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Eye : A Window To The Brain

Dr. Archana Tadwalkar, Dr. Harsha Pagad, Dr.Pooja Kumari, Dr Dipti Singh

***Corresponding Author:**
Dr Archana Tadwalkar
D.Y. Patil School of Medicine and Hospital, Nerul, Navi Mumbai

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Abstract

The present case report aims at reviewing the topographical localisation of the lesions / area affected by stroke by means of analysing the presenting ocular motor abnormalities in an emergency medical set up .
Three cases are described wherein the patient presented with an aggregation of discrete eye abnormalities classically aiding in the localisation of lesions in the brain.

Keywords: Parinaud Syndrome, Millard Gubler syndrome, Cranial nerve palsy

Introduction

Case History :

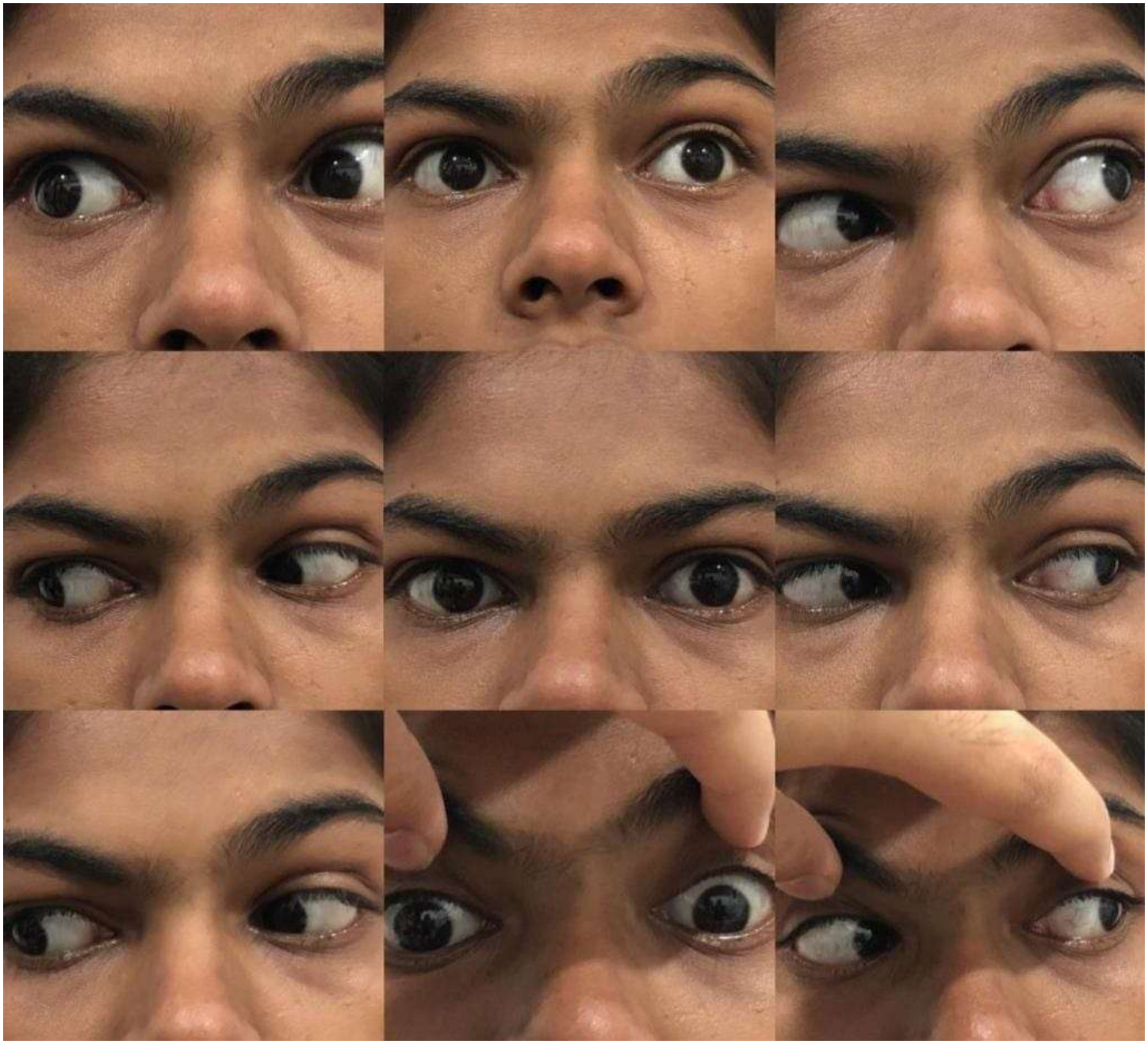
The first case is of a **18** year old girl who presented with acute onset of binocular diplopia from 1 week noticed on waking up from sleep especially when looking up and down .There was associated left sided headache and left eye pain.There was no history of projectile vomiting, transient visual obscurations,diminution of vision, redness or photophobia. There was no associated head trauma or any prior medical condition, fever.

