

Commentary

Isolated ocular motor cranial nerve palsy may be a challenging diagnostic process, sometimes without an obvious underlying cause. Among the main etiologies, one can consider: Metabolic, inflammatory (in the context of demyelination or not), infectious, traumatic, malignancy, or vascular.^[1]

Vascular etiology of isolated ocular motor cranial nerve palsy is generally of compressive nature (microvascular compression). In this issue of the *Journal of Neurosciences in Rural Practice*, our colleagues from Japan, Arishima and Kikuta, present a well-documented case of isolated abducens nerve palsy induced by vascular compression of vertebrobasilar dolichoectasia.^[2] Most of all, they have clearly demonstrated the importance of accurate neuroimaging to underscore the nonaneurysmal vascular compression. Actually, according to their review of literature, even though conventional computed tomography or magnetic resonance imaging (MRI) can show these pathological conditions in patients with isolated ocular palsy, cases without radiological abnormality can occur.

Currently, neurovascular compression or other vascular abnormalities around cranial nerves can be more easily demonstrated using magnetic resonance angiography (MRA); however, most of all specific sequences to evaluate brain blood vessel such as heavy

T2-weighted imaging (WI), constructive interference in steady state (CISS), and fast imaging employing steady-state acquisition (FIESTA) neurovascular compression or other vascular abnormalities around cranial nerves can be demonstrated. FIESTA sequence, for example, provides higher contrast resolution between cerebrospinal fluid, arteries, and the adjacent structures such as cranial and spinal nerves.^[3]

Among the ocular motor nerves, the fourth (trochlear nerve) and third (oculomotor nerve) ones are less likely a site of vascular involvement. On the other hand, the sixth nerve is commonly affected by aneurysmal or nonaneurysmal vascular abnormalities. In a large series of 109 patients with isolated third, fourth, and sixth cranial nerve palsies, 22 had cranial nerve III palsy, 25 had cranial nerve IV palsy, and 62 had cranial nerve VI palsy. Moreover, a cause other than presumed microvascular ischemia was identified in 18 patients, and the presence of one or more vascular risk factors (diabetes, hypertension, hypercholesterolemia, coronary artery disease, myocardial infarction, stroke, and smoking) was significantly associated with a presumed microvascular cause.^[4]

From a clinical perspective, binocular diplopia is a key feature in any patient with ocular motor nerve palsy. Binocular diplopia is defined as a double vision with both eyes opened. When one of the eyes is closed,

double vision usually remits. Exclusive monocular diplopia is generally of ophthalmologic nature rather than neurological, including astigmatism, keratoconus, pterygium, cataracts, among other causes.^[5]

The complete isolated third cranial nerve palsy (divergent strabismus, mydriasis, and ptosis) has generally and underlying posterior communicating artery (PCoA) aneurysm. This is different when one finds an incomplete third cranial nerve palsy, more likely of metabolic nature, for example, diabetes. MRI usually displays this type of aneurysm, notably those larger than 3 mm. Recently, a systematic review of literature and meta-analysis, including 11 relevant studies involving 384 patients with third nerve palsy caused by PCoA aneurysms at baseline, compared whether treating these patients with clipping or coiling could positively influence recovery. The authors found that the overall complete oculomotor nerve palsy recovery rate was 42.5% in the coiling group compared with 83.6% in the clipping group, suggesting that surgical clipping of PCoA aneurysms causing third nerve palsy achieves better outcomes recovery than endovascular coiling, particularly in the case of ruptured aneurysms.^[6]

A list of various etiologies, some of them very rare, is attributed to isolated trochlear nerve palsy. However, a retrospective review enrolling 32 patients showed that 56% had a microvascular etiology with diabetes mellitus, hypertension, or both being the most common risk factors. Furthermore, the prognosis for complete and spontaneous resolution of microvascular fourth nerve paresis was excellent, with 89% completely resolved within 10 months.^[7]

Finally, abducens nerve palsy is one of the most common isolated cranial nerve palsies with clear-cut etiology and straightforward management. However, there are several cases whose etiology and management are unclear, and vast mimics are possible. Microvascular ischemia in the context of diabetes mellitus, for example, is a frequent cause of acute isolated sixth cranial nerve palsy.^[8] Nonetheless, it is worthwhile to remember that the sixth nerve can be a false localizing sign, and that specific imaging, such as MRA or MRI-WI, CISS, FIESTA may need to address microvascular compression as showed in this issue in the Case Report by Arishima and Kikuta.^[2]

In conclusion, isolated ocular motor cranial nerve palsy should be carefully addressed, first, paying attention

to the clinical presentation, second, to the underlying etiology using whenever possible modern neuroimaging techniques, particularly to address microvascular compression, and finally, targeting clinical or surgical treatment to the appropriate cause.

Andre C. Felicio

Department of Neurology and Brain Institute, Hospital Israelita Albert Einstein, São Paulo, SP, Brazil

Address for correspondence:

Dr. Andre C. Felicio,
Hospital Israelita Albert Einstein, 627, Jardim Leonor,
São Paulo, SP, Brazil.
E-mail: cf.andre@gmail.com

References

1. Kung NH, Van Stavern GP. Isolated ocular motor nerve palsies. *Semin Neurol* 2015;35:539-48.
2. Arishima H, Kikuta K. Magnetic resonance imaging findings of isolated abducent nerve palsy induced by vascular compression of vertebrobasilar dolichoectasia. *J Neurosci Rural Pract* 2017;8:124-7.
3. Kulkarni M. Constructive interference in steady-state/FIESTA-C clinical applications in neuroimaging. *J Med Imaging Radiat Oncol* 2011;55:183-90.
4. Tamhankar MA, Biousse V, Ying GS, Prasad S, Subramanian PS, Lee MS, *et al*. Isolated third, fourth, and sixth cranial nerve palsies from presumed microvascular versus other causes: A prospective study. *Ophthalmology* 2013;120:2264-9.
5. Lee MS, Volpe NJ. Double Vision. *Curr Treat Options Neurol* 2001;3:383-8.
6. Gaberel T, Borha A, di Palma C, Emery E. Clipping versus coiling in the management of posterior communicating artery aneurysms with third nerve palsy: A systematic review and meta-analysis. *World Neurosurg* 2016;87:498-506.e4.
7. Khaier A, Dawson E, Lee J. Clinical course and characteristics of acute presentation of fourth nerve paresis. *J Pediatr Ophthalmol Strabismus* 2012;49:366-9.
8. Chi SL, Bhatti MT. The diagnostic dilemma of neuro-imaging in acute isolated sixth nerve palsy. *Curr Opin Ophthalmol* 2009;20:423-9.

This is an open access article distributed under the terms of the Creative Commons Attribution-NonCommercial-ShareAlike 3.0 License, which allows others to remix, tweak, and build upon the work non-commercially, as long as the author is credited and the new creations are licensed under the identical terms.

Access this article online	
Quick Response Code: 	Website: www.ruralneuropractice.com
	DOI: 10.4103/0976-3147.193566

How to cite this article: Felicio AC. Commentary. *J Neurosci Rural Pract* 2017;8:127-8.