



Silent Tumours, Loud Clues: Case Series of LR Palsy and Papilledema in Young Females

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ABSTRACT:

Unilateral sixth nerve palsy associated with papilledema is an alarming clinical finding that often indicates raised intracranial pressure. The purpose is to highlight uncommon presentations of sixth nerve palsy with papilledema as early indicative signs of space-occupying lesions in young females. We present two young female patients who reported experiencing a sudden onset of binocular diplopia and headaches. Ocular examination revealed limitations of abduction in the right eye, along with bilateral disc oedema. Neuroimaging identified significant intracranial pathology in both cases—one with an intracranial haemorrhage causing mass effect secondary to Ewing sarcoma and the other with cerebellopontine angle vestibular schwannoma causing hydrocephalus. These cases highlight the importance of thorough ophthalmic evaluation for complaints of headache, which could be a window to neurological lesions. It is crucial to conduct a thorough fundus evaluation and early neuroimaging in young patients, as prompt diagnosis is crucial in preventing permanent visual and neurological complications.

1. Introduction

Unilateral sixth cranial nerve palsy in association with papilledema constitutes a high-risk neuro-ophthalmic presentation that mandates prompt assessment for raised intracranial pressure (ICP). The abducens nerve is particularly vulnerable to intracranial hypertension owing to its prolonged intracranial trajectory, elevation along the clivus, and sharp curvature over the petrous apex prior to its entry into the cavernous sinus. When optic disc oedema is concurrently present, the probability of an intracranial mass lesion, obstructive hydrocephalus, or idiopathic intracranial hypertension increases considerably.

Posterior fossa lesions are particularly important in this context. Space-occupying lesions within the cerebellopontine angle, including vestibular schwannoma, may compress the fourth ventricle and impede cerebrospinal fluid outflow, leading to secondary hydrocephalus and raised ICP. In such cases, unilateral abducens nerve palsy may precede other focal

neurological deficits, increasing the risk of misdiagnosis as microvascular ischaemic neuropathy. The presence of true papilledema on fundoscopic examination distinguishes raised ICP from isolated ischaemic sixth nerve palsy and necessitates immediate neuroimaging.^{1,2}

Contrast-enhanced magnetic resonance imaging with venography remains the investigation of choice to exclude structural and venous causes. Early recognition is critical because persistent papilledema may progress to optic atrophy and permanent visual loss. Accordingly, unilateral sixth nerve palsy with papilledema should be approached as a neuro-ophthalmic emergency requiring systematic evaluation and timely intervention.¹⁻³

Case report 1

A 16-year-old female patient presented with complaints of double vision for eight days, which was sudden in onset and painless in nature and absent on closing one eye, increased on looking on the right side and decreased on turning the face towards the right side, along with the patient also had an associated throbbing headache,



sudden in onset and more on waking up with no relieving factor. The patient has two episodes of projectile vomiting. Patient is having history of left femur Ewing's sarcoma and underwent 4 cycles of chemotherapy. The patient's appetite was reduced, and sleep disturbances were noted. Cranial motor and sensory examinations were normal.

On ocular examination, unaided visual acuity was 6/6 in both eyes, colour vision was normal, and extraocular movement showed grade 2 limitations of abduction in the right eye on ductions. The rest of the extraocular movements were normal.



Figure 1: Normal primary gaze



Figure 2: Limitation of abduction in the right eye

Fundus examination revealed bilateral optic disc oedema with vessel tortuosity and an obliterated cup-to-disc ratio, suggestive of bilateral optic disc papilledema.

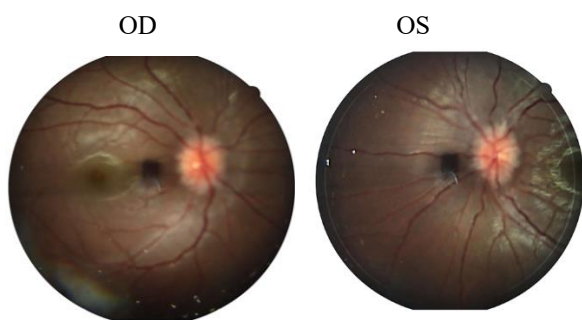


Figure 3: Bilateral Papilledema

The patient was referred to the neurosurgery department for further evaluation for raised intracranial pressure, on CT scan, a fairly well-defined hyperdense areas involving the right anterior temporal and left temporal regions with perilesional oedema causing mass effect and midline shift suggestive of intracranial bleed and raised intracranial pressure. This were due to secondary metastasis of Ewing sarcoma. Medical management was initiated with intravenous mannitol, and surgery was planned to reduce intracranial tension. On the subsequent 4 months follow up patient had full and free restoration of extraocular movements, with papilledema resolve.

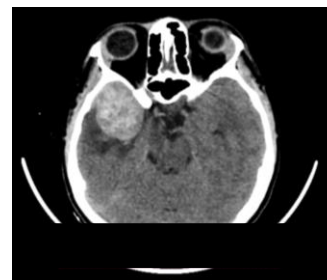
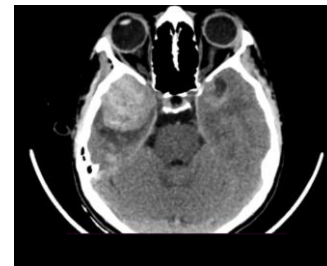


Figure 4: Fairly well-defined hyperdense areas (haemorrhagic area) involving the right anterior temporal and left temporal regions with perilesional oedema causing mass effect and midline shift Suggestive of intracranial bleed and raised intracranial pressure.

