LOCALIZED RIGHT CEREBRAL HEMISPHERE DYSFUNCTION, BEREAVEMENT AND SECONDARY MANIA

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Abstract: The authors report on a fully right-handed man without prior psychiatric history who developed mania secondary to vascular lesion in the right hemisphere immediately after bereavement of his wife. From literature consideration, it is suggested that bereavement is an effective trigger of mania and that right hemisphere dysfunction provides vulnerability for this effect. This case raises important question for both organic and psychological theories of causation of bipolar affective illness.

Index Terms

secondary mania, right cerebral dysfunction, bereavement, life events

CASE REPORT

Mr. A, a 61-year-old, right-handed man, had been in good health until the fall of 1991 when he suffered a cerebral infarction. Evidence of a right hemisphere infarction was obtained from CT and MRI (Fig. 1). An EEG was consistent with residual focal right hemisphere lesion (Fig. 2). He manifested a left hemiparesis and dysarthria, and began to walk with a stick.

In November 1994 Mr. A at age 60 was admitted to Kokubu Hospital for psychiatric care after a 10-day history of irritable mood and constant walking activity immediately after his wife's funeral. His wife had died of colon cancer and he had been chief mourner at the funeral. His sleep time had become shortened and fragmented. His speech was described by relatives as loquacious, loud, grandiose, and tangential. A mental status examination revealed that Mr. A was elated and irritable and had abortive episodes of tearfulness, with pressured speech and flight of ideas. His consiciousness level was not disturbed. He was diagnosed as suffering from manic-depressive illness, manic type, and lithium carbonate treatment was begun and bifemelane hydrochloride was administered. Since Mr. A did not respond to lithium carbonate treatment, lithium carbonate was discontinued after 4 weeks of administration, at which time his lithium level was 1.04 mEq/Liter. However, his symptoms abated 2 months later when he became euthymic. Mr. A has been symptom free in Kokubu Hospital until November of the following year, and he is waiting to move into a special nursing care home.

While he was euthymic, WAIS revealed verbal IQ 77, performance IQ 77 and total IQ 71.

Mr. A's episode of psychopathology met DSM-IV[1] criteria for manic episode. Despite

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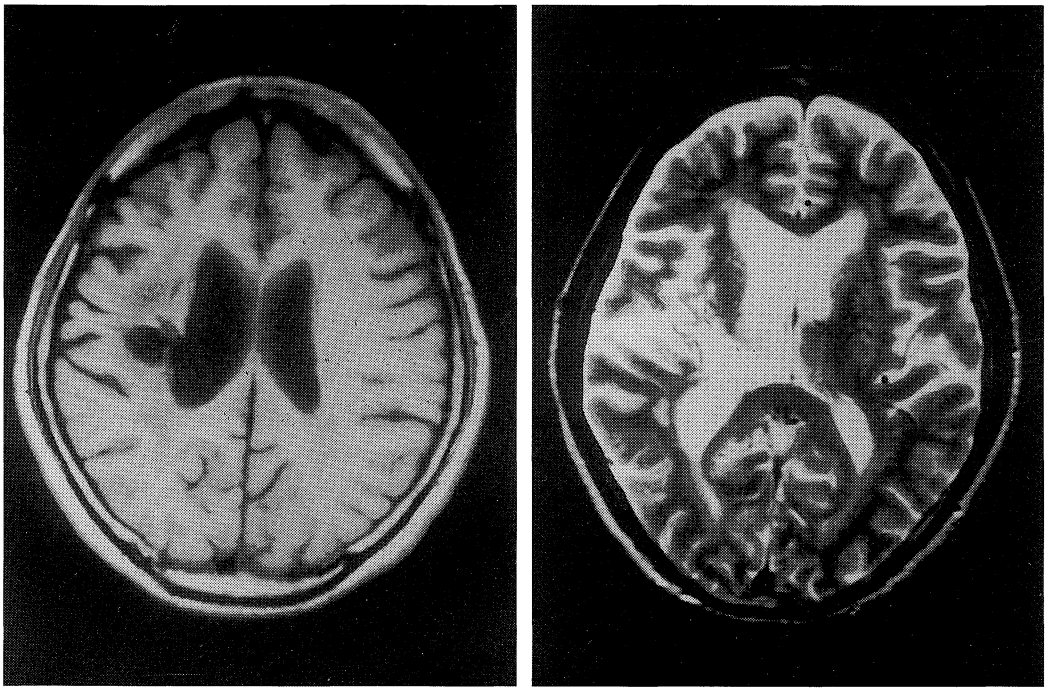

Fig. 1. MRI shows spacious lesion (T1 : right, T2 : left) in centrum semioval, corona radiata area, putamen and thalamus in right hemisphere.

extensive interviewing of Mr. A as well as many family members, we could find no history of affective disorder in him or his family.

DISCUSSION

We believe Mr. A's mania is not coincidental to his cerebral dysfunction. Although there are no infallible guidelines for determining whether the relationship between mood disturbance and general medical condition is etiological, several considerations provide guidelines in this area. One consideration is the presence of a temporal association between the onset, exacerbation, or remission of the general medical condition and mood disturbance. A second consideration is the presence of features that are atypical of primary mood disorders (e.g. atypical age at onset or course or absence of family history). The atypical onset of mania at age 60 without a personal or family history of affective disorder is suggestive of a causal relationship[1,2]. Mr. A also met Krauthammer and Klerman's operational criteria for secondary mania[1], and met the criteria for mood disorder due to right cerebral infarction, with manic features, on DSM-IV[3].

