

A RARE PRESENTATION OF HORNER'S SYNDROME

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History and Physical

We present the case of a 66 year old retired GP who presented with acute onset dysphagia, drooping of left eyelid and pupil asymmetry associated with left face and arm numbness.

He reported persistent non-productive cough over last few days and was complaining of mild neck pain.

Neurological examination in addition to Horner's syndrome, there was uvula deviation to left.

There was full range of eye movements and no ophthalmoplegia. There was no fatigability or features of myasthenia. Neck flexion and extension were strong. There was no facial weakness. Palatal movements were reasonable; the speech and language team had noted very slight asymmetry on phonation. His speech was good. Gait was normal with reasonable tandem gait. He had normal tone and power in his limbs. Reflexes were symmetrical. Plantar responses were down going. Pinprick sensation was normal in his limbs

Bloods

Atypical pneumonia screen (mycoplasma and legionella) was negative. ANA & pANCA were weakly positive but MPO and PR3 were negative. He was positive for lupus anticoagulant and was planned to repeat in 12 weeks for confirmation.

Chest X-ray: Normal heart size. Lung and pleural spaces were clear.

MRA

There is a short segment localised dissection seen in the left internal carotid artery subjacent to the skull base. It is seen on approximately 3 - 4 axial slices which suggest that the dissection is over a length of 2-2.5 cm. There is however no narrowing or obstruction of the left distal ICA. Normal calibre and flow is seen across the entire length of the left ICA up to the left carotid siphon

A diagnosis of spontaneous left carotid artery dissection was made possibly traumatic induced by cough. This is an uncommon case of isolated cranial nerve palsies. Commonly patients presents with stroke, headache, facial pain and Horner's syndrome with up to 16% having cranial nerve palsies.

The patient was also diagnosed with Villaret's syndrome.

Indication for Intervention

There was no indication for intervention in this case as endovascular and surgical repair of the internal carotid artery should be considered for patients with deteriorating or fluctuating neurologic symptoms resulting from thromboembolism or cerebral hypo perfusion refractory to conservative medical management and only if there is good collateral circulation.

We discussed the options between continuing on his Rivaroxaban or switching to an

antiplatelet medication. Studies such as the CADISS Trial have shown no benefit of one treatment over the other. Obviously, with anticoagulation there remains a small risk of extension dissection developing into neural haematoma and other adverse events caused by bleeding. However, on the flip side it is paroxysmal atrial fibrillation if we take him off his oral anticoagulation then he is at risk of embolic ischaemic stroke.

