Indian J. Psychiat. (1991), 33(1), 72—75

CASE REPORT: SCONDARY MANIA FOLLOWING ENCEPHALITIS : NEUROPSYCHOLOGICAL FINDINGS AND DIAGNOSTIC ISSUES

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SUMMARY

A 12 year old boy presented with the clinical picture of secondary mania. Neuropsychological examination revealed significant prefrontal disturbance. The overlap in the clinical presentation of secondary mania and frontal lobe syndromes is highlighted. At least some of the so called frontal lobe syndromes can be considered as secondary mania.

The role of neurologic and organic factors in the etiology of manic disorder has received increasing attention in recent times. Krauthammer and Klerman (1978) re-examined all cases reported in the English and French literature on manic symptoms secondary to drugs, infections, neoplasms, epilepsy and metabolic disturbances. Their criteria for diagnosis included : at least one week of predominantly elated or irritable mood, and at least two of hyperactivity, push of speech, flight of ideas, grandiosity, decreased sleep, distractibility and lack of judgement. Cases in which confusional state occurred were included only when the confusion was clearly temporally separated from the manic syndrome. Additional validity criteria were : a close temporal proximity of organic insult, negative premorbid history, predominantly negative family history and late age of onset.

DSM III R restricts the exclusion criteria to "not occurring exclusively during the course of delirium" (American Psychiatric Association, 1986). At present there is no clear reason for setting a fixed cut off point after which the appearance of mania would be considered "independent" from the brain injury (Starkstein, 1988).

Infections have infrequently been implicated in the etiology o secondary mania. Cases reported so far include those secondary to : Influenza (Steinberg et al., 1972), Q fever (Schwartz, 1974). St. Louis Type A encephalitis (Weisert and Hendrie, 1980), Polioencephalomyelitis (Subrahmanya et al., 1981), Neurosyphilis (Hoffman, 1982), Meningococal cryptococosis (Theinhaus and Khosla, 1984).

We report here the clinical and neuropsychological profile of a boy who developed an organic mental disorder (Secondary mania) following encephalitis.

Case Report

A. T., a 12 year old boy developed high grade (39.5°C—40.5°C) fever with headache, a few projectile vomitings, photophobia, and considerable prostration. For 3 days he was in a stupor like state with markedly diminished verbal and motor activity, minimal responsiveness to external stimuli, and double incontinence. Neck rigidity and general body stiffness were present. He was

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diagnosed as suffering from viral meningoencephalitis, and was treated symptomatically in a general hospital for 15 days. Following discharge, he was found to be quieter, not attending to instructions at school, and at times slightly confused.

Over the next 15 days he exhibited increasing behavioral problems. He was predominantly cheerful and hyperactive. Sleep was reduced. There were periods of abusive, assaultive and destructive behaviour, specially at night and resulting mostly from attempts by parents to curb his overactivity. He uncharacteristically insisted on talking in English only and not in his usual language. He often danced and sang before the T. V., at times took on postures associated with marshal arts, and more rarely muttered and laughed to self. He was also observed to joke inappropriately and played silly pranks on others.

During psychiatric consultation he was found to be alert, oriented, and denied having any problems. He was elated, hyperactive and distractible. Talk was limited to brief answers to specific questions. Serial mental status examinations revealed echoreactions, perseveration of speech and cognitive impairment. Investigations including blood counts, urinalysis, X-rays skull and chest, and C. T. scan were normal.

On Luria Nebraska Neuropsychological battery (LNN3) he showed perseveration on rapid sequential hand movement, graphaesthesia items, extemporaneous speech and spontaneous writing. Attentional problems and a tendency to answer impulsively and randomly was seen on spatial orientation items (Clock reading and Raven's items), in counting numbers or days of week backwards and in memorising a series of 7 words. Interference procedures diminished his ability to remember. He had gross problems in arithmetic. While writing he showed echolalic responses (one hundred and fifty eight was first written as 10058, and when asked to correct 1058, before finally correcting it). His ability to reason abstractly was limited, particularly on novel tasks where he had no over learned solution or strategy to depend on. Sequential thinking and planning skills were poor and he was unable to predict his own performance.

Scores on three clinical scales of LNNS were above the critical level. The summary scores showed the pathognomonic and lateralization scores to be within the critical level, though there was more impairment on the right side. There was a sifinificant profile elevation and impairment score was high.

