

# Persistent Severe Depressive Episode with Mood-Congruent Psychotic Features Associated with Left Temporal Ischemia

Nadia Ortiz, MSc,\* Jean-Marie Annoni, MD,† Dante Trojan, MD,\* Christel Alberque, MD,\* and Ariel Eytan, MD\*

**Objective:** The authors describe the case of a patient suffering from a persistent severe depressive episode with mood-congruent psychotic features following left temporal ischemia.

**Background:** Depression is a frequent disorder that may stem from psychosocial factors or occur after brain damage. Cases of poststroke depression with associated psychotic symptoms are rarely described in the literature, and, to our knowledge, no case of depression with psychotic symptoms in the presence of a focal lesion has yet been reported.

**Method:** History included psychiatric, neurologic, and neuropsychological as well as EEG data and CT scan and magnetic resonance images.

**Results and Conclusions:** Neurologic and psychiatric dimensions may be associated and difficult to disentangle, even when the patient presents the classic symptoms of a depressive disorder. This case underlines the importance of investigating possible neurologic signs or symptoms, even when the psychiatric presentation is prototypical of a determined mental disorder. This may be crucial for the patient's evolution.

**Key Words:** depression, stroke, psychotic symptoms, neuropsychiatry

(*Cog Behav Neurol* 2004;17:157–162)

Unipolar depression is the most prevalent and common of all mental disorders<sup>1</sup> and is known to affect approximately 6% of adults.<sup>2</sup> According to ICD-10<sup>3</sup> and DSM-IV,<sup>4</sup> typical depressive episodes are characterized by a change in previous functioning in conjunction with depressed mood and loss of interest or pleasure. Associated symptoms can be both

somatic (weight loss or weight gain, insomnia or hypersomnia, fatigue) and cognitive (diminished ability to think or concentrate, recurrent thoughts of death, feelings of worthlessness, excessive or inappropriate guilt). This well-established clinical picture fits into psychological models of depression, such as the psychoanalytic model (where it corresponds to aggressive feelings turned inward)<sup>5</sup> or the more recent cognitive model (where it is part of the so-called Beck cognitive triad of depression, ie, a negative view of self, of the world, and of the future).<sup>6</sup>

Between 14% and 19% of patients with a lifetime diagnosis of major depression may develop psychotic symptoms such as delusions or hallucinations.<sup>7</sup> In typical cases,<sup>8</sup> delusions involve ideas of sin, poverty, or imminent disasters or may relate to somatic themes such as body transformations or organ negation, known as Cotard syndrome.<sup>9</sup> Auditory or olfactory hallucinations may coexist. They usually entail defamatory or accusatory voices, rotting filth, or decomposing flesh. In psychotic depression feelings of guilt and of worthlessness may be increased compared with nonpsychotic depression.<sup>10</sup>

The onset of a depressive episode may result from several factors such as vulnerability, family history, or stress factors (external or endogenous), and specificity may vary. Personality seems to have a nonspecific effect on vulnerability,<sup>11</sup> and no specific association has been established between life events and depressive illness.<sup>12</sup> Cases of depressive episodes have also been described after strokes. The prevalence may be as high as 40%–50% in the acute postevent period and may reach 30% within 2 years of the vascular event.<sup>13</sup> When symptoms persist, poststroke depression (PSD) is frequently associated with increased disability, increased cognitive impairment, and poor rehabilitation outcomes.<sup>14</sup>

PSD was first correlated with left-hemisphere strokes and particularly with the left frontal region, but this association is now questioned.<sup>15</sup> In fact, there is conflicting evidence concerning the influence of the location of the brain lesion because in some studies no significant correlation was found between depression and stroke localization.<sup>16</sup> Shimoda and Robinson<sup>17</sup> propose a differential association between locations and early- or late-onset PSD to explain differences between authors. According to their results, during the acute phase of stroke, de-

Received May 21, 2003; revised May 24, 2004; accepted July 8, 2004.

\*Department of Psychiatry, Geneva University Hospital, Geneva, Switzerland; and †Department of Neurology, Geneva University Hospital, Geneva, Switzerland.

Reprints: Dr Ariel Eytan, MD, Department of Psychiatry, UPHA 5DL, Geneva University Hospital, 1211 Geneva 14, Switzerland (e-mail: Ariel.Eytan@hcuge.ch).

