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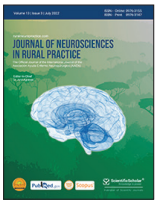

Case Report

Acute spasticity secondary to ischemic stroke involving superior frontal gyrus and anterior cingulate gyrus

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ABSTRACT

Spasticity is a velocity-dependent increase in muscle resistance associated with hypertonia after an acute stroke. Spasticity is expected to appear within a few weeks due to different mechanisms; we are reporting acute spasticity observed at the time of ischemic stroke involving the superior frontal gyrus and anterior cingulate gyrus. A healthy 64-year-old male patient suffered from subarachnoid hemorrhage secondary to aneurysmal rupture of the anterior communicating artery. He was referred to our center and treated with percutaneous transluminal coil embolization. In post-coiling, he developed acute left-sided weakness and spasticity. Magnetic resonance imaging brain showed an acute ischemic stroke involving the superior frontal gyrus and anterior cingulate gyrus. Patient follow-up showed improvement of weakness and spasticity. Therefore, acute spasticity can be related to a stroke involving the superior frontal gyrus and anterior cingulate gyrus.

Keywords: Spasticity, Ischemic stroke, Cingulate gyrus, Superior frontal gyrus, Complication, Case report

INTRODUCTION

Within the 1st weeks following an ischemic stroke, approximately 30% of patients develop spasticity.[1] It is a prevalent complication among stroke survivors. Spasticity is defined as an increase in muscle resistance to passive extension, accompanied by hyperreflexia and dependent on velocity.[2] In patients with post-stroke, spasticity may compromise mobility and functional capacity, resulting in an apparent decline in quality of life.[3] Upper motor neuron syndrome results from pyramidal fiber dysfunction caused by ischemic and hemorrhagic stroke. The patient could exhibit both positive and negative symptoms. Positive signs include clonus, flexor spasms, spasticity, rigidity, Babinski sign, and increased deep tendon reflexes. Negative signs include weakness, fatigue, coordination impairment, and loss of dexterity. Spasticity pathophysiology is not completely understood. There is an antagonist effect on the excitability effect of the anterior horn cells. This antagonist effect arises from the cortical motor regions and subcortical regions that facilitate the inhibitory effect of the descending dorsal reticulospinal tract, which originates from the ventromedial reticular formation in the brainstem. Not all cortical motor regions that enhance the inhibitory effect in the spinal cord have been investigated.[4,5] Supporting the finding of acute limb spasticity can occur simultaneously with ischemic stroke. Other studies have shown that hypertonic phenomena result from infarction of the anterior cerebral artery (ACA) or the pericallosal artery territory.[6-8] At the time of an ischemic stroke affecting the superior frontal gyrus and anterior cingulate gyrus, we observed acute spasticity.

CASE REPORT

A healthy 64-year-old male patient was referred to a hospital in Makkah City as a case of subarachnoid hemorrhage, secondary to the anterior communicating artery (ACOM) aneurysmal rupture with percutaneous transluminal coil embolization. On examination, the patient was conscious and following commands without neurological deficits. Computed tomography (CT) angiogram showed a saccular aneurysm originating from the ACOM, measured 1.1 cm × 0.7 cm × 0.9 cm [Figure 1].The patient underwent coiling of the aneurysm. In post-coiling, he developed a sudden onset of left-sided weakness associated with acute left-sided spasticity, flexed posturing in the left arm, with upgoing planter. Magnetic resonance imaging (MRI) brain showed an acute stroke involving multiple bilateral areas in right frontal parasagittal, right middle frontal gyrus, right pre-central gyrus, parietal parasagittal, left superior frontal,

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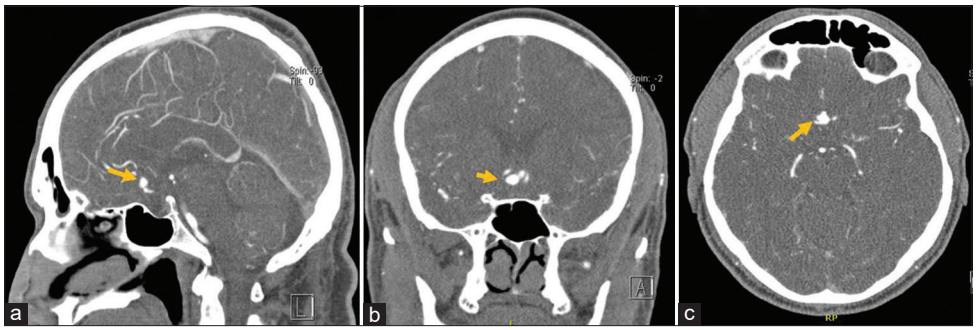

Figure 1: Saccular aneurysm originating from the anterior communicating artery measured 1.1 × 0.7 × 0.9 cm, the aneurysm is angulated posteriorly and superiorly. a: Sagittal view, b: Coronal view, c: Axial view of saccular aneurysm originating from anterior communicating artery.

and the body of the corpus callosum. The largest noted at the right frontal parasagittal area involving the cingulate gyrus [Figure 2].

