Behavioural Neurology (1997), **10**, 21–23

CASE REPORT

Manic pseudo-delirium – two case reports

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We wish to report two cases which illustrate a subtype of bipolar affective disorder and suggest that it should be termed Manic Pseudo-Delirium (MP-D) as it incorporates features of both mania and delirium. This will facilitate consistent recognition and characterization since mania remains the primary diagnosis and delirium is not thought to co-exist with functional psychoses. Extensive investigations were done to exclude underlying medical causes for the delirium. Possible explanations for MP-D are put forward for their heuristic value in the hope that further research will improve understanding of the pathophysiology of both mania and delirium.

Keywords: Delirium – Manic disorder – Brain diseases

INTRODUCTION

Delirium describes a clinical syndrome distinguished by a disturbance of consciousness and a reduced ability to maintain attention. It may be caused by a general medical condition, a substance and/or multiple medical conditions and/or substances (World Health Organization, International Classification of Diseases (ICD-10), 1992). In the absence of evidence of a specific organic factor, a nonorganic mental disorder, such as a manic episode, represents an exclusion criterion for the diagnosis of delirium (Diagnostic and Statistical Manual (DSM-IV), American Psychiatric Association, 1994). According to these diagnostic classifications, therefore, delirium may not co-exist with mania. However, in the early nineteenth century, Pinel described a state of mania with delirium in his attempt to classify what he termed as mental derangement (Hunter and McAlpine, 1970). Other reports include that of a condition labelled acute delirious mania by Luther Bell in 1849 (Bond, 1980) and subsequently named Bell's mania (Kraines, 1934).

Bell's mania was later categorized by Kraepelin (1921) as an extreme form of manic depressive psychosis. Leonhard (1961) described an independent category of atypical cycloid psychoses, one of which he termed an excited confusional psychosis; this should be distinguished from some confused states of mania. There may be some doubts about the nature of these early reports since physical and laboratory investigations which were available at the time may not have been able to exclude underlying organic pathology completely.

CASE REPORTS

Case 1. Mrs B is a 52 year old housewife with a long history of psychiatric illness (manic and schizomanic episodes). She presented with a two-week history of deteriorating self-care and signs of hypomania, such as decreased sleep, overactivity, racing thoughts and poor judgement. Compliance with her maintenance medication (trifluoperazine 5 mg daily) had been inconsistent.

She had a positive family history of severe mental illness (possible schizophrenia, details unknown) and was not known to drink alcohol or consume illicit drugs, or to have any chronic physical illnesses. She had an Intelligence Quotient (IQ) score of 80. On admission, physical examination including temperature, was entirely unremarkable. She was found to be very distractible, disorientated in time and place, overactive, perseverative in speech and she was experiencing auditory and visual hallucinations. After two weeks, there was an initial deterioration with incoherence, fluctuating consciousness and faecal incontinence. This persisted for three to four weeks in spite of unremarkable physical and laboratory investigations, including extensive neurological assessment. Full blood count, erythrocyte sedimentation rate, renal and liver function tests, antinuclear antibody, syphilis serology,

fasting and random blood glucose, creatine kinase, lipids, calcium, phosphate and thyroid function tests (T4, T3, TSH) were all within expected limits. Illicit drug screens were negative, urinalysis, chest X-ray and electrocardiography were normal. Computerized scanning (CT) of the brain showed no abnormality. In the acute phase, electroencephalography (EEG) demonstrated diffuse slowing but this returned to normal during the recovery period.

Increasing doses of trifluoperazine did not ameliorate the symptoms but the patient showed marked improvement when the medication was changed to chlorpromazine and haloperidol on an as required basis. She was later discharged in good health after two months on small doses of chlorpromazine and has remained well for over twelve months.

Case 2. Mrs E is a 58 year old divorced and retired nurse. She, too, had a long history of psychiatric illness (bipolar disorder). She was also diabetic (controlled on glibenclamide 5 mg daily) and hypothyroid, though well maintained on thyroxine. She had stopped taking her psychiatric medication (lithium carbonate) approximately four months prior to admission. She presented with a two-week history of deteriorating self-care and signs of hypomania. These included overactivity, decreased sleep, grandiose religious ideas and complex visual illusions and hallucinations, especially at night and sometimes accompanied by incontinence. There was a positive family history of affective disorder but no known alcohol or illicit drug consumption by the patient. Clinical examination revealed no abnormalities.

