

Mania following head injury: Case reports and neuropsychological findings

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ABSTRACT - Mania secondary to head injury is reported to be rare. Two cases of florid manic psychoses following head injury are reported along with neurological and neuropsychological investigations. Findings on the Luria Nebraska Neuropsychological Battery (LNNB) suggested residual cognitive deficits, predominantly of right hemisphere. Temporal proximity, clinical neurological findings, EEG changes and deficits on LNNB suggest a causal link between head injury and mania.

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Affective symptoms are frequently seen in cases of head injury (1). However, manic psychosis is rare. Bracken (2) on reviewing the available literature found only 20 cases of manic psychosis following head injury. Reiss et al. (3) using Krauthammer & Klerman's criteria for secondary mania (4) found only four such cases. They reported two more cases.

We are reporting two cases of mania following head injury. They both meet Krauthammer & Klerman's criteria for secondary mania.

Case 1

A 19-year-old student was first admitted as a psychiatric inpatient on November 20, 1985. He had had a fall from his bicycle into a ditch 11 days before admission. Immediately following the fall he had been unconscious for a few hours and was noticed to be bleeding from the nose. He regained consciousness and was observed to be irritable, abusive and wandering restlessly around. He had slept poorly, had no appetite and complained of giddiness.

There was no previous personal or family history of psychiatric illness. His birth was a full term normal delivery and his development had been normal. He was right-handed. His premorbid personality was well adjusted. On examination he was fully conscious and alert. He was globally oriented but distracted. Neurological examination did not reveal any focal deficit.

A mental status examination revealed an irritable individual with an increase in psychomotor activity. He was overtalkative. He expressed ideas of suspicion and persecution. He had auditory hallucinations. He was treated with chlorpromazine (CPZ). After 2 weeks he was transferred to a neurosurgical unit because of persistent vomiting. A routine skull X-ray and CT scan performed there were normal. He was treated conservatively and was referred back to the psychiatry unit. He continued to be treated on CPZ and was discharged after 7 weeks of hospitalization in an improved condition. Poor drug compliance at home resulted in a relapse of symptoms, and he required re-hospitalization within 2 weeks. He was treated for 2 months in hospital and discharged on CPZ in tablet form.

He did not come for subsequent follow-up. One year later he came to the hospital again. He was reported to have been completely normal in the intervening period without any drugs. He related a history of having spent money lavishly, going through a diminished sleep pattern, and behaving irritably and abusively. He was found to be having an acceleration in psychomotor activity. He was distracted. The tone and volume of his speech were increased. He displayed pressure of speech. His mood was elated and on occasion he became irritable. He had grandiose ideation.

An electroencephalogram (EEG), psychometry and a neuropsychological assessment were carried out. His EEG showed bi-temporal spike and slow wave dis-

charges on hyperventilation. This was seen more from the right than left temporal lobe. On Wechsler Adult Performance Intelligence scale (WAPIS) (5) his IQ was 87. The Bender-Gestalt Test (BGT) (6) scores were in the borderline range. The memory for Design Test (MFDT) (7) was normal.

LNNB profile showed mild to moderate deficits on motor, visual, arithmetic, memory and, intellectual scales. Disturbances of bilateral complex motor skills, three dimensional analysis and, spatial organization were present. He showed poor sensory recall and poor abstraction. No deficit was noted on tactile, impressive and expressive speech scales. The deficits were not of much localizing value though they revealed possible residual functional deficits of the right cerebral hemisphere.

He was treated with CPZ 700 mg a day. Later carbamazepine was added in a dose of 600 mg/day. He was discharged after 9 weeks. He was fully recovered at the time of discharge. He was followed up for a month after discharge and since then has been lost to follow-up.

Case 2

A 55-year-old official in a Nepali court was admitted to the same inpatient psychiatric unit on March 26, 1987. He presented with complaints of talking excessively, poor sleep pattern, eating erratically and spending money excessively for 2 months.

Nine months before admission he had been involved in a bus accident. He had sustained a head injury following which he had a brief period of unconsciousness lasting a few minutes. He was taken to a hospital where no focal neurological deficits were found. Four months later he developed severe headaches. There was vomiting and he developed gradual weakness of the right side of his body. His speech became difficult to understand and he had urinary incontinence. A CT scan was performed, which showed a left sub-dural haematoma. It was evacuated surgically and he made an uneventful recovery. He was completely well for the next 2 months after which his psychotic symptoms appeared.

There has been no personal or family history of any psychiatric illness. Premorbidly he was a sociable, affectionate, responsible and well-adjusted individual.

