Spasticity as a dilemma in acute stroke management in the emergency

Dear Sir,

Spasticity is considered a clinical marker of pyramidal tract involvement and is found in a stroke of chronic duration. The following is a case, in which spasticity was present as a presentation in the stroke of a few hours duration which was a dilemma in the acute stroke management in the triage about whether the occluded vessel was acutely closed or has been there since before.

A 70-year-old female presented with complaints of acute-onset left hemiparesis, dysarthria, and facial deviation for 5 h 40 min. Initial neurologic examination at the emergency revealed left-sided hemiplegia along with the left-sided facial weakness and dysarthria (NIHSS 13). The patient also had spasticity in both the left upper and lower limbs. The rest of her general and systemic examination was unremarkable.

Initial magnetic resonance (MR) imaging was suggestive of diffusion-weighted imaging (DWI) restriction at the right caudate head, putamen, and corona radiate (Fig. 1). MR angiography brain and neck vessels revealed the right internal carotid artery (ICA) complete occlusion with poor collaterals in the right middle cerebral artery (MCA) territory. There was a large area of DWI-fluid-attenuated inversion recovery mismatch involving the right ICA territory.

With a large area of the brain at risk and being eligible for endovascular mechanical thrombectomy, the patient was rushed to the cath lab for mechanical thrombectomy under monitored anesthesia. The right common carotid artery injection revealed occlusion at the origin of the right ICA (Fig. 2a). Subsequently, the left ICA injection revealed poor collaterals to the contralateral right MCA territory and right anterior carotid artery territory being supplied by leptomeningeal collaterals (Fig. 2b). With the help of ACE 68 aspiration catheter over 035 guidewire, the occlusion due to ICA clot was aspirated.

Subsequent contrast run revealed occlusion at the level of the proximal M1 segment of the right MCA which was further recanalized by aspiration technique, and final TICI 2b recanalization was achieved (Figs. 3 and 4). After an overnight observation in intensive care unit, a follow-up computed tomography scan next morning revealed mild subarachnoid bleed without any evidence of new infarct. The patient remained clinically stable throughout the period. His motor power improved to Grade 2/5 in the left upper and lower limb over the first 24 h. With dedicated physiotherapy, the patient continued to recover and achieved Grade 4/5 power on the left side. However, spasticity as initially documented on admission persisted throughout the course of his treatment.

This case highlights some very interesting clinical and radiological points: (a) The presence of spasticity during the

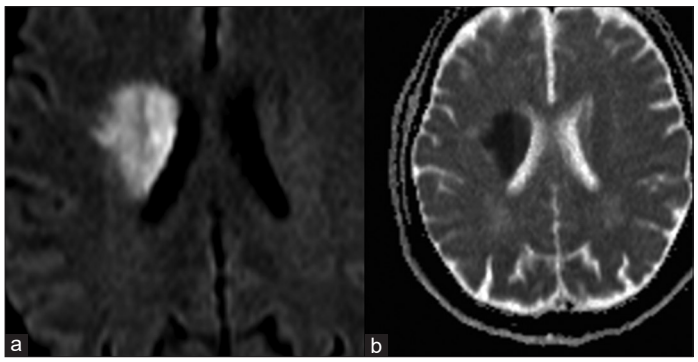

Figure 1: Magnetic resonance imaging diffusion-weighted imaging and ADC showing the right corona radiata and caudate body acute infarct

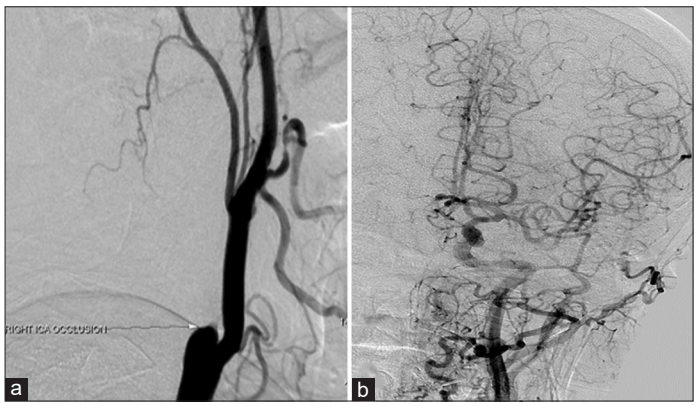

Figure 2: DSA image showing (a) the left internal carotid artery complete occlusion at its origin in the left common carotid artery injection; (b) poor collaterals into the right middle cerebral artery (MCA) territory by anterior carotid artery-MCA leptomeningeal collaterals

