

## Limbic Lesions in a Patient With Recurrent Mania

**SIR:** Secondary mania (mania after brain lesions) may be the result of injury to limbic or limbic-connected structures in the right hemisphere.<sup>1,2</sup> We describe the presence of limbic lesions in a patient with a 33-year history of recurrent unipolar mania.

### Case Report

A 57-year-old left-handed woman with a negative family history of psychiatric disorder was admitted for psychiatric care following a one-week history of irritable mood and psychomotor agitation. The patient had a 33-year history of recurrent unipolar mania (one manic episode per year, usually during the spring). The manic episodes were characterized by irritability, hyperactivity, inflated self-esteem, loud abusive pressured speech, mood-congruent paranoid delusions, overspending, and decreased need for sleep. She never had depression, and the manic episodes improved with lithium therapy, although lithium did not prevent the annual recurrence of mania. Clinical and neurological examinations, as well as laboratory studies (endocrine, metabolic, and EEG), were always negative. On admission, the patient was irritable and noncooperative, and she had pressured speech, flight of ideas, mood-congruent paranoid delusions, and insomnia. A psychiatric examination was carried out using a standardized instrument (the modified Present State Exam) and a Mania Rating Scale (MRS).<sup>3</sup> The patient met *DSM-III-R* criteria for a manic episode with mood-congruent psychotic features, and her MRS score was 19 (definite mania). She was prescribed lithium (900 mg/day) and thioridazine (100 mg/day) and showed a gradual improvement. An MRI scan (figure 1) revealed a left hemisphere arteriovenous malformation involving the amygdala, internal pallidum, and the pulvinar. Additional findings were a moderate left lateral

ventricular dilation, cerebellar vermal atrophy, and iron deposition in the left globus pallidus. A digital subtraction angiography confirmed the presence and location of the vascular malformation.

### Discussion

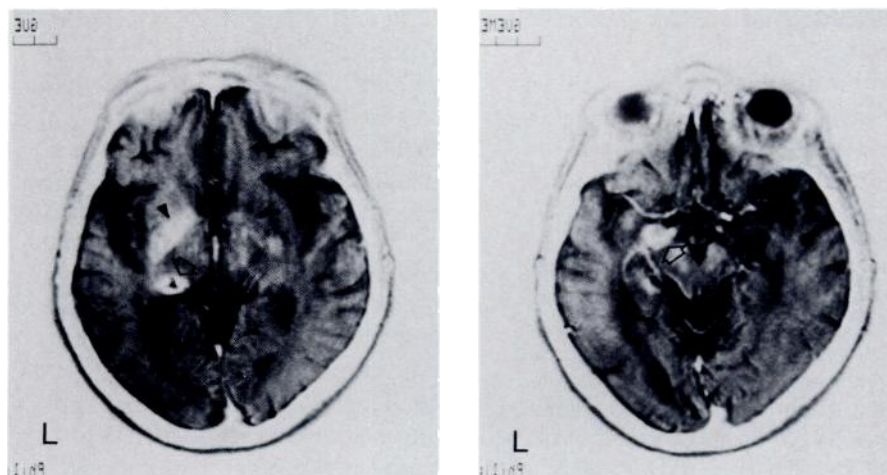
We presented a left-handed woman with a 33-year history of recurrent unipolar mania and a left hemisphere vascular malformation. Even though the early age of onset, the long duration of the psychiatric disorder, the pattern of symptom recurrence, and the presence of psychotic symptoms were suggestive of a functional disorder, an MRI study showed a focal lesion involving limbic and limbic-related areas of the left hemisphere (amygdala, internal pallidum, and pulvinar). While it could be argued that manic symptoms and the brain lesion were only related by chance, we have previously reported a high frequency of lesions to limbic or limbic-connected structures in patients with secondary mania.<sup>2</sup> Although we

have also reported a significant association of secondary mania with right hemisphere lesions,<sup>4</sup> the present patient had left hemisphere damage. This reversed cerebral lateralization may be related to atypical brain organization (she was left-handed). The influence of handedness and side of lesion in the production of secondary mania, however, remains to be systematically examined. To conclude, brain imaging in a subgroup of patients with affective disorders may reveal brain lesions that might be etiologically related.

