

CHOREA IN ACUTE STROKE TREATED WITH THROMBECTOMY: A CASE REPORT

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Background:

Stroke may be associated with different types of movement disorders, such as hyperkinetic syndromes (hemichorea–hemiballism, unilateral asterixis, limb-shaking, dystonia, tremor, myoclonus) and hypokinetic syndromes (especially vascular parkinsonism). Stroke results in early or delayed contralateral hemichorea or hemiballism in less than 1 percent of patients, although it is the most common cause of nongenetic chorea in the hospital population.

Chorea is a hyperkinetic movement disorder caused by excessive dopaminergic activity causing loss of inhibition to the pallidum. Vascular hemichorea/hemiballism can occur due to lesion in basal ganglia, thalamus, subcortical white matter and cortex. Most stroke lesions causing hemichorea/hemiballism involves posterolateral putamen.

Case summary:

A rare case of acute chorea after acute stroke is presented.

This 66-years-old, right handed Caucasian lady presented with weakness of her right arm and right leg with some speech problems, which resolved by the time she arrived in A&E. NIHSS on examination at the ED was 0 as there was no obvious focal sign apart from abnormal movement of the right side (arm and leg). The movement was deemed to be choreoathetoid. Her past medical history included AF without anticoagulation.

CT head was nil acute with an ASPECTS score of 10, confirmed by the eASPECTS software (Brainomix, Oxford). CT angiography of the carotids showed a hyperdense M2 segment of left MCA. The multidisciplinary team decided upon IV thrombolysis to be immediately followed by thrombectomy using aspiration. Thrombolysis with IV tPA (Alteplase) did not produce any noticeable improvement.

DSA selective injections demonstrate the M2 occlusion. Using the Penumbra aspiration device (Ace 68) 2 clots were removed with two aspirations. A small distal clot remained but TIMI/TICI 2b was achieved so it was expected that, over time, autolysis would resolve the remaining clot. 30 seconds after restoring blood flow the choreoathetotic movements ceased. The patient was brought to the intensive care unit for further monitoring, which was uneventful.

Discussion:

It was possible to advance an ACE 68 into a distal M2 branch. A CT perfusion scan was redundant in the presence of infarct volume as determined by e-ASPECTS. Only minimal established lacunar damage was later demonstrated on re-imaging as well as some haemorrhagic imbibition.

Conclusion:

This is a rare presentation of acute ischaemic stroke, not presenting with hypokinetic disorders but rather presenting with choreatic movement, where a basal ganglia branch was found to be occluded. Endovascular thrombectomy can be used in small vessel occlusion.