ONE AND A HALF SYNDROME:

an uncommon presentation of a common problem

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Introduction

One and a half syndrome (OAHS) is a rare disorder in which movement in both eyes is affected. It is characterized by conjugate horizontal gaze palsy in one eye (referring to the one conjugate palsy) and an internuclear ophthalmoplegia in the other (referring to the half a conjugate gaze palsy) (1).

Case summary

History of presenting complaint

49 year old female with a history of chronic dizziness presents to the emergency department after awakening with sudden worsening of her dizziness, double vision, headache and the inability 'to look to her right'.

Past medical history

Hypertension, Type 2 diabetes mellitus, Hyperlipidaemia, Obesity, Hypothyroidism, Osteoarthritis of knee, vitamin B12 deficiency

Social History

Current smoker, minimal alcohol intake.

Normally fit and independent. Works as receptionist

Medications

Losartan, betahistine, hydroxocobalamin, tramadol, atorvastatin, amitriptyline, citalopram, levothyroxine,

Physical examination

Observations: afebrile, BP 184/98, HR 80, RR=16, SpO2 97% RA, GCS 15

Exam: Ocular movements showed conjugate horizontal gaze palsy to her right eye with limited adduction in her left eye. Nystagmus was present when left eye abducted. Gross visual acuity was normal. Sensation, tone, power and reflexes were maintained in all limbs.

Laboratory tests

Blood tests were unremarkable aside from a raised lipid profile and blood glucose.

Imaging

MRI Brain: A tiny midline focus of restricted diffusion in the pontine tegmentum suggestive of acute lacunar infarct. Other intracranial structures appear normal (Fig.1).

Clinical Course

Patient treated for acute ischemic stroke and started on immediate oral antiplatelet therapy following a swallowing assessment

Admitted to the local stroke unit for further work up of her condition

ECG and cardiac telemetry showed normal sinus rhythm

Echocardiogram was unremarkable

Carotid dopplers showed no significant stenosis

Seen by diabetic specialist nurse who started on metformin and gliclazide for glycaemic control.

Stroke team increased dose of anti-hypertensives and her statin.

She was seen by therapy team whilst as an inpatient and once deemed therapy fit she was discharged home.

The patient was seen by orthoptics and fitted with prisms in her glasses to aid with her diplopia.

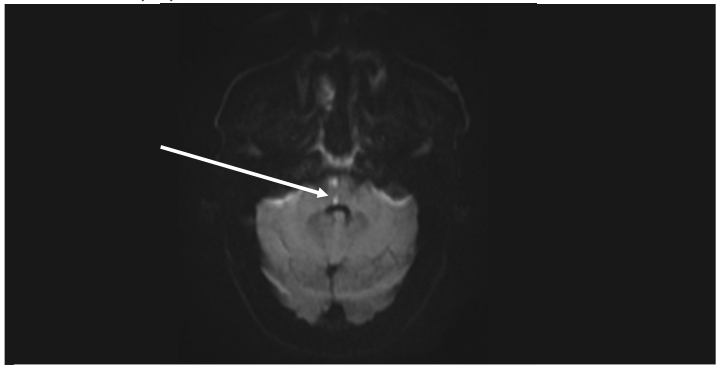

Figure 1: Diffusion Weighted MRI Brain showing infarct in pontine tegmentum

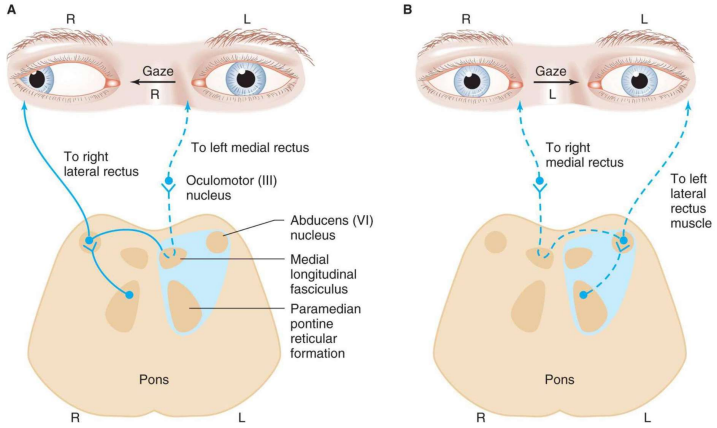

Figure 2: Visual pathways affected in OAHS during horizontal gaze (5)

Discussion

Any disease which can cause a lesion in the pontine region can account for the findings in OAHS. Lesions may be caused by inflammatory, traumatic, infiltrative, neoplastic and vascular processes. The most common etiology is vascular, mainly ischemic infarctions but may also include hemorrhages, aneurysms and arteriovenous malformations (2).

A pathological lesion in the dorsal pontine tegmentum involving the ipsilateral Medial Longitudinal Fasciculus (MLF), the ipsilateral Paramedian Pontine Reticular Formation (PPRF) and Abducens Nuclei may result in OAHS (Fig.2).

When initiating horizontal gaze, neuronal impulses from the PPRF project to the ipsilateral abducens nuclei. The abducens nuclei is vital for horizontal movement to the ipsilateral eye but also activates the contralateral MLF which carry fibres ultimately leading to the medial rectus in the contralateral eye (3).

Diagnosis of OAHS is made by clinically by full ocular motility exam and confirmed by radiological findings. MRI is the imaging modality of choice to identify the underlying neuropathic lesion (4).

Learning Point

Although this case shows an atypical presentation of cerebrovascular accident (CVA), this patient possessed many of the relevant risk factors for ischemic stroke. Alongside this, and the presence of sudden focal neurology, CVA should be foremost suspected and specific neuro-imaging should not be delayed.

References

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