



Isolated Medial Rectus Palsy as a Presentation of Midbrain Infarct

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ABSTRACT

Isolated ocular muscle palsy involving lateral rectus and superior oblique is very common due to individual nerve supply but isolated palsy of the remaining four ocular muscles is a very rare condition and most of the recorded cases are either due to midbrain infarct or local pathological causes. A 60 years female presenting with diplopia on left lateral gaze without any other neurological abnormality was diagnosed as isolated medial rectus palsy due to incomplete involvement of oculomotor nucleus by DWI imaging and fully recovered after treatment with antihypertensive and statin.

INTRODUCTION

Incomplete third CN lesion causing paresis rather than paralysis and affecting certain functions more than others, are more common than complete ones.^[1] Lesion involving oculomotor nucleus or the course of the nerve after it has split into its superior and inferior division are more likely to involve two or more extraocular muscles. But isolated medial rectus palsy is a rare condition due to involvement of ventral oculomotor subnucleus which can be confused with and must be distinguished from internuclear ophthalmoplegia by the absence of nystagmus in the abducting eye.

CASE REPORT

A 60yr old female presented with complain of sudden onset head reeling for last two days

associated with double vision mainly on looking towards left side. Patient was a known hypertensive for last 4 years and was on amlodipine and atenolol. No history of vomiting, headache, seizure, parosmia, anosmia, slurring of speech, dysphagia or weakness in any limbs. She is a nondiabetic and has no history of similar neurological illness in the past. On examination patient was conscious, oriented, PR=60/min, BP=120/80 mm Hg. Neurological examination revealed intact higher function, normal cranial nerve examination except failure of adduction of right eye on left gaze [Fig.1,2], no nystagmus, normal fundoscopy. Motor, sensory, reflexes and cerebellar functions were all intact. Routine investigation reports showed FBS=99mg/dl, Sr. cholesterol=239mg/dl, Sr Triglyceride=267mg/dl,

Sr. HDLC=39mg/dl, Sr. LDLC=136mg/dl. Initial CT scan of brain revealed no lesion. MRI was done later which showed hyperintensity on T2/FLAIR [Fig. 3] and DWI [Fig 4] in rostral midbrain paramedian plane at the level of superior colliculus suggestive of recent infarction. Patient was treated for hypertension and dyslipidemia with Amlodipine +Atenolol, Atorvastatin+Fenofibrate, Clopidogrel and physiotherapy. Patient condition started improving and was discharged on 5th day with the above medications. Patient recovered completely within 4 weeks.

DISCUSSION

The oculomotor, or third cranial nerve (CN III), arises from the oculomotor nuclear complex in the midbrain and conveys motor fibers to extraocular muscles, plus parasympathetic fibers to the pupil and ciliary body. These nuclear centers are situated in the periaqueductal gray matter just anterior to the aqueduct of Sylvius, at the level of the superior colliculi. A single midline structure, the central caudal nucleus, supplies the levatorpalpebrae muscles on both sides. There are 4 unpaired subnuclei.^[2] The sub nucleus for superior rectus muscle is situated dorsomedially and decussation of the fiber to the superior rectus takes place within the oculomotor nuclear complex. Thus, lesion affecting the nucleus may simultaneously involve ipsilateral superior subnuclei as well as crossing fibers resulting in bilateral superior rectus muscle palsy.^[3] Ventral to

superior rectus subnuclei lies the subnuclei for ipsilateral inferior rectus followed by intermediate subnuclei for ipsilateral inferior oblique and most ventrally situated subnuclei is for ipsilateral medial rectus. Third nerve nuclear lesion causes weakness of bilateral superior rectus muscle ,ipsilateral inferior oblique, medial rectus ,with bilateral partial ptosis [due to involvement of central caudal subnuclei supplying both levatorpalpebraesuperioris]. This case is a nuclear oculomotor palsy with unusual presentation due to location of infarct in rostral midbrain involving medius rectus subnuclei which is situated most ventrally and can be diagnosed with diffuse weighted imaging.^[4] Unilateral ocular palsy is commonly seen with lesions in the orbit like orbital wall fracture with muscle incarceration and rarely from third nerve nuclear lesion, though inferior oblique muscle palsy caused by involvement of intermediate subnuclei and isolated inferior rectus palsy due to ipsilateral involvement of dorsally situated subnuclei or fascicular lesion have been described.^{[5][6]} DWI is more sensitive than conventional T1/T2 MRI which may not demonstrate an infarct for 6 hours while increased DWI signal in ischemic brain tissue is observed within a few minutes after arterial occlusion. With use of DWI and other multimodality MRI the probability of picking up midbrain infarct causing isolated oculomotor palsies have increased

