RESEARCH PAPER

From Impact to Infarct: The Third Nerve Files! Mapping Oculomotor Palsy from Brainstem to Orbit

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ABSTRACT

Background: Third cranial nerve (oculomotor nerve) palsy is an important neuro-ophthalmic presentation that may result from diverse vascular, traumatic, compressive, and intracranial causes. The clinical distinction between pupil-sparing and pupil-involving palsy is particularly relevant because pupillary involvement may indicate a compressive lesion requiring urgent neuroimaging and intervention. This case series describes the varied clinical presentations and etiological associations of third nerve palsy encountered at a tertiary eye care center.

Aim: To describe the clinical presentation, pupillary involvement, neuroimaging findings, and etiological spectrum of third nerve palsy in a series of patients presenting to a tertiary eye care center.

Case Series: Ten patients with clinically diagnosed third nerve palsy were identified. The patients included six males and four females, with ages ranging from 22 to 71 years. The presentations included sudden or progressive ptosis, diplopia, ocular motility restriction, headache, retro-orbital pain, and, in some patients, loss of consciousness and vomiting. Diabetes and hypertension were documented among several patients with presumed ischemic third nerve palsy. Pupil-sparing palsy was observed in some cases, whereas others demonstrated complete pupil-involving third nerve palsy. Neuroimaging was normal in selected presumed microvascular or traumatic cases, while imaging identified post-traumatic orbital and intracranial abnormalities and sellar/suprasellar lesions in other patients. Two patients had pituitary macroadenoma-associated third nerve palsy. One patient had traumatic third nerve palsy with traumatic mydriasis following road traffic injury. Another patient presented with third nerve palsy secondary to a ruptured posterior communicating artery aneurysm with subarachnoid hemorrhage and underwent endovascular coil embolization.

Conclusion: This case series demonstrates the heterogeneous etiological spectrum of third nerve palsy and the importance of careful assessment of pupillary involvement, associated neurological symptoms, history of trauma and vascular risk factors, and appropriate neuroimaging. Although pupil-sparing presentations may occur in presumed ischemic neuropathy, pupil involvement can be associated with significant structural lesions, including pituitary macroadenoma and ruptured posterior communicating artery aneurysm. A systematic clinical and radiological evaluation is therefore essential for identifying potentially serious underlying causes and guiding appropriate management.

Keywords: *Third nerve palsy; oculomotor nerve palsy; ptosis; diplopia; pupil involvement; pupil sparing; microvascular ischemia; trauma; pituitary macroadenoma; posterior communicating artery aneurysm*

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INTRODUCTION

Third cranial nerve (oculomotor nerve) palsy is an important neuro-ophthalmic disorder characterized by ptosis, diplopia, and limitation of ocular movements due to dysfunction of the muscles supplied by the oculomotor nerve [1]. The clinical presentation may vary according to the extent of nerve involvement and whether the pupillary fibers are affected. Complete palsy may produce marked ptosis, ophthalmoplegia, and pupillary abnormalities, whereas partial or pupil-sparing presentations may have a more limited clinical profile [2].

The underlying causes of third nerve palsy are diverse. Vascular disorders, particularly in patients with diabetes and hypertension, may produce ischemic neuropathy. Trauma represents another important cause and may be associated with orbital fractures, intracranial injury, or other post-traumatic structural abnormalities [3]. Compressive lesions involving the cavernous sinus, orbital apex, sellar and suprasellar regions, or posterior communicating artery may also produce third nerve

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dysfunction. The presence of pupil involvement, particularly in an acute presentation, is clinically important because it may indicate a compressive etiology requiring urgent investigation [4].

The cases in the present series demonstrate this broad clinical and etiological spectrum. The source records patients with presumed ischemic third nerve palsy associated with diabetes and hypertension, traumatic third nerve palsy following road traffic accidents or blunt ocular trauma, third nerve palsy associated with pituitary macroadenoma, and a life-threatening presentation secondary to a ruptured posterior communicating artery aneurysm with subarachnoid hemorrhage [5].

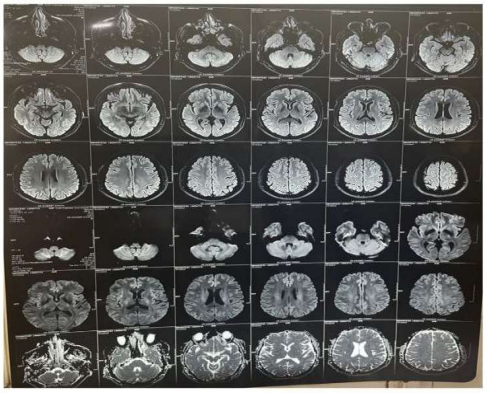
Recognition of the clinical pattern and systematic evaluation of associated neurological findings and pupillary status are therefore essential in patients presenting with third nerve palsy. **Therefore, it is of interest to describe the varied clinical presentations, pupillary findings, neuroimaging abnormalities, and etiological spectrum of third nerve palsy observed in this case series from a tertiary eye care center.**

CASE SERIES

A total of 10 patients with clinically documented third cranial nerve palsy were included in this case series. The patients demonstrated considerable variation in age, presenting symptoms, associated systemic and traumatic history, pupillary involvement, neuroimaging findings, and presumed underlying etiology. The cases ranged from presumed microvascular ischemic neuropathy in patients with diabetes or hypertension to traumatic nerve palsy, orbital injury, pituitary macroadenoma, and ruptured posterior communicating artery aneurysm with subarachnoid hemorrhage.