Copyright © 2004 by Lippincott Williams & Wilkins

pressive features may correlate with a lesion in the left frontal region, whereas in the long term, they correlate with lesions closer to the occipital pole of the left hemisphere. The anterior-to-posterior gradient should be different between early- and late-onset PSD, but lesion location is significantly linked to depressive pathology. Then, because of this important variability, depression symptoms should be examined in all patients after a stroke.<sup>18</sup>

PSD is often difficult to assess because neurologic stroke symptoms may be confounded with depressive symptoms (eg, neurologic apathy in frontal lobe syndromes).<sup>18</sup> In the fourth version of the DSM,<sup>4</sup> PSD is classified in the category of "Mood disorder due to general medical condition." The following subtypes are described: 1, with depressive features; 2, with major depressive-like episode; 3, with manic features; and 4, with mixed features. This implies that mood disorder is the direct physiological consequence of a general medical condition<sup>18</sup> without specifying depressive symptoms like psychotic features.

The presence of psychotic symptoms in PSD is therefore rarely studied or described, or they are part of a confusional state. Low et al, for example, described a patient who developed, after an acute infarct of the left posterior cerebral artery, a confusional state with very labile behavior, changing from severely agitated, restless, and noisy (shouting, screaming) to being quiet and docile.<sup>19</sup> In degenerative disease the frontotemporal structures have been associated with behavior alteration including affective and psychotic symptoms, for example, in frontotemporal dementia or Alzheimer disease.<sup>20</sup> However, to our knowledge no case of a classic depressive state with psychotic symptoms congruent to mood in the presence of a focal lesion has been previously reported.

The current report describes a patient who presented the classic pattern of a severe depressive episode with mood-congruent psychotic features but whose psychiatric history started after a left temporal ischemia and lasted for 5 years without significant evolution.

### CASE REPORT

MR, a patient of Mediterranean origin, had lived in Switzerland for 30 years and worked as a kitchen assistant. He was married and had an adult son. He had no prior personal or familial psychiatric history. According to family observations, at the age of 46, while he was unwillingly separated from wife and son, he presented with psychotic symptoms of rapid onset (delusions and thought disorder) accompanied by memory impairment, apathy, anhedonia, social withdrawal, and lack of interest in personal hygiene. He was addressed for outpatient psychiatric care.

Six months later, symptoms were persisting, and he was admitted for the first time to a psychiatric inpatient care unit for a major depressive episode accompanied by psychotic symptoms. Depressed mood, psychomotor retardation with reduced

