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Horner syndrome in fibromuscular dysplasia without carotid dissection

A 62-year-old female was referred for right-sided lid ptosis and periorbital pain of a 1-month duration. She denied diplopia or any significant fluctuations in her ptosis, which had begun acutely 1 month before. She denied any history of trauma and had not noticed anisocoria previously. The patient had a complex history of chronic rhinosinusitis requiring endoscopic debridement with multiple revisions, the most recent of which was 6 weeks before her presentation. An episode of preseptal cellulitis heralded that operation and further complicated the etiology of her periorbital pain.

Upon ophthalmologic examination, her visual acuity was 20/20-1 OD and 20/30 OS. Unilateral right-sided ptosis was evident with an unequal upper marginal reflex distance (right 1 mm, left 5 mm) and asymmetric palpebral fissures (right 6 mm, left 10 mm). Levator function was symmetric (14 mm OU), and there was no fatigable component to the ptosis. Pupils were anisocoric (right 4.5 mm, left 6 mm) with a brisk response to light (right 2.5 mm, left 4 mm) although, interestingly, the anisocoria was not more prominent in the dark. There was no relative afferent defect. No periorbital swelling, facial rashes, or other skin findings were noted, and the remainder of the ocular examination was unremarkable.

To further evaluate for Horner syndrome (HS), 2 drops of 0.5% apraclonidine were instilled in each eye. This resulted in reversal of the anisocoria and improvement in ptosis, confirming a diagnosis of HS. Subsequent magnetic resonance imaging (MRI) of the brain showed only chronic small vessel disease and sinus pathology (mucosal thickening in the bilateral frontal, maxillary, and sphenoid sinuses and ethmoid air cells and small fluid levels in the maxillary sinuses) consistent with prior disease but no acute parenchymal infarct, hemorrhage, or mass. Brain and neck magnetic resonance angiography (MRA) showed no flow-limiting stenosis, aneurysm, or dissection. Neck MRA, however, showed ectatic distal internal carotid arteries (ICA), the right greater than the left, and multiple areas of narrowing and dilatation of the ICA and vertebral arteries bilaterally (Figs. 1 and 2). These findings are consistent with multifocal fibromuscular dysplasia (FMD).[1] Given the patient's smoking history, a lower neck mass or apical lung tumour affecting the right sympathetic chain was also considered, but follow-up imaging to the level of T2 revealed no such lesion. The neck MRI also confirmed the absence of ICA dissection. Although HS from an unidentified cause, with only coincidental radiological findings, could not be excluded, the more severe right-sided disease was the most likely etiology of the unilateral HS.

FMD is a nonatherosclerotic, noninflammatory vascular disease that may result in arterial stenosis, occlusion, aneurysm, or dissection.[1] A histological classification system, based on the affected vascular layer, was published by Harrison and McCormack in 1971, but the American Heart Association's proposed system, based on angiographic patterns, is more appropriate in this case.[2] Intimal fibroplasia causing focal arterial constriction characterizes "focal FMD," whereas multifocal disease, exhibited in our patient by the multiple areas of narrowing and dilation, indicates medial fibroplasia.[1] Traditionally, FMD was seen as a disease of young, premenopausal women but, although 90% of FMD cases occur in females, our 62-year-old patient was a typical age for presentation based on the most recent literature. In fact, the average age on diagnosis is 51.9 (13.4) years, with a range from 5 to 86 years.[3]

Secondary hypertension caused by FMD-related renovascular disease is a well-documented entity that can result in end-organ damage from hypertensive emergency and/or chronic hypertension, but FMD is also an important cause of cerebral aneurysm and subarachnoid hemorrhage as well as mesenteric ischemia and infarct.[4,5] The renal arteries are most commonly affected with involvement in as many as 75% of FMD cases; in the largest series, however, 32% had vertebral or ICA involvement, as in this case.[6,7]

HS is an uncommon manifestation of cerebrovascular FMD; it was the presenting sign in only 4.7% of patients in the U.S. Registry for FMD. Although it is not uncommon to have a carotid dissection due to FMD or for a dissection to present with HS, our review of the literature found no cases of FMD presenting as HS without concurrent ICA dissection.[3,8] Positive apraclonidine tests have been reported in HS caused by lesions of all 3 segments (central, preganglionic, and postganglionic) of the sympathetic pathway.[9] As such, radiologic studies could not be targeted on a particular nerve segment and a comprehensive imaging workup was initiated.