Case 1

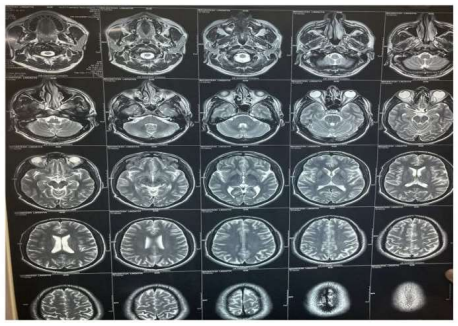
A 71-year-old male, a known case of diabetes mellitus for 5 years, presented with sudden-onset drooping of the left upper eyelid associated with diplopia. On ocular examination, complete left third cranial nerve palsy was identified, with sparing of the pupil [6]. Neuroimaging did not demonstrate any significant abnormality. In view of the acute presentation, background history of diabetes, complete third nerve involvement, and pupil-sparing pattern in the absence of an identifiable structural lesion on neuroimaging, a diagnosis of presumed microvascular ischemic third nerve palsy was made [7].



Case 2

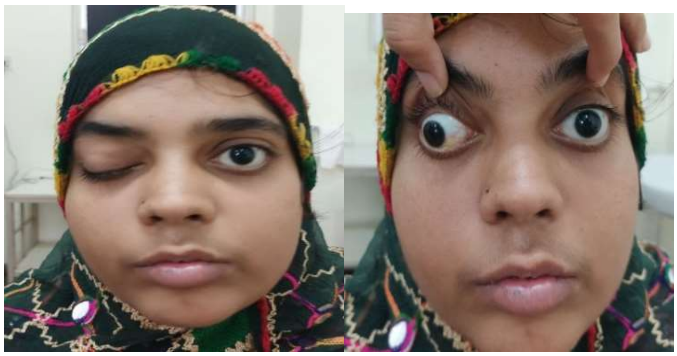
A 38-year-old female, with diabetes mellitus for 3 years and a past history of tuberculosis 10 years previously, presented with gradual and progressive drooping of the right upper eyelid. She had associated retro-orbital pain of 15 days' duration. Examination revealed complete right third cranial nerve palsy with involvement of the pupil [8].

Unlike the previous case, the clinical presentation was progressive rather than sudden and was accompanied by retro-orbital pain. Neuroimaging was reported as normal. Despite the presence of pupillary involvement, the documented clinical diagnosis was microvascular ischemic third nerve palsy [9].



Case 3

A 26-year-old female had a history of road traffic accident 1 year prior to presentation. The accident resulted in head injury followed by right-sided hemiparesis. She subsequently developed acute-onset drooping of the right upper eyelid. Ocular examination revealed complete right third cranial nerve palsy with pupillary involvement [10]. Neuroimaging demonstrated findings suggestive of a possible post-traumatic orbital apex syndrome. The documented diagnosis was ischemic third nerve palsy. This case was notable for the preceding significant head trauma, associated hemiparesis, acute ptosis, pupil involvement, and radiological suspicion of orbital apex involvement [11].

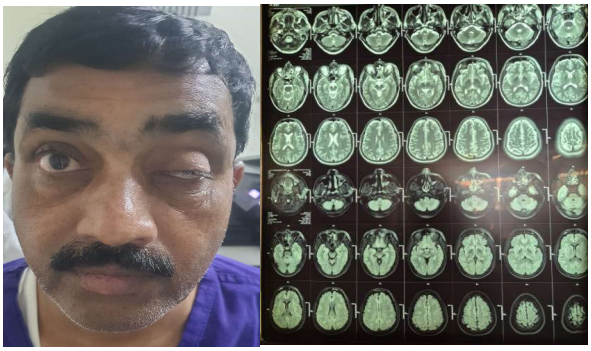


Case 4

A 30-year-old male presented with a history of road traffic accident 8 months earlier, which had resulted in head injury. He subsequently developed acute-onset right upper eyelid drooping associated with double vision involving the right eye. Examination demonstrated complete right third cranial nerve palsy with pupillary involvement [12]. Neuroimaging revealed diffuse axonal injury following trauma. The documented diagnosis was ischemic third nerve palsy. The case represented a post-traumatic presentation in which the third nerve palsy occurred in the setting of previously documented intracranial traumatic injury [13].

Case 5

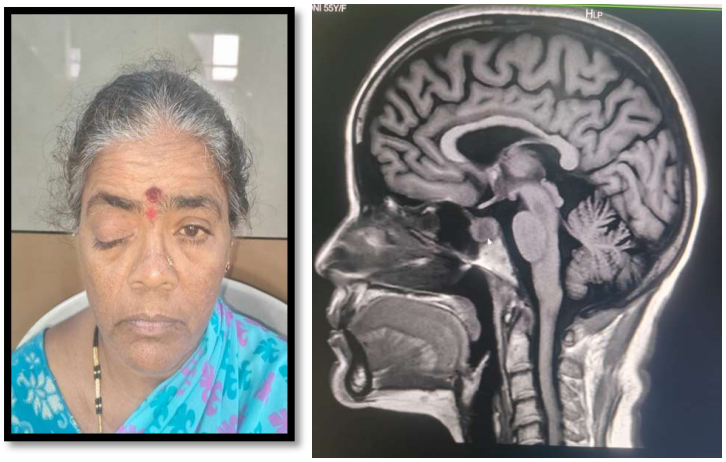
A 56-year-old male, a known case of both hypertension and diabetes mellitus for 4 years, presented with sudden-onset drooping of the left upper eyelid associated with diplopia in the left eye. Examination revealed complete left third cranial nerve palsy without pupillary involvement, indicating a pupil-sparing presentation [14]. Neuroimaging demonstrated hypertensive ischemic changes. Based on the clinical presentation, vascular risk factors, pupil-sparing pattern, and radiological evidence of hypertensive ischemic changes, a diagnosis of ischemic third nerve palsy with pupil sparing was documented [15].