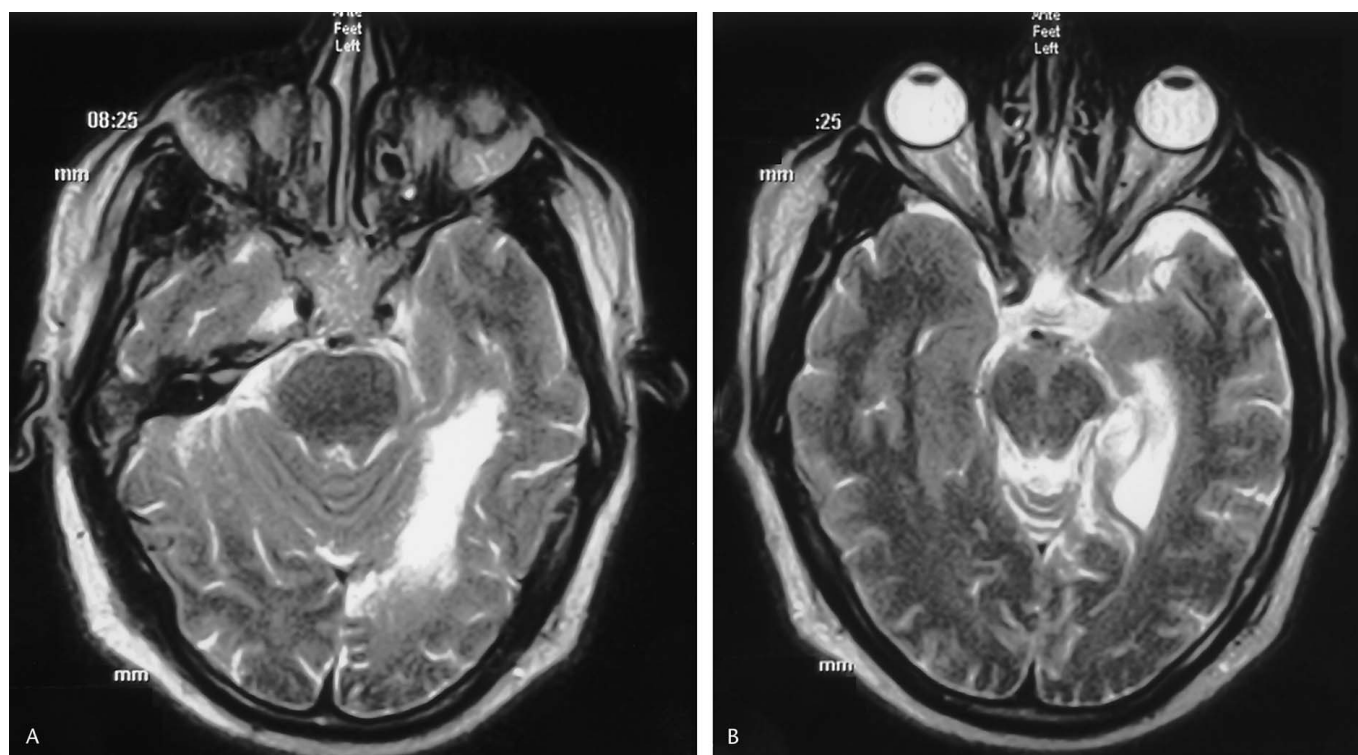
facial expression, and suicidal ideations were present. Nosognosia was incomplete, and important attentional variations were also noted. MR's speech was perseverative and centered on delusional ideas of ruin (he thought he was broke and that the ceiling of his flat would collapse). He described irrational body transformations (said hair was disappearing from his body, his beard was like wire, he was swollen, that he looked like a monster) and had strong guilt feelings (he felt he was punished by God because he had been with prostitutes, because he was the Devil). No obvious neurologic signs were present except for memory impairment. In 2 months, tricyclic antidepressants (clomipramine) and atypical antipsychotics (risperidone) slightly decreased the depressive symptoms.

Three months later, he was hospitalized again for severe depression with psychotic symptoms. His symptoms were very much like those of the previous hospitalization. The lack of evolution suggested the need for more specific neurologic (neuroradiologic and neuropsychological) investigations. A CT scan showed a corticosubcortical hypodensity in the internal temporal lobe, which was the consequence of an infarction in the territory of the left distal posterior cerebral artery. Magnetic resonance imaging confirmed a status of post-cerebrovascular accident in the posterior part of the left parahippocampal gyrus sparing the amygdala and with no signs of a new cerebrovascular event (Fig. 1). The electroencephalogram was not contributory. An echo-Doppler of the carotid and vertebral arteries was normal. The only cardiovascular risk factor found was a dyslipidemia, corrected by atorvastatin.

Three neuropsychological evaluations were conducted (Table 1). MR's first assessment was done about a year after the first psychiatric symptoms. MR complained of speech and memory difficulties. We observed a moderate dysarthria, altered oral expression (word-finding impairment), difficulties in visual recognition, executive dysfunction (poor verbal and nonverbal fluency, and reduced mental flexibility and psychomotor speed), and a severe encoding deficit (assessed with Rey Auditory Verbal and Visual Design Learning Tests) with temporal disorientation. A second evaluation conducted about 1.5 years after the first psychiatric symptoms showed a positive evolution for focal signs of cognitive dysfunction such as language, praxis, gnosis, and calculation difficulties. On the other hand, memory and executive functions remained impaired without any significant change. We assessed MR again 2.5 years after the first symptoms and found no evolution concerning neuropsychological aspects.

Scores on the Hospital Anxiety and Depression Scale<sup>21</sup> were significant for both depression and anxiety and did not vary over time. During the last assessment, MR's scores in the Beck Depression Inventory<sup>22</sup> were compatible with severe depression.

