

CASE REPORT

Can mild head injury cause ischaemic stroke?

L Shaffer, P M Rich, K R E Pohl, V Ganesan

Arch Dis Child 2003;**88**:267–269

Five cases of ischaemic stroke preceded by minor head trauma in children are described. All patients had striatocapsular infarction. Three had no cerebrovascular abnormality; two had turbulent flow in the proximal middle cerebral artery. None of the patients had evidence of arterial dissection or any other risk factors for stroke. All made an excellent neurological recovery. Possible mechanisms include mechanical disruption to the flow in the perforating branches of the middle cerebral artery, intimal trauma and subsequent thrombosis, or arterial spasm induced by trauma. The specific susceptibility in affected children remains unexplained; both genetic and environmental factors (for example, previous chickenpox) may be implicated.

Ischaemic stroke is an important clinical problem in childhood affecting around 5/100 000 children each year. Although detailed investigation usually reveals risk factors for stroke in the majority of affected children,¹ there remains a small group in whom none are identified. We have observed a distinctive clinical and radiological pattern of stroke in a number of children, in whom striatocapsular infarction was preceded by mild head injury. We will describe some illustrative cases and discuss possible underlying mechanisms.

All the patients described here were investigated with brain magnetic resonance imaging (MRI), magnetic resonance angiography (MRA) of the circle of Willis and carotid and vertebral arteries in the neck (down to the level of the mid-common carotid artery, around C7), cardiac echocardiography, and extensive screening for haematological or biochemical abnormalities according to a recommended protocol.² All results were negative unless specifically mentioned and all patients were neurologically and developmentally normal prior to the stroke. None had any external signs of trauma elsewhere in the body or signs suggestive of non-accidental injury (such as retinal haemorrhages).

CASE 1

A 1 year old girl was seen to fall 12 inches from a sofa onto a thinly carpeted floor. She cried immediately but did not lose consciousness. Within a few minutes she was noted not to be using her left hand. Her parents thought this was the result of a soft tissue injury. However, by the following day she was also not using her left leg. Clinical examination confirmed a left hemiparesis. MRI of the brain three days later showed an infarct in the right lentiform nucleus and corona radiata. MRA showed normal intracranial and cervical vessels. She made a gradual improvement and three months later was neurologically normal.

CASE 2

A 1 year old boy was walking across the floor when he lost his balance and fell over, hitting his head on the floor. There was no loss of consciousness. Within a few minutes he began

drooling out of the left side of his mouth and was not moving his left arm and leg. A computed tomography (CT) scan of his brain later that day showed an infarct in the right putamen. Brain MRI confirmed an infarct of the right lentiform nucleus and corona radiata. MRA of the intracranial and cervical vessels was normal. He made a complete clinical recovery within three months of the initial event.

CASE 3

This patient was a 14 month old girl who was born at 36 weeks gestation with a birth weight of 2 kg. She was nasogastrically fed for the first four days of life but subsequently made normal developmental progress. At the time of her presentation her elder brother had chickenpox, which she subsequently developed eight days later. She was witnessed pulling herself to stand by the sofa, when she lost her balance and fell over, hitting her head. She immediately cried and her mother tried to comfort her. Within 24 hours of the head injury she was not able to move her left arm or leg and was unable to sit or crawl. Brain MRI showed an infarct of the right basal ganglia and corona radiata. MRA of the intracranial and cervical vessels was normal. Her motor impairment began to resolve within a week. She has minimal pyramidal tract signs in her left side.

CASE 4

A 4 year old girl fell off the sofa, hitting her head on a carpeted floor. There was no loss of consciousness. However, within a few minutes she developed intermittent episodes of left sided weakness involving her leg, arm, and face, lasting from 30 seconds to 30 minutes. During the episodes she was noted to have brisk reflexes and an upgoing plantar response on the left side. Consciousness was not impaired during the episodes and she was well in between. Brain MRI showed an infarct involving the posterior limb of the right internal capsule. MRA showed a focal area of turbulent flow (suggestive of narrowing) in the distal right middle cerebral artery (MCA) only. She was found to be homozygous for the thermolabile methylene tetrahydrofolate reductase gene mutation but had a normal random level of total plasma homocysteine. She had had shingles two months prior to these events. She had no residual neurological signs and has had no further events.