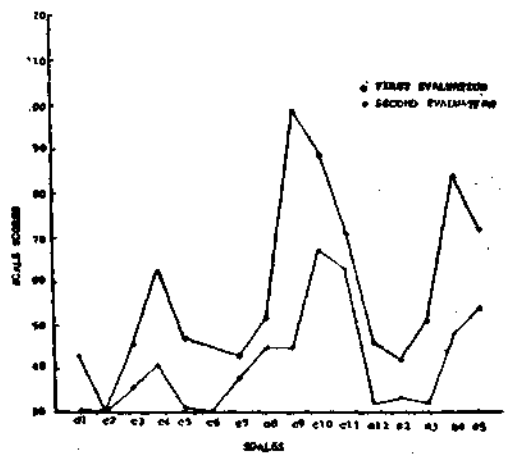

Fig. 1—Clinical and summary scores on Luria Nebraska Neuropsychological battery at first and second evaluation.

A. T. was treated with upto 20 mg of haloperidol per day. As the behavioral problems decreased, cognitive retraining was initiated. Parents worked as cotherapists. All behavioral symptoms remitted within 6 weeks. He showed gradual improvement in cognitive functions over the two months stay in hospital and then at monthly follow ups. Haloperidol was gradually reduced and

stopped. After a gap of 3 months, a reassessment on LNNB revealed some impairment in memory and intelligence, though none of the clinical and summary scales were above the critical level (Figure I). By another 3 months he had made complete recovery and function at premorbid levels.

Discussion

This patient had high grade fever along with headache, vomitting, photophobia, prostration, neck rigidity, general body stiffness, and disturbance in consciousness. In close temporal association he developed a psychiatric syndrome characterized by a predominantly elated mood, hyperactivity, distractibility, decreased sleepand lack of judgement. These features along with a negative past and family history of any psychiatric illness would satisfy the criteria for secondary mania (organic affective disorder, mania type—DSM III R). Encephalitis can be judged as the etiological factor causally related to the production of the organic psychiatric syndrome. However, a viral etiology is merely presumed on account of the general features of the illness. This is true of a large number of cases, particularly sporadic ones, where opportunities for extensive virological investigations are not available (Lishman, 1987).

On the other hand, the presence of inattention, hyperactivity, perseveration, affective dyscontrol (euphoria with outbursts of irritability, inappropriate jokes and silly pranks) and impaired judgement may suggest the diagnosis of a frontal lobe syndrome (Organic personality syndrome DSM III R). A good deal of debate has surrounded the issue o the uniqueness of frontal lobe syndromes, the range of symptoms to be included and the mechanisms by which they come about (Lishman, 1987). The personality of the patient is more profoundly and obviously affected. The present case highlights the overlap in the clinical presentation of secondary mania and frontal lobe syndrome. Lesions of either frontal lobe or limbic system have frequently been involed in the explanation of affective changes after brain lesions, and Starkstein et al. (1988) have recently presented a brief review of frontal lobe involvement in affective symdromes.

On neuropsychological assessement significant organic impairment were found in this case. Though there was no lateralization impairment was more marked on the right. The scores on arithmetic, memory and intelligence were well above the critical level signifying a prefrontal disturbance (Golden, 1981). The frontal areas are thought to be involved in the pathophysiology of primary mania (Taylor and Abrams, 1986; Gruzelier et al., 1988). Starkstein et al. (1988) have suggested that the damage to structures functionally connected to the orbitofrontal cortex, mainly in the right hemisphere, is associated with secondary mania. We suggest that at least some of the so-called frontal lobe syndromes may in fact be secondary manias. In the area of "functional" disorders in our classification systems, the diagnosis of disorders of affect takes precedence over the diagnosis of a personality syndrome, in a hierachical fashion. The same should apply to the realm of organic mental disorders.

Psychiatric disturburbances have frequently been reported in cases with encephalitis, and young children are supposed to be at special risk (Lishman, 1987). However, even the occurrence of manic depressive illness in children has been a moot point (Youngerman and Canino, 1978). It is not surprising then to find only few reports on mania secondary to encephalitis. Shukla et al. (1988) have highlighted significant failure on part of clinicians to recognize organic

factors in mania. Secondary mania may occur at either extremes of ages (Khanna and Borde, 1989). A high degree of suspicion will lead us to identify more cases and help us in in resolving the many complex problems in the field of organic mental dirorders.

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