

Letter to the Editor

Depression Cured by Stroke

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We recently observed a patient who recovered from a chronic treatment-resistant depression following an infarction to a discreet area of the right frontal cortex. To our knowledge, this is the first reported case in which a mood disorder improved in such a manner.

Mrs. A. is a 60-year-old divorced Hispanic female. She has had a long history of depression going back at least 14 years. She has been treated with tricyclic antidepressants and lithium augmentation with no success. She has also received phenelzine, fluoxetine, and escitalopram at various times without results. This individual has also been treated with high doses of bupropion as well as mirtazapine. Her treatment has been totally resistant to these conventional therapies at therapeutic levels. She has undergone a series of eight bilateral electroconvulsive treatments with only temporary response lasting approximately 10 days. Psychotherapy was of no use to her.

At the age of 56, this patient suffered a cerebral infarction with the primary location of injury being in the right orbitofrontal cortex approximately 1.5 cm anterior to the inferior cingulate gyrus. This lesion measured approximately 0.5 cm³ in size. She did not suffer any physical problems from this event, but it was noted that her mental status changed abruptly after the stroke. Instead of being depressed, the patient was cheerful. She was not disinhibited or confused. She was alert and oriented to person, place, and time. Mrs. A. could read and write normally. There was no evidence of psychosis. Subsequently, she continued to be free of depressive symptoms at all further visits. No neurological focal signs were detected and no seizures have occurred. The patient is now three years post-stroke and continues to be happy and pleased with her life. She has not required any psychiatric medications since her stroke. It would appear that the ischemic episode, which occurred in the right frontal cortex, has had a profound effect on her mood, resulting in sustained neutral mood and appropriate affect.

We believe this is the first description of a case in which an affective disorder actually improved after an infarction. Documentation of affective change following cerebral vascular events is known to the literature (1, 2). In at least one case, euphoria was related to increased perfusion of the left frontal area (2). Our patient presents with an elimination of dysphoria following decreased perfusion in the right frontal cortex. Robinson has reported an increase in serotonin receptor binding in cases of right hemispheric infarction (3). It appears that, generally speaking, a balance of activity in the frontal poles may be needed to produce affective stability.

The history of treatment of psychiatric disorders by interruption of frontal lobe circuits is long and controversial (4, 5). Although we do not endorse radical intervention for psychiatric problems, we feel that it is important to mention this case in which an individual experienced remarkable improvement following the natural disruption of white matter pathways from this region.

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