Right Internal Carotid Artery Thrombosis Presenting as Bilateral Ptosis with Bilteral Cerebellar Signs

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Abstract- Mid brain is often affected in patients with embolic stroke occurring in the posterior circulation usually with concomitant involvement of other structures as the pons,thalamus and the cerebellum.Here we are presenting a rare case of internal carotid artery thrombosis with hyperhomocystenemia presented with bilateral ptosis with mid brain infarction in a 40yr old non hypertensive and non-diabetic farmer.

Index Terms- Bilateral ptosis,posterior cerebral circulation,mid brain infarction,internal carotid artery thrombosis.

I. INTRODUCTION

The third cranial nerve occulomotor nerve inervates the medial, inferior and superior recti, inferior oblique,levatorpalpebraesuperioris and the iris sphincter. Total palsy of the oculomtor nerve causes ptosis, a dilated pupil, and leaves the eye " down and out" because of the unopposed action of the lateral rectus and superior opblique[1].

The oculomotor nucleus complex lies in the midbrain and consists of :

1. A midline unpaired Structure called the central caudal nucleus that supplies the levatorpalpebrae muscle on both sides. 2 Four lateral paired subnuclei that innervate the superior, inferior, and medial rectus, as well as the inferior oblique muscles. 3. The Edinger – Westphal nucleus, which contains preganglonic, parasympathetic neurons whose axons project to the ciliary ganglion and ultimately control pupillary constriction and accommodation. A third nerve palsy caused by a nuclear lesion is rare. When it occurs, it produces specific deficits in both eyes because of the anatomy of the nucleus complex : 1. Superior rectus subnucleus : Axons from one superior rectus (SR) subnucleus results in bilateral superior rectus palsy 2. Central caudal nucleus : Unpaired and supplies both levatorpalpebrae muscles, thus, a lesion in the nucleus causes bilateral ptosis. Located in the most caudal part of the oculomotor nuclear complex, so it may be selectively affected (i.e. bilateral ptosis may be the only manifestation of a nuclear third nerve palsy), or it may be selectively spared. 3. Medial rectus subnuclei: lie in three different locations; thus, an isolated medial rectus palsy (unilateral or bilateral) without other muscle involvement cannot be as nuclear third nerve palsy. 4. Edinger-Westphalnucleus: spread throughout the rostral half of the oculomotor nucleus complex ; thus, the pupil may be spared in lesions affecting the caudal half of the complex, but when the pupil is involved, both pupils are affected (i.e.bilateral internal ophthalmoplegia). This summary was taken from Chp 12, Nuclear and infranuclear Ocular Motor Disorders. Wong AM (Ed) Eye Movement Disorders, Oxford University Press, 2008[2].

Posterior circulation is composed of the paired vertebral artery, basilar artery & paired posterior cerebral arteries. These major arteries give rise to short & long circumferential branches that supply the cerebellum, medulla, pons, midbrain, thalamus, hippocampus and medial temporal & occipital lobes. PCA syndrome usually result from atheroma or emboli at the top of basilar artery, finbromusculardysplaisa or vertebral artery dissection.

Posterior cerebral artery encircles the midbrain close to the oculomotor nerve at the level of tentorium and supplies the inferior part of temporal lobe and the occipital lobe(Marinkovic et al 1987).Many small perforating arteries arise from the proximal portion of PCA to supply the mid brain,thalamus,hypothalamus and geniculate bodies. Sometimes a single perforating artery supplies the medial part of each thalamus and both sides of mid brain.

In about 15 percent of individuals the PCA is direct continuation of POCA its main blood supply then coming from ICA rather than basilar artery[3].

The occulomotor nerve travels between the superior cerebellar artery and posterior cerebral artery to enter the cavernous sinus. In subarachnoid space, the nerve lies adjacent to the posterior communicating artery and the tip of the basilar artery. Therefore, aneurysms of these arteries often compress the third nerve[4].

