SHORT REPORT

Oral dyskinesias associated with bilateral thalamo-capsular infarction

O Combarros, A Gutiérrez, J Pascual, J Berciano

Abstract
Involuntary mouthing movements indistinguishable from neuroleptic-induced tardive dyskinesia followed stroke in a woman whose computed tomographic (CT) scan showed bilateral thalamo-capsular infarction.

The commonest involuntary mouthing movements are those induced by chronic neuroleptic treatment as a form of tardive dyskinesia. Dyskinesias restricted to the oral region may present as manifestations of the blepharospasm-oromandibular dystonia syndrome; they rarely occur in the elderly or with Huntington's disease. We report a patient whose involuntary movements of the tongue were the presenting manifestation of bilateral thalamo-capsular ischaemia.

Case report
A 68 year old woman suddenly noticed continuous involuntary mouthing movements and slurred speech. Next morning she awoke with weakness in the left arm and loss of balance. She had no serious past illnesses and she had not been given any neuroleptic medication. On admission, a week after onset of the symptoms, the patient presented with continuous and repetitive writhing tongue movements with the tongue on the floor of the mouth (fig 1). These involuntary movements were readily suppressed when the patient put food in her mouth or when she talked. She could keep the tongue protruded for about 30-60 seconds without it darting back into the mouth. She also made occasional lip pursing, chewing and grimacing movements. No choreic movements were detected in the upper face, limbs or trunk. Her speech was dysarthric. There was slight left central facial weakness and moderate weakness of the left upper limb. Gait was ataxic with a tendency to fall to the left. Severe cerebellar ataxia on the left shin test was evident. Tendon reflexes were increased and the plantar response was extensor on the left. Sensation and mental status were normal. General examina-

Figure 1  Sequential photographs showing writhing tongue movements.
tion was normal with blood pressure of 140/85 mm Hg and regular pulse.

Routine laboratory tests and ECG were normal. In the third week after onset, the plain CT outlined a poorly defined radiolucent right thalamic lesion (fig 2). CT immediately after injection of contrast revealed bilateral enhancing lesions, larger on the right, involving the posterior internal capsule and lateral thalamus, with some extension into the posterior lenticular nucleus and inferior corona radiata on the right side. A delayed CT 90 minutes after contrast injection showed more prominent enhancement in the same areas (fig 2).

At the end of the third month the patient's involuntary movements had largely ceased, the left arm had completely recovered its strength, and the left leg was still dysmetric. At this time noncontrast CT showed a residual hypodensity on the lateral right thalamus.

Discussion
The involuntary mouthing movements in our patient were remarkably similar to neuroleptic-induced tardive dyskinesia. Characteristically, patients with tardive dyskinesia show repetitive stereotyped movements of tongue writhing or twisting when it's on the floor of the mouth, but they can hold their tongue protruded for any duration without involuntary retraction. The patient's clinical course with sudden onset and near complete recovery over weeks was that of stroke. In our case, plain CT during the third week after onset showed a poorly defined unilateral hypodensity, but contrast infusion revealed bilateral enhancement of the lateral thalamus and posterior internal capsule. The site of these lesions corresponds with the area of supply of the anterior choroidal artery as reconstructed by Damasio and Graff-Radford et al on diagrams of regular CT cuts. Bilateral anterior choroidal artery infarction, as described here, is rare and does not usually result in pseudobulbar mutism.

Oral dyskinesias are a very unusual accompaniment to focal ischaemic lesions. Meige's syndrome can result from infarcts of the corpus striatum or of the mesencephalic region. An association between abnormal mouthing movements and infarction in the mouth area of the cerebellar vermis has been suggested. Oral dyskinesias have not been mentioned in published series of thalamic infarcts. Bilateral thalamo-capsular ischaemia might be considered as a rare cause of involuntary mouthing movements indistinguishable from neuroleptic-induced tardive dyskinesia.

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