
CASE REPORT**Cerebellar Infarction & Migraine: Uncommon Presentation of a Common Disease**

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Abstract:

Migraine is a common disease in older pediatric population. It is uncommonly associated with cerebellar infarction. The patient presents with headache, symptoms and signs of raised intracranial pressure and cerebellar ataxia. There are very few case reports of cerebellar infarction in migraine in pediatric age group. We could not find any Indian case report, after a thorough literature search. First such Indian case is presented and discussed.

Keywords: Cerebellar Infarction, Childhood Migraine, Basilar Artery.

Introduction:

Migraine is a common disease in older pediatric population. Children usually present with some form of headache with or without other associated symptoms or aura. It is uncommonly associated with cerebellar infarction. This is due vasoconstriction of the vertebrobasilar system. The patient presents with headache, symptoms and signs of raised intracranial pressure and cerebellar ataxia. One such case is presented.

Case Report:

Pruthviraj Thakur, a 10 years old boy studying in class 5 was brought for generalized headache since 2 days that began while attending the school. Next day this was followed by giddiness, diplopia, vomiting and inability to stand.

There was no history of convulsions, altered sensorium, head injury, fever, rash or ear discharge. There was no history of any medication.

There was history of generalized headache on and off since last one year. It was not associated with giddiness or vomiting or any form of aura. It used to subside either spontaneously or after pain relievers and rest. Occasionally there was loss of school days.

The father had history of headache which is suggestive of migraine.

There was no other contributory past or family history.

On general examination the child was conscious, co-operative and well-oriented.

Pulse was 90/min, regular, good volume. Both carotids and peripheral pulses were normal. The blood pressure was 94/64 mm of mercury in right upper limb in supine position. There was mild pallor and mild dehydration. Ear, nose and throat examinations were normal. The skeletal system was normal.

The higher functions, cranial nerves and sensory system were normal.

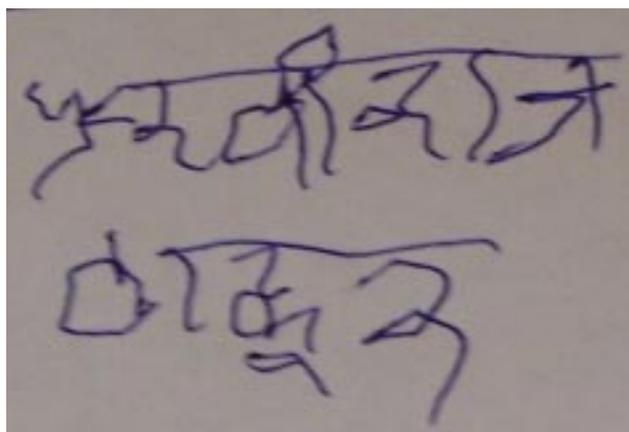
On examination of motor system bulk, nutrition and tone of all limb muscles were normal.

The boy had unstable gait and was unable to stand. The power of all groups of muscles in all four limbs was normal. The deep tendon reflexes were sluggish on the right side. The plan-

tar reflex could not be elicited on right side. Cerebellar signs were present on right side viz: limb ataxia, positive finger nose and knee heel test, gait ataxia, rebound phenomenon and dysdiadocokinesis. He had dysarthria and his handwriting had become illegible.

With this presentation the possible etiology

Fig. 1: Handwriting on admission



considered was vascular, post infectious or a mass lesion.

Complete blood count, peripheral smear, erythrocyte sedimentation rate, and routine urine examination were normal. The bleeding time was 1 minute 30 seconds, clotting time was 3 minutes 15 seconds, prothrombin time was 12 seconds and activated partial thromboplastin time was 35 seconds. Chest roentgenogram and colour doppler of heart were normal. M.R.I. of brain revealed cerebellar infarction. M.R. angiography was normal.

With symptoms related to vertebro-basilar circulation, cerebellar signs, family history of migraine, normal hematology and coagulation workup, cerebellar infarct on M.R.I. and normal M.R. angiography, diagnosis of basilar type migraine was made.

The boy was treated with tablet flunarizine once

Fig. 2: T1 weighted image (sagittal)