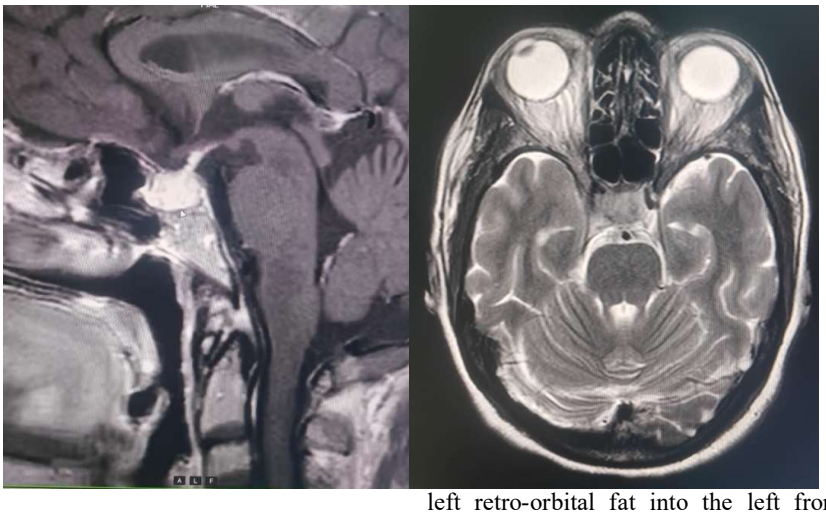


Case 6

A 55-year-old female, a known case of type 2 diabetes mellitus, presented with drooping of the right eyelid for approximately 1½ months. The ptosis was described as sudden in onset, painless, and progressive, eventually resulting in complete occlusion of the eye for approximately 1 week before presentation. The patient also reported a right-sided dull, aching, continuous, non-radiating headache. Restriction of right ocular movements had been present for approximately 1 month [16].

Neuroimaging revealed a 1.4 × 2.6 × 1.1 cm heterointense sellar mass lesion, considered likely to represent a pituitary macroadenoma. The orbital anatomy and optic nerve were reported to be normal. The clinical diagnosis documented was right isolated, pupil-involving, complete third cranial nerve palsy with grade I nuclear sclerosis. The etiological possibilities considered in the source record included diabetic ischemic neuropathy, vasculitis, and pituitary macroadenoma [17]. Thus, this case presented an important diagnostic overlap between a vascular risk profile and a structural sellar lesion.



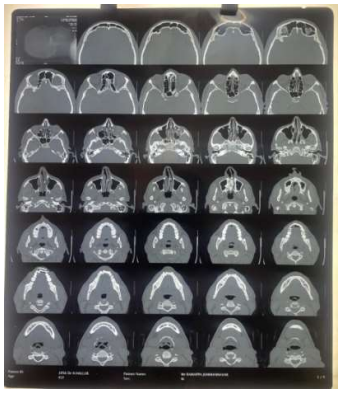


Case 7

A 42-year-old male presented 20 days after sustaining blunt trauma to the left eye caused by a whip. Following the injury, he developed acute-onset pain and diffuse swelling of the left eye, associated with drooping of the eyelid and double vision. Clinical examination revealed complete third cranial nerve palsy without pupillary involvement, representing a pupil-sparing presentation [18].

Neuroimaging demonstrated a comminuted displaced fracture involving both the roof and floor of the left orbit. The orbital roof fracture was associated with herniation of left retro-orbital fat into the left frontal and maxillary sinuses. The documented diagnosis was ischemic pupil-sparing third nerve paresis secondary to blunt trauma with associated enophthalmos [19].

Objective assessment of the anterior-posterior diameter by the two-ruler method showed a measurement of 15 mm in the right eye and 9 mm in the left eye, documenting marked asymmetry between the two eyes. This case therefore demonstrated a combination of significant orbital trauma, structural orbital fractures, enophthalmos, and complete pupil-sparing third nerve paresis [20].

