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STRIATAL TOE SIGN IN A 49 YEAR OLD MALE NIGERIAN HYPERTENSIVE WITH THALAMIC STROKE: A RARE PRESENTATION

Ernest.O.Nwazor[1*], Ikechukwu Chukwuocha[2], Anthony C. Anyanwu[3]

[1,2,3] Department of Medicine, Federal Medical Centre, Owerri, Imo State
Corresponding author
*Dr .Ernest. O. Nwazor
Dept of Medicine
Federal Medical Centre, Owerri, Imo State

ABSTRACT

INTRODUCTION

Movement disorders following a stroke are commonly associated with damage to the basal ganglia and thalamus.[1] Damage to deep brain nuclei, such as thalamus, from stroke is thrice more likely to result in movement disorders than when the same occurs in more peripherally located structures.[2] Amongst the various movement disorders described following a thalamic stroke is the striatal toe, which is an apparent extensor response, without fanning out of other toes unlike what is usually seen corticospinal tract lesions.[3]

Striatal toe sign, in connection with lesions located in the caudate nucleus and putamen, was initially described by Charcot and Purves-Stewart.[4] However, it is not well established that a pathology affecting these two structures is all that is needed to produce this deformity. It can also occur in a number of other non stroke-related neurodegenerative conditions, such as dystonia and parkinsonism.[5]

Striatal toe is an uncommon finding in most patients who suffer a hemiparetic stroke, and so its presence is highly suggestive of damage to deep brain structures.

Case Report

We report a 49 year old male known hypertensive referred to Neurology Clinic on account of recent history of stroke. On evaluation, he had associated slurring of speech but no facial weakness, dysphagia or double vision. He had no limb weakness, even though he had experienced that at the onset of his illness. However, of interest to him was persistent extensor deformity of his right big toe following stroke. This affected his movement significantly and caused him a lot of discomfort. Apart from this, he also experienced continuous abnormal sensory symptom, described as peppery, on the same right side of the body, including his face.

His past medical history was only significant for transient ischemic attack, which happened to him six months earlier.

He was not regular with his medications for hypertension and rarely visited hospital for routine health check up. He did not have type 2 diabetes mellitus and had no significant history of smoking and alcohol use.

Neurological examination was significant for persistent extension of the right big toe (figure 1) as well as impaired gait. Other aspects of his motor and sensory functions were essentially intact.

Available laboratory work up showed normal complete blood count, serum electrolytes, urea and creatinine, serum calcium and phosphate. Electrocardiography showed sinus rhythm with mild left axis deviation. Left ventricular hypertrophy was not noted. Serum lipid profile showed mostly normal parameters except for slightly elevated total cholesterol. Non contrast Brain Computerized Tomography scan revealed small non enhancing hypodensity in the left thalamus (figure 2)

He received drugs for blood pressure control and also for secondary stroke prevention. For his right hemisensory central neuropathic symptom, he received Pregabalin as well. In addition, he was give Clonazepam and Baclofen tablets for his striatal toe. As at the time of his last review, he had not experienced any significant improvement regarding his toe deformity and would have benefitted from botox injection, if not for its prohibitive cost and unavailability.

