

Authors: M.C. Acciarri¹, S.M. Angelocola¹, F. Girelli¹, C. Rinaldi¹, P. Di Bella¹, F. Logullo², L. Provinciali¹

¹ Neurological Clinic, Department of Experimental and Clinical Medicine, Marche Politecnico University, Ancona, Italy

² Neurology Operating Unit, Hospital of Macerata

BACKGROUND

Neuropathy of the third cranial nerve is characterized by the presence of deficits in the vertical and lateral inward eye movements, divergent strabismus, crossed diplopia, eyelid ptosis, and mydriasis. When neuropathy is partial it may have only a few of these events; especially if it is concerned the upper branch, only the superior rectus muscle and the elevator eyelid muscle can be affected. The main causes are microvascular injury, trauma, compression, immunological and inflammatory damage. The prognosis depends on the cause, that once solved, leads to a spontaneous recovery within a few months; rarely strabismus and ptosis need correction with prismatic lenses, botulinum toxin or surgery.

CASE REPORT

A 74 years-old woman has come to our attention because of the appearance, a few weeks before, of diplopia in vertical and oblique look upwards to the right and of right eyelid ptosis, that cannot be modified by fatigue maneuvers.

In the medical history there wasn't significant diseases, except for a mild hypertension in treatment. Physical examination revealed a deficit of the right elevator eyelid muscle and the right upper rectus muscle, normal and reagent pupils and the absence of an evident strabismus: this findings were compatible with a partial neuropathy of the right oculomotor nerve.

Laboratory tests had not found anything significant; the anti-AChR antibodies were negative and the repetitive nerve stimulation did not confirmed the diagnostic hypothesis of Myasthenia Gravis with ocular onset.

MRI of the brain with Angio-MRI study (Fig. 1) has instead revealed a 15 mm saccular aneurysm with wide collar of the vertex of the basilar artery, causing compression of the right cerebral peduncle and the right ponto-mesencephalic junction. The angiography of cerebral vessels (Fig. 2) confirmed the presence of the aneurysm that was treated with the endovascular placement of a flow diversion stent with complete exclusion of the aneurysm from the cerebral circulation.

