Evolution of Technetium-99m-HMPAO SPECT and Brain Mapping in a Patient Presenting with Echolalia and Palilalia

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A 78-yr-old woman presented with transient echolalia and palilalia. She had suffered from Parkinson's disease for 2 yr. Routine laboratory examination showed hypotonic hyponatremia, but was otherwise unremarkable. Brain mapping revealed a bifrontal delta focus, more pronounced on the right. Single photon emission computed tomography (SPECT) of the brain with technetium-99m labeled d,l hexamethylpropylene-amine oxime (^{99m}Tc-HMPAO), performed during the acute episode showed relative frontoparietal hypoactivity. Brain mapping performed after disappearance of the echolalia and palilalia, which persisted only for 1 day, was normal. By contrast, SPECT findings persisted for more than 3 wk. Features of particular interest in the presented patient are the extensive defects seen on brain SPECT despite the absence of morphologic lesions, the congruent electrophysiologic changes and their temporal relationship with the clinical evolution.

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Echolalia is a pathologic language behavior characterized by contextually inappropriate repetition of verbal stimuli uttered by the interlocutor (*1*). Palilalia, on the other hand, entails the involuntary repetition twice or more of one's own terminal sounds, words, phrases, or sentences (*2*). Actual topographic knowledge of associated lesions in echolalia and palilalia is mostly based on postmortem studies of patients with dementia, especially Pick's disease (*1,2*). We report on the evolution of ^{99m}Tc-HMPAO SPECT and brain mapping in a patient presenting with transient echolalia and palilalia.

PATIENT REPORT

A 78-yr-old woman was admitted because of spatial and temporal disorientation and excitation with verbal aggression. She had suffered from Parkinson's disease for 2 years, with more pronounced symptoms on the left. The patient had refused all medication (LDOPA 100 mg, benserazide 25 mg tid, and biperideenchlorohydrate 2 mg 2.5/day) several days before admission.

Routine laboratory examination demonstrated a low serum sodium, 124 mEq/liter; a normal potassium, 4.3 mEq/liter; a low chloride, 89 mEq/liter, and a normal bicarbonate, 26 mEq/liter. Serum osmolality was 272 mOsm/kg; urinary osmolality, 713 mOsm/kg; and urinary sodium, 14.4 mEq/24 hr. Other laboratory examinations including thyroid hormone levels were normal.

Neurologic examination on admission demonstrated a slight rest tremor and coghwheel rigidity, more pronounced on the left; vivid myotatic reflexes and a Babinski sign located on the left. The patient was somnolent and confused.

The echolalia concerned complete phrases or parts of sentences. Sometimes, phrases of the examiner were grammatically adapted by the patient. When commands were given, the patient only repeated them; but when the examiner insisted, they were correctly performed. In several instances the patient echoed what she was saying more than 20 times. Spontaneous speech was limited to "medical" topics, including the words *Parkinson* and *death*. It was further noted that this multilingual patient involuntarily switched from one language to another.

On the fifth day, the patient showed hypokinesia and mutism, with only slight mimic and motor responses to verbal stimuli. Bilateral Babinski signs and disinhibition reflexes, including snout, masseter and palmomental reflexes, were present.

On the 14th day after admission, her mental status had normalized. Because of increased extrapyramidal symptoms LDOPA treatment was restarted. The patient was discharged 3 wk after hospitalization.

Complementary Examinations

Computed tomography (CT) scan of the brain after contrast injection on Day 4, magnetic resonance imaging (MRI) on Day 14, and echo-Dopplertomography of the carotid vessels were unremarkable. On Day 5, an electroencephalogram (EEG) demonstrated a diffuse slow background activity of a δ,α-rhythm with a mean amplitude of 40 μV. There were numerous monomorph discharges of a delta activity of 2.93 Hz with an amplitude of 200 μV, more pronounced in the frontal region. No graphoepileptic changes were noted.

Brain mapping of 3-sec epochs of stationary δ discharges revealed a reproducible and consistent delta focus of 426.8 μV with a peak frequency of 2.93 Hz, more markedly on the right side (Fig. 1). This focus was also detected by quantified analysis of the diffusely slowed background activity.

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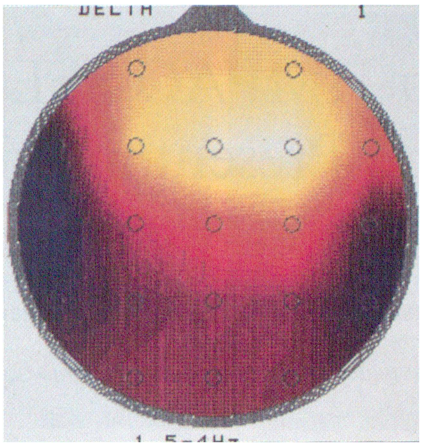

FIGURE 1. Brain mapping on Day 5: demonstration of a frontal deltafocus, more markedly on the right side.