Over 5 years, MR spent 22 months in several psychiatric units. Schizoaffective disorder was ruled out because a period of at least 2 weeks of delusions or hallucinations in the absence



**FIGURE 1.** Transverse MRI sections of the left temporal lesion at 14 mm distance (transverse 7-mm-thick fast spin echo T<sub>2</sub>) 2 years after the first psychiatric symptoms. The amygdala is at least partly preserved. No other lesion has been found across the brain.

of prominent mood symptoms was never objectified.<sup>4</sup> Moreover, the patient did not exhibit all criteria A for schizophrenia, and the psychotic features were clearly mood congruent. The patient was thus treated for a major depressive disorder, but remission of symptoms was very slight despite multiple pharmacological treatments [tricyclic antidepressants (clomipramine), selective serotonin reuptake inhibitors (citalopram), and venlafaxine, in monotherapy or associated with different antipsychotics (haloperidol, risperidone, clozapine, olanzapine)]. Electroconvulsive therapy (ECT) could have been considered at that stage. However, this treatment is not allowed by our local laws.

The last hospitalization occurred in a mixed medical psychiatry unit. More specific investigations were undertaken to define the cardiovascular risk factors, and a patent cardiac foramen ovale (PFO) was discovered. This was considered to be a probable cause of his paradoxical embolism and stroke because a strong association has been shown between these factors.<sup>23</sup>

## DISCUSSION

This report describes a patient who presented the classic pattern of severe depressive episode with mood-congruent psychotic features. This episode was associated with a left temporal ischemia discovered after neuroradiologic investi-

gations. Moreover, during the first year after the onset of psychiatric symptoms, MR suffered from cognitive dysfunction and particularly severe memory impairment congruent with temporal lesions. Partial cognitive recovery was observed in the following months, but psychiatric symptoms persisted.

This case has led us to question the relationship between MR's brain lesion and psychiatric symptoms and to propose the following explanations.

First, we could suppose that MR presented a classic pattern of depression independently of the stroke, perfectly fitting the ICD and DSM diagnostic features<sup>3,4</sup> and the typical psychodynamic and cognitive models of depression. There were no significant clinical signs of neurologic impairment (except cognitive impairment) at his first hospitalization. The only clinical sign, ie, memory impairment, was not investigated or characterized, and the infarction was only found months later, after neuropsychological examination and brain imaging. The neuropsychological symptoms that evolved concerned focal signs that could be explained by the lesion location (language, praxis) while memory and executive impairment remained as frequently observed in depressed patients.<sup>24</sup> In addition, MR presented stress factors from several domains (social, familial) that could elicit a depressive state leading to melancholia with severe symptoms such as delusions.

**TABLE 1.** Neuropsychological Assessment Scores

	<b>First Examination (10 months)</b>	<b>Follow-ups (20 months + 30 months)</b>
Language		
Boston Naming (of 34)*	Pc < 1	Pc 5
Calculation (written) (correct/total)	Impaired (1/3)	Normal (3/3)
Praxis		
Constructive (correct/total)	Impaired (0/2)	Normal (2/2)
Gestual (correct/total)	Impaired (2/5)	Normal (4/5)
Buccolingual (correct/total)	Impaired (1/7)	Normal (6/7)
Gnosis		
Visual (correct/total)	1/2 impaired	2/3 impaired
Spatial	Right neglect	Normal
Executive functions		
Categorical fluency (animals)	Pc 2	Pc 7
Design fluency (5-point test)	Pc 2	Pc 2
Color Trails Test (time A/time B)	Pc < 1/Pc < 1	Pc < 1/Pc < 1
Verbal memory		
RAVLT (trials 1–5)	Pc < 1	Pc < 1
RAVLT (recognition)	Pc < 1	Pc < 1
RAVLT (delayed recall)	Pc < 1	Pc < 1
Visuospatial memory		
RVDLT (trials 1–5)	Pc < 1	Pc < 1
RVDLT (trial 5)	Pc < 1	Pc < 1
RVDLT (delayed recall)	Pc < 1	Pc < 1

\*The Boston Naming 34 items is a shortened French version of original test.  
RAVLT, Rey Auditory Verbal Learning Test; RVDLT, Rey Visual Design Learning Test;  
Pc, percentile.

