tal models of seizure generation. Curr Opin Neurol 1998; 11:123–127
5. Nestler E, Hyman S, Malenka R: Molecular Neuropharmacology: A Foundation for Clinical Neurosciences. New York, McGraw-Hill, 2001

Poststroke Hallucination Delusion Syndrome

To the Editor: Neurology and psychiatry are a continuum separated historically by Descartes.[1] Here we discuss a patient of poststroke hallucination delusion syndrome, a syndrome rare in literature.[2,3] The factors leading to atypical psychosis are not well understood. The role of the seizure following a stroke needs to be further ascertained.

Case Report

Mr. N, a 42-year-old right handed hypertensive male patient, presented with vivid hallucinations and delusions of paranoia and control of 2–3 months' duration in 2004. He complained of an existing person for him who would "follow him trying to harm him," "shout his name from his window," and "see and smile from the roadside while chasing him." His wife denied that such things were happening to him. His social and professional life was grossly affected. Neither he nor his relatives had a history of psychiatric disorder.

In 2002, he developed right-sided hemianopia with acute nonhemorrhagic infarct in posterior cerebral artery territory involving the left occipital lobe. In January 2003, he was admitted for slurring of speech and left arm weakness. Immediate magnetic resonance imaging (MRI) of the brain showed hyperacute non-hemorrhagic distal right middle cerebral artery territory infarct and chronic infarct in occipital lobes. He was sucessfully thrombolysed with complete clinical recovery.

Six months later, following thrombolysis, he developed partial seizure followed by generalized tonic clonic convulsions. His laboratory workup did not reveal any abnormality. Cardiac function was normal. Computerized tomography (CT) showed dilatation of the lateral ventricle. With no history (personal or family) of psychiatric illness, and with history of preceding stroke and later a seizure, lack of insight for his illness and persistent hallucinations and delusions, diagnosis of secondary hallucination delusion syndrome was made. He responded to high dose phenytoin sodium and sodium valproate.

Discussion

There is little description of atypical psychosis following stroke.[2] Why a few patients, and not many, develop schizophreniform disorders following stroke remains unanswered.[2]

Here, our patient had strokes, seizure, and then acute onset hallucination and delusion. His lack of insight for his illness was remarkable. He had no history of psychiatric illness in the past or in family. His neuroradiological scans suggested ventricular dilatation.

It can be hypothesized that the stroke lesion site generates continuous electrical activity which is the organic substrate for his psychotic phenomenon. Our patient responded well to a high dose of phenytoin sodium, probably by modifying the underlying electrical activities, further suggesting the role of seizure activity in such disorders.

We thank Dr. Ruchika Chhibar and Dr. Bharat Shah for useful comments while preparing the manuscript.

Nishant Kumar Mishra, M.B.B.S.
Department of Neurology, University Hospital (CHUV), Lausanne, Switzerland; Department of Neurology, Lilavati Hospital and Research Center, Mumbai-50, India
Shirish Hastak, M.D., D.M.
Department of Neurology, Lilavati Hospital and Research Center, Mumbai-50, India

References

1. Pert CB: Molecules of cognition: the science behind mind-body medicine. New York, Simon and Schuster, 1997
2. Rabins PV, Starkstein SE, Robinson RG: Risk factors for developing atypical schizophreniform psychoses following stroke. J Neuropsychiatry 1991; 3:6–9
3. Levine DN, Finkelstein S: Delayed psychoses after right temporoparietal stroke/trauma: relation to epilepsy. Neurology 1982; 32:267–273

Late-Life Anxiety

To the Editor: Anxiety disorders often begin in early adulthood[1] and an initial onset of these disorders after the age of 60 is usually associated with a traumatic event.[2] Numerous factors can predispose this population to high levels of anxiety, including medical illness, psychosocial changes, depression, and dementia.[1,3] The symptoms are similar to those in younger patients,[1,4] but they are usually associated with unfavorable prognosis in the elderly.[1]

Anxiety disorders in the elderly are often underdiagnosed and, when treatment is provided, benzodiazepines are overused and antidepressants are underused.[5]

The use of benzodiazepines is usually effective but is associated with increased risk of cognitive impairment, sedation, falls, and fractures.[6] Based on the safety, efficacy, and high rate of comorbid depression,[4] the serotonergic antidepres-

sant medications are preferred to benzodiazepines as a first-line of treatment for anxiety disorders in the elderly.[6] Psychotherapy, particularly cognitive behavior therapy, is often effective in these disorders as well.[6]

We reviewed symptoms of three cases in which onset of anxiety symptoms developed after age 60 as a result of having a medical procedure. They were highly functioning individuals and anxiety symptoms led to impairment of their social and occupational life. They were all successfully treated with selective serotonin reuptake inhibitor medications without any side effects and achieved the overall level of functioning.