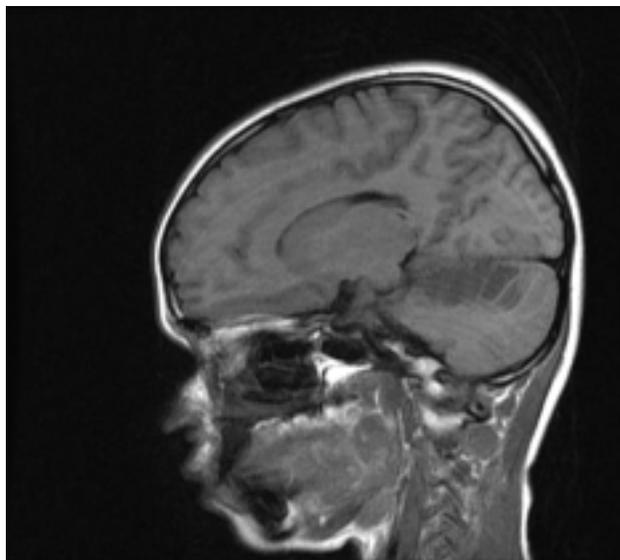
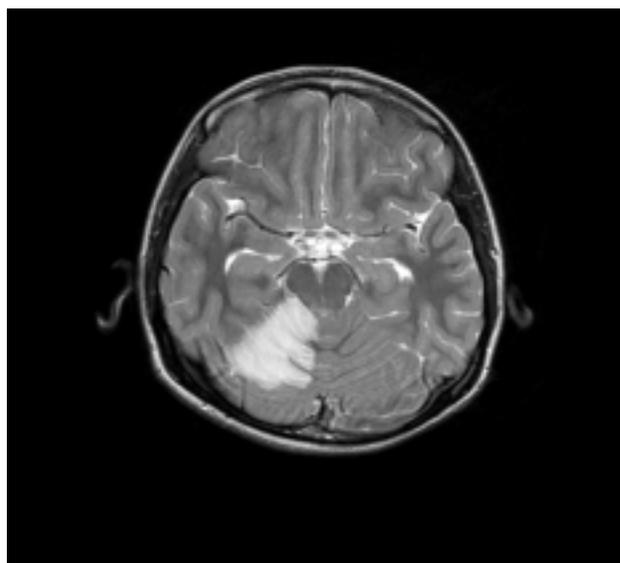


Fig. 3: T2 weighted image (axial)

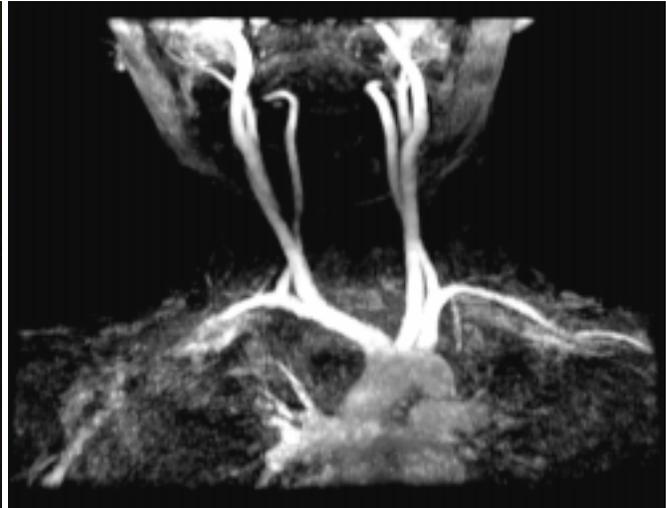
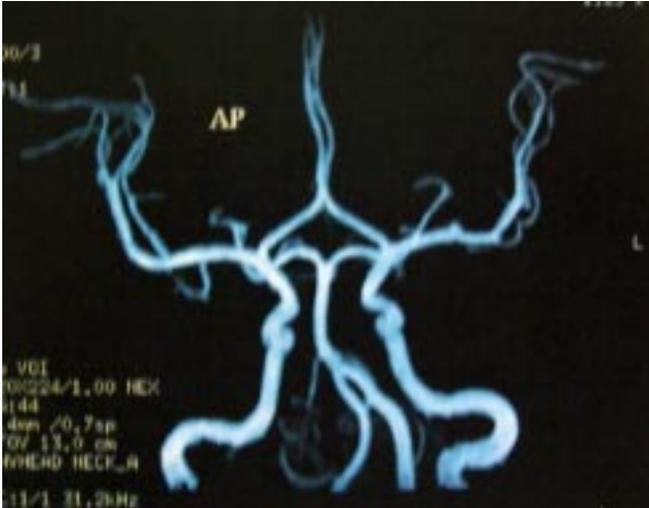


a day and symptomatically (i.v. fluids to correct and maintain hydration, ondansetron and paracetamol) as well. There was no neurological deterioration while in hospital. On follow up examination at 6 months and at one year, there was a marked improvement in cerebellar signs viz. finger nose, knee heel test, rebound phenomenon and dysdiadocokinesis.

