Eight-and-a-half syndrome: an unusual presentation of brainstem infarction

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Case report

A 68-year-old man known to have non-insulin-dependent diabetes, hypertension and hypercholesterolemia presented with a 2-day history of abnormal sensation on the left side of his face and associated weakness of his right arm and leg. The symptoms presented with a sudden onset and had a stuttering course over the next 2 days at which point he presented to hospital.

Initial examination revealed a left lower motor neurone VII nerve type weakness (Figure 1) with associated upper motor neurone type hemiparesis of his right upper and lower limbs. Further eye examination revealed total left horizontal gaze paresis and partial right horizontal gaze paresis with limitation of adduction of the right eye. Right eye abduction and vertical eye movements were preserved (Figures 2–4). Jump saccades were present on right gaze and absent on left gaze.

His initial investigations revealed normal electrolytes, blood profile and liver functions. An autoimmune screen, ESR and treponemal antibody testing did not reveal any abnormality either. Diffusion weighted magnetic resonance imaging revealed a left caudal pontine infarct (Figure 5). Further CT angiography did not reveal any evidence of fresh vertebro-basilar thrombi or vertebral dissection.

On the basis of his presentation and his neuroimaging, a diagnosis of Eight-and-a-half syndrome was put forward. The patient continues to undergo neuro-rehabilitation on the Stroke ward.

Discussion

Eight-and-a-Half syndrome is the combination of ipsilateral lower motor neuron VIth and VIIth nerve palsy, inter-nuclear ophthalmoplegia and ipsilateral gaze paralysis. The syndrome is a combination of ipsilateral seventh lower motor neurone paresis associated with partial horizontal gaze palsy. The affected nuclei may include either

- combination of the ipsilateral VIth and VIIth nerve nucleus and the ipsilateral medial longitudinal fasciculus or
- combination of ipsilateral VIth and ipsilateral paramedian pontine reticular formation along with the ipsilateral medial longitudinal fasciculus.

This results in complete ipsilateral horizontal gaze paresis and partial gaze paresis of the opposite eye (spared contralateral abduction). The condition is often caused by a lesion (vascular or demyelinating) in the dorsal tegmentum of the caudal pons. In our patient, magnetic resonance imaging revealed a left caudal pontine infarct involving the para pontine reticular formation, medial longitudinal fasciculus and VII nerve nucleus. We present this case to highlight the fact that interpretation of eye movement...
Figure 1. Left lower motor facial paralysis.

Figure 2. Straight gaze.
Figure 3. Left gaze.

Figure 4. Right gaze.

Figure 5. Left pontine infarct on MRI (arrow).
abnormalities in brainstem strokes can be difficult yet fascinating.

References