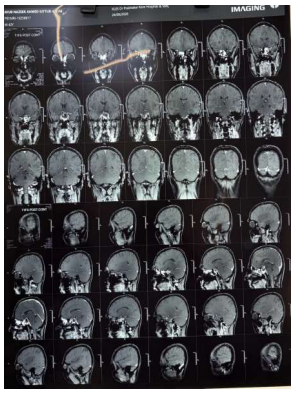


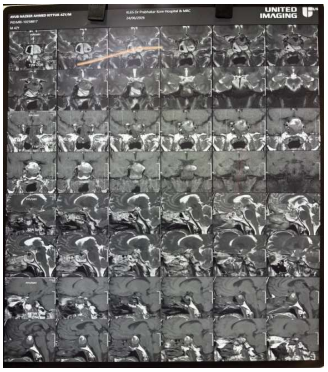
Case 8

The patient was apparently well until 5 days before presentation, after which headache developed. The headache was described as a feeling of heaviness involving the whole head, of moderate to severe intensity, non-radiating, insidious in onset, and gradually progressive. There was no preceding aura. The headache was associated with 2–3 episodes of vomiting and was not described as having specific aggravating or relieving factors. This was followed by an episode of loss of consciousness lasting approximately 18 hours [21].

Following the episode of loss of consciousness, the patient's attendants noticed drooping of the left upper eyelid. The ptosis was insidious in onset and gradually progressive, progressing to complete closure of the eyelid on the third day after onset. There was no documented diurnal variation or specific aggravating or relieving factor for the ptosis [22].

Neuroimaging demonstrated a hypoenhancing, lobulated lesion in the sellar and suprasellar region producing expansion of the sella. The lesion measured approximately 1.7 × 2.4 × 2.4 cm in anteroposterior, mediolateral, and craniocaudal dimensions and was reported as suggestive of a pituitary macroadenoma. The documented diagnosis was left eye pupil-involving third cranial nerve palsy secondary to pituitary macroadenoma. The combination of progressive headache, vomiting, prolonged loss of consciousness, progressive complete ptosis, and a sellar-suprasellar mass represented a significant intracranial presentation of third nerve palsy [23].

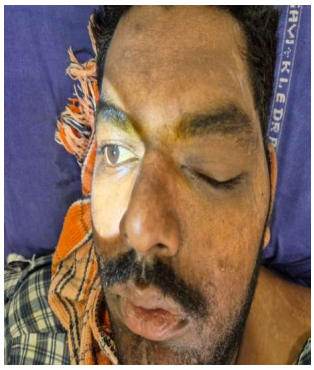


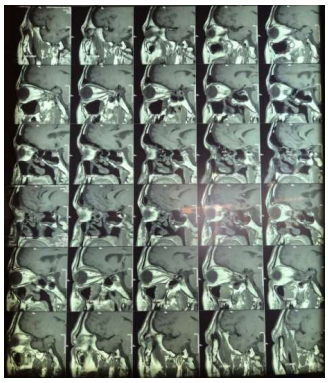


Case 9

A 22-year-old male had sustained a road traffic accident 8 days before presentation. The traumatic event resulted in grade III splenic injury and hemothorax. After regaining consciousness, the patient developed complete drooping of the left upper eyelid. In contrast to several other patients in the series, he did not report diplopia [24].

Examination revealed complete left third cranial nerve palsy associated with traumatic mydriasis. Neuroimaging was reported as normal. A diagnosis of traumatic left third cranial nerve palsy was made. The case illustrates that significant third nerve dysfunction may occur following major trauma even when subsequent neuroimaging does not demonstrate a specific intracranial abnormality [25].



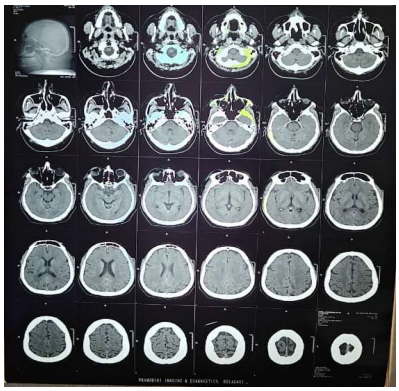


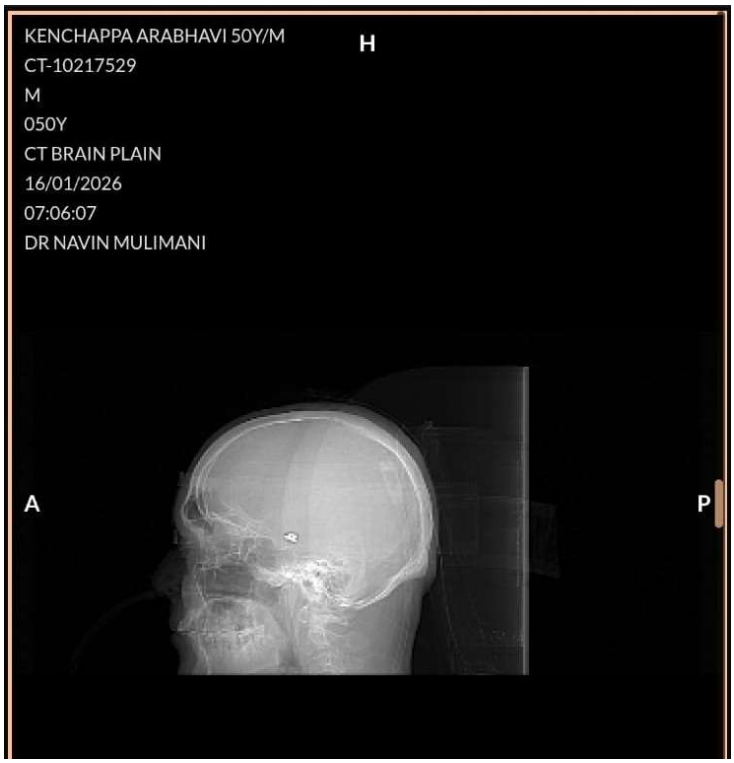
Case 10

A 50-year-old male presented with sudden-onset headache, left eye ptosis, an episode of loss of consciousness, and three episodes of vomiting, all occurring within 1 day of presentation. He was initially taken to an outside hospital, where he was intubated and shifted to the intensive care unit. CT of the brain performed at the outside facility demonstrated grade II subarachnoid hemorrhage. High-resolution CT of the chest showed findings suggestive of aspiration pneumonitis [23].