On examination he was fully conscious, alert and oriented. He had increased psychomotor activity. He was voluble with pressure of speech. He had ideas of grandeur and his mood was elated. No perceptual abnormality was noted. He lacked insight. A neurological examination revealed a minimal right upper-motor-neuron facial paresis, right-sided nerve deafness and an equivocal right plantar response.

His EEG showed background asymmetry with mild

suppression of activity on the left side. There were high amplitude sharp and slow wave discharges from the left mid temporal (T_3) area. Hyperventilation revealed spike discharges from the right fronto-temporal area.

On WAPIS, his IQ was 97 and he had scores suggesting organicity on BGT and MFDT. LNNB revealed gross deficits on motor, visual and memory scales. There was general slowness, perseveration and, impairment of complex bilateral movements. He was unable to draw simple geometric figures and had visuo-spatial disturbance and difficulty in three dimensional analysis. A mild deficit in comprehending complex grammatical sentences along with difficulty in mental arithmetic calculations was also present. Both verbal and non-verbal memory were impaired and he had poor abstraction. These findings suggested bilateral involvement of the cerebral hemispheres though comparatively more deficits of the right-hemisphere were noted.

He was treated with 30 mg of haloperidol a day. It was gradually reduced to 15 mg a day at the time of discharge. The patient was discharged after 4 weeks, in an improved state. He has been followed up since discharge and has remained well.

Discussion

The cases reported here have significant closed head injury. There appears to be a temporal and causal link between the head injury and mania. In the first case the psychosis supervened without any euthymic period after head injury. Temporal proximity between head injury and mania, negative past and family history of affective illness support the diagnosis of secondary mania. The abnormal EEG and abnormal LNNB profile even after 1 1/2 years are also corroborative evidences for secondary mania. However, a possibility remains that the head trauma may have precipitated a bipolar disorder in a predisposed patient.

In the second case head injury seems to be a direct causal factor. There is an atypical age of onset, absence of past and family history of affective illness and unequivocal signs of associated brain damage.

On the neuropsychological assessment there were similar deficits in both the cases, though it was much more extensive in Case 2. Both patients had comparatively more right hemispheric dysfunction. Right hemispheric lesions leading to affective illness have been reported by

Jampala & Abrams (9), Forrest (10) and Lishman (1). The cases reported here are similar to those reported by Cohn et al. (11), Deshimaru et al. (12), Sinanan (13), Khanna et al. (14), Reiss et al. (3) and Bracken (2).

The follow-up period in most of the previously reported cases are brief. The 1 1/2 year follow-up in our first case suggests a recurrence of an affective psychosis.

In conclusion, both the cases reported suggest head injury as a possible causal factor in mania. Though in the first case factors like age of onset and subsequent course of illness raise a possibility of the unmasking of a latent affective illness. A long term follow-up of such cases and a matched/controlled study with functional affective psychoses may help in the elucidation of the course of mania following head injury.

References

1. Lishman W A. Brain damage in relation to psychiatric disability after head injury. *Br J Psychiatry* 1968;114:373-410.
2. Bracken P. Mania following head injury. *Br J Psychiatry* 1987;150:690-692.
3. Reiss H, Schwartz C E, Klerman G L. Manic syndrome following head injury: Another form of secondary mania. *J Clin Psychiatry* 1987;48:29-30.
4. Krauthammer C, Klerman G L. Secondary mania. *Arch Gen Psychiatry* 1978;35:1333-1339.
5. Ramalingaswami P. Measurement of intelligence among adult Indians. New Delhi: National Institute of Educational Research and Training, 1975.
6. Bender L. A visual motor gestalt test and its clinical use. American Orthopsychiatric Association, Research Monograph No. 3, 1938.
7. Graham F K, Kendall B S. Memory for designs test: Revised general manual. Perceptual and motor skill 1960;2:147-188.
8. Golden C J, Hammeke T A, Purisch A D. A manual for the Luria Nebraska Neuropsychological Battery. Los Angeles, California: Western Psychological Services, 1980.
9. Jampala V C, Abrams R. Mania secondary to right and left hemisphere damage. *Am J Psychiatry* 1983;140:1197-1199.
10. Forrest D V. Bipolar illness after right hemispherectomy. *Arch Gen Psychiatry* 1982;39:817-819.
11. Cohn C K, Wright J R, Devan R A. Post head trauma syndrome in an adolescent treated with lithium carbonate: Case report. *Dis Nerv System* 1977;38:630-631.
12. Deshimaru M, Miyakawa T, Suzuki T. An autopsy case of head injury with a manic-depressive state. *Brain Nerve (Tokyo)* 1977;29:787-790.
13. Sinanan K. Mania as a sequel to a road traffic accident. *Br J Psychiatry* 1984;144:330-331.
14. Khanna S, Srinath S. Symptomatic mania after head injury. *Can J Psychiatry* 1985;30:236-237.

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