Artery of foramen rotundum: guilty or innocent?
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DESCRIPTION
Hemifacial spasm (HFS) is defined as involuntary, irregular clonic or tonic movement of muscles innervated by the ipsilateral seventh cranial nerve.1 While HFS is often attributed to a cross-compressive effect of an artery or arteries at the root exit zone of the facial nerve, there are other aetiologies that should be considered in the differential diagnosis.

A woman in her late 60s visited a hospital due to a headache and involuntary muscular contractions of the left face. Symptoms started 3 years ago, initially affecting the left eyelids and then involving the ipsilateral lower facial muscles with gradually increased intensity and frequency of spasms during the last 6 months. Her medical history was negative except for hypertension. Physical examination did not reveal any abnormality other than HFS.

Brain MRI showed no associated neurovascular lesions. Cerebral angiography revealed an occlusion of the left cervical internal carotid artery (ICA). However, the artery of foramen rotundum anastomosed with the cavernous ICA and supplied the collateral flow (figure 1).

Hyperactive facial motor nucleus has been suggested as a possible mechanism of HFS associated with vascular migraine. Cluster headache is postulated to be associated with an abnormal interaction between trigeminal afferents and cranial parasympathetic efferents.1 The trigeminal nerve in conjunction with the artery passes the foramen rotundum, and the excitability of facial motoneurons in HFS is modulated by afferent trigeminal inputs.2 3 The presence of collateral circulation via the artery of foramen rotundum may give an opportunity to have a benign neurological course but may exacerbate the HFS.

Learning points
▸ While hemifacial spasm (HFS) is often attributed to a cross-compressive effect of an artery or arteries at the root exit zone of the facial nerve, there are other aetiologies that should be considered in the differential diagnosis.
▸ Hyperactive facial motor nucleus has been suggested as a possible mechanism of HFS associated with vascular migraine. The trigeminal nerve in conjunction with the artery passes the foramen rotundum, and the excitability of facial motoneurons in HFS is modulated by afferent trigeminal inputs. The presence of collateral circulation via the artery of foramen rotundum may exacerbate the HFS.

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REFERENCES