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**FIGURE 1.** Inversed T2-weighted axial MRI scan showing an arteriovenous malformation involving (left) the left pulvinar (outlined arrow) and (right) the left amygdala (outlined arrow). Note the iron deposition in the left internal pallidum (left, solid arrow).



## References

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## Schizophrenia Spectrum Disorder and Limbic Epilepsy

*SIR:* Schizophrenia spectrum disorders (SSDs) were first noted among the family members of schizophrenics. Now, many of the *DSM-III-R* cluster A personality disorders are considered to be within this "spectrum."<sup>1</sup> However, before SSDs are considered unique syndromes that occur only in relation to functional psychiatric disorders, SSD researchers should consider interictal psychopathology seen in some patients with limbic epilepsy (LE). Waxman and Geschwind<sup>2</sup> emphasized certain characteristic symptoms (e.g., viscosity, aggression) in LE patients. Yet, a range of conditions and much individual variation occur.<sup>3</sup>

Within a recent series of LE patients,<sup>4</sup> we have found several cases that fulfill *DSM-III-R* criteria for schizotypal personality disorder.

### Case Reports

*Patient 1.* The patient, a 35-year-old man, suffered a head injury at age 20. At age 24, partial complex and major motor seizures began. EEGs revealed left temporal lobe and bilateral anterior temporal lobe foci. Since age 32, the patient has complied with anticonvulsant medication irregularly, due to his belief

that he can control his seizures by frequent masturbation.

The patient is circumstantial and digressive. His affect is exaggerated and inappropriate. He admits to vague auditory hallucinations and intense sexual fantasies. He has few acquaintances and no friends. The patient has come to the attention of psychiatrists often in the last two years, and various diagnoses have been used to describe his symptoms (e.g., atypical psychosis, acute paranoid schizophrenia, and unspecified personality disorder).

*Patient 2.* The patient, a 20-year-old man, was delivered as the second twin after a difficult 18-hour labor. Complex partial, absence, and major motor seizures began at age 6. EEGs revealed bilateral anterior temporal lobe foci.

Over the last few years, the patient has gone to great lengths to develop alternative identities. He uses these identities to confuse people he believes are prejudiced against him. He fears that others are conspiring to discredit and insult him by making his problems known to everyone. He has no social contacts. When one inquires about his multiple identities, he becomes angry, digressive, vague, and tangential. Several diagnoses have been used to describe his symptoms (e.g., paranoid disorder, intermittent explosive disorder, and organic personality disorder).

### Discussion

Approaches that are used in the study of SSDs and LE have much to offer each other. The etiologies and pathogenic mechanisms of schizophrenia and SSDs are unknown. For LE, the etiology is often known, and mechanisms to explain symptom production may be more readily investigated. Certain anatomical or neurochemical mechanisms may be common between these disorders.<sup>3</sup> One way to determine shared mechanistic elements would be to compare responses to pharmacological challenges (e.g., neuroleptics and amphetamines). These drugs have been shown to improve and exacerbate,

respectively, the symptoms of SSD patients.<sup>5</sup>

For LE research, the subgrouping of patients using *DSM-III-R* personality disorder criteria would aid studies of biological correlates of psychopathology (e.g., seizure focus location, degree of brain tissue destruction). Longitudinal studies of selected *DSM-III-R* subgroups may yield further insights. For example, in LE patients with schizotypal *DSM-III-R* personality disorder, is there a progression of symptoms with time toward frank psychosis? Such a progression would lend further credence to the notion that certain SSDs are transitional forms, and a longitudinal study would provide a valuable opportunity for prospective study of the mechanisms of psychosis development.

## References

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