SHORT REPORT

Seasonal cyclothymia to seasonal bipolar affective disorder: a double switch after stroke

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Abstract

The appearance of bipolar affective disorder after stroke depends on the presence of two factors: a predisposing factor of either genetic loading or subcortical atrophy, and a lesion of specific corticolimbic pathways involving the right hemisphere. Whether cyclothymia and seasonal affective disorder further predispose to post-stroke affective disorder is not clear. A case is described which highlights these issues. The aetiological factors, pathophysiology, and diagnosis are discussed.

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Secondary mania is thought to be a rare consequence of stroke and is only reported anecdotally in the literature.¹ Although the incidence of bipolar affective disorder decreases with age,² the mortality rate for late onset bipolar affective disorder exceeds the community base rate for this age group³ and for geriatric depressed patients.⁴ Poststroke mania is thought to depend on the presence of two factors⁵: (a) a predisposing factor of either genetic loading or subcortical atrophy, and (b) a lesion of specific corticolimbic pathways involving the right hemisphere. The infrequency of the combination of these factors may explain the relative rarity of poststroke mania.

We report a case of poststroke bipolar affective disorder, in which symptoms "converted" to syndrome and their respective seasonal patterns "switched". This has not been described previously.

Case history

An 88 year old retired man was referred from a home for elderly people with a one week history of sudden onset of grandiose speech, sexual advances towards female residents, undue cheerfulness, increased socialisation, pressure of speech, and decreased sleep. He had not received treatment for any physical or mental illness until 1989 when he experienced a left sided hemiplegia with ipsilateral supranuclear facial palsy. Brain CT at the time showed

some enlargement of the third and lateral ventricles with widening of the cerebral sulci, extensive patchy low density changes in the white matter of both cerebral hemispheres, and in the right internal capsule extending into the corona radiata, and focal lacunar type infarcts in the right corona radiata extending to the left external capsule. Six months later he had another mild stroke, details of which were not available.

Three months after the first stroke in November 1989 he experienced a manic episode which remitted after three weeks of treatment with haloperidol. He had three further manic episodes, in December 1990, October 1991, and October 1993, each lasting for three to four weeks. Each was associated with memory impairment, and both deficits recovered after treatment with haloperidol or thioridazine.

He had experienced a depressive episode in July 1989, when he was treated with dothiepin (50 mg at night). This had to be withdrawn after 10 days when he began to exhibit symptoms of hypomania. A second depressive episode occurred in March 1991 which responded to fluoxetine within four weeks (without a hypomanic switch).

Premorbidly he was a moody person whose "mood and personality varied regularly with the seasons". In the summers, he was flirtatious, cheerful, sociable, and spent money erratically. In the winters he became withdrawn, isolated, and spoke much less. His son and daughter in law recalled witnessing, almost with predictable regularity, these mood swings for 40 and 26 years respectively. His family considered these as vagaries in his personality, never serious enough to warrant medical attention.

In the spring of 1978, immediately after his wife's death, he withdrew from his children and friends, spoke little, neglected himself, and slept poorly for two to three weeks. He "pulled himself through" without any treatment and soon remarried. There was no family history of mental illness and he had no psychiatric history.

He recovered from the index episode after treatment with haloperidol and sodium valproate. Investigations disclosed no new abnormality. He was discharged back to the old

people's home where he died of an unrelated chest infection three weeks later.

Discussion

Before his stroke, the patient manifested persistent mood instability, involving periods of mild depression and mild elation, thereby attracting an International Classification of Diseases version 10 (ICD 10) diagnosis of cyclothymia.⁶ The consistent seasonal pattern of his subaffective episodes was remarkable, and has not been reported previously to our knowledge. These subaffective episodes changed after his strokes in two ways: firstly, into full-blown bipolar affective disorder (ICD 10)⁶; and secondly, the seasonal pattern of the now affective episodes switched from the polarity of his previously subaffective episodes.

Cyclothymic temperaments along with dys-thymic and hyperthymic temperaments are thought to represent putative developmental pathways to bipolar affective disorder; whereas inhibited or anxious-phobic temperaments seem related to non-bipolar outcomes.⁷ Tricyclic antidepressant drugs⁸ are recognised triggers which can convert cyclothymia to bipolar affective disorder. Little is known about other converting factors.

Seasonal affective disorder has been reported after right sided lesions involving the temporal and frontal lobes.⁹ Two types (A and B) of seasonal affective disorder have been described.¹⁰ Type A is seasonal affective disorder with fall-winter depression with or without spring-summer mania or hypomania, and type B is seasonal affective disorder with spring-summer depression with or without fall-winter mania or hypomania. Both types are thought to show consistent times of onset and remission.¹⁰ Our patient manifested a type A pattern of cyclothymia before the stroke, which changed to a type B pattern afterwards (manic episodes: two in the fall, one in winter and depressive episodes: one in spring and one in summer).

He developed bipolar affective disorder after a right hemispheric lesion. Brain CT showed widespread bilateral atrophy of the white matter. The existence of cerebral atrophy has been identified as a factor increasing the vulnerability to mania after stroke.^{2,11} By contrast with poststroke depression, in which anterior left hemispheric lesions are more commonly re-

ported, poststroke bipolar affective disorder is more often associated with right hemispheric lesions especially involving right orbitofrontal and basotemporal cortices, the thalamus, and the head of the caudate nucleus.⁵ Brain CT had shown lesions in the right corona radiata and in the left external capsule. A single lesion may have extended from the right hemisphere to the left, or he may have had two separate infarcts. In either case, he had sustained at least one right sided stroke.

The first appearance of mania in elderly people is not thought to reflect the presence of a unique bipolar affective disorder, but rather a conversion factor, especially an organic disorder affecting CNS function, acting in concert with a predisposition to affective illness.¹² Such a model fits with this case, but does not explain the switching of the seasonal pattern of the mood swings.

In conclusion, we present a case of late onset bipolar affective disorder in which a right hemispheric stroke extending to the subcortical areas precipitated a seasonal bipolar affective disorder in a person predisposed to cyclothymia and cerebral atrophy, and also reversed the previous seasonal pattern of the premorbid mood swings.

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