

increasing the awareness of physicians and patients of the early signs of PD can improve the current under-recognition and misdiagnosis of PD.⁵ Since patients are often not aware that adhesive capsulitis can be an early presentation of PD, they will not voluntarily discuss it with their physician and thus, delay the appropriate treatment of their condition. As such, physicians should take the initiative to screen for early PD symptoms when no other causes of frozen shoulder can be elucidated.

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TO THE EDITOR

Parinaud's Syndrome Due to Migraine

A 28-year-old Caucasian patient has been treated for two years by his general practitioner (GP) for history of migraine. He has throbbing headache associated with nausea, vomiting, photophobia and phonophobia. He presented with new onset history of two months duration of recurrent, severe and unilateral headache associated with double vision and similar migraine elements he had in the past. When he was examined in the Eye department, he was found to have a head tilt to left to compensate for double vision. His visual acuity was recorded at 6/5 bilaterally, the fundi were normal and there was no ptosis. He has sluggish pupillary reaction to light and accommodation examination provoked convergence nystagmus. He has impaired upward eyes movement. The Hess chart suggested weakness of the left superior rectus and right medial rectus. He was eventually referred to our neurology department for the problem of diplopia. His neurological examination confirmed eye signs and a diagnosis of Parinaud's syndrome were made. His routine vasculitis, autoimmune screen and lactic acid blood tests were all normal apart from a slightly high triglyceride level of 1.8 mmol/L.A. His brain imaging including CT and MRI scans were normal. In the light of clinical presentation and normal investigations which excluded other causes, in our opinion, the most likely cause of his Parinaud's syndrome was migraine.

DISCUSSION

Parinaud's syndrome is diagnosed when a lesion in the superior tectal area causes a combination of supra-nuclear upgaze palsy, retraction convergence nystagmus and light- near dissociation. Recognised causes of Parinaud's syndrome include tumour, haemorrhage, infarction, vascular malformation, demyelination and viral infection. In the absence of all other explanations migraine was thought to be most reasonable cause. Permanent ocular paralysis is very rare but it was described in some patients who experienced recurrent episodes of diplopia and headache. The disorder described could be classified under the rubric ophthalmoplegic migraine. The mechanism of

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ophthalmologic migraine is not clear. It has been suggested that oedema in the wall of the internal carotid artery results in compression of the oculomotor and trigeminal nerves. This theory has not been supported by angiogram findings.¹ In a recent case report, enhancement and thickening of the cisternal portion of the oculomotor nerve associated with ophthalmoplegic migraine has been described.² Neither theory explains occasional development abducens and trochlear palsy. Currently ophthalmologic migraine is considered to be a type of recurrent demyelinating cranial neuropathy.³

CONCLUSION

Migraine is associated with transient neurological deficit such as parathesia, hemiplegia and oculomotor palsy. In rare cases, these deficits become permanent and require further investigations, as in our patient, who developed permanent Parinaud's syndrome.^{4,5}

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