POST TRAUMATIC HYPERACTIVE DELIRIUM

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SUMMARY

Hyperactive delirium following head injury is a common problem during the early recovery phase. Twenty-nine patients who evinced hyperactive delirium were prospectively followed up during their stay in the hospital. Compared with controls, alcohol dependence was significantly more among these patients. Occurrence of delirium was related to the generalized cerebral disturbances due to diffuse damage in acceleration injuries and due to metabolic or post-seizure disturbances in contact injuries. Follow-up of these patients showed that psychiatric problems were more common among them.

Delirium manifests by global impairment of cognitive functions. Post traumatic delirium is the commonest behavior problem during the early recovery period and is of significance in the prediction of the late outcome (Levin et al., 1982). Clouding of consciousness and memory impairment, at least of a limited extent accompany the recovery process following head injury and two types of post traumatic delirious syndromes are discernible against the background of disturbed cognition (Bond, 1985). Lipowski (1975) observed that hypoactive and hyperactive types of delirium reflect decreased or increased activity of the arousal system. The former is related to frontal lobe damage and a poor long-term outcome (Sabhesan and Natarajan, 1988). In contrast, patients with hyperactive delirium evince fluctuating mental phenomena. Physical restraint may even be needed to prevent further injuries and their wandering from the wards (Levin and Grossman, 1978). The management problem due to and the prognostic significance of the syndrome emphasize the practical need for a detailed study of its clinical correlates.

The present study is concerned with the phenomenology of the post traumatic hyperactive delirium and the clinical correlates of the syndrome.

MATERIALS AND METHODS

The study was conducted in the Trauma Ward, Department of Neurosurgery, Govt. Rajaji Hospital, Madurai. A total of 174 patients admitted between September 1984 and June 1985 were followed up by a team of neurosurgeon, psychiatrist, psychologist and social workers, daily during the period of hospitalization. Patients who evinced features of hyperactive delirium during the recovery period were included in the study. The following definitions were used:

i. Post traumatic delirium and alcohol dependency were defined according to DSM III criteria (A.P.A., 1980). As mentioned previously, increased psychomotor activity was a precondition for inclusion.

ii. Severity of injury was defined by the duration of coma. It was mild, moderate, and severe if it took less than one hour, one to six hours and more than six hours respectively, to reach a...
Glasgow Coma Scale score of eight (Teasdale and Jennett, 1974).

iii. Post traumatic amnesia was defined as the lapse of time after the injury when continuous memory was re-established (Jennett, 1977).

iv. Injuries were classified as acceleration injuries and contact injuries, according to the biomechanics of the injury (Gennarelli and Thibault, 1985).

Clinical indices of injury were compared against those of the rest of the head injured patients who did not evince delirium during the early phase. Twentynine patients were included in the hyperactive delirious group and the control included 145 patients. Appropriate statistical tests were used to analyse the data.

RESULTS

The characteristic feature of the delirium was the changing phenomenology which varied from day to day and from hour to hour. Because of the changed sleep-wake rhythm, the patients were described as more disturbing at nights. Global cognitive impairment, memory disturbances and secondary consequences such as disorientation were noticed in all patients. Visual hallucinations accompanied by fear and panic were present in 19 patients, and two of them described short commanding phanomas in addition. One patient complained of tactile hallucinations. Transient persecutory delusions were noted in 17 patients, and in nine patients aggression and violent behaviour were noted. Onset of delirium was one to three days after the injury and was cotemporary with recovery of coma except in those with assault injuries and in those with clinically identifiable delirium tremens. Personal and clinical variables of delirious patients were compared against those of the controls as given in Table 1. Alcohol dependency was significantly associated with the occurrence of hyperactive delirium.

| TABLE-1. Comparison of clinical variables of delirious patients and of controls |
|-------------------------------------|-----------------|-----------------|
|                                    | Delirious patients  | Control group  |
|                                    | (n = 29)               | (n = 145)          |
| Age (in yrs)                       | 14 < 30            | 73 > 30         |
|                                   | 6 to 45            | 46 < 45         |
|                                   | 9 > 45             | 28 < 45        |
|                                    | X² = 2.11, N. S.   |                 |
| History of alcohol dependency     | Present 19          | Absent 10       |
|                                   | 43                   | 102              |
|                                    | X² = 12.03, p < 001 |                 |
| Cause of head injury              | Traffic accident 17 | Assault 5       |
|                                   | 69                   | 46               |
|                                   | Fall 7              | 22               |
|                                    | X² = 2.18, N. S.   |                 |
| Severity of unconsciousness       | Mild 6              | Moderate 7      |
|                                   | 32                   | 28               |
|                                   | Severe 6            | 22               |
|                                    | X² = 0.09, N. S.   |                 |
| Early seizures:                   | Present 4           | Absent 25       |
|                                   | 10                   | 135              |
|                                    | p = 0.19 (Fisher's exact prob.) |
| Early neurological deficits       | Present 7           | Absent 22       |
|                                   | 21                   | 24               |
|                                    | X² = 1.09, N. S.   |                 |