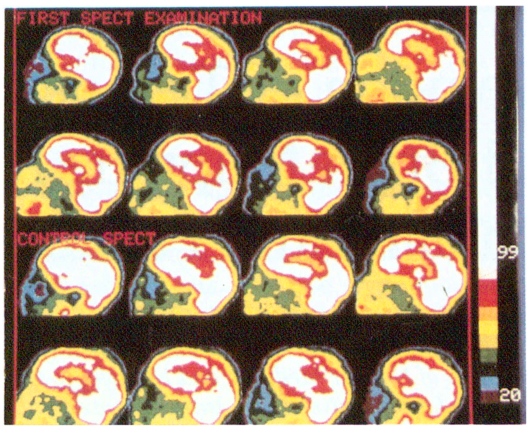

FIGURE 2. First two rows: ^{99m}Tc-HMPAO SPECT of the brain on Day 4, performed during exacerbation of the symptomatology. Sagittal slices are depicted progressing from right to left, with the nose oriented towards the left side of the picture. This SPECT examination shows a bifrontoparietal defect, larger on the right. Last two rows: control ^{99m}Tc-HMPAO SPECT of the brain on Day 19 after disappearance of echolalia and palilalia. The bifrontoparietal defect is now small when compared with the first examination.

On Day 4, brain SPECT examination was performed using 15 mCi of ^{99m}Tc-labeled hexamethylpropyleneamineoxime (Amersham, UK) and a single head rotating gammacamera (Orbiter 75, Siemens, FRG) with a low-energy all-purpose collimator interfaced to an Elscint computer. The study was initiated 10 min post-radiopharmaceutical injection. A total of 64 projection images were acquired over a 360° rotation, 30 sec/frame, radius of rotation 15 cm. The reconstruction was performed using filtered backprojection with a Shepp–Logan–Hanning filter.

Transaxial slices were reoriented parallel to the orbitomeatal axis and coronal and sagittal plane images were created. SPECT of the brain demonstrated a large bifrontoparietal defect, larger on the right (Fig. 2). A repeat brain SPECT on Day 11 showed the same disturbances. Control brain mapping on Day 15, however, was normal. A third SPECT on Day 19 showed a smaller bifrontoparietal defect.

DISCUSSION

Echolalia has been reported to occur in a variety of neurologic and psychiatric syndromes and diseases. It has been noted in the presence of a lesion involving the posterior part of the temporal cortex with preservation of the midtemporal convolution, the inferior frontal speech area and the arcuate fasciculus, connecting the two (*3*). Palilalia has been noted in epilepsy (*4*) and during electrical stimulation of the inferior part of the ascendent parietal and especially frontal convolutions of the left hemisphere and the foot of the third frontal convolution of the right hemisphere (*4*). It has also been described during stimulation of the internal side of the first frontal convolution (*4*).

Finally, it has also been noted in postencephalitic Parkinson and pseudobulbar syndromes (*2*). In these cases, it is usually associated with diffuse pallidostriatal disease (*2*).

In the patient presented, SPECT was performed because of a normal CT scan and MRI in the presence of major acute neuropsychological changes. SPECT demonstrated a large bifrontoparietal defect. Because of the size and the confirmation of the defect by repeat SPECT, an accidental nature of the finding can be excluded. Also, although not completely overlapping, the focus of brain mapping pointed to the same location. Brain mapping is the topographic representation of the quantified analysis of the bioelectrical activity of the brain.

In both echolalia and palilalia, frontal lobe involvement has been demonstrated, but their simultaneous occurrence represents an exception. In this patient, symptomatology proved transient and upon re-examination no neuropsychological findings suggestive of frontal lobe type dementia were found. Finally, a vascular event or seizure disorder seems improbable because of the history and complementary examinations.

The patient presented suffered from Parkinson's disease. Few investigators have reported on the occurrence of changes in regional cerebral blood flow in patients with Parkinson's disease (*5–8*). The predominant symptomatology, lateralization, and treatment may play a role in the pattern found but have remained unchanged during the evolution of SPECT findings in our patient.

Tentatively, the acute interruption of the parkinsonian medication or, more likely, the hyponatremia caused the symptoms. Hyponatremia was attributed to extrarenal loss and resolved after 1 week under proper treatment. Hypothetically, the functional changes demonstrated may have been secondary to a metabolic or toxic encephalopathy, based on the assumption that these parameters indirectly might reflect local brain metabolism.

CONCLUSIONS

Echolalia and papilalia may be related to functional changes in the frontoparietal regions. Two functional im-

aging techniques, ^{99m}Tc-HMPAO SPECT and mapping of the brain, revealed focal dysfunction, whereas morphologic imaging techniques, such as CT scan and MRI did not detect any structural lesion. In the patient presented, the electrophysiologic changes temporally correlated well with the clinical evolution while brain SPECT remained abnormal. Finally, subclinical dysfunction as demonstrated by perfusion SPECT may persist for weeks.

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