Fig 1 |

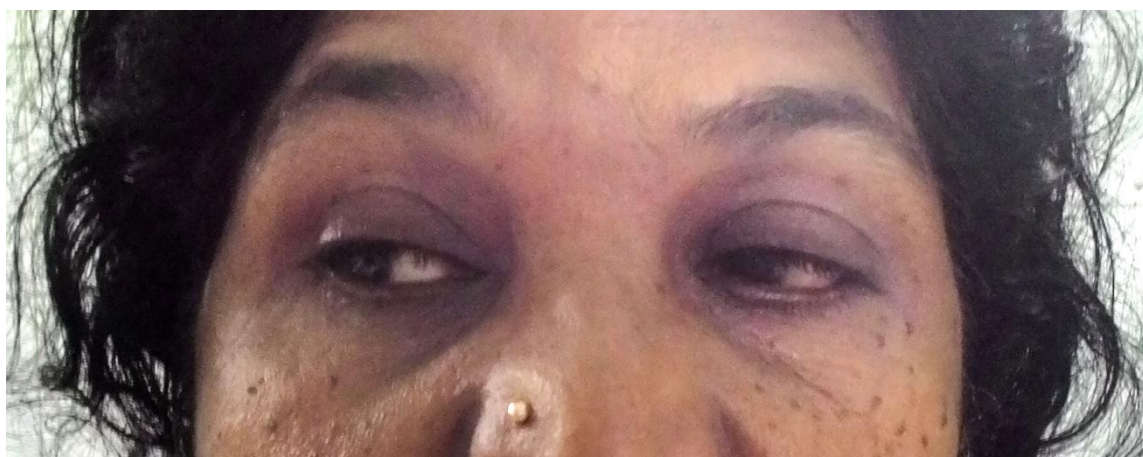


Fig 2



Fig 3

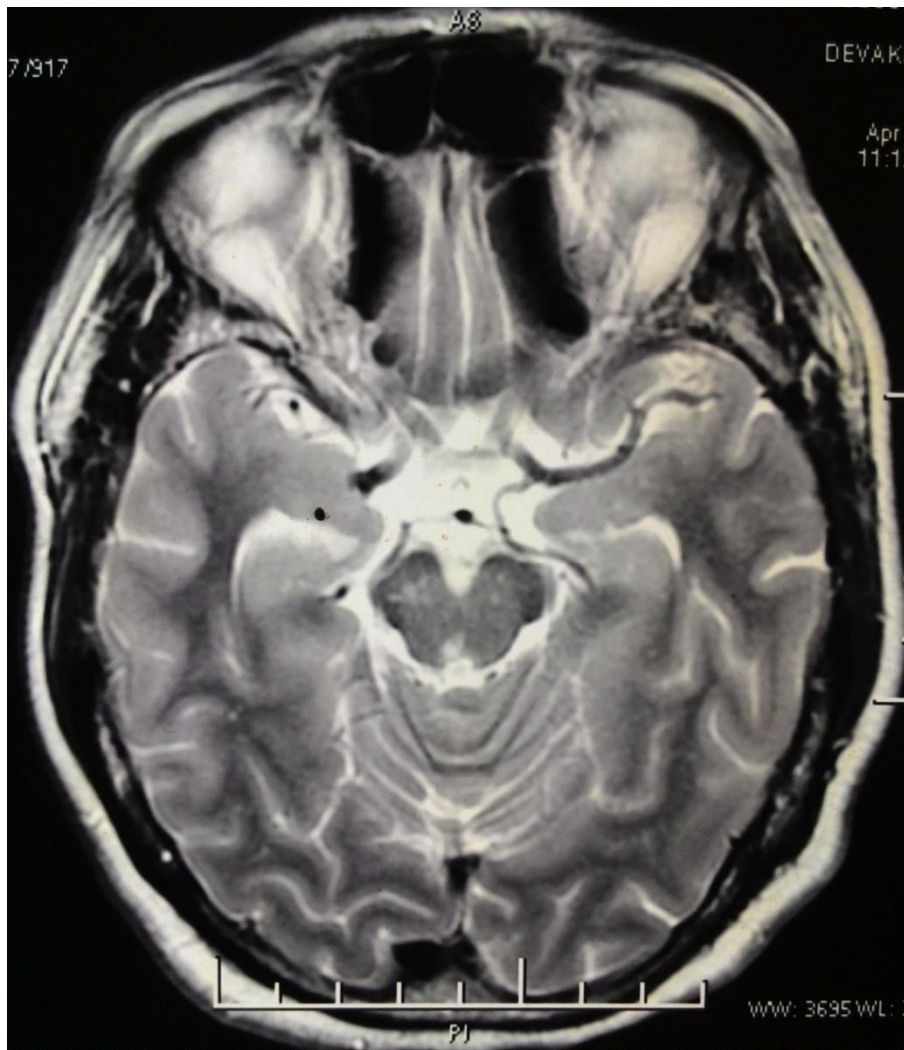
