Journal of Chemical Health Risks
www.jchr.org
JCHR (2023) 13(6), 3165-3168 | ISSN:2251-6727

Post Traumatic Basal Ganglia Infarction in Children of Eastern India

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(Received: 07 October 2023 Revised: 12 November Accepted: 06 December)

KEYWORDS
Posttraumatic Basal Ganglia infarction, MRI and MCA

ABSTRACT

Introduction: Post traumatic infarction in children is an uncommon event after head injury in children, makes for less than 2% of all pediatric ischemic strokes.

Aims: This case series aims to study the clinical profile of patients with posttraumatic basal ganglia infarction, imaging abnormalities. Associated risk factors and to assess outcome.

Material and Methods: We report 14 cases of posttraumatic basal ganglia infarction. All patients were children below 13 years of age. Majority of them had minor head injury after fall, only two patients presented after road traffic accident (RTA). All patients were investigated according to protocol with CT head; both magnetic resonance angiography and a small number of patients had magnetic resonance imaging (MRI) procedures. Carotid Doppler and 2-D echocardiography was performed to rule out embolic source. Screening for haematological or biochemical abnormalities was done by complete blood count, coagulation profile, ESR, CRP. RA factor, ASO titers etc. All patients were treated conservatively. Outcome was assessed with Glasgow outcome scale. Patients were followed up to 6 months.

Results: Nine of the 14 cases were men, and five were women. The mean age was3 years (range: 9 months to 12 years). 12 of them had minor head injury after fall, only two patients presented after RTA. None of the patients had previous history of seizures or heart disease. Two patients had history of fever preceding fall.

Conclusion: Most of the patients may not have any predisposing conditions but it is mandatory to exclude other causes of ischemia like embolic source in heart, acute traumatic arterial dissections or any thrombophilic states. Most of the patients will have complete recovery attributable to childhood neuronal plasticity.

INTRODUCTION

Post traumatic infarction in children is an uncommon event after head injury in children, makes for less than 2% of all pediatric ischemic strokes [(1)]. It is usually followed by minor head injury. Even though pathogenesis is less clear, it is supposed to be due to stretching and vasospasm of lenticulostriate or perforating arteries. It is usually followed by minor head injury. Although viral infections, hematologic abnormalities with hypercoagulability and vascular disorders can predispose to infarction, though majority

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of children with posttraumatic infarction may not have any predisposing factors.

AIMS

This case series aims to study the clinical profile of patients with posttraumatic basal ganglia infarction, imaging abnormalities, associated risk factors and to assess outcome.

MATERIAL AND METHODS

We report 14 cases of posttraumatic basal ganglia infarction. All patients were children below 13 years of age. Majority of them had minor head injury after fall, only two patients presented after road traffic accident (RTA). All patients were investigated according to protocol [(2)] with CT head; both magnetic resonance angiography (MRA) and a small number of patients had magnetic resonance imaging (MRI) procedures. Carotid Doppler and 2-D echocardiography [(2)] was performed to rule out embolic source. Screening for hematological or biochemical abnormalities [(2)] was done by complete blood count, coagulation profile, ESR, CRP. RA factor, ASO titers etc. All patients were treated conservatively. Outcome was assessed with Glasgow outcome scale. Patients were followed up to 6 months.

RESULTS

Nine of the 14 cases were men, and five were women. The mean age was3 years (range: 9 months to 12 years). 12 of them had minor head injury after fall, only two patients presented after RTA. None of the patients had previous history of seizures or heart disease. Two patients had history of fever preceding fall.

Seven patients had vomiting after injury, 5 had loss of consciousness for less than 5minutes. One patient also had history of seizure. All patients presented with hemiparesis & pyramidal tract signs like brisk jerks, extensor plantars on affected side. All but one had UMN facial nerve palsy on the side of hemiparesis. Average latency for onset of neurological deficit was one hour (range: 10minutes - 4hours). Dysarthria was present in three patients at the time of presentation. None of the patients had evidence of external injuries.