The patient was diagnosed with pupil-involving third cranial nerve palsy secondary to a ruptured posterior communicating artery aneurysm associated with subarachnoid hemorrhage. Given the underlying vascular pathology, he underwent complete endovascular coil embolization of the posterior communicating artery aneurysm under general anaesthesia using the balloon-remodeling technique. Following the procedure, the patient was extubated and had no new neurological deficits. Postoperative improvement was documented [24].

This case represented the most acute cerebrovascular presentation in the series and demonstrated the association of pupil-involving third nerve palsy with a ruptured posterior communicating artery aneurysm and subarachnoid hemorrhage [25].

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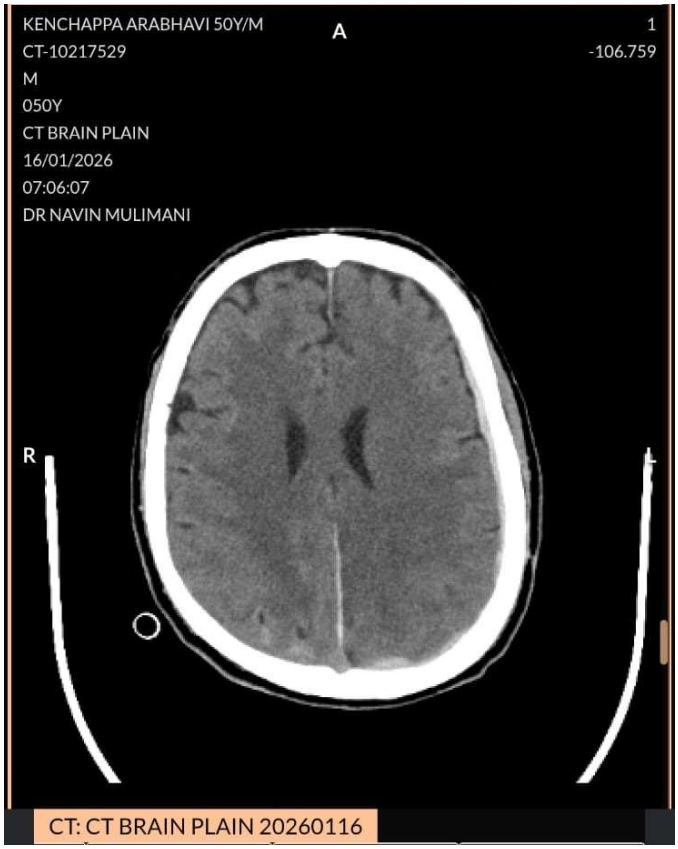
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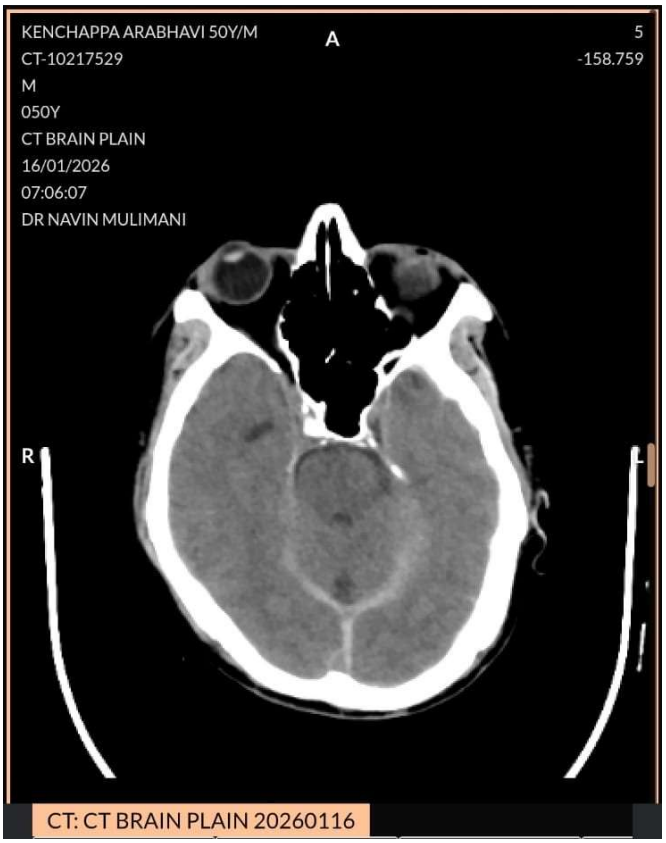
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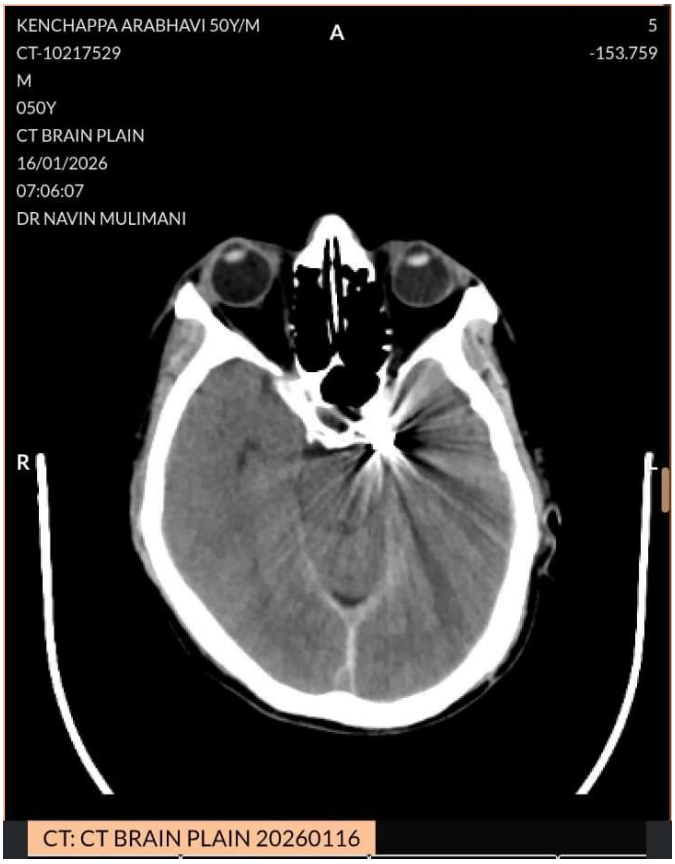
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Overall clinical spectrum

