation to the emergence of the delusions was noted. Open injuries were never associated with the delusions. If the head injury acts as a non-specific stress, patients in both the open and closed groups should have developed the delusions. An independent factor peculiar to closed injuries, such as diffuse neural dysfunction, might increase the patients' vulnerability to the delusions. The long PTA period in most of the patients and their significantly longer mean PTA compared to the controls, indicate that increasing severity of the injury added to the individual's vulnerability. Evidences

for lateralisation of neural damage favour that delusions were more often associated with left sided damage. Recent studies on organic delusions by Cummings (1985) conclude with similar inferences, but, delusions as a part of affective syndromes were often related to right sided damage. A statistical inference is not possible with the available information. Detailed studies with the recently evolved neuro-imaging techniques at the time of the delusions may help in clarifying such a association. Similarly, alcohol abuse can contribute either by its associated metabolic abnormality or through previous neuropathological changes. The present study hints at the association between them during the early phases, but definite conclusions require a large sample size.

### Conclusion

Though the delusional disorders have been apparently classified arbitrarily aetiology, the degree of permanence, methods of management and the residua after head injury and delusional state justifies the classification. Delusional disorders after head injury have always been stated to occur in constitutionally predisposed individuals. Bond (1985) mentions, ".....perhaps the ease with which individuals develop paranoid delusions in association with disturbed consciousness due to traumatic injury varies, presumably as a result of differences in constitutional susceptibility". A generalized cortical dysfunction as in closed injuries, increased severity of dysfunction as evidenced by long PTA, a left sided involvement (except in those with an affective colouring) and a metabolic aberration as in an alcohol abuser could be factors which tilt the patient's vulnerability towards disordered thinking in the form of delusions.

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