Post-Traumatic Middle Cerebral Artery Occlusion

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The development of hemiparesis and other focal cerebral signs in a patient with recent head injury often suggests a subdural, epidural or intracerebral hematoma, or cerebral contusion. However, occasionally these signs may be related to the occlusion of a large cerebral artery. Although obstruction of the carotid artery after head trauma or non-penetrating injuries of the neck has been described adequately in the literature, only a few instances of post-traumatic middle cerebral artery occlusion have been recorded. In this paper we are reporting 3 cases of middle cerebral artery occlusion that followed closed head injuries, and have reviewed 9 similar cases from the literature.

Case Reports

Case 1. A 54-year-old woman was hospitalized in 1963 following an automobile accident. She complained of headaches, chest pain and nausea.

On examination there were multiple contusions, abrasions and lacerations. Physical signs of a left pneumothorax, confirmed by chest roentgenogram, were present. On the second hospital day the patient became stuporous and developed a left hemiparesis and left hemisensory syndrome. The left plantar response was extensor. Her level of consciousness gradually deteriorated and by the 5th hospital day she was comatose.

Carotid arteriography (Fig. 1) indicated complete occlusion of the horizontal portion of the right middle cerebral artery, distal to the origin of the lenticulostriate branches. The right anterior cerebral artery was displaced to the left. Some collateral circulation entered the right middle cerebral artery via the right anterior cerebral artery.

Despite steroids, hypothermia, urea and other supportive measures the patient remained comatose. By the 9th hospital day the right pupil dilated and decerebrate phenomena appeared.

Operation. A right temporal craniectomy was performed. The brain was soft, swollen and edematous. There was no intracranial hematoma.

Following surgery the patient remained comatose and died on the 20th hospital day.

Postmortem Examination. At autopsy there was infarction and edema of the right cerebral hemisphere. In addition there was uncal herniation and brain stem hemorrhage. The right middle cerebral artery was completely patent. Multiple sections of the middle cerebral artery were made in an attempt to demonstrate a recanalized thrombosis, dissecting aneurysm or other abnormality, but no such lesion was found.

The transient nature of the middle cerebral artery occlusion has been discussed elsewhere.37

Case 2. A 41-year-old housewife was involved in an automobile accident, and struck her head and neck on the dashboard. She was unconscious for 10 minutes. Later she complained of intermittent headaches and pain in the front of her neck. One week after the accident she was brought to the hospital because of the sudden development of difficulty in speaking and moving the right arm and leg. There was no alteration in consciousness.

Examination. General physical examination was normal except for healing facial lacerations and neck abrasions. She was alert but severely dysphasic. Although able to follow some simple commands, she could not name most objects. The cranial nerves were intact except for a right central facial weakness. A right hemiparesis, most pronounced in the arm, was present. There was moderate right hyperreflexia. The right plantar response was extensor. Sensory examination was normal. Routine laboratory studies were normal. Skull and chest roentgenograms were negative. Lumbar puncture gave normal results.

On the first hospital day left carotid arteriography was performed (Fig. 2). There was marked irregularity and narrowing of the internal carotid artery in its cervical and supraclinoid portions. The left middle cerebral artery was occluded just distal to the origin of the lenticulostriate branches. In later films the left middle cerebral artery and its branches were visualized in retrograde fashion from the distal branches of the anterior cerebral artery.

Two weeks after admission the patient left the hospital much improved. Minimal dysphasia and
FIG. 1. Case 1. Right carotid arteriogram demonstrating occlusion (arrow) of the horizontal portion of the middle cerebral artery. A. Anteroposterior view. B. Lateral view.
right hemiparesis were present at the time of discharge. Six months later the patient was readmitted to the hospital. The neurological examination was negative. This time the left carotid angiogram (Fig. 3) was normal; there was complete visualization of the left middle cerebral artery.