The 10 cases demonstrated a broad spectrum of presentations and presumed etiologies. Pupil-sparing palsy was documented in Cases 1, 5, and 7, whereas pupil involvement was documented in Cases 2, 3, 4, 6, 8, 9, and 10. Diabetes was present in Cases 1, 2, 5, and 6, while hypertension was documented in Case 5. Traumatic antecedents were present in Cases 3, 4, 7, and 9. Structural sellar lesions suggestive of pituitary macroadenoma were demonstrated in Cases 6 and 8, while Case 10 had a ruptured posterior communicating artery aneurysm with subarachnoid hemorrhage. Normal neuroimaging was reported in Cases 1, 2, and 9, whereas other cases demonstrated orbital, traumatic intracranial, sellar, or vascular abnormalities.

DISCUSSION

Third nerve palsy is a clinically important neuro-ophthalmic manifestation with a broad spectrum of underlying causes [1]. The present case series illustrates this heterogeneity through 10 patients with complete third nerve palsy associated with different vascular, traumatic, sellar, and cerebrovascular conditions [2,3]. The patients ranged from 22 to 71 years, and the presenting manifestations included ptosis, diplopia, ocular motility restriction, headache, retro-orbital pain, vomiting, and loss of consciousness. The variation in presentation and neuroimaging findings emphasizes that third nerve palsy should not be considered a single etiological entity [4-6].

Microvascular ischemia was documented or considered in several patients, particularly those with diabetes or hypertension. Case 1 was a 71-year-old diabetic male with sudden-onset ptosis and diplopia, complete third nerve palsy, pupil sparing, and normal neuroimaging [7-9]. Case 5 similarly involved a 56-year-old male with diabetes and hypertension, sudden ptosis and diplopia, pupil-sparing complete third nerve palsy, and hypertensive ischemic changes on imaging [8]. These presentations are consistent with the clinical pattern documented in the source material for presumed ischemic third nerve palsy [10-12].

However, pupillary status was not uniform among patients considered to have ischemic disease. Case 2, a 38-year-old diabetic female, had complete third nerve palsy with pupil involvement despite normal neuroimaging, and the documented diagnosis was microvascular ischemic third nerve palsy [13,14]. This finding demonstrates that pupil involvement alone did not establish a single etiological diagnosis within this series and reinforces the importance of interpreting pupillary findings together with the complete clinical and radiological picture [15,16].

Trauma represented another prominent association. Cases 3, 4, 7, and 9 had histories of road traffic accident or blunt ocular trauma. Case 3 had previous head injury with right-sided hemiparesis and subsequent complete pupil-involving third nerve palsy, with imaging suggesting possible post-traumatic orbital apex syndrome [17,18]. Case 4 had previous head injury and imaging evidence of diffuse axonal injury. Case 7 demonstrated an orbital structural injury, including comminuted displaced fractures of the orbital roof and floor with herniation of retro-orbital fat, and was associated with pupil-sparing third nerve paresis and marked enophthalmos. Case 9 developed complete traumatic third nerve palsy with traumatic mydriasis after a road traffic accident, despite normal neuroimaging [19,20].

The sellar and suprasellar region was another important anatomical site represented in the series. Case 6 had isolated complete pupil-involving third nerve palsy with a heterointense sellar lesion measuring 1.4 × 2.6 × 1.1 cm, considered likely to be a pituitary macroadenoma. The documented differential included diabetic ischemic neuropathy, vasculitis, and pituitary macroadenoma [21]. Case 8 demonstrated a larger hypoenhancing lobulated sellar and suprasellar lesion measuring approximately 1.7 × 2.4 × 2.4 cm, with expansion of the sella and a diagnosis of pituitary macroadenoma-associated pupil-involving third nerve palsy. The associated headache, vomiting, prolonged loss of consciousness, and progressive ptosis further demonstrated the importance of considering an intracranial structural cause in patients with evolving neurological symptoms [22,23].