Catheter-based angiography is the diagnostic gold standard for FMD. Although MRA has been well validated for cases of renal FMD with a sensitivity of 97% and specificity of 93%, no such studies have confirmed the utility of MRA in cerebrovascular FMD.[1,10] In this case, carotid artery dissection had to be considered given the ipsilateral pain in conjunction with HS, and MRI and MRA are adequate means of assessing carotid or vertebral arterial dissection.[11,12] Interestingly, the patient had been seen regularly over the prior 2 years by

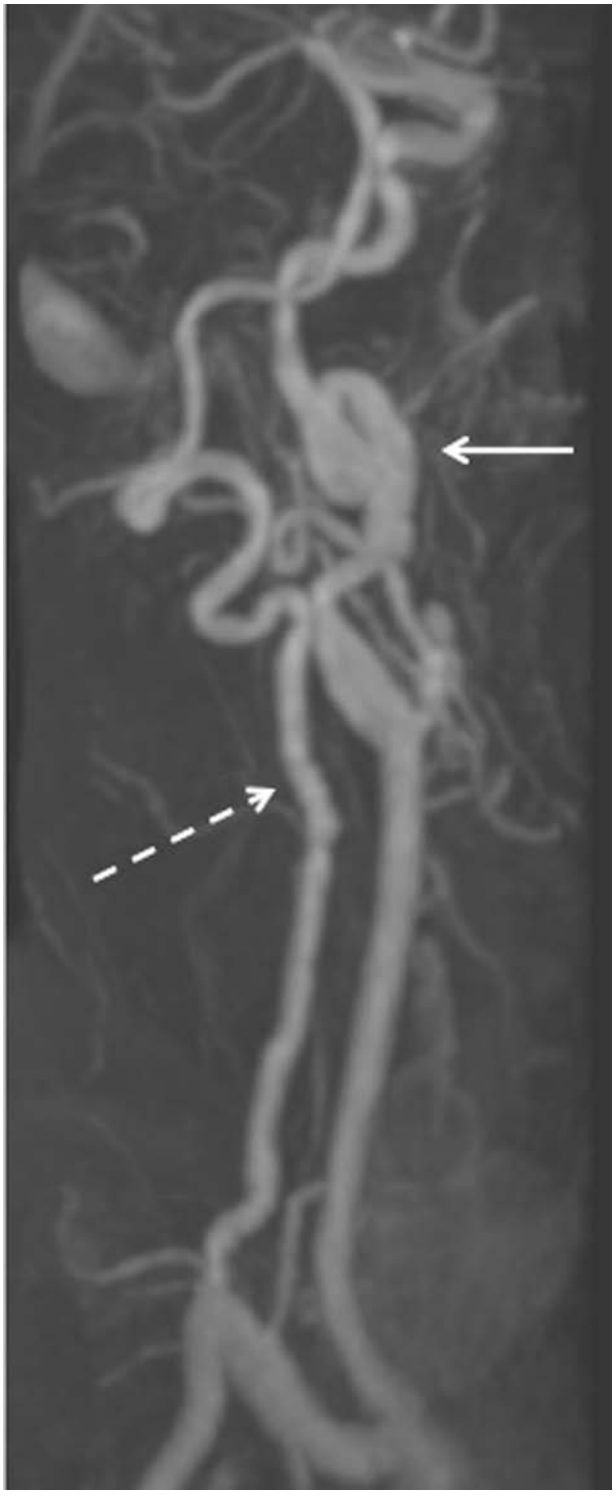

Fig. 1—Magnetic resonance angiography of right neck vessels. The figure shows multifocal irregularity in right vertebral artery (dashed arrow) and irregularity and ectasia in right internal carotid artery (solid arrow).

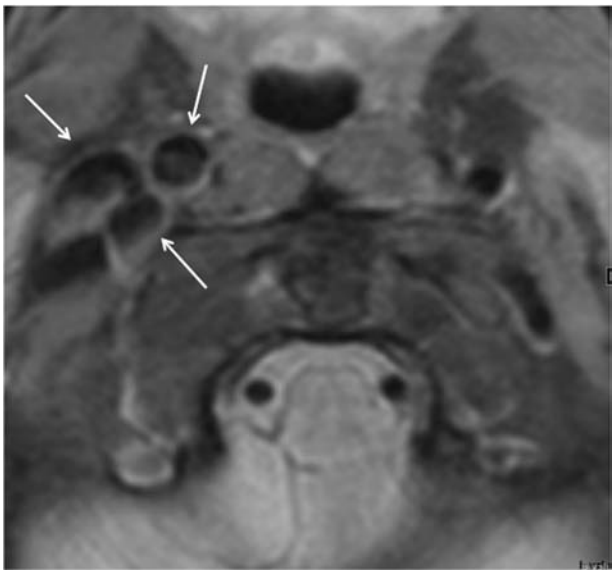

Fig. 2—Axial fat-saturated proton density image. Image at the level of the solid white arrow in the previous figure shows multiple loops in the right internal carotid artery, as expected from the magnetic resonance angiography, but no evidence of a dissection, which would produce wall thickening and signal hyperintensity.