Case 3. A 46-year-old man was involved in an automobile accident. His head struck the steering wheel and he sustained a left frontal scalp laceration. There was no loss of consciousness or apparent cervical trauma. After the injury he complained of persistent headaches. Five days later he had several transient episodes during which he experienced difficulty in speaking and numbness of the right hand. Over the next few weeks he felt quite well. Four weeks after the accident he entered the hospital because of the recurrence of difficulty with speech.

Examination. On admission the general physical examination was negative except for a bruit over the left carotid artery. He was alert but severely dysphasic. The cranial nerves were intact except for right central facial weakness. There was right hemiparesis, most marked in the arm. The deep tendon reflexes were normal. Sensation was intact.

Routine laboratory studies were normal. Skull and chest roentgenograms were negative. The electroencephalogram was normal. Brain scan demonstrated an area of increased radioactive uptake in the left fronto-parietal area. Lumbar puncture was normal except for a spinal fluid protein of 61 mg%. Two weeks after admission, left carotid arteriography showed slight irregularity and narrowing of the internal carotid artery at its origin, and partial occlusion of the left middle cerebral artery (Fig. 4). Later films in the series demonstrated some retrograde filling of the middle cerebral artery via the left anterior cerebral artery. Follow-up angiography 1 month later showed no change. The defect in the cervical portion of the internal carotid artery appeared to be more pronounced.

Physical examination 6 months later showed...
only minimal improvement of the patient’s neurological deficit.

Discussion

Nine cases of middle cerebral artery occlusion attributed to non-penetrating head injuries have been adequately described in the literature. In 1942 DeVeer and Browder reported the case of a 42-year-old man who developed stupor and hemiplegia 12 hours after falling 20 feet. At autopsy an occlusion of the middle cerebral artery was found. Similarly, middle cerebral artery thrombus was demonstrated at autopsy in Verbiest and Calliauw’s patient, who became comatose 24 hours after she was thrown out of an automobile. Jacobsen and Skinhoj stated that of 36 patients with angiographic evidence of middle cerebral artery occlusion 4 had a history of head trauma 1 to 6 days before the manifestations of occlusion. Frantzen et al. reported 2 cases occurring during childhood and Duman and Stephens added 3 instances of post-traumatic middle cerebral artery occlusion. Bushart reported a patient who developed psychomotor attacks 1 year after a head injury; 20 years after the onset of seizures, a right carotid arteriogram revealed middle cerebral artery occlusion.

Our 3 cases and those described in the literature are summarized in Table 1. We are excluding Bushart’s case because of the long interval between the head injury and the carotid angiogram.

Clinical Features

The ages of the patients varied from 6 to 67 years. Eight of the 12 cases were under 45, significantly younger than is usually seen with spontaneous middle cerebral artery occlusions. Many of the patients had suffered relatively minor head injuries, often with no loss of consciousness. The interval from the time of injury to the onset of occlu-
sive symptoms varied from several hours to 2 weeks but occurred within 24 hours in 6 patients, and in all but 1 within 8 days.

Signs and symptoms were similar to that of spontaneous middle cerebral artery occlusion. As indicated in Table 1, some of the patients were comatose. In addition, decerebrate phenomena were observed several times. Thus the clinical picture of post-traumatic middle cerebral artery occlusion may simulate that of cerebral contusion or post-traumatic intracranial hematomas.

Radiographic Findings

All x-ray films of the skull were normal. Ventriculography carried out in 2 cases demonstrated a ventricular shift to the opposite side in one and slight dilatation of the ventricles in the other.

Angiography was performed in 10 cases. The interval from the onset of focal neurological signs to angiography varied from 12 hours to 6 weeks. An occlusion of the main stem or horizontal portion of the middle cerebral artery was present in 8 of the 10 angiographically verified cases. Occlusion of the more distal middle cerebral artery ("branch occlusion") occurred in 2 instances.