No abnormalities were discovered in spite of exhaustive physical and laboratory investigations, as had applied to Case 1. In particular, a range of thyroid function tests was carried out; these included challenge tests and they indicated that she was receiving optimal replacement therapy: electrolyte and renal function tests were all normal while fasting, postprandial and random blood glucose tests showed that her diabetes was well controlled. CT and Magnetic Resonance Imaging (MRI) of the brain also revealed no abnormalities. EEG investigation exhibited diffuse slowing with poorly formed alpha rhythm during the acute phase of the presentation which returned to normal when she recovered.

Her symptoms of delirium, such as fluctuating consciousness, visual hallucinations, disorientation, confusion and incontinence, lasted six weeks. Dramatic improvement in this case was associated with the use of sulpiride, an atypical neuroleptic which is a selective dopamine antagonist, at a dose of 400 mg twice daily. This replaced the initial selection of phenothiazines and butyrophenones. She was discharged after ten weeks in good health on sulpiride, lithium carbonate, glibenclamide and thyroxine and has remained well for over twelve months.

DISCUSSION

The cases described illustrate features of both mania and delirium. Both exhibited diffuse abnormalities on EEG testing in the acute phases of their illness which improved in parallel with their mental state. Both could be described as suffering from acute delirious mania, initially defined as a time-limited illness marked by sudden onset, severe insomnia, loss of appetite, paranoia, disorientation, emotional lability and bizarre hallucinations and delusions (Kraines, 1934; Bond, 1980). The modern organic concept of delirium associates it with infection, intoxication or physical disease (ICD-10, 1992; DSM-IV, 1994) but it appears that manic illness may also present with symptoms of delirium. In Case 2, the presence of known medical disorders was a likely aetiological factor but extensive medical investigation did not support this. Thyroid function was exhaustively reviewed in an attempt to identify any syndromes that may have contributed to the symptoms of delirium. In addition, review of the medical notes in both cases revealed similar, self-limiting and less severe episodes in the past.

Aggressive neuroleptic medication seemed to aggravate the condition initially although, in Case 1, this was eventually therapeutic. The anticholinergic effects of neuroleptic medication may be implicated (Ital and Fink, 1966) or perhaps these sequelae may also be due to the neurotransmitter interactions indirectly triggered by antipsychotic medication (Tune *et al.*, 1982; Rovner *et al.*, 1988). Delirium is generally regarded as a condition that results from diffuse brain dysfunction. Confusion, disorientation and impaired reality testing are cardinal features and there are disruptions in the sleep-wake cycle and psychomotor activity (Lipowski, 1987; Taylor and Lewis, 1993). However, focal right hemisphere lesions have been found to cause subacute agitated delirious states (Krasucki and Gaviria, 1994) in addition to enduring manic and hypomanic disturbances of mood (David and Cutting, 1990). Right hemisphere hyperfunction has also been postulated in hypomania and mania (Robinson *et al.*, 1988). No lateralized structural damage to the brain was detected in either case with brain imaging, yet manic disorders share some of the features of delirium listed above and may even share some underlying pathophysiological processes. Furthermore, at the cognitive level, manic

subjects exhibit some of the same disruptions in vigilance that are the hallmark of delirium (Van Sweden, 1986).

Nevertheless, a presentation akin to delirium occurring in mania is not common, although it may be underdiagnosed. It is likely that certain factors predispose to this presentation, such as increasing age (both our cases were in their fifties) and premorbid low IQ (Case 1), suggesting that reduced cognitive reserve may also be implicated as it is in depressive pseudodementia (Mahendra, 1985) which could be viewed as an analogue of MP-D. Co-existing metabolic disorders, as in Case 2, are also likely to be relevant even if they appear to be under control. Finally, anticholinergic activity of medication has been implicated as a cause of delirium in a number of clinical groups, including the elderly and schizophrenic patients (Tune *et al.*, 1982; Rovner *et al.*, 1988). Phenothiazines have considerable anticholinergic potential, so they are prime suspects in causing confusional states, perhaps exacerbating the delirium in Case 2. Clinicians should be wary of diagnosing MP-D where such agents are employed but not all cases can be explained in this way.

In summary, we recommend description of this clinical entity as MP-D so that clinicians may recognize the primacy of mania in this presentation. This diagnosis should not preclude exhaustive investigations for demonstrable organic pathology (hence the qualifier – pseudo). Features of this subtype of mania seem to include: 1. history of manic depressive illness; 2. prominent manic symptoms; 3. signs and symptoms of delirium; 4. family history of mental illness; 5. absence of physical causes of delirium; 6. faecal incontinence. In addition, there is a tendency for the disorder to recur but with full resolution if the florid illness can be managed with appropriate supportive care, in which selective dopamine antagonists may play an important role. Dysfunction of the right hemisphere may provide a plausible unifying hypothesis.

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(Received 6 October 1995; accepted 28 November 1996).