II. CASE REPORT

A 40 year old male patient presented with altered sensorium inability to raise both eye lids, unable to talk and the weakness of the both lower limbs which was sudden in onset. On examination patient was aphasic, responding to commands unable to open the eyes.

Past History

No history of head trauma, hypertension, CAD, diabetes, and previous neurological problems.

Personal history

Smoker,occasionalalcoholic,no history of drug abuse.

General Examination

Lean male with pulse 76/min regular and all peripheral pulse felt,arterial wall not thickened, BP – 130/70 mm Hg.

Patient was aphasic, responding to commands unable to open the eyes.

Neurological examination

Bilateral Ptosis bilateral dilated fixed pupils with eye balls deviated down wards and out ward,fundus is normal.All other cranial nerves are normal. Motor system examination – bulk normal,hypotonia in all four limbs, power 3/5 in all limbs, corneal conjunctival reflexes not elicitable, all other superficial reflexes present. Deep tendon reflexes 2 + no involvuntary movements,co-ordination and sensory system could not be examined. No meningeal signs ,skull and spine normal.Patient managed conservatively,hissensorium,speechimproved,past pointing ataxia and ptosis are still persisting.After 4 weeks of treatment,power improved 3/5 in all limbs. Patient was able to stand and sit with support.Tone increased in all limbs.Patient was swaying eitherside on standing and sitting with support.Bilateral cerebellar signs like past pointing,heel knee test,truncal ataxia are present.Rombergs couldn't be tested.