Case report 2

A 23-year-old female presented with complaints of diminished vision in both eyes and double vision for the past 12 days, associated with headache. She also reported limitation of movement in the right eye. She had no known systemic comorbidities. However, she had associated hearing loss and right-sided facial nerve palsy.



Figure 5: Normal primary gaze



Figure 6: Limitation of abduction in the right eye



On ocular examination, her unaided visual acuity was 3/60 in both eyes. There was a limitation of abduction in the right eye, while other extraocular movements were normal. She experienced binocular diplopia, which resolved when she closed either eye. Lagophthalmos was present in the right eye due to facial palsy, though Bell's phenomenon was intact. The rest of the anterior segment examination was within normal limits.



Figure 7: loss of nasolabial fold on the right side suggestive of right-sided facial nerve palsy

The fundus examination revealed bilateral disc oedema accompanied by flame-shaped haemorrhages and retinal oedema surrounding the discs, which is suggestive of papilledema. Visual fields showed an enlarged blind spot. Facial examination showed the absence of the right nasolabial fold, consistent with facial nerve palsy. All other cranial nerve functions were within normal limits.

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Figure 8: Bilateral Papilledema

The patient was referred for evaluation of raised intracranial pressure. A CT scan revealed a well-defined hypodense soft tissue lesion in the right cerebellopontine angle, causing mass effect on the brain stem, ipsilateral cerebellar lobe and fourth ventricle with adjacent perilesional oedema. There was effacement of the fourth ventricle, compression of the right cerebellar hemisphere and brainstem, and evidence of trans tentorial herniation, suggestive of raised intracranial tension.

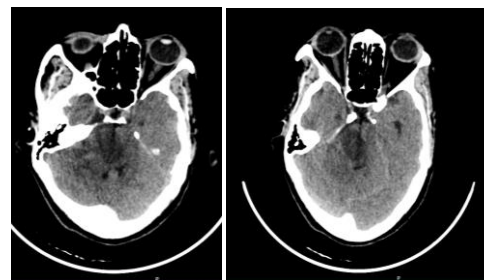
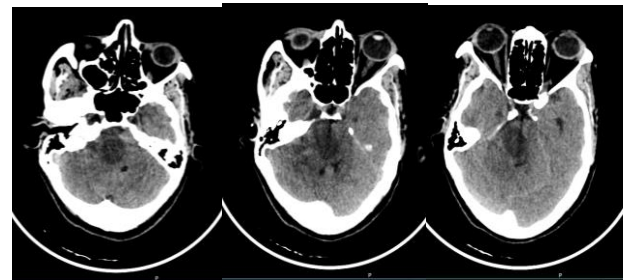


Figure 9: Fairly well-defined extraaxial hypodense mass lesion with adjacent perilesional oedema involving the right cerebellopontine angle, causing mass effect and mild obstructive hydrocephalus.

A multidisciplinary approach was adopted, and the patient was referred to the neurosurgery and otorhinolaryngology departments for further management. She was started on intravenous mannitol for control of intracranial pressure along with symptomatic treatment for headache and pain. Unfortunately, the patient declined further intervention and chose to be discharged against medical advice; no additional treatment was undertaken.

DISCUSSION

Unilateral sixth cranial nerve palsy in a young patient requires prompt evaluation, as the likelihood of underlying intracranial pathology is significantly higher in this age group compared to older individuals with vasculopathy risk factors. Isolated abducens nerve palsy in young patients should not be presumed to be



microvascular in origin. Early neuroimaging, preferably contrast-enhanced MRI of the brain, is essential to exclude intracranial mass lesions.^{4,5} Delay in diagnosis may result in progression of neurological deficits and irreversible visual or neurological damage.

The sixth cranial nerve is particularly susceptible to raised ICP and is often the earliest nerve affected.⁴ This vulnerability is attributed to its long intracranial course. Originating from the dorsal pons, it ascends along the clivus within the subarachnoid space, passes through Dorello's canal at the petrous apex, and then enters the cavernous sinus before innervating the lateral rectus muscle.⁴ Because of this long and relatively fixed pathway, increased ICP can stretch or compress the nerve, especially with downward displacement of the brainstem, making sixth nerve palsy a classic "false localising sign."⁴

Clinically, sixth nerve palsy presents with limitation of abduction of the affected eye, resulting in horizontal diplopia that worsens on ipsilateral gaze and esodeviation in the primary position.^{4,5} In cases of sustained intracranial hypertension, bilateral involvement may occur. When unilateral sixth nerve palsy is associated with headache or papilledema, raised ICP must be strongly suspected.^{6,7} Intracranial tumours such as glomus jugular tumours and pineal epidermoid cysts have been reported to present initially with papilledema or isolated lateral rectus palsy.^{6,7}

Although many cases demonstrate spontaneous recovery, a considerable proportion of young patients may harbour significant intracranial lesions.⁵ Therefore, early recognition, detailed ocular and neurological examination, and timely neuroimaging are crucial for appropriate intervention and improved prognosis.^{5,8}

CONCLUSION

Unilateral sixth nerve palsy occurring with papilledema should always raise concern for raised intracranial pressure and possible underlying intracranial pathology. The cases described highlight that even in young patients presenting primarily with diplopia, significant intracranial disease may be present. Careful fundus examination and recognition of papilledema play a crucial role in guiding further evaluation. Early neuroimaging is therefore essential to identify the underlying cause and initiate appropriate management. Timely diagnosis and multidisciplinary care are important to prevent progression of neurological deficits and avoid permanent visual loss.

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