The frequent occurrence of depression after brain injury has been reported in the literature for many years[4-7]. From previous literature concerning post-stroke mood disorders, there have been consistently significant associations between left anterior brain injury and major depression[8]. In contrast to this frequent occurrence of depression after ischemic brain injury, mania after brain damage has been only rarely reported[9]. These anecdotal case reports have suggested that socondary manic patients are older than "primary" manic patients[1], have low frequency of positive familial history[1], and have lesions primarily within the right

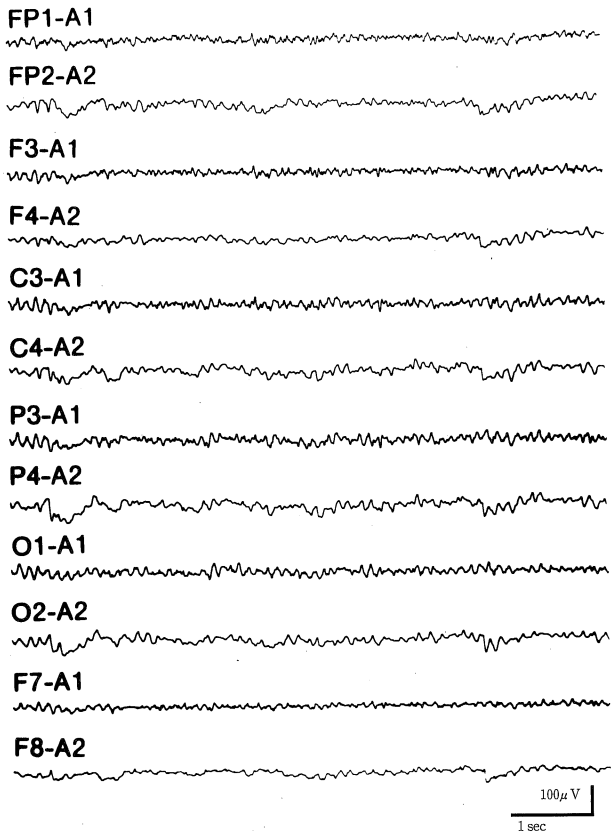

Fig. 2. EEG shows irregular low voltage slow waves in right hemisphere, esp. parietal and occipital areas, compared with the other side.

hemisphere[10]. These suggestions have never been confirmed since Jampala and Abrams[11] presented a case of a 52-year-old fully left-handed man without prior psychiatric history who developed mania secondary to vascular lesion of the left hemisphere, and discounted the role of the location of the lesion in the pathogenesis of secondary mania. Robinson et al[9] compared a consecutive series of secondary mania patients who followed focal brain lesion with a consecutive series of patients with post-stroke major depression and patients without affective disturbance, and then demonstrated that mania after brain injury was strongly associated with lesions involving areas of the right hemisphere that were connected with the limbic system, whereas post-stroke major depression was more commonly associated with left hemisphere lesions involving mainly anterior cortical subcortical lesions.

How might the relatively rare occurrence of secondary mania be explained? The fact that mania is much less frequent than depression after brain injury might suggest that another variable (or variables) besides the side on which the lesion occurs is involved. Robinson et al also hypothesized that either a genetic loading or a pre-existing subcortical atrophy might play an etiological role, at least in the subgroup of patients with secondary mania[9].

In addition, although right hemisphere lesions are significantly associated with secondary

mania, it should be pointed out that a life event stress, "bereavement," appeared to be a crucial factor in the development of post-lesion affective syndrome in this case. The proximity of the event and the onset of illness limits doubts which might be cast on causal association. Reports of manic illnesses closely following emotional events are remarkably scarce. While the connections between life events and schizophrenia[12] and depression[13] have been thoroughly investigated, and causal models proposed[14], life events as precipitants of mania have been little discussed. Reports of specific cases showing an association between bereavement and mania are very uncommon.

In their reviews of psychiatric sequealae of bereavement, neither Parkes[15] nor Clayton[16] mention mania. Scotti and Scotti[17] reported three cases, two of whom had preceding histories of diagnosed bipolar affective illness. Rickarby[18] reported four cases of mania following bereavement, but only one bereavement was contiguous, and in the other three cases, the illness occurred between two and 12 years after the bereavement. Ambelas[19] reported five cases in which bereavement had occurred in the preceding four weeks, three of these having had no previous history of illness. Rosenman[20] reported a case in which bereavement was contiguous and it was a trigger from preexisting depression to mania.

In each of the five cases in which mania followed bereavement, Ambelas[19] found that admission occurred at least one week after the bereavement. As three of these five cases had no previous history of affective illness, it was suggested that such losses are especially potent stressors, but require some days to be effective. Post et al[21] found that patients with very rapid onset of mania had a history of more previous episodes of affective illness than those who became manic more gradually. From this or previous findings, Stoddard et al[22] hypothesized (by analogy with on-off phenomena in Parkinson's disease) that repeated episodes of illness established a facilitated pathway by which rapid changes in mood could occur, and that repeated episodes of affective illness sensitized the patient so that environmental effects might become more effective triggers of mood change. The case reported here suggests that bereavement was an effective trigger of mania, but that right hemisphere dysfunction provided vulnerability for the effect.

The paradoxical nature of the response-that an event which should produce grief produces mania-is consistent with other retrospective examinations or event-related mania. However, the paradox of this manic response remains intriguing and raises important questions for both biological or organic and psychological theories of causation of bipolar affective illness.

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