



Research Article

A CASE REPORT OF RIGHT LATERAL RECTUS (LR) PALSY IN A TERTIARY CARE HOSPITAL

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ABSTRACT

This case report explores the presentation, diagnosis, and management of a 60-year-old female patient with right Lateral Rectus (LR) palsy, a manifestation of abducens nerve dysfunction. The patient, with a history of Type 2 Diabetes Mellitus, presented with Giddiness, Blurred vision, and Facial palsy. Initial investigations revealed elevated random blood glucose levels. Neuroimaging, specifically brain MRI, confirmed chronic small vessel ischemia as the underlying cause.

The patient initially declined hospital admission but was prescribed a combination of medications. Subsequent follow-ups revealed worsening double vision and blurred vision over the following week. Further treatment involved a three-day hospital stay, during which intravenous Methylprednisolone was administered. The patient responded positively, and left abducens nerve palsy completely resolved within one week.

The discussion highlights the prevalence of microvascular causes in adults, differing from pediatric trends, and emphasizes the importance of considering various etiological factors such as Diabetes, Hypertension, and Immunologic damage. The study advocates for thorough Neuroimaging in cases lacking clear risk factors or suggestive clinical findings. The conclusion underscores the significance of proper follow-up, especially for initially idiopathic cases, to facilitate a more precise diagnosis based on evolving clinical manifestations.

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INTRODUCTION

Cranial nerve VI palsy, known as "Sixth Nerve Palsy," "Abducens Nerve Palsy," or "Lateral Rectus Palsy," is a neurological condition impacting the sixth cranial nerve's function. It is relatively rare, particularly among healthy adults. The abducens nerve, or the sixth cranial nerve, regulates the lateral rectus muscle responsible for outward eye movement (abduction). When this nerve is affected, it can give rise to various specific symptoms and issues [1]. Abducens nerve palsy is recognized as the most prevalent ocular motor nerve palsy [2]. A thorough examination, involving imaging studies, is essential due to potential serious underlying causes, including intracranial mass, elevated intracranial pressure, or stroke. Indeed, isolated cranial nerve palsies are among the more frequent reasons for Neuroimaging. MRI has proven effective in revealing lesions and conditions that might have otherwise gone unnoticed [3]. Following the age of 50, vascular diseases are the most frequently identified causes. A lesion will lead to an ipsilateral deficiency in abduction and increased esotropia at a distance [4]. Originating near the seventh cranial nerve in the pons, the abducens nerve exits the brainstem and traverses the subarachnoid space, following the

route along the skull known as the clivus. Progressing further, the nerve reaches the petrous apex of the temporal bone within the basal skull before entering the cavernous sinus [5].

The abducens nerve provides innervation to the ipsilateral lateral rectus muscle, governing horizontal eye movement. Therefore, when the sixth cranial nerve is impacted, deviations manifest exclusively in the horizontal plane. In instances where solely the isolated peripheral nerve is affected, there are no associated vertical or torsional movements [6]. Having the longest intracranial course among all cranial nerves, the abducens nerve plays a primary role in ipsilateral eye abduction. In instances of abducens nerve palsy, the affected nerve is incapable of transmitting signals to the lateral rectus muscle, leading to the inability to abduct the eye and subsequent horizontal diplopia. In situations involving central nervous system defects, the localization of the sixth nerve tract can be determined based on characteristic findings associated with each type of lesion [7]. Involvement of the sixth nerve nucleus leads to ipsilateral gaze palsy. The lack of contralateral adduction aids in distinguishing a nuclear lesion from a fascicular or non-nuclear lesion. Elevated intracranial pressure, causing the stretching of the sixth nerve, can produce

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a false localizing sign, indicating abducens nerve palsy as the nerve crosses the clivus. Additionally, abducens nerve palsy may arise from Post-Viral Syndrome in Pediatric and adolescent populations, while in adults, it may manifest as Ischemic Mononeuropathy [8].

CASE REPORT

A 60-year-old female patient presented to the Neurology department as an outpatient with complaints of giddiness and blurred vision. She had a past medical history of Type 2 Diabetes Mellitus for 2 months, at the time of the patient's visit, her Random Blood Glucose Level is 299 mg/dl and her medication was Tab. Metformin-500mg (BD), Tab. Prednisolone-10mg (BD), Tab. Pantoprazole + Domperidone 40 mg/30 mg(BD) Tab. Calcium + Vit D3-500 mg/250 IU (OD), Tab. Cefixime 200mg(BD), Inj. Human Insulin Actrapid 6U (BD). This information can be considered for her initial assessment in the Neurology outpatient department.

After the patient came to the neurology department for a review, she presented complaints of facial palsy and reported no pain. Her random blood glucose level was 516 mg/dL. The patient has been diagnosed with Right Lateral Rectus (LR) Palsy Syndrome based on the findings from a brain MRI report. The brain's MRI was performed employing T1 and T2 weighted sequences across various planes, utilizing a quadrature head coil. The impression from the MRI indicates the presence of chronic small vessel ischemia. The physician advised her to be admitted to the hospital.



Fig.1 A clinical photograph of a patient displays the right lateral rectus while in a right lateral gaze.



Fig.2 A clinical image of a patient demonstrates normal left lateral gaze with the eyes.