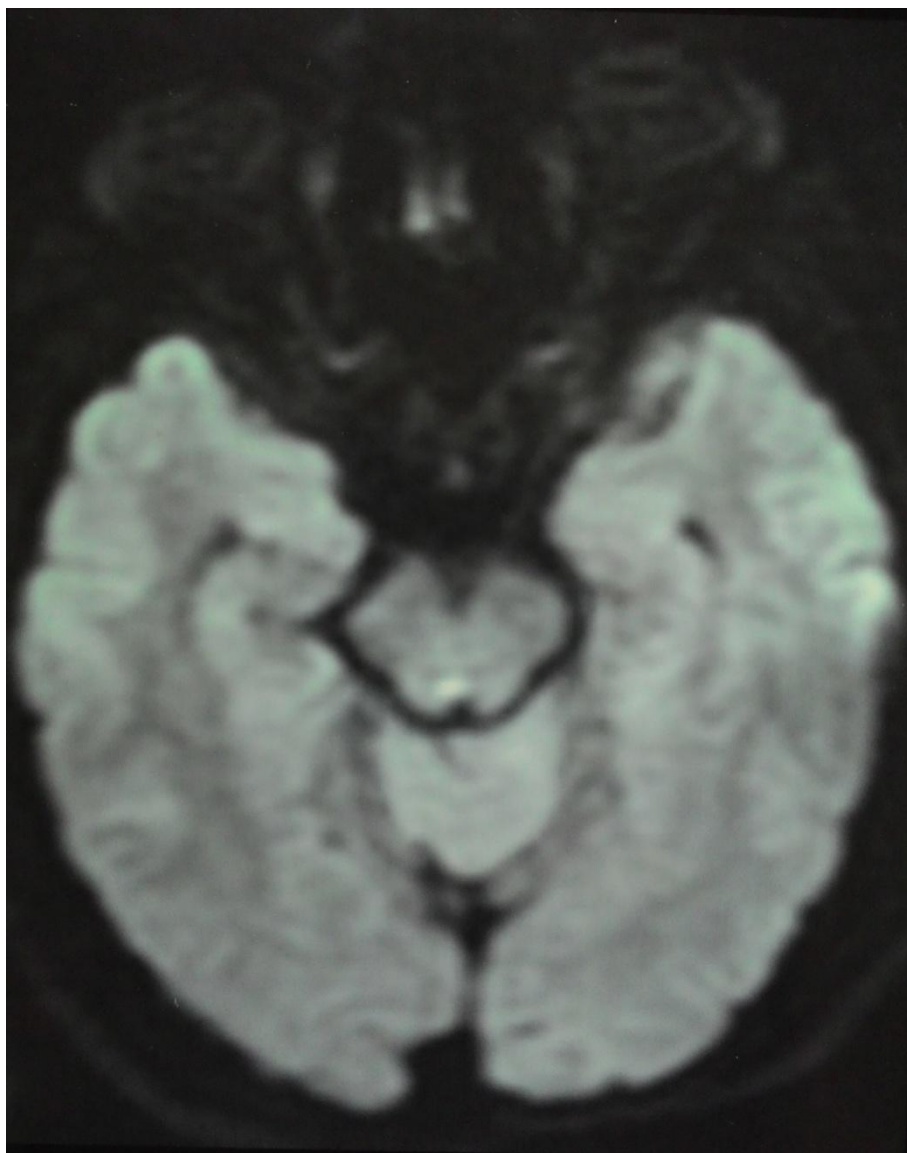


Fig 4



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