The most critical presentation in the series was Case 10, in which pupil-involving third nerve palsy occurred in association with a ruptured posterior communicating artery aneurysm and grade II subarachnoid hemorrhage. The acute presentation with headache, ptosis, loss of consciousness, and vomiting was followed by neuroimaging confirmation of subarachnoid hemorrhage [24]. Definitive treatment was performed with endovascular coil embolization using balloon-remodeling technique, followed by extubation without new neurological deficits and documented postoperative improvement. This case underscores the potentially life-threatening nature of acute third nerve palsy when associated with intracranial vascular pathology [25].

Overall, the series demonstrates that third nerve palsy may occur in the setting of systemic vascular disease, trauma, orbital injury, sellar lesions, and intracranial aneurysmal disease. Pupil-sparing and pupil-involving patterns were both represented, while neuroimaging ranged from normal examinations to clearly demonstrable orbital, sellar, suprasellar, traumatic, and vascular abnormalities. The findings support a systematic approach incorporating onset and progression of symptoms, pupillary examination, ocular motility assessment, vascular and traumatic history, associated neurological manifestations, and appropriate neuroimaging. The diversity of cases in this series highlights the importance of identifying the underlying cause rather than treating third nerve palsy solely as an ocular motility disorder.

CONCLUSION

Third nerve palsy has a heterogeneous clinical and etiological presentation and may arise from microvascular ischemia, trauma, orbital injury, sellar lesions, and intracranial vascular pathology. In this case series of 10

patients, both pupil-sparing and pupil-involving complete third nerve palsies were observed, with clinical manifestations ranging from isolated ptosis and diplopia to headache, vomiting, loss of consciousness, and associated traumatic or neurological abnormalities. Neuroimaging played an important role in identifying underlying structural abnormalities, including orbital fractures, diffuse axonal injury, pituitary macroadenoma, and ruptured posterior communicating artery aneurysm. The series emphasizes that careful assessment of pupillary status, systemic and traumatic history, associated neurological features, and appropriate neuroimaging is essential for determining the underlying cause and guiding timely management of third nerve palsy.

REFERENCES

1. Schroeder RM, Stunkel L, Gowder MTA, Kendall E, Wilson B, Nagia L, Eggenberger ER, Van Stavern GP. Misdiagnosis of Third Nerve Palsy. J Neuroophthalmol. 2022 Mar 1;42(1):121-125. doi: 10.1097/WNO.0000000000001010. Epub 2020 Sep 23. PMID: 32991390; PMCID: PMC9179086.
2. Chan Chung C, Wen DW, Chew TLT, See SJ. Incomplete Third Nerve Palsy Due to a Carotid-Cavernous Fistula. Neurologist. 2023 Jan 1;28(1):49-53. doi: 10.1097/NRL.0000000000000437. PMID: 35442935.
3. Rosso M, Ramaswamy S, Sucharew H, Vagal A, Anziska Y, Levine SR. Isolated Third Cranial Nerve Palsy in Pituitary Apoplexy: Case Report and Systematic Review. J Stroke Cerebrovasc Dis. 2021 Sep;30(9):105969. doi: 10.1016/j.jstrokecerebrovasdis.2021.105969. Epub 2021 Jul 22. PMID: 34303962.
4. Radia M, Stahl M, Arunakirinathan M, Kadhim M. Examination of a third nerve palsy. Br J Hosp Med (Lond). 2017 Dec 2;78(12):C188-C192. doi: 10.12968/hmed.2017.78.12.C188. PMID: 29240513.
5. Khalili MR, Roshanshad A, Vardanjani HM. Botulinum Toxin Injection for the Treatment of Third, Fourth, and Sixth Nerve Palsy: A Meta-Analysis. J Pediatr Ophthalmol Strabismus. 2024 May-Jun;61(3):160-171. doi: 10.3928/01913913-20231120-02. Epub 2023 Dec 19. PMID: 38112391.
6. Nagendran ST, Lee V, Perry M. Traumatic orbital third nerve palsy. Br J Oral Maxillofac Surg. 2019 Jul;57(6):578-581. doi: 10.1016/j.bjoms.2019.01.029. Epub 2019 May 31. PMID: 31155398.
7. Nelson LB. A Surgical Option for Complete Third Nerve Palsy. J Pediatr Ophthalmol Strabismus. 2019 Jan 23;56(1):9. doi: 10.3928/01913913-20181212-01. PMID: 30673101.
8. Vaphiades MS, Roberson GH. Imaging of Oculomotor (Third) Cranial Nerve Palsy. Neurol Clin. 2017 Feb;35(1):101-113. doi: 10.1016/j.ncl.2016.08.009. PMID: 27886888.
9. Arya Pv A, Madathanapalli KA, Tenezaca FC, Wang A. Temporal arteritis presenting as third nerve palsy - a case report and review of literature. Rheumatol Int. 2024 Oct;44(10):2245-2251. doi: 10.1007/s00296-024-05604-6. Epub 2024 May 13. PMID: 38739224.
10. Iwasaki M, Nishizawa T, Iida E, Arioka H. Third cranial nerve palsy due to COVID-19 infection. BMJ Case Rep. 2023 May 3;16(5):e255142. doi: 10.1136/bcr-2023-255142. PMID: 37137545; PMCID: PMC10163548.
11. Kameda-Smith M, Pai A, Jung Y, Khan D, Adile AA, Hui K, Martyniuk A, Larrazabal R, Algird A, Klurfan P, Gunnarsson T, Farrokhyar F, van Adel B. Third Nerve Palsy Due to Intracranial Aneurysms and Recovery after Endovascular Coiling. Can J Neurol Sci. 2022 Jul;49(4):560-568. doi: 10.1017/cjn.2021.145. Epub 2021 Jun 24. PMID: 34167603.
12. Bolton N, Gushchin A, Williams KM. 2. Acquired third nerve palsy. Dis Mon. 2021 May;67(5):101131. doi: 10.1016/j.disamonth.2021.101131. Epub 2021 Mar 5. PMID: 33678417.
13. Villamil F, Caffaratti G, Ruella ME, M Giovannini SJ, Cervio A. Postoperative Third Nerve Palsy after Endoscopic Endonasal Transsphenoidal Surgery for Pituitary Adenoma: Elucidating its Mechanism. Neurol India. 2023 May-Jun;71(3):522-526. doi: 10.4103/0028-3886.378673. PMID: 37322750.
14. Nichani P, Montanera WJ, Micieli JA. Third nerve palsy due to influenza A infection. Can J Ophthalmol. 2022 Feb;57(1):69. doi: 10.1016/j.jcjo.2021.02.035. Epub 2021 Mar 23. PMID: 33766487.
15. Habek M, Brinar VV, Ozretić D. Third nerve palsy. J Emerg Med. 2010 Sep;39(3):e131-3. doi: 10.1016/j.jemermed.2007.12.037. Epub 2008 Aug 30. PMID: 18762403.
16. Wen LY, Wah LP, Mohamad NF, Singh S, Toong LY. Isolated third nerve palsy: Lessons from the literature and 4 case studies. J Fam Pract. 2023 Mar;72(2):E1-E7. doi: 10.12788/jfp.0563. PMID: 36947782.
17. Shakrawal J, Bhatnagar KR, Srivastava K. COVID-19 - associated third nerve palsy. Indian J Ophthalmol. 2021 Oct;69(10):2913-2915. doi: 10.4103/ijo.IJO_1619_21. PMID: 34571694; PMCID: PMC8597522.
18. Balik AÖ, Özgültekin A, Aksoy Y. Transient bilateral third cranial nerve palsy following traumatic perimesencephalic subarachnoid haemorrhage. Br J Hosp Med (Lond). 2023 Jan 2;84(1):1. doi: 10.12968/hmed.2022.0341. Epub 2023 Jan 5. PMID: 36708344.