Displacement of the anterior cerebral artery to the opposite side was seen in 1 instance. Collateral circulation with retrograde filling of the middle cerebral artery via the anterior cerebral artery was noted in 3 cases. Three patients had associated defects in the cervical portion of the carotid artery; one also had abnormalities in the supraclinoid portion of this vessel.

Follow-up angiograms, obtained in six
### TABLE 1

**Summary of main clinical features of cases of post-traumatic middle cerebral artery occlusion**

<table>
<thead>
<tr>
<th>Author</th>
<th>Age (yrs.)</th>
<th>Sex</th>
<th>Initial Injury</th>
<th>Interval Between Injury and Onset of Final Neurological Signs</th>
<th>Signs and Symptoms</th>
<th>Arteriographic Findings</th>
<th>Follow-Up Arteriogram or Autopsy</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>DeVeer and Browder (1942)</td>
<td>42</td>
<td>Male</td>
<td>Fell 60 ft. to street below. Struck head. Loss of consciousness 60 minutes.</td>
<td>12 hours</td>
<td>Stupor, left hemi-plegia.</td>
<td>Autopsy 3 days following trauma. Occlusion of middle cerebral artery. Possible dissecting aneurysm. Ischemic infarction cerebral hemisphere.</td>
<td>Died</td>
<td></td>
</tr>
<tr>
<td>Jacobsen and Skinhoj (1959)</td>
<td>11</td>
<td>Male</td>
<td>Fell on stairs.</td>
<td>1 day</td>
<td>Somnolent. Left homonymous hemianopsia, left hemiparesis.</td>
<td>Occlusion main stem of middle cerebral artery.</td>
<td>Follow-up arteriogram 15 months after injury. demonstrated “main stem of middle cerebral artery replaced a distance of 5 mm. by several small vessels” and complete occlusion of the anterior cerebral artery.</td>
<td>Mental changes, left hemianopsia and spastic hemiparesis (18 months after injury).</td>
</tr>
<tr>
<td>Jacobsen and Skinhoj (1959)</td>
<td>14</td>
<td>Male</td>
<td>Sword thrust on cheek.</td>
<td>12 hours</td>
<td>Decreased level of consciousness, left hemiparesis.</td>
<td>Occlusion main stem of middle cerebral artery.</td>
<td>Follow-up arteriogram 8 days after injury normal.</td>
<td>Recovered.</td>
</tr>
<tr>
<td>Frantzen et al. (1961) Case 1</td>
<td>14</td>
<td>Male</td>
<td>Hit head on wooden bridge after fall into water. Loss of consciousness.</td>
<td>2 days</td>
<td>Right hemiplegia.</td>
<td>&quot;Complete occlusion of middle cerebral artery and, in addition, small defects of internal carotid artery in neck.&quot;</td>
<td>Follow-up arteriogram 9 months after injury normal.</td>
<td>Mental changes. Slight spastic hemiparesis 8 months after injury.</td>
</tr>
<tr>
<td>Frantzen et al. Case 8</td>
<td>6</td>
<td>Male</td>
<td>Struck head on play pen. No definite loss of consciousness.</td>
<td>Several hours</td>
<td>Vomiting, drowsiness, left hemiparesis, papilledema.</td>
<td>Branch occlusion of middle cerebral artery.</td>
<td>Follow-up arteriogram 1 month after injury showed &quot;more abundant, but not normal, outflow into the Sylvian vessels.&quot;</td>
<td>Moderate hemiparesis 8 months later.</td>
</tr>
<tr>
<td>Duman and Stephens Case 2</td>
<td>18</td>
<td>Male</td>
<td>Struck on right eye during fight. Brief loss of consciousness.</td>
<td>2 weeks</td>
<td>Headaches, drowsiness, left homonymous hemianopsia, left hemiplegia.</td>
<td>Occlusion main stem of middle cerebral artery.</td>
<td>Improved but residual spastic hemiparesis.</td>
<td>Residual hemiparesis.</td>
</tr>
<tr>
<td>Duman and Stephens Case 3</td>
<td>60</td>
<td>Male</td>
<td>Automobile accident. Dazed and confused.</td>
<td>3 days</td>
<td>Sudden left hemi-plegia.</td>
<td>Occlusion main stem of middle cerebral artery.</td>
<td>Normal middle cerebral artery at postmortem 11 days after injury. Cerebral swelling and infarction. Brain stem hemorrhages.</td>
<td>Died</td>
</tr>
<tr>
<td>Hollis et al. (1959) Case 1</td>
<td>54</td>
<td>Female</td>
<td>Automobile accident. Multiple contusions. Pneumothorax.</td>
<td>44 hours</td>
<td>Right hemiplegia, coma, decerebration.</td>
<td>Occlusion main stem of middle cerebral artery.</td>
<td>Follow-up arteriogram 6 months after injury normal.</td>
<td>Recovered.</td>
</tr>
<tr>
<td>Hollis et al. Case 2</td>
<td>41</td>
<td>Female</td>
<td>Thrown forward in car, striking face. Loss of consciousness 10 minutes.</td>
<td>7 days</td>
<td>Headaches, dysphasia, right hemiparesis, right Babinski.</td>
<td>Occlusion main stem of middle cerebral artery. Narrowing of the cervical and supraclinoid portions of the internal carotid artery.</td>
<td>Arteriogram 6 weeks after injury unchanged except more pronounced defect internal carotid artery.</td>
<td>No major improvement. Residual aphasia and spastic hemiparesis.</td>
</tr>
</tbody>
</table>
patients, were completely normal in 3 cases. In the 4th case there was a "more abundant, but not normal outflow into the Sylvian vessels."