Patient did not report any neurological symptoms. Her best corrected visual acuity was 6/6, N6 in both eyes with a correction of -1.25 dc @180. Ocular motility evaluation revealed a -4 limitation of elevation and -2 limitation of depression in both eyes. There was presence of convergence retraction nystagmus, Colliers sign in both eyes.There was light near dissociation in the left eye.

We went ahead with the neuroimaging in this patient with a clinical diagnosis of Parinaud's syndrome. MRI brain revealed a tiny acute non hemorrhagic infarct in midbrain around peri aqueductal grey matter. MRI orbit was normal. Patient was extensively investigated for any thrombophilic disorders, autoimmune diseases causing vasculitis, complete cardiac evaluation for any source of emboli but all were unyielding.

The definitive cause of infarct in this young patient could not be discerned. Patient was treated with low molecular weight heparin and antiplatelet agents. Improvement in depression after 2 weeks but elevation limitation had remained. Further follow ups were only by telephonic conversation due to COVID pandemic when the parents reported marked improvement and resolution of diplopia.

Fig1: 9 Gaze demonstration



The second case is of a 75 year old male with complaints of blurring of vision in both eyes past 1 year, drooping of left eye past a year with limitation of movements in both eyes. There was past history of stroke a year ago following which the complaints started . He was a known diabetic and hypertensive on oral medications for the same. His visual acuity was counting fingers at 2 m in both eyes, not improving. Anterior segment evaluation showed grade 3 nuclear sclerosis in both eyes .Intraocular pressure measured by applanation tonometry was 12 mmHg in both eyes. Pupillary evaluation was as follows. Right eye pupil was round, reactive to light. Left pupil was mid-dilated. The anisocoria increased in bright illumination. There was no afferent pupillary defect. The Fundus examination was unremarkable. External examination showed presence of bilateral mild ptosis. There was 30 degree left exotropia on Hirschberg corneal reflex test. Extraocular motility examination revealed -3 limitation of elevation in right eye and -4 limitation of adduction, elevation and depression in the left eye suggestive of a nuclear third nerve palsy.

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MRI brain dated a year back showed well defined focal areas of hyperintense on T2W1 in midbrain suggestive of acute infarct.