19. Thompson B, Kerr N, Bell A, Graves E, McGregor A. Congenital Ocular Neuromyotonia with Partial Third Nerve Palsy. J Binocul Vis Ocul Motil. 2019 Jan-Mar;69(1):13-17. doi: 10.1080/2576117X.2018.1563451. Epub 2019 Feb 26. PMID: 30806169.

20. Donaldson L, Mandell DM, Iizuka M, Margolin E. Third Nerve Palsy Secondary to Compression by Dolichoectatic Posterior Communicating Artery. Can J Neurol Sci. 2022 May;49(3):463-464. doi: 10.1017/cjn.2021.133. Epub 2021 Jun 14. PMID: 34121641.

21. Arrigoni F, Rombetto L, Redaelli D, Mancarella G, Polenghi F, Salati R, Romaniello R, Peruzzo D, Bianchi PE, Piozzi E, Mazza M, Magli A. Congenital isolated unilateral third nerve palsy in children: the diagnostic contribution of high-resolution MR imaging. Neuroradiology. 2023 Apr;65(4):865-870. doi: 10.1007/s00234-022-03106-5. Epub 2022 Dec 29. PMID: 36580093.

22. Thurtell MJ, Longmuir RA. Third nerve palsy as the initial manifestation of giant cell arteritis. J Neuroophthalmol. 2014 Sep;34(3):243-5. doi: 10.1097/WNO.0000000000000116. PMID: 24667773.

23. Aygit ED, İnal A, Ocak OB, Celik S, Fazıl K, Yildiz BK, Taskapili M, Gokyigit B. Simplified approach of Gokyigit's technique for complete cranial nerve third palsy. Int Ophthalmol. 2019 Jan;39(1):111-116. doi: 10.1007/s10792-017-0794-x. Epub 2017 Dec 23. PMID: 29274025.

24. Chaurasia S, Sharma P, Kishore P, Rasal A. Surgical strategy for third nerve palsy with aberrant regeneration: Harnessing the aberrant power. Indian J Ophthalmol. 2021 Apr;69(4):910-917. doi: 10.4103/ijo.IJO_1701_20. PMID: 33727458; PMCID: PMC8012919.

25. Wen Y, Shen T, Yan J. Larger Than Larger: Huge Amounts of Recession-Resection Surgery for Third Nerve Palsy. J Pediatr Ophthalmol Strabismus. 2023 May;60(3):184-194. doi: 10.3928/01913913-20220428-03. Epub 2022 Jun 27. PMID: 35758195.