A

Fig. 4: M.R. Angiogram

B



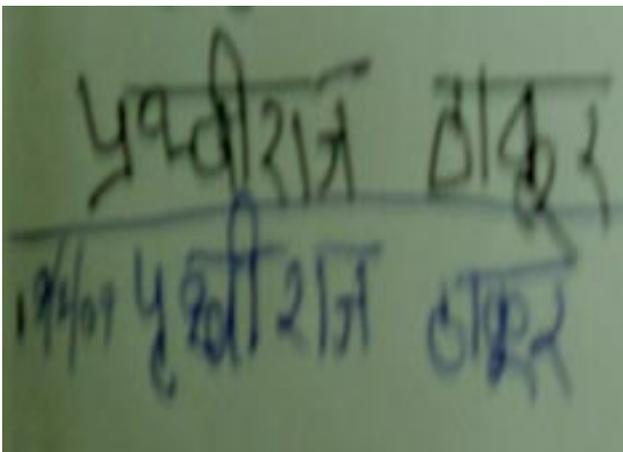
There was no rebound phenomenon or dysdiadochokinesis. The hand writing was more legible. His speech had also improved. Due to financial constraints the thrombophilia profile was not done.

Discussion:

Cerebellar infarction and infarctions of the posterior circulation of brain i.e. vertebrobasilar system are uncommon in pediatric population as compared to infarctions in anterior circulation [1]. Gait ataxia and vomit-

ing are the two cardinal findings in cerebellar infarction. Classical cerebellar signs such as limb incoordination and dysmetria are uncommonly seen [2]. The chief causes of cerebellar infarction in childhood as found in literature are idiopathic, trauma, congenital vascular and cervical spine anomalies, sepsis and dehydration. Migraine is a rare cause and was first reported in 1982 by Harbaugh et al [1, 3, 4]. Migraine is characterized by episodic headaches that may be moderate to severe in intensity, focal in location, have a throbbing quality, and may be associated with nausea, vomiting, light sensitivity and sound sensitivity [5]. Migraine is found to be a significant risk factor for posterior circulation infarction [6]. The diagnosis of migrainous infarction is based on the abrupt onset of a neurologic deficit during a migraine attack associated with evidence of cerebral infarction on neuroimaging [7]. The mechanisms underlying migraine attacks remain fairly unknown, although accumulating data have demonstrated that it is a primary brain disorder. In 1945, Leao and Morrison have suggested for

Fig. 5: Handwriting 7 months later



the first time that Spreading Depression (SD) could be the pathophysiology of migraine.

Spreading depression (SD) consists of a spreading wave of depolarization associated with a reduction of the cortical activity that lasts for minutes. SD is accompanied by an initial hyperperfusion, followed by prolonged and pronounced spreading hypoperfusion. The genetically hyperexcitable brain in migraine probably facilitates paroxysms of SD-like phenomena initiating the cascade of events ultimately leading to the attacks [8].

Basilar artery migraine presents with symptoms related to vasoconstriction of the basilar, vertebral, or posterior cerebral arteries. Stroke is an uncommon complication of childhood migraine but occasionally has been reported to complicate basilar artery migraine [2]. Cerebellar infarction occurs due to severe and prolonged vasoconstriction. The most commonly affected territory is superior cerebellar artery. Cerebellar infarction can result in cytotoxic and vasogenic edema. Being a posterior fossa lesion, rise in intracranial pressures can be fatal due to brain stem compression. Close monitoring is required for early identification of this complication. C.T. scan may not pick up acute infarcts. M.R.I. is a more sensitive modality for early detection of infarct, especially diffusion weighted images.

Majority of cases are managed by medical treatment. Progressive deterioration of consciousness is an indication for surgical intervention. Surgical intervention includes external ventricular drainage and decompressive craniectomy of posterior fossa [9]. One view is that if there is direct brain stem compression, suboccipital craniectomy and posterior

fossa decompression is indicated. If there is acute obstructive hydrocephalus then external ventricular drainage is indicated [1]. However Heros RC is of the view that the operation of choice is a suboccipital decompression carried through the foramen magnum to relieve tonsillar herniation and removal by suction of the grossly infarcted cerebellar tissue. Ventricular drainage should not be used alone without prompt suboccipital decompression because of inadequate relief of the direct compression of the brainstem and the risk of upward herniation with ventriculostomy [10]. Because of the unusual association of stroke and migraine in childhood, other etiologies for infarction have been sought in the case reported here. The child's blood pressure has been normal. There has been no evidence of congenital heart disease and the echocardiogram has not demonstrated cardiac source for emboli. Angiography has documented no structural anomaly of cerebral vessels, no arterial malformation, and no evidence of vasculitis. There has been no evidence of hematologic disorders which might lead to abnormal coagulation. The boy has been followed up over one year and is found to have neurological improvement.

Conclusion:

Migraine is commonly thought to be acutely disabling during attacks with no long-term consequences. Cerebellar infarction in migraine is a rare complication. Early recognition of migraine and awareness of complications can reduce morbidity. Cerebellar infarction in migraine in children is sparsely reported. To our knowledge there are no Indian reports. Ours is probably the first Indian report.

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