Fig 2: Pupil RE: CCRTL LE: Mid dilated fixed

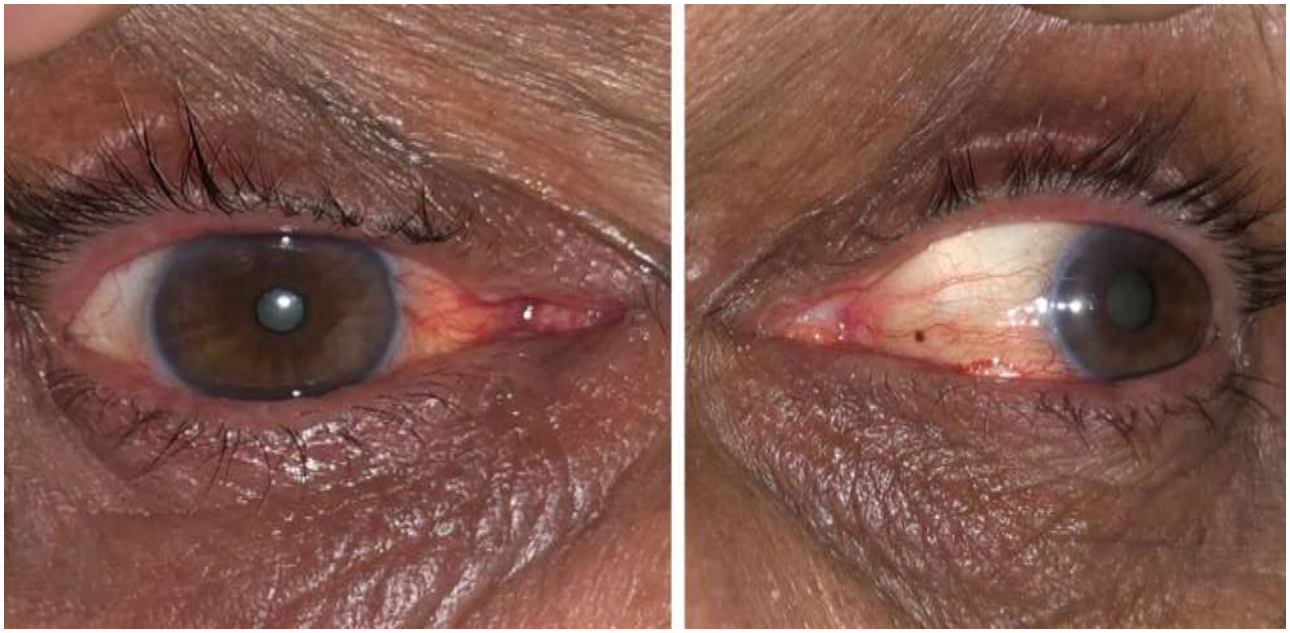

Fig 3: 9 gaze demonstration

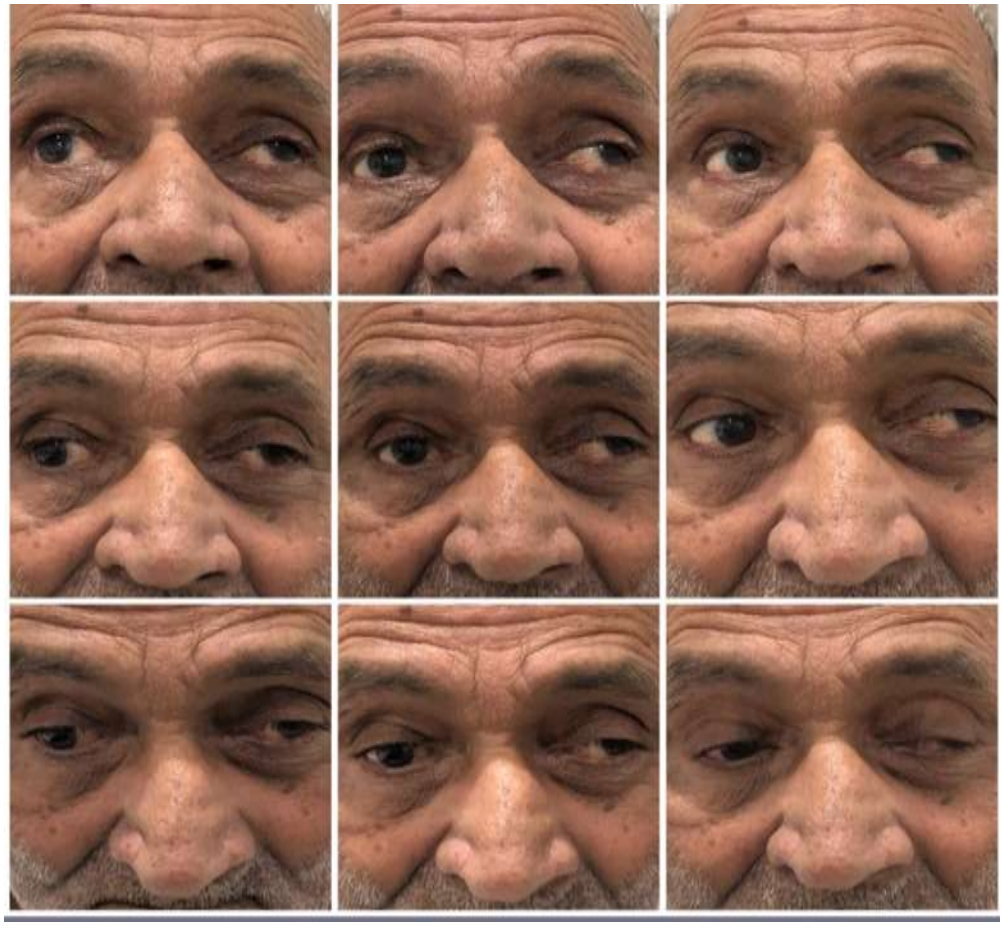

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Fig 4: Intorsion caused by superior oblique on looking down

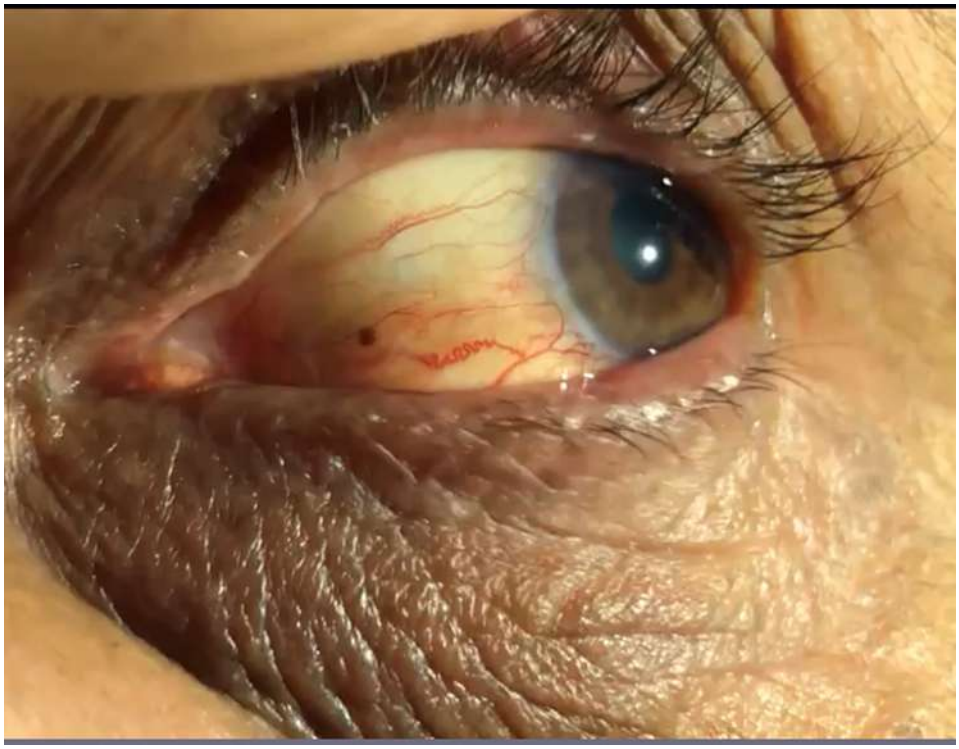
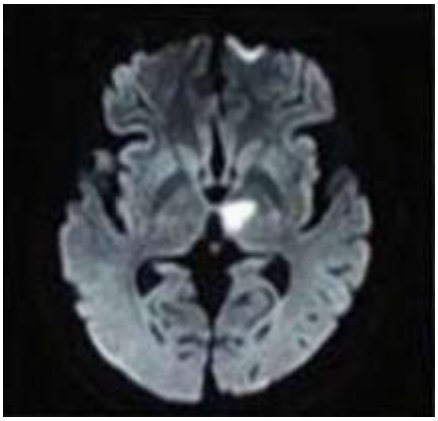


Fig 5: MRI Brain: Well defined focal areas of hyperintensity on T2WI in MidBrain(L>R) Suggestive of Acute Infarct



The third case is of a 70 year old male who presented with sudden onset of weakness in upper and lower limbs and binocular diplopia on looking to the left side. His best corrected visual acuity was 6/9 in right eye and 6/36 in left eye. Pupils were miotic with sluggish reaction to light. Patient was pseudophakic in both eyes. Dilated fundus evaluation was normal. Extraocular motility examination showed limitation of adduction in right eye with abduction limitation in left eye suggestive of left gaze palsy. MRI brain done was s/o acute non hemorrhagic infarct at the pontomedullary junction. MR Angiography showed narrowing in the proximal part of the basilar artery just distal to its origin. Pt was a known diabetic on oral medications. So a diagnosis of Millard Gubler Syndrome was made with ipsilateral gaze palsy and contralateral hemiparesis.