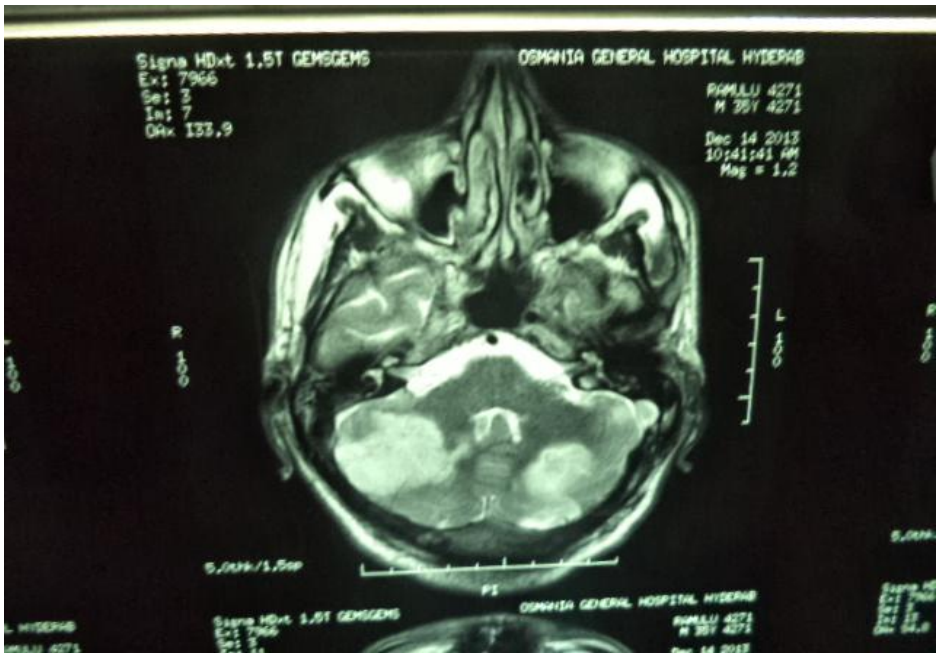
Investigations

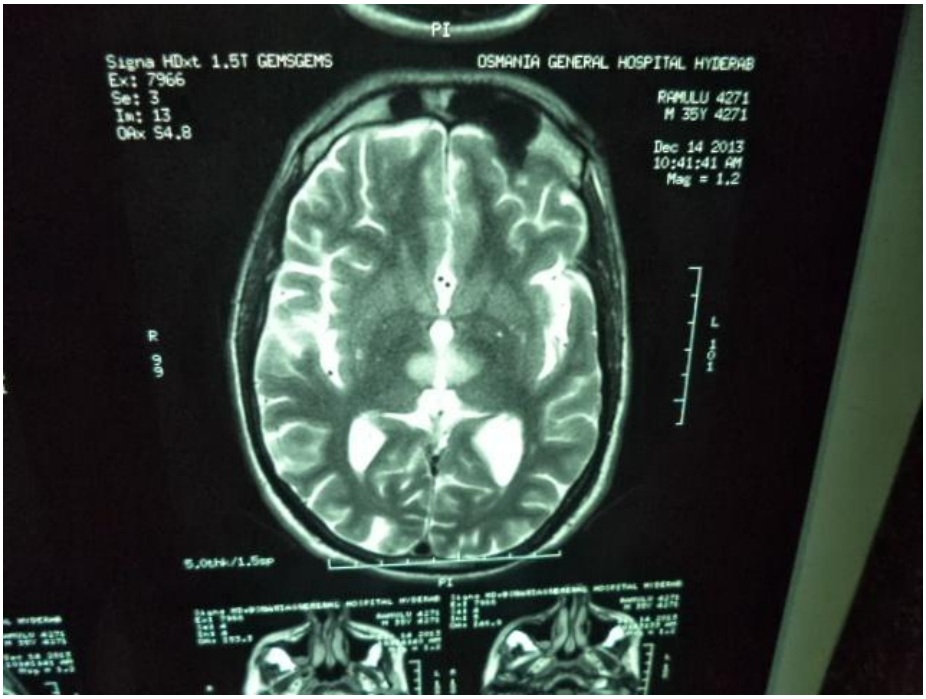
Complete Blood Picture: Hb:13.3,Wbc:6400, Neutrophils:63, Lymphocytes:32, Monocytes:3, Eosinophils:2, Platelets:adequate, ESR:30, RBS:90,Serum urea:20,Serum creatinine:1, Serum electrolytes:Sodium:138, Potassium:3.6

Complete Urine Examination : Albumin:nil, Sugar:nil, Bile salts, Bile Pigments :nil, 1to 2 epithelial cells/hpf, 10 to 20 pus cells/hpf, HIV and HBSAG negative, PT , Test:15 sec, Control:14 sec, INR:1.1, Lipid Profile -Total cholesterol:135, Hdl:23, Ldl:98, Vldl:14, Triglycerides:69, Ultrasound Abdomen:normal study, ECG:WNL, 2D ECHO:no rwma,EF:60, Good LV systolic function, Grade1 diastolic function, Good RV function, Normal valves, Normal sized chambers, No pericadial effusion/clot/vegetation, **Carotid Doppler**:**Thrombosis involving right ICA,CCA,and Proximal ECA, Serum Homocysteine Levels :21.87(3.7 to13.9micromol/litre),**Protein c,protein s,antiphospholipid antibody,factor mutations,factor 1X factor X are normal.

CTScan Brain:Ill definedhypodensities in bilateral thalami,right high parietal region,bilateral cerebellar hemisheres.

MRI BRAIN:Acute infarcts midbrain, in medial thalami,bilateral cerebellar hemisperes,right temporal ,rightoccipital region.Right CCA, ICA ,ECA thrombosis, MRV:normal study.

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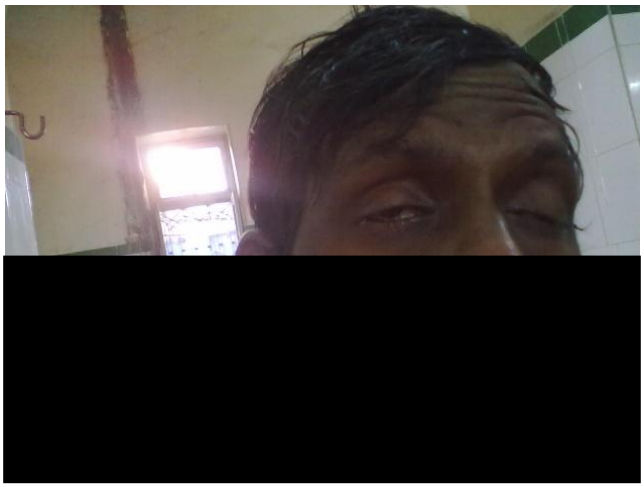

