Can mild head injury cause ischaemic stroke?

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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, 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. 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 had 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...
fat saturation through the neck showed no evidence of inti-
mal 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 hemi-
paresis caused by striatocapsular infarction following mild
head trauma. While minor bumps to the head are a very com-
on 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 exten-
sive 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 clin-
cal 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. 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 recogni-
tion of this association in the past. These alternative diagnoses
should not be made without detailed clinical and radio-
logical evaluation.

Previous reported cases have relied on CT scans for diagno-
sis, 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
adolescents and should be actively excluded, particularly
where there is a history of trauma. Although none of the
patients described here had conventional cerebral angio-
graphy, 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. These vessels originate from the MCA
trunk at an acute angle and follow a recurrent course before
penetrating the anterior perforated substance. There is, there-
fore, 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.
This may mechanically disrupt distal blood supply or may lead to intimal trauma and subsequent intracranial
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.4, 9

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 hemipare-
sis, would be consistent with intermittent arterial spasm.
Previous reports of cases with reversible imaging changes13 as well as experimental evidence that trauma to the middle cerebral
artery can lead to arterial spasm16 would also support this
hypothesis. In a possible mechanism, it is possible that a 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.2 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 fac-
tor in the vulnerability of young children to this form of basal
ganglia damage.

If head injury is sufficient to cause transtentorial hernia-
tion, compression of the thalamoperforating arteries by the
descending brain may lead anterolateral thalamic infarction.11
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
local neurological deficits.13, 14 An extreme example is a malig-
nant 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 neuro-
logical sequelae following mild head injury may be genetically
determined in some individuals.15 It is therefore possible that
the patients described here have an underlying genetic suscepti-
bility 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.16 Although
case 4 could represent an instance of post-varicella vasculopa-
thy, 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 ves-
sel 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, anterior cerebral 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. 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.

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