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Fig 6: 9 gaze demonstration

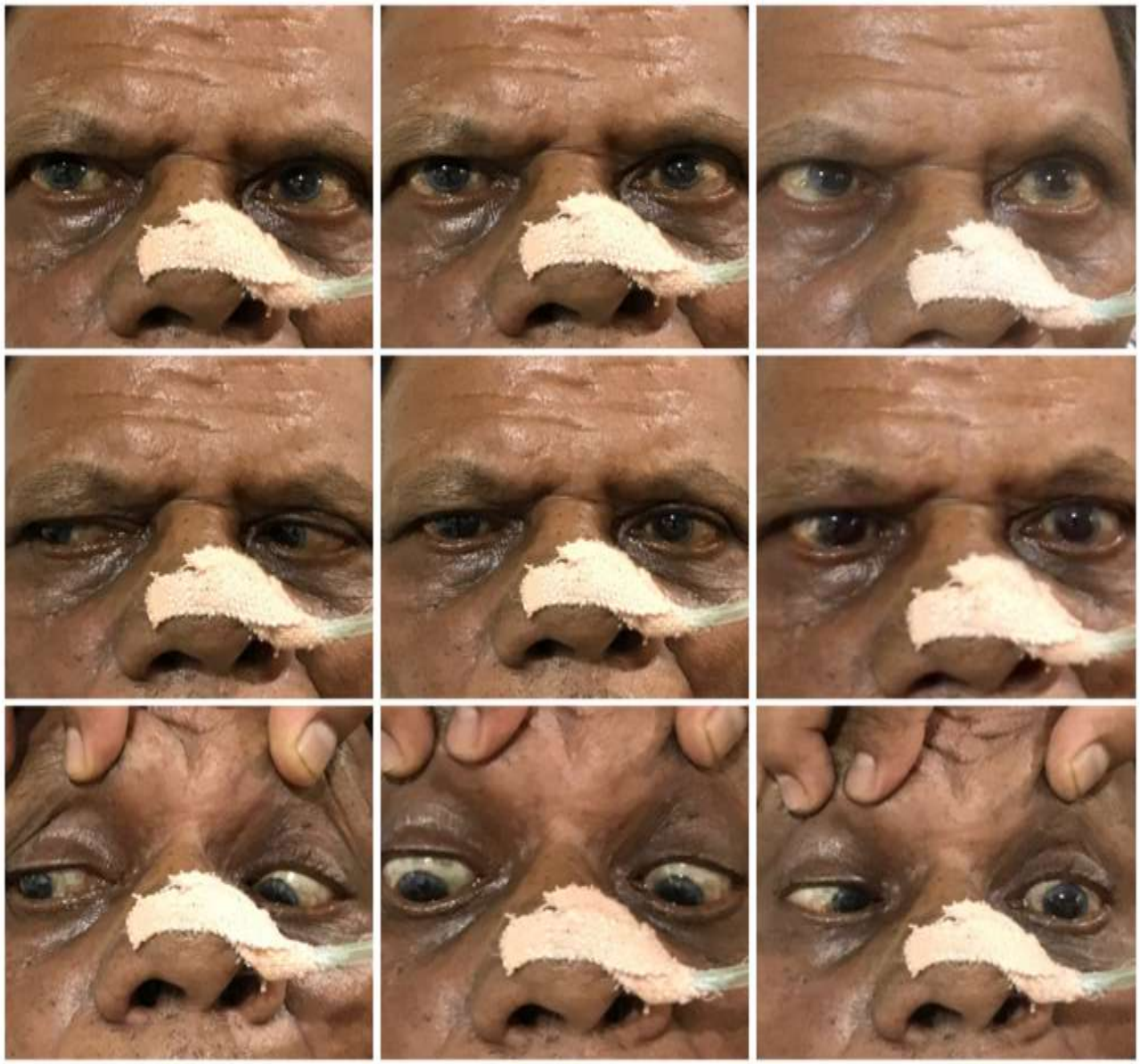
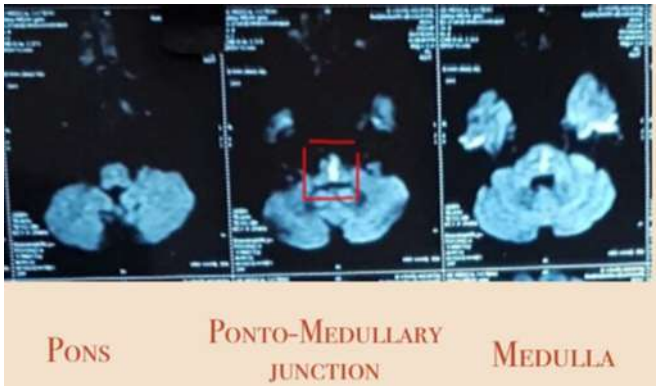