The first case was a 61-year-old male who worked in graphic art. He developed severe neck pains and a magnetic resonance imaging scan (MRI) of cervical spines was recommended. After having the MRI, he developed recurrent unexpected panic attacks and anxiety about being in a closed place. Commuting to work caused marked distress and he subsequently avoided traveling in a bus, train, or car, and his daily activities were restricted. He initially refused to consider any medications that might limit his creativity. He agreed to a trial of sertraline, 50 mg/day, which was increased gradually to 100 mg. He noted significant improvement in intensity and frequency of his panic attacks and regained the ability to use the public transportation without any fear.

The second case involved a 61-year-old female who was a medical technician.

She suffered gastrointestinal reflux disease and underwent diagnostic upper endoscopy. Since then, she had been feeling anxious, and had poor concentration, frequent unpredicted panic attacks, and anxiety about being in a crowd. She could not resume her work and preferred to stay home to avoid situations that might provoke her anxiety. She was prescribed sertraline and was maintained on 150 mg/day. She reported lower anxiety level, became comfortable in public, and decided to look for a part-time job.

The third case was a 75-year-old male. He was a retired photographer and developed minor neurological deficits. Computed tomography scan (CT) of the head was conducted. After the image study, he started to have periods of intense fear and excessive worry cued by his presence in places from which escape might be difficult. He isolated himself at home, stopped going to church services and the senior citizen center, and suffered depression. He showed significant symptom response on paroxetine, 40 mg/day.

Nahla A. Mahgoub, M.D.
Psychiatry, Weill Medical College of Cornell University, New York
Jahandar Saifollahi, M.D.
Melinda Lantz, M.D.
Geriatric Psychiatry, Beth Israel Medical Center, New York

References

1. Sadock BJ, Sadock VA: Geriatric psychiatry, in Kaplan and Sadock's Synopsis of Psychiatry: Behavioral Sciences/Clinical Psychiatry, 9th ed. Philadelphia, Lippincott, Williams & Wilkins, 2002, pp 1318–1337
2. Flint AJ: Management of anxiety in late life. J Geriatr Psychiatr Neurol 1998; 11:194–200
3. Rangaraj J, Pelissolo A: Anxiety disorders in the elderly: clinical and therapeutic aspects. Psychol Neuropsychiatr Vieil 2006; 4:179–187
4. Flint AJ, Gagnon N: Diagnosis and management of panic disorder in older patients. Drugs Aging 2003; 20:881–891
5. Flint AJ: Epidemiology and comorbidity of anxiety disorders in later life: implications for treatment. Clin Neurosci 1997; 4:31–36
6. Lenze EJ, Pollock BG, Shear MK, et al: Treatment considerations for anxiety in the elderly. CNS Spectr 2003; 8(S3):6–13

Aripiprazole Diminishes Cannabis Use in Schizophrenia

To the Editor: Aripiprazole is the first partial agonist dopaminergic D_2 with clear antipsychotic effect. Many schizophrenic patients will develop comorbid substance abuse. Cannabis consumption may worsen psychotic symptoms of schizophrenic patients.[1] We describe the case of a schizophrenic patient whose use of cannabis and related problems disappeared after treatment with aripiprazole.

Mr. A, a 33-year-old Caucasian patient with schizophrenia, has since his mid-20s been treated with olanzapine, 20 mg/day, and escitalopram, 10 mg/day. He did reasonably well with this regimen but was apragmatic. He frequently used cannabis every day (urine screen for tetrahydrocannabinol [THC] was positive). He was moderately obese (BMI: 28) but had no other medical problems. He lived in a community house, in which nurses ensured that he was compliant with treatment. His Brief Psychiatric Rating Scale (BPRS) score was 52. Before his mid-20s, the patient was treated successively for different classic antipsychotic conditions (i.e., haloperidol, bromperidol, and pimozide) and he reported increased cannabis abuse concomitant with these regimens.

Aripiprazole, 15 mg/day, was added to his treatment regimen. After 1 week, the olanzapine dose was decreased to 10 mg. One week later, olanzapine was discontinued. After 5 weeks, escitalopram treatment was discontinued because the pa-