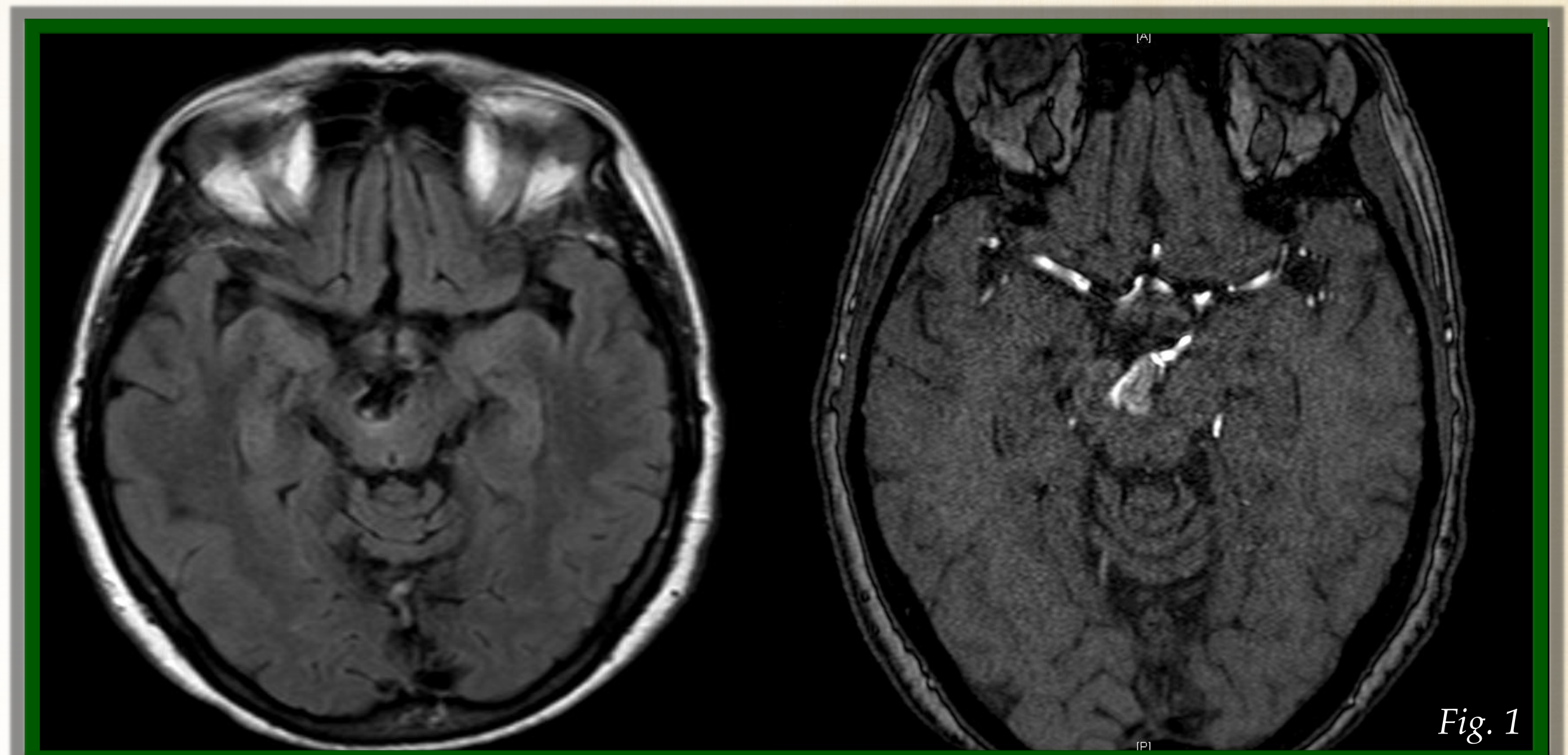


Fig. 1

The patient started a dual antiplatelet therapy (clopidogrel 75 mg and acetylsalicylic acid 100 mg every day) for three months and then only with acetylsalicylic acid 100 mg every day for one year. The cerebral Angio-CT scan performed after three months have confirmed the complete exclusion of the aneurysm from the cerebral circulation. Clinically persists right eyelid ptosis; diplopia is absent.

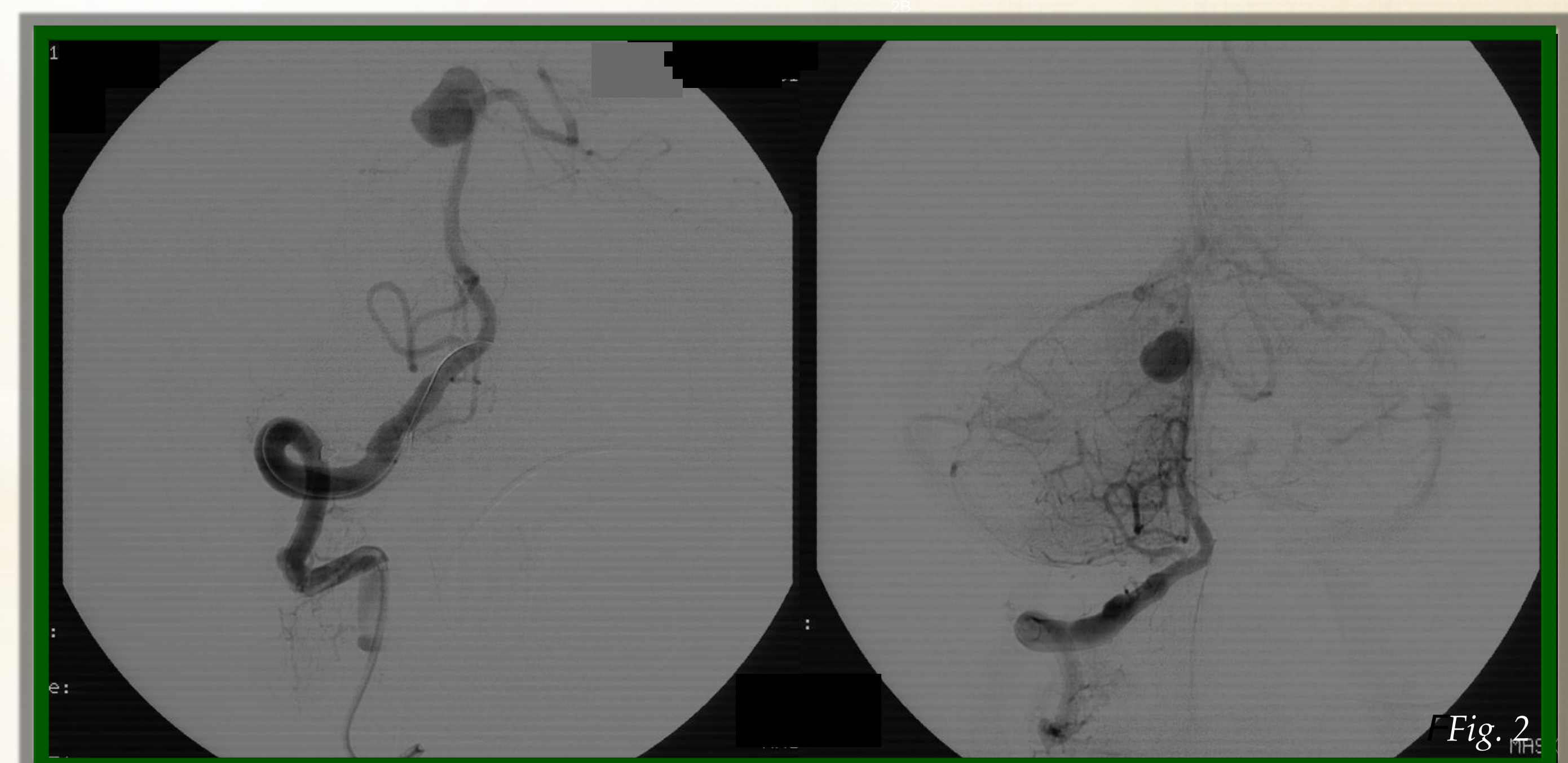


Fig. 2

CONCLUSIONS

This case report describes a possible cause of partial compressive neuropathy of the third cranial nerve, that can mimic the clinical features of an ocular Myasthenia.

REFERENCES

- Shimizu M. et al. Third nerve palsy due to local inflammation associated with vascular compression: a case series. *J Neurol Sci.* 2016 Aug 15; 367:365-7
- Binyamin TR. et al. Resolution of third nerve palsy despite persistent aneurysmal mass effect after flow diversion embolization of posterior communicating artery aneurysms. *J Clin Neurosci.* 2016 Sep; 31:207-9.