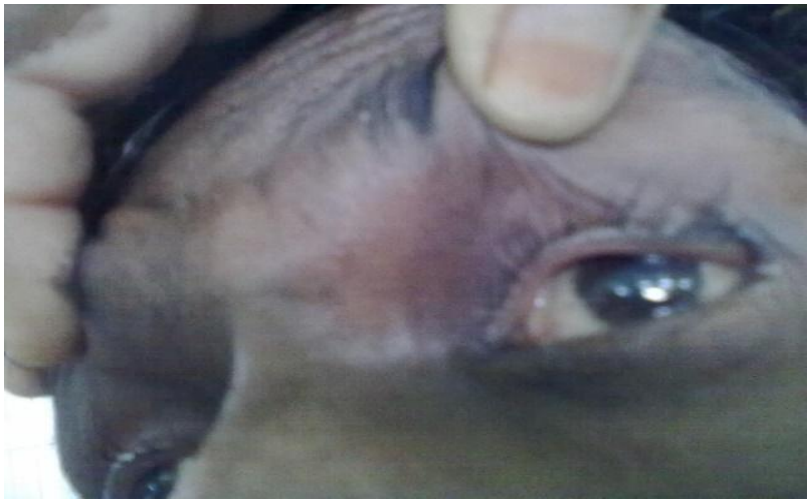

III. DISCUSSION

The midbrain is supplied by the posterior cerebral artery (PCA), basilar artery, superior cerebellar artery, and anterior choroidal artery. The midbrain is often affected in patients with embolic stroke occurring in the posterior circulation, usually with the concomitant involvement of other structures, such as the pons, thalamus, and the cerebellum. Infarction limited to the midbrain is rare. The reported prevalence of pure midbrain infarction varies from 0.7% to 2.3% [5,6]Ptosis could be caused by oculomotor nerve palsy in the patients with midbrain infarction. In several cass, bilateral ptosis showed the clinical characteristics of midbrain infarction [7,8].

The internal carotid artery bifurcates into the anterior cerebral artery and the large middle cerebral artery. Other branches include the ophthalmic artery the posterior communicating artery anterior choroidal artery. The posterior communicating artery (PoCA) passes back to join the first part of the posterior cerebral artery, so contributing to the circle of Willis. Tiny branches supply the adjacent optic chiasm, optic tract, hypothalamus, thalamus, and midbrain [9].

The clinical picture of internal carotid occlusion varies depending on whether the cause of ischemia is propagated thrombus, embolism, or low flow. The cortex supplied by the MCA territory is affected most often. With a competent circle of Willis, occlusion may go unnoticed. If the thrombus propagates up the internal carotid artery into the MCA or embolizes it, symptoms are identical to proximal MCA occlusion. Sometimes there is massive infarction fo the entire deep white matter and cortical surface. When the origins of both the ACA and MCA are occluded at the top of the carotid artery, abulia or stupor occurs with hemiplegia. Hemianesthesia, and aphasia or anosognosia. When the PCA arises from the internal carotid artery (a configuration called a fetal posterior cerebral artery), it may also become occluded and give rise to symptoms referable to its peripheral territory)[10].

We are presenting case of severe bilateral ptosis that occurred with mid brain infarction due to hyperhomocystenemia, right common carotid artery, external carotid artery and right internal carotid artery thrombosis, in which the patient was presented with altered sensorium and unable to open his eyes. On conservative management patient sensorium improved, was able to walk with difficulty but Ptosis is persisting. Patient was on regular follow up. 6 months following presentation also ptosis has not improved at all, but power and cerebellar symptoms have improved. Patient is able to perform activities of daily living and mobility with minimal assistance.He was on lipid lowering drugs

and antiplatelet agents. Patient was referred to ophthalmology department for surgical correction of ptosis.

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