However, the persistence of severe cognitive impairment is rather uncommon in depressed patients,<sup>25</sup> and the pattern of memory deficits is also unusual: an encoding deficit is present, whereas a retrieval deficit is generally associated with endogenous depression.<sup>26</sup> Moreover, despite the prototypical pattern of depression, the evolution of symptoms is rather uncommon. Depression with psychotic symptoms is a severe form of depression that requires both antidepressant and antipsychotic treatment. Short-term evolution is favorable, although relapses are frequent. In MR's case the depressive episode did not significantly evolve for 5 years despite biologic and nonbiologic in- and outpatient treatments. Findings suggest a relationship between left hemisphere damage and SSRI treatment resistance.<sup>27</sup> According to the neuropsychological pattern of memory impairment and the clinical evolution of MR, the psychopathological diagnosis alone seems insufficient to explain all the symptoms presented as well as their evolution.

A second hypothesis could therefore be to consider closely the implications of the brain structural dysfunction. We

may suppose that MR presented a vascular disease leading to cognitive impairment and associated with psychiatric symptoms. Sporadic vascular cognitive impairment includes various disorders<sup>28</sup> such as cognitive impairment related to stroke, multiple cortical infarcts, multiple subcortical infarcts, or silent infarcts. PSD can result from different vascular causes and changes in the brain. In subcortical ischemic vascular dementia, the onset is frequently insidious, and temporal relations among the cognitive syndrome, brain imaging features, and evidence of cerebrovascular disease may not be clear. The psychiatric profile in vascular mild cognitive impairment has not been established, but there is a strong relationship between cerebrovascular disease (particularly white matter lesions) and depression. Moreover, these lesions generally affect the thalamocortical projections and may predispose to the development of PSD. The cognitive vascular impairment resulting (attentional impairment, executive dysfunction, and depression) is generally associated with disruption of frontosubcortical circuits.<sup>28</sup> The depressive symptoms showed by MR as well as their onset could suggest an early subcortical ischemic vascu-



lar disease. However, other aspects do not support this hypothesis: the frontosubcortical circuits were spared, MR was only 46, and he did not present multiple infarcts. Moreover, psychotic symptoms are rarely found in subcortical ischemic vascular disease. They are present in Lewy-body disease or frontotemporal dementia, but again the localization of MR's lesions differed in that they concerned the internal temporal lobe, sparing the thalamus and the amygdala.

A third hypothesis should be considered more closely: the specificity of the lesion. That is to consider the depression with psychotic symptoms presented by MR as a direct physiological consequence of a stroke in the left internal temporal lobe. We could then assume that the main clinical manifestations of the cerebrovascular accident (CVA) were the psychiatric symptoms and the presence of clinically relevant memory impairment. Although we have no neuroradiologic data on the acute phase, the subsequent regression of focal neuropsychological signs (anomia and apraxia) as well as the absence of perilesional atrophy on the first brain scan (performed the first year after the onset of symptoms) suggest a short delay between the stroke and the expression of psychiatric symptoms. We should then accept the possibility of a close association between the specific focal lesion and both depression and psychosis. Major depression following a stroke is common and may affect up to 40% of patients.<sup>13</sup> When its specific evaluation is not limited by neurologic deficits, symptoms described are very similar to those of functional depression.<sup>29</sup> However some authors<sup>30</sup> have described a different pattern between endogenous and poststroke depression. In the latter, higher scores on catastrophic reactions, hyperemotionalism, and diurnal mood variations and lower scores on suicide and anhedonia are found. Moreover, there is presently no consensus concerning the specificity of lesions in patients who develop depression after a CVA.<sup>15,16</sup> Some authors have shown that left cortical and subcortical lesions, particularly involving the frontal lobe, are significantly more frequent among patients with major depression.<sup>31</sup> The fact that MR's lesion is more posterior does not exclude the existence of a correlation between the localization of MR's infarction and the particular pattern of depression observed. Furthermore, research that has studied the localization of brain lesions in PSD has not taken into account the variety of depressive symptoms found in these patients; in particular, few descriptions concern psychotic symptoms in poststroke depression.

