Letters to the Editor

Transient Abnormal Behavior After Pontine Infarction

To the Editor:

In a recent issue of Stroke, Andersen et al reported patients with poststroke pathological crying associated with bilateral pontine or hemispheric lesions, and suggested that an involvement of the serotonergic system may be related to their symptoms. They also reported a patient with a unilateral pontine lesion who had transient pathological crying. We have observed a patient who exhibited transient abnormal behavior after acute unilateral pontine infarction, which may also be attributable to the derangement of serotonergic neurotransmission in the brain.

A 54-year-old hypertensive man suddenly developed dysarthria followed by a series of unusual behavior. He called his wife repeatedly to his room, but would not let her in when she came. When he was later asked why, he said "I suddenly became suspicious and angry about her infidelity, but I don't know why. I've had a relatively good marital life so far." He also became irritable, impulsive, and dysphoric. He called his friends and asked them to join him in drinking alcohol. Normally, he did not enjoy drinking and had not had alcohol for 1 year prior to this episode. When his friends declined, he went out alone to a bar and became intoxicated. That night, he brought home a pork cutlet and devoured it, saying repeatedly, "I have to eat this," even though he had had his dinner just a few hours before. The next day, he had difficulty writing because of his weak and clumsy right hand. He felt excessively fatigued, lost his appetite, and continued to be suspicious about his wife. At one time, he found a dirty, used gas range discarded by his neighbor outside the house and brought it home, saying, "Let's use it." When later asked about this, he said "I really don't know why I did that." When the patient was examined at our hospital the next day, he was irritable and impulsive though alert and oriented. There was moderate dysarthria, mild right lower facial palsy, and slight weakness and ataxia of the right arm. The results of routine laboratory tests were all within normal limits. Brain magnetic resonance imaging showed an infarction in the left pons (see Figure). After admission to the hospital, the patient did not exhibit definitely abnormal behavior. However, at 6-month follow-up, he remained slightly more impulsive and less tolerant than before.

This patient had dysarthria—clumsy hand syndrome due to a paramedian pontine infarction. The coincidental appearance of the bizarre behavior and its improvement over a few days suggest that this transient abnormal behavior was also related to the stroke. Drake et al reported 2 patients with pontine base infarction who had manic symptoms including grandiosity, sleeplessness, irritable mood, and hyperactivity. Our patient's symptoms, which included suspicion of his wife's infidelity, bizarre behavior, irritability, and impulsiveness, do not conform to mania, but are consistent with acute transient psychosis. These were probably not due to a stress reaction to his stroke, because they started before he realized that he had suffered one.

There is accumulating evidence that altered serotonin metabolism is related to the pathogenesis of psychic disturbances. Low levels of 5-hydroxyindoleacetic acid, a serotonin metabolite, in the cerebrospinal fluid are related to agitation, delusion, sadness, or suicidal ideas. In the human brain stem, raphe nuclei, which contain neurons synthesizing serotonin, are located from the medulla to the midbrain. The principal ascending fibers arise from serotonin cell bodies in the dorsal nucleus and the superior reticular nucleus.
central nucleus, a rostral extension of the pontine raphe nuclei. In our patient, magnetic resonance imaging revealed a paramedian upper pontine lesion, which might have involved the superior central nucleus or the ascending serotonergic fibers. The consequent derangement of serotonin neurotransmission may be associated with the psychotic symptoms seen in this patient.

Response

It is well known that bizarre behavior may accompany occlusive vascular disease of the rostral basilar artery and infarction of the midbrain, thalamus, and portions of the temporal and occipital lobes, usually without prominent motor dysfunction. Kim et al report a case of transient abnormal behavior after a paramedian left pontine infarction, with symptoms of dysphoric mood, irritability and impulsiveness, and suspiciousness. The case illustrates a complex behavior that, although it is not uncommon, cannot be distinguished from the large group of heterogeneous conditions reported with rostral brain stem infarctions. It is therefore highly speculative to attribute such a complex behavior, although transient, to derangement of a single neurotransmitter system. However, part of the behavior described, namely the loss of control of temper (ie, aggression and irritability), might be attributable to derangement of serotonin metabolism because drug trials with the selective serotonin reuptake inhibitor citalopram has been effective in the treatment of such emotional disturbances in demented patients.3

We find it very important and illuminating to attempt to localize single symptoms such as uncontrolled crying or laughing, uncontrolled temper, sleeplessness, central pain, and amnesia after stroke, and to explore which neurotransmitter systems might be involved. This approach makes possible specific treatment, which is very much needed in rehabilitation to improve quality of life after stroke.

References

3. Carpenter MB, Sutin J. Apo E Phenotype in Atheromatous Plaques

To the Editor

The association of apolipoprotein (apo) E4 alleles and cerebrovascular disease has been discussed in some recent articles in Stroke.1,2 According to the latest epidemiological research, there is an increased frequency of apo E4 phenotype in patients affected by multi-infarct dementia, which should not be surprising since high apo E4 level increases cardiovascular risk. These patients constitute an interesting reference group because of their increased risk. It must be remembered that the three apo E variants—apo E2, apo E3, and apo E4—have different bonding capacities with low-density lipoprotein (LDL) (apo E has the greatest and apo E2 the least), so homozygotes for apo E2 are at risk of cardiovascular disease because of their increased incidence of type III hyperlipidemia, and homozygotes for apo E4 are at greater risk because the more efficient bond of apo E4 to the LDL receptor causes reduced activity of the receptor in hepatocytes and an increase in levels of cholesterol and triglycerides.

It is possible to identify which of the three apo E alleles is present in carotid atheromatous fibrous plaques by using a method that amplifies a portion of genomic DNA from the plaques. We analyzed 35 carotid plaques and 5 femoral ones. One carotid plaque showed an apo E4/E2 allele and the other 39 plaques (98%) showed an apo E3/E3 homozygote isoform. Our conclusion is that apo E alleles in plaques are nearly all E3/E3, in a frequency that is not different from that in a control population (whose DNA, for the study of apo E, is taken from peripheral blood rather than from plaques). It is important in assessing these findings to consider the technique of genetic phenotyping used; also, incomplete separation of bands or uncertain interpretation and identification of the same could lead to erroneous classification of the alleles.

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