CASE 5

A 6 year old girl was seen to fall down a small flight of five steps by her parents. She bumped her head at the bottom but appeared uninjured, with no impairment of consciousness. Around six hours later she was noted to have developed weakness of her right arm and leg. By the next morning she had developed an expressive dysphasia and right facial weakness. Brain MRI showed an infarct affecting the basal ganglia and external capsule on the left. Axial T1 weighted MRI with

Abbreviations: CT, computed tomography; MCA, middle cerebral artery; MRA, magnetic resonance angiography; MRI, magnetic resonance imaging

fat saturation through the neck showed no evidence of internal carotid artery dissection. Intracranial MRA showed attenuation of signal in the proximal left MCA with reduced filling of the distal branches. She has a mild residual hemiparesis and ongoing expressive language difficulties.

DISCUSSION

We have described a series of young children with acute hemiparesis caused by striatocapsular infarction following mild head trauma. While minor bumps to the head are a very common occurrence in childhood, cerebral infarction is an exceedingly rare sequela. It should also be noted that this sequence is an unusual mechanism of childhood stroke and should only be made after systematic exclusion of other causes. The cases described here were identified during the course of a detailed analysis of around two hundred cases of ischaemic stroke in childhood. The possibility of more extensive trauma than that which was reported, for example as a result of non-accidental injury, appears unlikely, given the nature of the brain lesion and the lack of other supportive clinical and radiological evidence. It is possible that the fall leading to the head injury could have been the first presenting symptom of a neurological event. It is difficult to definitively rule this out; however, the head injury was witnessed in all the patients and their parents clearly observed the emergence of the neurological signs following a brief interval. Although it is important not to rely purely on witness accounts when considering the possibility of non-accidental injury, as previously stated this diagnosis appeared unlikely in the clinical contexts described.

Striatocapsular infarction after mild head trauma in children, either followed by acute stroke or with a delayed presentation of secondary dystonia (caused by basal ganglia infarction), has previously been described by several groups.^{3–10} Given that most of the patients described here made an excellent neurological recovery, it is possible that in the past some would have been classified as having “complicated migraine” or “acute infantile hemiplegia” as the presence of cerebral infarction would not have been apparent without brain imaging. There may, therefore, have been under recognition of this association in the past. These alternative diagnoses should not now be made without detailed clinical and radiological evaluation.

Previous reported cases have relied on CT scans for diagnosis, and major arterial pathologies, in particular arterial dissection, were not excluded. There has been no previous information about the intracranial or cervical vessels in these patients. Three of five patients in the present group had normal intracranial vessels on MRA and none had evidence of internal carotid artery dissection. Arterial dissection may account for up to 20% of cases of stroke in children and adolescents¹¹ and should be actively excluded, particularly where there is a history of trauma. Although none of the patients described here had conventional cerebral angiography, the MRI and MRA studies carried out were adequate to exclude dissection.

Features common to all these cases are the young age of the patients and the mild nature of the trauma. However, given the frequency of mild head injury in young children, the pathophysiology of stroke resulting from mild head trauma deserves further consideration. The basal ganglia (caudate nucleus, putamen, and globus pallidus) and internal capsule are supplied by the lenticulostriate branches of the MCA. These are functional end arteries and therefore the territory they supply is vulnerable to ischaemia as a result of disruption to arterial supply.¹² These vessels originate from the MCA trunk at an acute angle and follow a recurrent course before penetrating the anterior perforated substance.¹³ There is, therefore, a redundant extracerebral segment. Any motion of the brain may lead to disruption of the vessel between the

mobile extracerebral portion and the fixed intracerebral portion.³ This may mechanically disrupt distal blood supply or may lead to intimal trauma and subsequent thrombosis. Thrombosis and subsequent vascular occlusion would explain the relatively delayed onset of symptoms in case 3 as well as in some other cases described in the literature.^{6–8}

However, this mechanism may not explain the rapid time course observed in some patients. The clinical course of case 4 with recurrent episodes of transient but reversible hemiparesis, would be consistent with intermittent arterial spasm. Previous reports of cases with reversible imaging changes⁵ as well as experimental evidence that trauma to the middle cerebral artery can lead to arterial spasm¹⁴ would also support this hypothesis. It is possible that, in a susceptible child, a mild blow to the head may lead to MCA spasm, and that crying, with consequent reduction in pCO₂, may exacerbate arterial constriction. This could lead to thrombosis and produce the imaging appearances of arterial narrowing observed in cases 4 and 5.

