Abstract: We describe a case of adult onset simple phonic tic after subcortical stroke involving left caudate nucleus. In the acute phase of stroke the patient presented a mild right clumsiness with complete recovery one week after onset. Within 3 weeks after stroke the patient noticed the gradual onset of involuntary simple phonic tic consisting of an “a” sound which persists. The patient did not present motor tics or the typical Tourette syndrome co-morbidities, such as attention deficit or obsessive-compulsive disorder. © 2008 Movement Disorder Society

Key words: phonic tic; subcortical stroke; Tourette syndrome; caudate nucleus.

Motor and phonic tics are most frequently due to Tourette Syndrome (TS). There are, however, many other causes of tics, such as various acquired and congenital neurological and neuropsychiatric disorders, or exposure to a range of drugs. Acute brain injury such as stroke, central trauma, and peripheral injury has been associated with a variety of movement disorders, most frequently dystonia and tremor. Although subcortical stroke has been described as a cause of TS, stroke as a cause of simple phonic tic has not been reported.

We report an adult patient who presented a simple motor phonic tic three weeks after acute left caudate stroke.

CASE REPORT

A 71-year-old man, right-handed, ex-smoker, and with hypertension as a vascular risk factor, was admitted to the hospital due to suddenly suffering a slight clumsiness on his right side. The neurological exam showed a mild right clumsiness and a gait disturbance. On admission a CT scan did not show anomalies with the exception of a previous lacunar infarct in the right lenticular nucleus. The magnetic resonance imaging (MRI), which included a diffusion sequence, performed in the first week after stroke onset showed an acute lacunar infarct located in the tail of the left caudate nucleus (see Fig. 1).

During hospitalization the patient improved, and after seven days from admission recovered.

Within three weeks after the stroke he noticed the onset of involuntary “a” sound with no premonitory symptoms. He observed that stress exacerbated the phonic tic. He was able to suppress it voluntarily for only very short periods of time and then experienced an urge. Because of the interference of the tic, the prosody of the patient speech was not normal. The patient did not present the phonic tic during sleep. No motor tic was observed and he denied exposure to any medication or illicit drugs. Personal and family history for tics or behavioral disorders was negative.

Except for the phonic tic, the results of the patient’s physical and neurological examinations were normal, as were the neuropsychological tests. The MRI showed an acute ischemic lesion involving the left caudate nucleus and some previous lacunar strokes. Functional imaging using photon emission computerized tomography (SPECT) highlighted abnormal patterns of blood flow and metabolism in the left basal ganglia, and in the motor cortex of the left frontal lobe.

The phonic tic only partially responded to pimozide treatment, which the patient discontinued due to severe somnolence.

DISCUSSION

Tics are abrupt, repetitive movements (motor tics), or sounds (phonic tics), that are commonly preceded by a premonitory sensation of an extreme tension discomfort or other sensory phenomena.

Motor and phonic tics are usually seen in children, most frequently due to TS, but there are many other causes of tics. In a study evaluating tics and co-existing disorders, ~9% of the sample had tics associated with an effect on the basal ganglia; and stroke represented 1.2% of these.

In one of the few large prospective studies evaluating the involuntary abnormal movements following a stroke, it was observed that the frequency of these movements was ~3.7%. Chorea was the commonest movement although dystonia, tremor, and parkinsonism were also observed, but not tics.

Although the specific etiology and pathogenesis of tics cannot always be determined, in many cases of tics not related to TS a clear anatomical-clinical relationship can be established. The association of tics with well-documented lesions involving the basal ganglia suggests that these subcortical structures play an important role in the genesis of...
secondary tics. Stroke affecting the basal ganglia has been reported as producing symptoms otherwise typical of TS. We have shown, however, that an isolated simple phonic tic could be a consequence of subcortical stroke involving the basal ganglia, specifically the caudate nucleus. In the literature there is a case report of an isolated simple phonic tic related to multiple sclerosis.

Studies have demonstrated that reduced caudate nucleus volumes might be a good candidate marker for a trait abnormality in the structure of the basal ganglia in people with TS compared with healthy subjects. It has also been shown that caudate volume in children with TS can predict symptom severity of tic and obsessive-compulsive symptoms in early adulthood.

Functional MRI studies during periods of suppression have shown decreased neuronal activity in the ventral globus pallidus, putamen, and thalamus, and increased activity in cortical areas normally involved in the inhibition of unwanted impulses. Previous neuroimaging studies of TS using PET (positron emission tomography) have shown decreased metabolic activity in the caudate and thalamic areas and studies using SPECT have also identified frontal lobe metabolic differences between TS and healthy subjects. The fact that there is some evidence linking sound production and excitability changes in the motor cortex is of interest.

In our case, brain MRI showed an acute lesion in the left basal ganglia specifically in the tail of the caudate nucleus suggesting that the tic was secondary to this lacunar stroke. The SPECT, however, showed abnormal patterns of blood flow, not only in the left basal ganglia but also in the motor area of the left frontal lobe. It is possible, therefore, that disinhibition of the cortico-striatal-thalamic-cortical circuitry plays a role in its pathogenesis.

FIG. 1. Diffusion weighted magnetic resonance imaging (DW MRI) of the patient showing an acute stroke in left caudate nucleus.