Fig 7: MRI Brain: Acute non hemorrhagic infarct at the pontomedullary junction



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Discussion

Case1: Parinaud Syndrome

French ophthalmologist, Henri Parinaud, first described Parinaud syndrome in the late 1800s. Parinaud described it in a series of case reviews of patients with disturbances of associated eye movements and gaze paralysis. He attributed the cause of this condition to a lesion of the quadrigeminal area. This condition is variously known as the Sylvian aqueduct syndrome, dorsal midbrain syndrome, Pretectal syndrome, and Koerber-Salus-Elschnig syndrome.[1]

The classic triad of conjugate up gaze paralysis, convergence-retraction nystagmus and light-near dissociation was only present in 65% of cases. Pineal neoplasms remain an important etiological consideration. Conservative management approaches for ocular symptoms are sufficient in most cases although surgical treatment of up gaze palsy can be a useful option in refractory cases.[2]

Case 2: The Third Cranial Nerve Palsy

Third nerve palsies can result from lesions located anywhere from the oculomotor nucleus to the termination of the third nerve in the extraocular muscles within the orbit, and may be the herald manifestation of underlying neurological emergencies such as intracranial aneurysm, pituitary apoplexy, and giant cell arteritis.[3]

Nuclear third nerve palsy can result from ischemia, infective causes, or tumors

The topographic arrangement of oculomotor subnuclei within the midbrain is responsible for the varying clinical presentations in nuclear, fascicular ,or infra nuclear lesions.[4]

Case 3- MILLARD GUBLER SYNDROME

Millard-Gubler syndrome (MGS), also known as facial abducens hemiplegia syndrome or the ventral pontine syndrome. It is a classical crossed brainstem syndromes characterized by a unilateral lesion of basal portion of the caudal *pons* involving fascicles of abducens (VI) and the facial (VII) cranial nerve, and the pyramidal tract fibers.[5]

Components of MGS include ipsilateral weakness of the eye on abduction (VI nerve, Ipsilateral facial muscle weakness (VII nerve), Contralateral hemiparesis or hemiplegia of upper and lower extremities (pyramidal tract involvement).

Conclusion

The various parts of the brain like cortex , basal ganglia , cerebellum and brainstem are involved in the generation and control of eye movements . The signals of this complex neural network finally converge on the ocular motoneurons of the brainstem. Infarct or hemorrhage at any level of the oculomotor system may give rise to a broad spectrum of eye movement abnormalities . These eye movement abnormalities when recognised at the right time will have high localizing value .

Our case series proves this beyond doubt . All our patients had classic eye movement abnormalities which aided in topographical localisation of brain lesions even before the results of neuroimaging were available.Recognition of the patterns and characteristics of abnormal eye movements observed is important in understanding the roles of each neural structure and circuit in ocular motor control as well as in localizing the offending lesion.

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