The reasons why this syndrome appears to be confined to young children are not immediately clear. The anatomical relationship between the lenticulostriate arteries and the trunk of the middle cerebral artery changes from fetal life, through childhood and adulthood.¹⁵ The angle between the main MCA and both the medial and lateral lenticulostriate perforators is more acute in younger individuals. It is possible that this alters the relationship between the fixed and mobile portions of the perforating vessels and may be a relevant factor in the vulnerability of young children to this form of basal ganglia damage.

If head injury is sufficient to cause transtentorial herniation, compression of the thalamoperforating arteries by the descending brain may lead anterolateral thalamic infarction.¹⁶ However, this mechanism is unlikely in the patients described here as the head injury was minor in all cases and their clinical courses are not compatible with the presence of significant intracranial mass effect. Moreover, none of the patients had lesions in the appropriate vascular territory.

The anatomical features discussed may be contributory but do not explain the specific susceptibility in affected children. Other adverse neurological consequences have been described following similarly mild head injury in children. Examples include attacks of migraine, encephalopathy, seizures, and focal neurological deficits.^{17–18} An extreme example is a malignant syndrome of delayed cerebral oedema and coma. It has recently been shown that some individuals with this syndrome have mutations in the CACNA1A calcium channel subunit gene, suggesting that vulnerability to adverse neurological sequelae following mild head injury may be genetically determined in some individuals.¹⁹ It is therefore possible that the patients described here have an underlying genetic susceptibility to arterial spasm or intimal disruption following mild trauma.

Two of these patients (patients 3 and 4) had had recent varicella infection. The relationship between *Varicella zoster* and cerebral arteriopathy affecting proximal large intracranial arteries in childhood is now clearly established.²⁰ Although case 4 could represent an instance of post-varicella vasculopathy, the time course in this child suggests that the acute symptoms were caused by some additional factor related to the cranial trauma. It is possible that, in addition to large vessel vasculopathy, previous infection with *Varicella zoster* could sensitise the cerebral arteries in some children, increasing their susceptibility to developing arterial thrombosis or spasm following mild head trauma.

The reproducibility of the pattern of infarction, temporal relation with the trauma, and exclusion of other causes of stroke, in particular arterial dissection and cardiac sources of embolism, leads us to conclude that this association is not merely coincidental. Clearly the mechanisms discussed above

remain speculative. However, improved recognition of cases may shed further light on environmental and genetic factors determining individual susceptibility and on the pathophysiological mechanisms involved.

Kieslich *et al* recently described eight cases of cerebral infarction following minor head trauma in children.²¹ The posterior circulation was involved in two cases, and six patients had residual disabilities. The spectrum of abnormalities and sequelae may therefore be wider than seen in our cases.

ACKNOWLEDGEMENTS

This work was undertaken in part by Great Ormond Street Hospital for Children NHS Trust which received a proportion of its funding from the NHS Executive. The views expressed in this publication are those of the authors and are not necessarily those of the NHS Executive.

.....

Authors' affiliations

L Shaffer, V Ganesan, Neurosciences Unit, Institute of Child Health, University College, London WC1N 1EH, UK
P M Rich, Department of Neuroradiology, Great Ormond Street Hospital for Children NHS Trust, London WC1N 3JH, UK
K R E Pohl, Department of Paediatric Neurology, Guys Hospital, London SE1 9RT, UK

Correspondence to: Dr V Ganesan, Lecturer in Paediatric Neurology, The Wolfson Centre, Mecklenburgh Square, London WC1N 2AP, UK; v.ganesan@ich.ucl.ac.uk