* Only major groups were considered for comparison of cause of injury.

** Severity of coma was compared only in patients with acceleration injuries in whom Glasgow Coma Scale was applicable.

Four patients developed clinically identifiable delirium tremens. Two had suffered assault injuries and two others from acce-
Post traumatic hyperactive delirium

Post-traumatic injuries. Coma was short in these patients. One or two days after recovery from coma, they lapsed into delirium which was typical of delirium tremens. Leaving out these patients, history of alcohol dependence was still found to be significantly related to hyperactive delirium ($X^2 = 7.44$, d.f. = 1, $p < .01$). Among the five patients with assault injuries, two had developed delirium tremens and all other (three) patients suffered from early seizures, following which they developed delirium.

Post-traumatic amnesia was measured in all patients, except those who suffered from contact injuries, delirium tremens, amnestic syndrome and secondary psychoses. Among those in the delirious group, length of PTA ranged from 3 days to 31 days, with a median of 14 days. The mean PTA of these patients (17.78 days) was significantly longer compared to the controls ($t = 2.67$, d.f. = 105, $p < .01$). Duration of delirium ranged from one to eight days, with a median of four days. Duration of delirium did not show significant correlation with either that of coma ($r = .33$, d.f. = 15, N.S.), or that of PTA ($r = .35$, d.f. = 16, N.S.).

Psychiatric syndromes were observed during the subacute phase of recovery in 11 of the delirious patients. Compared to 16 patients among the controls, occurrence of the behaviour problems was significantly more ($X^2 = 11.36$, d.f. = 1, $p < .01$).

Discussion

Phenomenology of the delirium indicated that the clinical features were in no way different from organic delirium due to other causes. Three patients absconded from the wards during this phase in response to perceptual disturbances and transient delusions in the context of disorientation. Only three patients were administered psychopharmacological drugs for the management of violent behaviour (These patients were not included in computations involving duration of delirium). The course of the syndrome was marked by spontaneous self limitation even in the absence of other treatment measures.

Comparisons with the controls indicated that age, cause of injury, seizures and neurological deficits did not significantly correlate with the occurrence of delirium. Severity of coma did not predict either the occurrence or the delirium. Increased length of PTA in these patients pointed to the severe functional disorganization of the brain (Ommaya and Gennarelli, 1974). Lack of significant correlation between the length of PTA and duration of delirium signified that the recovery processes were unrelated.

Alcohol dependency was significantly related even when cases of delirium tremens were excluded. The exclusion was possible only when there was sufficient interval of normalcy between the injury and onset of delirium. In other patients with prolonged traumatic confusional state, the possibility of alcohol withdrawal superimposing over post trauma recovery could not be ruled out. Increased predisposition of alcoholic brain to the development of delirium could be another contributory factor.

Two patients with hyperactive delirium developed post traumatic amnestic syndrome subsequently though classical description of Wernicke-Korsakoff syndrome was one of hypoactive delirium (Victor et al., 1971). Energy requirements of hyperactive state were vastly different from those of normal recovery (Powiertowski, 1971). The additional metabolic strain during the delirium on the thiamine depleted alcoholic could have precipitated the syndrome (Sabbesan et al., 1989).

Global disturbance was central to the pathophysiology of delirium. In acceleration injuries, diffuse injury was associated with global dysfunctions. But, among the five patients with assault injuries, the injury was predominantly focal. Two of them developed delirium tremens and in
the rest seizures preceded the onset of delirium. Among the 59 patients with contact injuries, none developed hyperactive delirium in the absence of metabolic or post-seizure disorganization of cerebral functions.

Increased occurrence of psychiatric consequences in these patients indicated their vulnerability to other behaviour problems. Delirium could be used as a clinical predictor of behaviour sequelae during the later recovery.

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