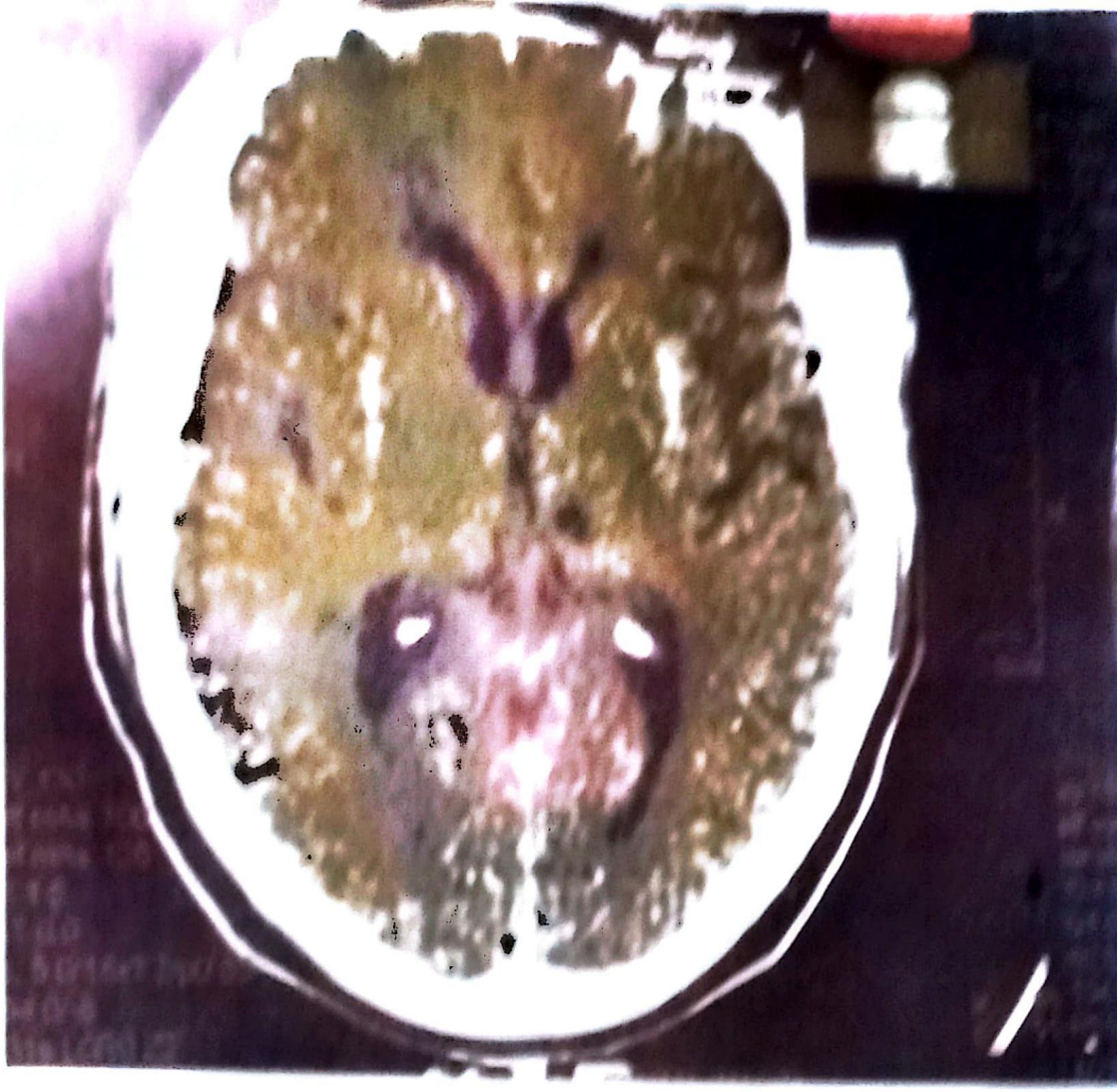

Figure 2

DISCUSSION

Striatal toe is a classical clinical finding in extrapyramidal disorders, and usually associated with a lesion occurring in the caudate nucleus and putamen,[1] and rarely seen in a thalamic stroke, like in our patient.

Thalamus is an important subcortical structure comprising various nuclei, which act as relay stations between the cerebral cortex, brainstem, cerebellum and spinal cord. It is divided into anterior, lateral and medial parts by a Y-shaped internal medullary lamina. The lateral part is further subdivided into ventral and dorsal nuclei.[6] The ventral nucleus receives projections from the output structures of globus pallidus interna and subthalamic nucleus and projects them to the motor cortex, cerebellum and spinal cord while the intralaminar nucleus influences the activity of corpus striatum. These structures are part of the circuitry that plays a major role in movement. A vascular lesion, such as stroke, to any of these nuclei can result in a motor disorder.[7]Studies on striatal foot have suggested an extrapyramidal origin and can be confused, sometimes, with dystonia, babinski sign or psychogenic striatal toe.[8] Striatal toe deformity is usually fixed and even present during sleep while dystonia commonly begins with activity and can be associated with tremors. In contrast, babinski toe sign involves fanning and flexion synergy of other muscles in the same leg in response to a stimulus.[9] Functional or psychogenic striatal toe is usually accompanied by passive plantar flexion of the first toe and can be painful. To further confirm functional etiology, forced dorsiflexion of the 2nd and 5th toe results in reciprocal plantar flexion of the first toe, with associated incongruous gait unlike striatal toe from a structural cause.[10]Treatment involves administration of botox toxin injection, with the dose guided by electromyographic activity in the affected muscles. Drugs, like Baclofen and Benzodiazepnes, are also useful, especially in a striatal toe associated dystonia and parkinsonism.[11]

CONCLUSION

Striatal toe is a rare feature of thalamic lesions and should be looked out for in patients presenting with thalamic stroke. Early detection and appropriate treatment of this condition might improve gait difficulty in the affected patients

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