However, she was not willing to stay in the hospital. Consequently, the following medications were prescribed: Tab. Metformin + Glimipride-500/1mg (BD), Tab. Prednisolone-10mg(BD), Tab. Pan D-40/30mg(BD), Tab. Calcium + Vit D3-500 mg/250 IU (OD), Inj. Human Insulin Actrapid 12U (BD).

After spending five days as an outpatient in the Neurology department, the patient returned for further treatment. During this visit, she complained of experiencing double vision for the past one and a half months, and this symptom had worsened over the past week. Furthermore, she noted having blurred vision. The patient did not have a history of limb weakness or other cranial nerve deficits, and she also had no history of vomiting, fever, or dysuria.

The patient reported no recent travel, viral illnesses, or head injuries. Additionally, there were no similar occurrences of

headaches or attacks in the family history. The review of systemic and rheumatological systems revealed no significant findings.

Investigation

In the course of the examination, the patient demonstrated alertness and orientation, while their vital signs remained within the expected range. During the ocular examination, the only notable findings were a right lateral rectus palsy and left-sided horizontal nystagmus. The pupils were equal in size, round, and responsive to both light and accommodation. There was no evidence of proptosis, ptosis, or facial numbness. Fundoscopy did not reveal any signs of papilledema. Visual acuity, as well as assessments of other cranial nerves and upper and lower motor neuron function, were all found to be normal.

On investigation, full blood count into Complete Blood Count(CBC), blood count displayed raised WBC's (7840 million/ml) with a left sided neutrophil shift (80.4%), normal lymphocytic count (13.2%), normal haemoglobin (14.8 gm/dl) and normal platelets level (3.3 L/cu.cm). No elevated inflammatory markers (CRP 0.2 mg/L; ESR 20). Random blood glucose levels (516 mg/dl). Infectious work up including Lyme and syphilis serologies and CSF analysis were not performed, because patient was not travel any other places.

Differential Diagnosis

The sixth cranial nerve (abducens nerve) controls the lateral rectus muscle. Damage to this nerve, often due to various causes like head injury, vascular issues, tumors, or inflammation, can result in lateral rectus palsy.

Treatment

The following medication was initiated for a duration of three days.

- Inj. Methylprednisolone 1 gm/day IV at a rate of 50 ml/hr.
- Inj. Thiamine 200 mg IV (once daily).
- Inj. Methylcobalamin 1000 mcg (once daily).
- Tab. Pantoprazole + Domperidone 40 mg/30 mg IV (BD)-IV (twice daily).
- Tab. Calcium + Vitamin D3 - 500mg/250 IU (twice daily).
- Tab. Metformin + Glimipride + Voglibose - 500/1/0.2mg (twice daily).
- Inj. Human Insulin Actrapid 12U (twice daily).

Outcome and Follow Up

She returned to a normal state 48 hours after the initiation of Inj. Methylprednisolone 1 gm IV for 50 ml/hr. She underwent a three-day hospital admission. After completing the three doses of Inj. Methylprednisolone 1 gm, she was discharged. During the one-week follow-up, her left abducens nerve palsy had completely resolved.

DISCUSSION

The study assessed the clinical features and eventual outcomes of patients presenting with isolated abducens nerve paralysis. When considering the age of patients admitted to neurology clinics with this condition, microvascular causes emerged as the most prevalent etiological factors, differing from trends observed in pediatric and neurosurgical clinics. In this study, we unveiled both the commonalities and distinctions in

clinical and prognostic factors between microvascular and other causes[12].In cases of orbital syndrome associated with sixth nerve palsy, one would anticipate the involvement of the optic nerve, possibly presenting as papilledema or optic atrophy. In contrast, in cases of isolated sixth nerve palsy syndrome, as observed in our case, it is commonly linked to vascular factors such as diabetes, hypertension, migraine manifestation, or immunologic damage, capable of affecting the nerve at any point along its length [3]. A conceivable explanation for diabetes being linked to a higher percentage of isolated sixth nerve palsy may stem from the elevated incidence of type II diabetes in India, contrasting with the United States—14.3% versus 8.2%.[9,10]. Isolated sixth nerve palsy can result from diverse factors, such as diabetes, hypertension, and recent viral infections. There is also a possibility of intermittent recurrence in isolated involvement. Abducens nerve palsy can be induced by ophthalmic migraine, with involvement manifesting either centrally or peripherally. When imaging yields mixed findings, identifying the precise type of palsy becomes challenging. Immunologic damage to the sixth nerve is potential at any point [11]. Moreover, instances of abducens nerve palsy resulting from trauma and malignancy are relevant to neurosurgery clinics, and introducing a challenge in achieving a homogeneous distribution among etiological causes. Therefore, conducting diverse clinical studies encompassing various scenarios of abducens nerve paralysis cases would yield more objective data. [13].

CONCLUSION

In cases where there are no risk factors, a suggestive history, or positive laboratory and clinical findings, neuroimaging becomes a valuable diagnostic tool for identifying the specific cause of sixth nerve palsy.

Although various causes are acknowledged, abducens nerve palsy represents a relatively frequent occurrence of isolated ocular nerve palsy. The incidence of cases labeled as idiopathic is on the rise; nevertheless, it is crucial to recognize that some cases deemed idiopathic may reveal a defined pathogenesis through detailed a comprehensive history and diligent follow-up. It is imperative to conduct proper follow-up for patients, especially those initially classified as idiopathic, as the course of the illness may evolve, and a more precise diagnosis can be established based on a comprehensive history and physical examination findings.

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