CT brain was done after 4 months, showing a right frontal parasagittal ischemic lesion involving the superior frontal gyrus and cingulate gyrus [Figure 3]. On follow-up after 4 months, the weakness and spasticity improved and the patient was able to walk independently.

DISCUSSION

As previously stated, spasticity is a velocity-dependent increase in muscle tone resulting from the excitability of the muscle stretch reflex.[2] Spasticity is expected to manifest acutely or over the course of several months to a year after a stroke.[9,10] The frontal lobe of the cortex contributes to the pyramidal tract, the premotor and supplementary motor areas aid in the sequencing and control of all voluntary movements, and the anterior cingulate cortex acts as a cortical suppressant; these regions all play a significant role in muscular activity and tone.[11] Several connections between the cingulate cortex and motor areas have been mentioned, known to participate in monitoring and detection, response selection, and attention control. Moreover, the anterior cingulate gyrus is involved in pain perception, autonomic function, memory, social and effective interactions. During movement, a number of motor regions in the dorsal cingulate sulcus are active, including clusters in the dorsal midcingulate sulcus that connect to the dorsal striatum, premotor cortex, and pre-central gyrus.[8]

Earlier studies attempted to explain the pathophysiology of spasticity as the dorsal reticulospinal tract, which originates from the ventromedial reticular formation, is the principal inhibitory pathway for spinal reflex activity, resulting in spasticity in patients with acute brain injuries. Some patients with acute stroke develop spasticity in less than two weeks as a result of subcortical basal ganglia injury, but not as a result of cortical lesions.[8]

Similar case series and case reports involving a total of six patients with ACA, stroke, early spasticity, and MRI findings including the cingulate gyrus provide support for the presented case. However, further studies are required to clarify the pathophysiology of acute onset spasticity.[11,12]

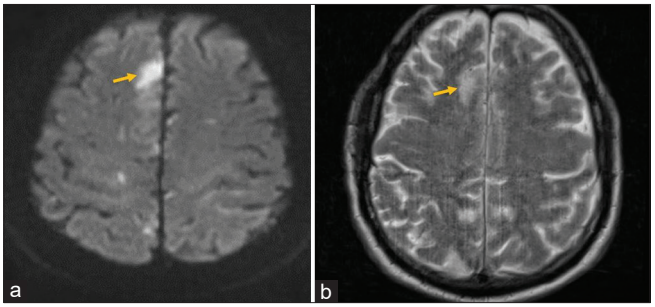

Figure 2: (a) Diffusion-weighted imaging showing multiple bilateral areas of acute ischemic stroke with the largest one noted at the right frontal parasagittal area. (b) High T2 signal intensity in multiple areas, the largest one being the right frontal parasagittal area. A-diffusion restriction along the right frontal parasagittal area most likely represent acute ischemic stroke.

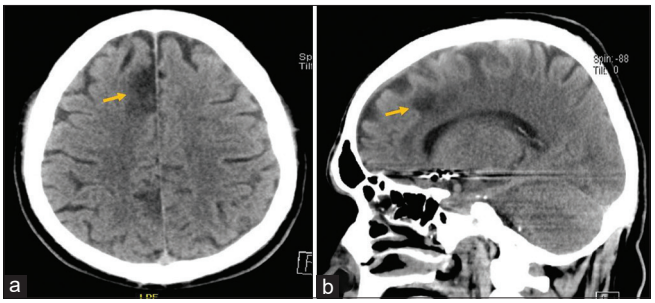

Figure 3: (a) Axial plane showing the right frontal parasagittal ischemic area. (b) Sagittal plane showing superior frontal gyrus and cingulate gyrus ischemic lesions. CT brain without contrast axial and sagittal view showing right frontal parasagittal area hypodensity most likely representing acute ischemic stroke.

CONCLUSION

The future studies to explore the role of the superior frontal gyrus and anterior cingulate gyrus in the pathophysiology of acute spasticity are suggested.

Declaration of patient consent

Patient's consent not required as patient's identity is not disclosed or compromised.

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Nil.

Conflicts of interest

There are no conflicts of interest.

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