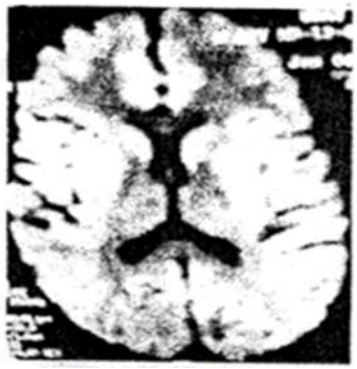
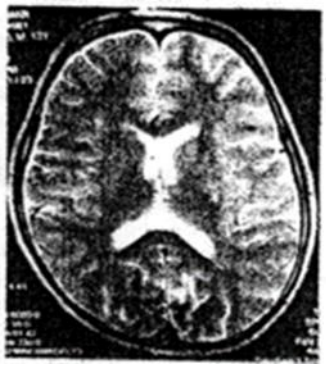


Fig. 1: Left putamen & internal capsule Infarction in 1 year old boy developed after fall from bed. Hemiparesis developed within 10minutes after fall.

Imaging showed infarction in putamen, may or may not involving internal capsule in 11patients, rest 3 patients had infarction in CT scans revealed hypodensity in putamen or caudate suggestive of infarction. MRI brain performed in 4 patients showed hypointense changes in basal ganglia region in TIWI, hyperintense on T2WI with restricted diffusion in DWI, MRA was normal. Hemiparesis developed within 10minutes after fall. Hematological Investigations revealed leukocytosis in 2 patients, elevated CRP levels in 4 patients. Rest all screening hematologic investigations were normal in all patients. Carotid Doppler & 2-D echocardiography was normal in all patients. All except one patient recovered completely within 6 weeks. One patient developed dystonia after recovery of hemiparesis.



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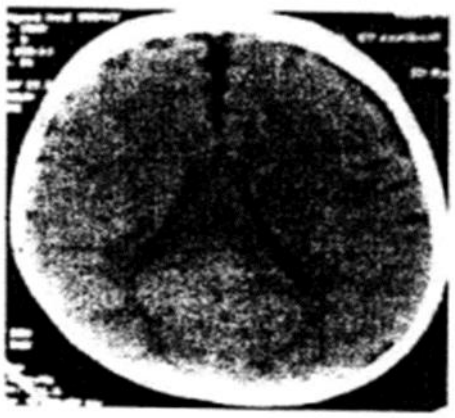

Fig. 2: MRI brain of 12years old boy after history of fall from height, T2WI shows hyperintensity in left putamen & restriction diffusion in diffusion weighted images.

DISCUSSION

Even though minor head injuries due to frequent falls are common in children, post traumatic striatocapsular infarction in children is an uncommon event. It manifests as acute contralateral hemiparesis and facial palsy caused by upper motor neuron type or later with development of secondary dystonia [3-11], Dysarthria may be present in few patients. Pathophogenesis of stroke brought on by a minor head trauma is less clearly understood. The MCA's lenticulostriate branches serve the internal capsule [12] and the putamen, globus pallidus, and caudate nucleus of the basal ganglia. Due to artery disruption [13], the region supplied by these functioning end arteries is susceptible to ischemia. These arteries have an acute angle of origin from the MCA trunk, and they travel in a recurrent path before entering the anterior perforated material [12]. If there is any brain movement, this redundant extracerebral component could get disrupted between the fixed intracerebral portion and the mobile extracerebral portion. After minor trauma, these perforating branches may 1) disrupt leading to acute immediate neurodeficit 2) have intimal trauma and subsequent thrombosis [8] leading to relatively delayed onset if symptoms as seen in few cases of our series. 3) Undergo intermittent vasospasm [14] leading to recurrent episodes of transient but reversible hemiparesis. MCA spasm after trauma may be exaggerated by reduction in pCO2 following crying [1]. Children are more predisposed to posttraumatic striatocapsular infarction due to following anatomical relationships 1.The acute angle forming the medial and lateral lenticulostriate perforators and the major MCA more so in the case of lateral punctators) 2. Shorter lateral perforators, more vulnerable to stretch 3. In youngsters, the temporal lobes are not completely covered by the sphenoid bone [4] since it is still developing. Predisposing greater motion of brain than skull base, further increasing stretch on perforators. Other factors which may predispose to posttraumatic ischemia could be post infectious vasculopathies, vasculitis, hypercoagulable states like thrombophilia, protein C or S deficiency, antithrombin III deficiency. But majority of children with posttraumatic infarction may not have any predisposing factors. Previous infection with Varicella zoster's [15] or herpes can sensitize the cerebral arteries, increasing their vulnerability for artery spasm or thrombosis after minor head trauma. So, previous history of fever is important to rule out this predisposition. Previous history of fever in two patients of our series may have predisposed them for infarction. A very rare genetic disease characterized by diffuse cerebral edema and coma is caused by a mutation in the CACNA1A gene, which codes for a structural protein for the. Because of a hereditary predisposition to intimal disruption after minor trauma or arterial spasm, calcium channels [16] may be predisposed to infarction. Most of the patients recover completely within one week to three months duration. Rapid recovery may be attributed to the resolution vasospasm. Also, childhood neuronal plasticity [16] will contribute to complete recovery inspite full blown. striatocapsular infarction.