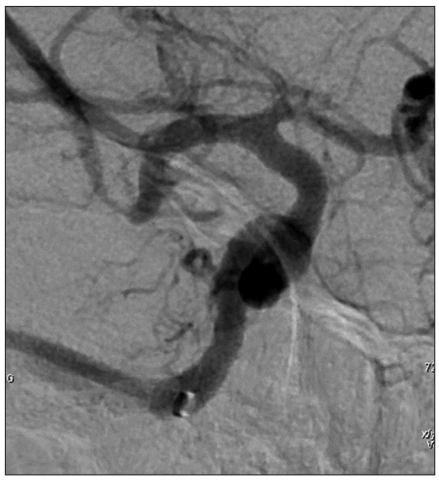

Figure 3: Post-aspiration thrombectomy of M1 occlusion achieving recanalization

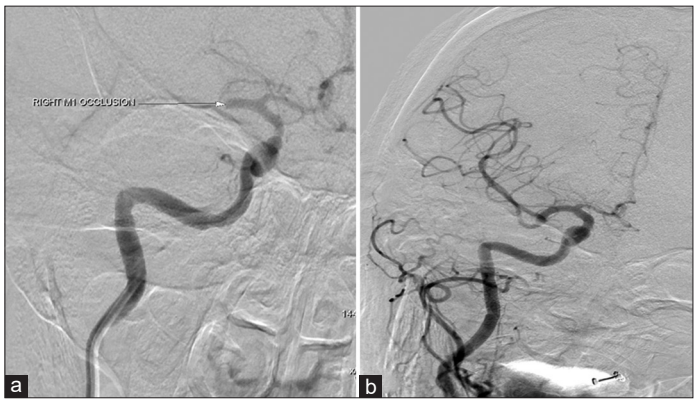

Figure 4: (a) Recanalization of proximal internal carotid artery (ICA) occlusion reveals the right middle cerebral artery M1 segment occlusion in subsequent injection; (b) DSA image showing recanalization of proximal ICA as well as distal M1 occlusion finally

initial assessment created confusion about the timeline of the symptoms. The examining physician wondered if the carotid occlusion was chronic and the patient might have had previous strokes which could explain the spasticity on the left side. (b) Furthermore, if the ICA occlusion is considered to be chronic as mentioned above, there remains a dilemma about the treatment approach in terms of mechanical thrombectomy which would be the treatment of choice in a similar acute setting but would not be done in the chronic setting. (c) The importance of detailed history taking (family was sure that there was no stiffness in the past). Imaging also showed a poor collateral status involving the affected territory which also supports the acuteness of the episode. Such points, if missed, could deprive the patient of the endovascular treatment.

The word "spasticity" is derived from the Greek word "spasticus," which means "to pull or to tug." In 1980, Lance defined it as "a motor disorder characterized by a velocity-dependent increase in tonic stretch reflexes (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex, as one component of the upper motoneuron syndrome" [1]. The exact mechanism is not completely understood but is primarily believed due to the reduction of spinal inhibitory mechanisms. Spasticity is more often found in the flexor muscles of the upper limb (fingers, wrist, and elbow flexors) and in the extensor muscles of the lower limb (knee and ankle extensors).

The degree of severity in spasticity can vary widely, from mild, episodic spasms, or stiffness to a significant problem causing constant pain and limited mobility. It can be associated with various disorders such as multiple sclerosis, stroke, cerebral palsy, spinal cord, and brain injuries, and neurodegenerative diseases affecting the upper motor neuron, pyramidal, and extrapyramidal pathways [2]. Usually, spasticity is expected to develop only weeks after an acute ischemic lesion. Studies have shown that 39% of patients with first-ever stroke are spastic after 12 months [3]. Left untreated, it gives rise to many problems, such as pain, spasms, limb contracture, and deformity.

A retrospective study done by Barlow found that spasticity can be present in acute stroke involving gray matter regions of the insula, basal ganglia, and thalamus [4]. Even white matter tracts involvement, including the pontine crossing tract, corticospinal tract, internal capsule, corona radiata, external capsule, and the superior fronto-occipital fasciculus, was also found to be significantly associated with acute spasticity.

Thus, the learning point from the above case is that spasticity does not always signify old stroke, sometimes acute stroke can also present with spasticity. A careful detailed history, along with additional information from the imaging, can lead to proper diagnosis and management of such stroke and can prevent long-term morbidity and mortality.

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REFERENCES

1. Lance JW. In: Feldman RG, Young RR, Koella WP, editors. Symposium Synopsis in Spasticity: Disordered Motor Control. Chicago: Symposia Specialistsp. 485-94.
2. Bavikatte G, Gaber T. Approach to spasticity in general practice. Br J Med Pract 2009;2:29-34.
3. Watkins CL, Leathley MJ, Gregson JM, Moore AP, Smith TL, Sharma AK. Prevalence of spasticity post stroke. Clin Rehabil 2002;16:515-22.
4. Barlow SJ. Identifying the brain regions associated with acute spasticity in patients diagnosed with an ischemic stroke. Somatosens Mot Res 2016;33:104-11.

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