colleagues in otolaryngology without previous mention of ptosis. HS may have suddenly manifested between clinic visits because the FMD simply disrupted enough of the ICA plexus to prevent sympathetic innervation. However, the patient had also been started on 37.5 mg of venlafaxine daily, a selective serotonin-norepinephrine reuptake inhibitor (SNRI), just 6 days before being evaluated for HS. SNRIs have been suggested to cause mydriasis because of their adrenergic effects, weak anticholinergic activities, or increased levels of serotonin.[13] Venlafaxine has been implicated in case reports for unmasking HS, likely because of the aforementioned systemic effects on neurotransmission.[14] This relative increase in sympathetic tone may precipitate a more pronounced anisocoria by causing contralateral mydriasis (as the denervated side is unaffected) and making HS more apparent to either the patient or clinician.

Although hypertension is the most common presenting sign of FMD, hypertension itself has not been implicated in the development of FMD, and its role in spontaneous cervical artery dissection is unclear, at best.[1,15–17] Most importantly, our patient exhibited HS without evidence of dissection. Although venlafaxine appears to have a small, sustained effect on blood pressure at high doses (>300 mg/day), not only was our patient taking a much smaller dose than necessary to significantly elevate blood pressure in the acute phase of therapy (initiation to 6 weeks), but also hypertension was not likely a primary process in the HS.[15,18] Although an interesting correlate, SNRI therapy in our patient was more likely to make the anisocoria more discernable than to cause progression of the disease itself.

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Chronic mucocutaneous candidiasis presenting as *Candida* endophthalmitis

A 38-year-old female from Montreal, Canada, consulted for sudden, painless vision loss in her left eye 5 days before presentation. Her medical history was known for bipolar disease, hypothyroidism, and clinically diagnosed truncal tinea versicolor (Fig. 1).

Her history was largely unremarkable except for occasional ingestion of beef tartar. She denied any chills or fever and was afebrile upon physical examination. At initial visit, her visual acuity was 20/20 OD and counting fingers OS. Slit-lamp examination of the left eye showed 1+ cells and 1+ flare in the anterior chamber as well as 2+ vitreous cells and 1+ vitreous haze as per the Standardization of Uveitis Nomenclature working group grading scheme.[1] Dilated fundus examination revealed an exophytic white lesion in the macula (Fig. 2).

A clinical diagnosis of panuveitis caused by probable ocular toxoplasmosis OS was established. Baseline uveitis workup was initiated while empirical therapy with pyrimethamine (25 mg PO daily), folinic acid (10 mg PO every second day), and clindamycin (300 mg PO QID) was started on the day of the initial presentation. The patient also received routine topical uveitis treatment (prednisolone 1% q1hr, dexamethasone ointment 0.1% qHS, and homatropine 2% BID) in her left eye and oral prednisone 60 mg daily, started 48 hours after the initiation of antiprotozoal treatment. Her initial workup was notable for mild absolute eosinophilia and negative *Toxoplasma gondii* serology (immunoglobin [Ig]M and

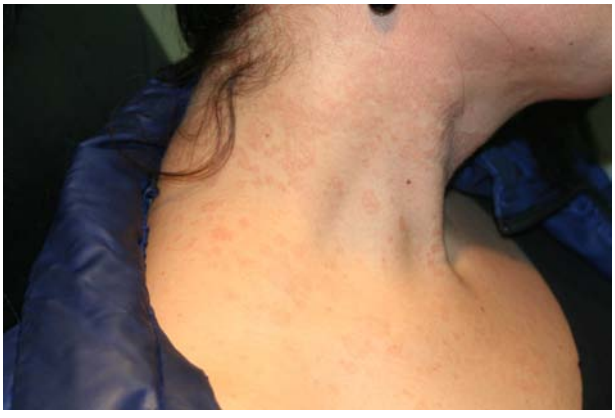

Fig. 1—Tinea versicolor lesions of neck.