Accepted 25 March 2002

REFERENCES

- 1 **Lanthier S**, Carmant L, David M, *et al*. Stroke in children. The coexistence of multiple risk factors predicts poor outcome. *Neurology* 2000;**54**:371–8.
- 2 **Kirkham FJ**. Stroke in childhood. *Arch Dis Child* 1999;**81**:85–9.
- 3 **Maki I**, Akimoto H, Enomoto T. Injuries of the basal ganglia following head trauma in children. *Childs Brain* 1980;**7**:113–23.
- 4 **Dharker SR**, Mittal RS, Bhargava N. Ischaemic lesions in basal ganglia in child after minor head injury. *Neurosurgery* 1993;**33**:863–5.
- 5 **Muthukumar N**. Basal ganglia-internal capsule low density lesions in children with mild head injury. *Br J Neurosurg* 1996;**10**:391–3.
- 6 **Brett EM**, Hoare RD, Sheehy MP, *et al*. Progressive hemidystonia due to focal basal ganglia lesion after mild head trauma. *J Neurol Neurosurg Psychiatry* 1981;**44**:460.
- 7 **Demierre B**, Rondot P. Dystonia caused by putamino-capsulo-caudate vascular lesions. *J Neurol Neurosurg Psychiatry* 1983;**46**:404–9.
- 8 **Krauss JK**, Mohadjer M, Braus DF, *et al*. Dystonia following head trauma: a report of nine patients and review of the literature. *Mov Disord* 1992;**7**:263–72.
- 9 **Jaap Kapelle L**, Willemse J, Ramos LMP, *et al*. Ischaemic stroke in the basal ganglia and internal capsule in childhood. *Brain Dev* 1989;**11**:283–92.
- 10 **Okuno T**, Takano T, Ito M. Infarction of the internal capsule in children. *J Comput Tomogr* 1980;**4**:770–4.
- 11 **Williams LS**, Garg BP, Cohen M, *et al*. Subtypes of ischemic stroke in children and young adults. *Neurology* 1997;**49**:1541–5.
- 12 **Yonas H**, Wolfson SK, Dujovny M, *et al*. Selective lenticulostriate occlusion in the primate. A highly focal cerebral ischemia model. *Stroke* 1981;**12**:567–72.
- 13 **Umansky F**, Gomes FB, Dujovny M, *et al*. The perforating branches of the middle cerebral artery. A microanatomical study. *J Neurosurg* 1985;**62**:261–8.
- 14 **Symon L**. An experimental study of traumatic arterial spasm. *J Neurol Neurosurg Psychiatry* 1977;**30**:497–505.
- 15 **Kodama N**, Suzuki J. Cerebrovascular moyamoya disease. Third report: The study of aging of the perforating branches and the possibility of collateral pathways. *Neurologia Medico-Chirurgica* 1974;**14**:55–67.
- 16 **Endo M**, Ichikawa F, Miyasaka Y, *et al*. Capsular and thalamic infarction caused by tentorial herniation subsequent to head trauma. *Neuroradiology* 1991;**33**:296–9.
- 17 **Haas DC**, Sovner RD. Migraine attacks triggered by mild trauma and their relation to certain post-traumatic disorders of childhood. *J Neurol Neurosurg Psychiatry* 1969;**32**:548–54.
- 18 **Snoek JW**, Minderhond JM, Wilmink JT. Delayed deterioration following mild head injury in children. *Brain* 1984;**107**:15–36.
- 19 **Kors EE**, Terwindt GM, Vermeulen FL, *et al*. Delayed cerebral edema and fatal coma after minor head trauma: the role of the CACNA1A calcium channel subunit gene and relationship with familial hemiplegic migraine. *Ann Neurol* 2001;**49**:753–60.
- 20 **Chabrier S**, Rodesch G, Lasjaunias P, *et al*. Transient cerebral arteriopathy: a disorder recognised by serial angiograms in children with stroke. *J Child Neurol* 1998;**13**:27–32.
- 21 **Kieslich M**, Fiedler A, Heller C, *et al*. Minor head injury as a cause and co-factor in the aetiology of stroke in childhood: a report of eight cases. *J Neurol Neurosurg Psychiatry* 2002;**73**:13–16.