CONCLUSIONS

Post traumatic striatocapsular infarction in children is an uncommon event after head injury in children. Few anatomical relationships leading to stretching of lenticulostriate perforators from middle cerebral make children vulnerable to infarction of basal ganglia & internal capsule. Post infectious vasculopathies, hypercoagulable states, rare genetic syndromes may increase susceptibility for infarction. However, further predisposing environmental and genetic factors have to be studied to make pathogenesis clearer. Most of the patients may not have any predisposing conditions but it is mandatory to exclude other causes of ischemia like embolic source in heart, acute traumatic arterial dissections or any thrombophilic states. Most of the

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patients will have complete recovery attributable to childhood neuronal plasticity. However more detailed study with more number of patients and long term follow-up and multicentric approach will be an option for future to get more elaborate and conclusive results.

REFERENCES

1. Shaffer L, Rich PM, Pohl KR, Ganesan V. Can mild head injury cause ischaemic stroke?. Archives of disease in childhood. 2003 Mar 1;88(3):267-9.
2. Kirkham FJ. Stroke in childhood. Arch Dis Child 1999:81:85-9.
3. Maki Y, Akimoto H, Enomoto T. Injuries of basal ganglia following head trauma in children. Pediatric Neurosurgery. 1980 Mar 4;7(3):113-23.
4. Dharker SR, Mittal RS, Bhargava N. Ischemic lesions in basal ganglia in children after minor head injury. Neurosurgery. 1993 Nov 1;33(5):863-5.
5. Muthukumar N. Basal ganglia-internal capsule low density lesions in children with mild head injury. British journal of neurosurgery. 1996 Jan 1;10(4):391-4.
6. Brett EM, Hoare RD. Progressive hemi-dystonia due to focal basal ganglia lesion after mild head trauma. Journal of Neurology, Neurosurgery, and Psychiatry. 1981 May;44(5):460.
7. Demierre B, Rondot P. Dystonia caused by putamino-capsulo-caudate vascular lesions. Journal of Neurology, Neurosurgery & Psychiatry. 1983 May 1;46(5):404-9.
8. Krauss JK, Mohadjer M, Braus DF, Wakhloo AK, Nobbe F, Mundinger F. Dystonia following head trauma: a report of nine patients and review of the literature. Movement disorders: official journal of the Movement Disorder Society. 1992;7(3):263-72.
9. Kappelle LJ, Willemse J, Ramos LM, van Gijn J. Ischaemic stroke in the basal ganglia and internal capsule in childhood. Brain and Development. 1989 Jan 1;11(5):283-92.
10. Okuno T, Takao T, Ito M, Konishi Y, Mikawa H, Nakano Y. Infarction of the internal capsule in children. Journal of Computer Assisted Tomography. 1980 Dec 1;4(6):770-4.
11. Landi A, Marotta N, Mancarella C, Marruzzo D, Salvati M, Delfini R. Basal ganglia stroke due to mild head trauma in pediatric age-clinical and therapeutic management: a case report and 10 year literature review. Italian journal of pediatrics. 2011 Dec;37(1):1-7.
12. Umansky F, Gomes FB, Dujovny M, Diaz FG, Ausman JI, Mirchandani HG, Berman SK. The perforating branches of the middle cerebral artery: a microanatomical study. Journal of neurosurgery. 1985 Feb 1;62(2):261-8.
13. Yonas H, Wolfson Jr SK, Dujovny M, Boehnke M, Cook E. Selective lenticulostriate occlusion in the primate. A highly focal cerebral ischemia model. Stroke. 1981 Sep;12(5):567-72.
14. Symon L. An experimental study of traumatic arterial spasm. J Neuro! Neurosurg Psychiatry 497:30:497-505.
15. Bodensteiner JB, Hille MR, Riggs JE. Clinical features of vasculopathy associated with primary varicella infection. Am J Dis Child. 1992;146:100.
16. Anderson V, Catroppa C, Morse S, Haritou F, Rosenfeld J. Functional plasticity or vulnerability after early brain injury?. Pediatrics. 2005 Dec 1;116(6):1374-82.