We could then consider MR's psychotic symptoms independently. Psychotic symptoms have already been associated with left temporal dysfunction in neurologic illnesses, for example, in patients with epilepsy.<sup>32</sup> In dementia with Lewy bodies, the presence of hallucinations seems also to correlate with depletion of acetylcholine in the temporal cortex.<sup>33</sup> In the case of focal internal temporal lesions after stroke, psychotic symptoms are also described. Bouvier-Peyrou and colleagues<sup>34</sup> have considered the implication of left paralimbic structures in

a particular form of delusional misidentification syndrome and have discussed a type of psychotic symptom following brain damage, ie, a delusion of the self shifted in time. However, in all these cases temporal dysfunction seems to be associated with a more generalized brain dysfunction. Low et al<sup>19</sup> describe a poststroke patient whose behavior was altered (agitation, restless and noisy behavior, shouting) after an infarct in the territory of the left posterior cerebral artery. Social withdrawal, delusions, and psychomotor retardation were not present as in MR, and the behavior of Low's patient also showed signs of a confusional state. In addition, small old infarcts were also discovered in other subcortical structures.

MR's symptoms are therefore particularly interesting in that the patient presents specific psychotic symptoms (without confusional state or agitation) in the presence of a unique focal lesion. Moreover, a specific link can be made between the localization of stroke and psychotic symptoms.

In conclusion, our case brings converging evidence suggesting that both psychosis and depression were mainly related to the stroke. Psychosis was certainly associated with injury to the paralimbic neuromatrix, and the PSD could have been facilitated both by the left lateralization of the injury and by several factors such as vulnerability or stress factors (external or endogenous).

In addition, this case underlines the importance of exploring possible somatic anomalies even when a common mental disorder is identified. Here, the patient was eventually operated for his PFO, thus preventing the recurrence of a stroke.

## ACKNOWLEDGMENTS

The authors thank Barbara Whitaker-Clinch, clinical and research psychologist, for her help with the English version of the manuscript and the reviewers for their precious help through scientifically constructive comments.

## REFERENCES

1. Kessler RC, McGonagle KA, Zhao S, et al. Lifetime and 12-month prevalence of DSM-III-R psychiatric disorders in the United States. *Arch Gen Psychiatry*. 1994;51:8-19.
2. Regier DA, Boyd JH, Burke JD, et al. One-month prevalence of mental disorders in the U.S. Based on five epidemiologic catchment area (ECA) sites. *Arch Gen Psychiatry*. 1988;45:977-986.
3. World Health Organization. *The ICD-10 Classification of Mental and Behavioural Disorders: Clinical Descriptions and Diagnostic Guidelines*. Geneva: WHO, 1992.
4. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders, 4th ed*. Washington DC: American Psychiatric Association, 1994.
5. Freud S. *Grief and Melancholia. Standard Edition of the Complete Works of Sigmund Freud*. London: Hogarth Press, 1953.
6. Beck AT, Rush AJ, Shaw BF, et al. *Cognitive Therapy of Depression*. New York: Guilford Press, 1979.
7. Ohayon MM, Schatzberg AF. Prevalence of depressive episodes with psychotic symptoms in the general population. *Am J Psychiatry*. 2002; 159:1855-1861.
8. Akiskal HS. Mood disorders: clinical features. In: Kaplan HJ, Sadock BJ,

