CASE REPORT

Isolated III cranial nerve palsy: a surprising presentation of an acute on chronic subdural haematoma

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SUMMARY

Many aetiologies have been associated with isolated oculomotor nerve palsies. They are ischaemic microangiopathy, posterior communicating artery aneurysm, uncal herniation, neoplasia, traumatic and inflammatory conditions. We report the case of a patient who presented with left oculomotor cranial nerve palsy with an associated large volume left acute on chronic subdural haematoma. Coincidentally, this woman was also found to have a recent history of herpes zoster ophthalmicus.

BACKGROUND

We believe that this case displays a presentation with multiple possible differential diagnosis. Excluding the life-threatening cause from benign cause is essential in treatment decision. One cannot assume the third nerve palsy is caused by the recent herpes zoster ophthalmicus (HZO) attack and further investigation is warranted to exclude other causes.¹⁻⁴

CASE PRESENTATION

The patient was an 84-year-old woman with medical history of hypertension, hypercholesterolaemia and Alzheimer's disease. She suffered a recent attack of left HZO 8 weeks prior. She is not a diabetic. She was referred from a tertiary ophthalmology centre with a 2-day history of complete ptosis of her left eye. She did not have any headache or other symptoms of intracranial hypertension.

On examination, she was alert but not orientated to time and place due to her dementia. She had complete ptosis of her left eye (figure 1). Her left eye was in a 'down and out' position (figure 2). Adduction, upward and downward gaze was impaired on the affected eye. Her left pupil was 5 mm in size as opposed to 2 mm on the right and

Figure 2 Left oculomotor palsy (eye deviated downward and outward).

both were reactive. There was no cutaneous lesion on her face in the distribution of the left V1 dermatome. Other cranial nerves examination was unremarkable. She was otherwise neurologically intact without any motor or sensory deficits.

In light of the acute unilateral oculomotor palsy, a CT angiogram (CTA) was performed. A posterior communicating artery aneurysm was not detected. Surprisingly there was an acute on chronic subdural haematoma (SDH) measuring 16 mm extending over the left frontoparietal convexity with a midline shift of 12 mm (figure 3). A left parietal mini craniotomy was performed and the haematoma was successfully evacuated. She made good postoperative recovery. Despite this, her left oculomotor palsy persisted.

INVESTIGATIONS

CT angiogram.

DIFFERENTIAL DIAGNOSIS

- ▶ SDH with uncal herniation causing the third nerve palsy
- ▶ HZO
- ▶ Ischaemic microangiopathy

TREATMENT

Left parietal mini craniotomy.

DISCUSSION

We report an interesting presentation of a patient with large volume SDH causing mass effect. We outline three potential causes for her unilateral oculomotor palsy.

First, SDH with significant mass effect have been reported in the causation of isolated oculomotor nerve palsies.⁵⁻⁸ A proportion of these cases

Figure 1 Left oculomotor palsy (complete ptosis).

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Figure 3 Acute on chronic subdural haematoma of 16 mm maximum depth extending over left frontoparietal convexity (red arrow) with 12 mm midline shift.

demonstrated rapid resolution of the third nerve function following evacuation of the clot. Our patient did not recount any symptoms suggestive of intracranial hypertension perhaps due to age-related cerebral atrophy.

Second, HZO although uncommon, has been reported to cause ophthalmoplegia with the oculomotor nerve being the most commonly affected.^{9–10} Its mechanism is still unclear. Proposed theories include extension of inflammation to ocular nerves via sensory ramifications of V1 nerve,¹¹ direct extension of the virus,¹² microinfarction from occlusive vasculitis,¹³ chronic inflammatory cell infiltration of long posterior vessels and nerves¹⁴ and a demyelination process.^{15–16} HZO-induced ophthalmoplegia usually manifests 9–10 days after the onset of rash.¹⁷ Our patient was affected by HZO 8 weeks prior to her presentation making it less likely as the primary cause of her cranial nerve palsy.

It has been shown that risk factors of developing ischaemic microvascular ocular palsy include diabetes mellitus, age more than 50 years, hypertension, dyslipidaemia and smoking.¹⁸ With chronic history of hypertension and hypercholesterolaemia our 84-year-old patient could have suffered an ischaemic microvascular oculomotor nerve palsy.

Learning points

- ▶ To acknowledge the multiple aetiologies of third nerve palsy.
- ▶ Herpes zoster ophthalmicus is a rare cause third nerve palsy; therefore, CT angiogram is warranted to exclude other life-threatening diagnosis.
- ▶ Subdural haematoma with mass effect poses immediate threat to life and therefore assumed to be the primary aetiology in this case.

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