- eds. *Comprehensive Textbook of Psychiatry*, 6th ed. Philadelphia: Lippincott Williams & Wilkins, 1995:1123–1152.
9. Cotard J. Du délire hypocondriaque dans une forme grave de mélancolie anxieuse. Mémoire lu à la Société médico-psychologique dans la séance du 28 juin 1880. *Ann Med Psychol*. 1880;4:168–174.
  10. Thakur M, Hays J, Krishnan KRR. Clinical, demographic and social characteristics of psychotic depression. *Psychiatry Res*. 1999;86:99–106.
  11. Hirschfeld RMA, Shea MT, Holzer CE III. Personality dysfunction and depression. In: Honig A, Van Praag HM, eds. *Depression: Neurobiological, Psychopathological and Therapeutic Advances*. Chichester: John Wiley & Sons, 1997:329–341.
  12. Jenaway A, Paykel ES. Life events and depression. In: Honig A, Van Praag HM, eds. *Depression: Neurobiological, Psychopathological and Therapeutic Advances*. Chichester: John Wiley & Sons, 1997:280–295.
  13. Nasreddine ZS, Cummings JL. Depression in neurological diseases. In: Honig A, Van Praag HM, eds. *Depression: Neurobiological, Psychopathological and Therapeutic Advances*. Chichester: John Wiley & Sons, 1997:312–325.
  14. White EM, Mulsant BH. Post stroke depression: epidemiology, pathophysiology, and biological treatment. *Biol Psychiatry*. 2002;52:253–264.
  15. Carson AJ, MacHale S, Allen K, et al. Depression after stroke and lesion location: a systematic review. *Lancet*. 2000;356:122–126.
  16. Pohjasvaara T, Leppävuori A, Siira I, et al. Frequency and clinical determinants of poststroke depression. *Stroke*. 1998;29:2311–2317.
  17. Shimoda K, Robinson RG. The relationship between poststroke depression and lesion location in long term follow-up. *Biol Psychiatry*. 1999;45:187–192.
  18. Carota A, Staub A, Bogousslavsky J. Emotions, behaviours and mood changes in stroke. *Neurology*. 2002;15:57–69.
  19. Low JA, Yap KB, Chan KM. Posterior cerebral artery territory infarct presenting as acute psychosis. *Singapore Med J*. 1999;40:702–703.
  20. Bathgate D, Snowden JS, Varma A, et al. Behaviour in frontotemporal dementia, Alzheimer's disease and vascular dementia. *Acta Neurol Scand*. 2001;103:367–378.
  21. Snaith RP, Zigmond AS. The Hospital Anxiety and Depression Scale. *Acta Psychiatr Scand*. 1983;67:361–370.
  22. Beck AT, Ward CH, Mendelson M, et al. An inventory for measuring depression. *Arch Gen Psychiatry*. 1961;4:561–571.
  23. Mas JL, Zuber M. Recurrent cerebrovascular events in patients with patent foramen ovale, atrial septal aneurysm, or both and cryptogenic stroke or transient ischemic attack. French Study Group on Patent Foramen Ovale and Atrial Septal Aneurysm. *Am Heart J*. 1995;130:1083–1088.
  24. Beblo T, Hermann M. Neuropsychological deficits in depressive disorders. *Fortschr Neurol Psychiatr*. 2000;68:1–11.
  25. Kuny S, Stassen HH. Cognitive performance in patients recovering from depression. *Psychopathology*. 1995;28:190–207.
  26. Burt DB, Zembar MJ, Niederehe G. Depression and memory impairment: a meta-analysis of the association, its pattern and specificity. *Psychol Bull*. 1995;117:285–305.
  27. Spalletta G, Guida G, Caltagirone C. Is left stroke a risk factor for selective reuptake inhibitor antidepressant treatment resistance? *J Neurol*. 2003;250:449–455.
  28. O'Brien JT, Erkinjuntti T, Reisberg B, et al. Vascular cognitive impairment. *Lancet Neurol*. 2003;2:89–98.
  29. Paradiso S, Ohkubo T, Robinson R. Vegetative and psychological symptoms associated with depressed mood over the first two years after stroke. *Int J Psychiatry Med*. 1997;27:137–157.
  30. Gainotti G, Azzoni A, Marra C. Frequency, phenomenology and anatomical-clinical correlates of major post stroke depression. *Br J Psychiatry*. 1999;175:163–167.
  31. Robinson RG. Poststroke depression: prevalence, diagnosis, treatment and disease progression. *Biol Psychiatry*. 2003;54:376–387.
  32. Marsh L, Sullivan EV, Morell M, et al. Structural brain abnormalities in patients with schizophrenia, epilepsy, and epilepsy with chronic interictal psychosis. *Psychiatry Res*. 2001;108:1–15.
  33. Ballard C, Holmes C, McKeith I, et al. Psychiatric morbidity in dementia with Lewy bodies: a prospective clinical and neuropathological comparative study with Alzheimer disease. *Am J Psychiatry*. 1999;156:1039–1045.
  34. Bouvier-Peyrou M, Landis T, Annoni J. Self-duplication shifted in time: a particular form of delusional misidentification syndrome. *Neurocase*. 2000;6:57–63.