The repeat arteriogram in another patient showed some filling of the Sylvian vessels and an occlusion of the anterior cerebral artery. In the last patient there was no significant change demonstrated by follow-up angiography.

It was interesting to note the subsequent clearing of the cervical carotid defect in one patient and the normal angiogram seen in a patient who previously had combined cervical and supraclinoid carotid defects. The filling defect in the cervical carotid artery in a 3rd case appeared to be more pronounced at the time of repeat arteriography.

Autopsy Findings

Postmortem examination was made on 4 patients in the series. The patient of DeVeer and Browder was found to have an obstruction of the middle cerebral artery by a thrombus at the origin of the vessel. Separation of the intima and the internal elastic lamina from the media was present, suggesting that the occlusion had actually been due to a dissecting aneurysm. There was ischemic infarction with swelling and softening of the involved cerebral hemisphere.

Verbiest and Calliauw found the middle cerebral artery completely blocked by a thrombus in their patient. There was massive edema and softening of the cerebral hemisphere with herniation of the hippocampal gyrus.

Duman and Stephens case had an obstruction at the origin of the middle cerebral artery by a dissecting aneurysm, which extended 1 cm. along the course of the vessel. There was ischemic infarction with swelling of the cerebral hemisphere.

In our Case 1 the angiographically occluded middle cerebral artery was patent at autopsy and there was no evidence of thrombus, dissecting aneurysm, or injury to the arterial wall. In addition to cerebral infarction and swelling, there were also brain stem hemorrhages secondary to increased intracranial pressure.

Pathogenesis

The pathogenesis of middle cerebral artery occlusion following non-penetrating head trauma is not clearly understood, especially since relatively few cases have been examined at autopsy. Several possible explanations exist:

1. Emboli from the cervical portion of the internal carotid artery. Post-traumatic thrombus formation in the cervical portion of the internal carotid artery may result from direct contusion of the arterial wall or shearing or stretching of the vessel by sudden hyperextension of the neck. Embolization of fragments of the mural thrombus to the middle cerebral artery readily occurs. If the carotid artery is atherosclerotic, an injury may disrupt the intima and mobilize clots in the vicinity of the atheromatous mass with subsequent embolization. Intravascular clots in the distribution of the middle cerebral artery have been found at autopsy in patients who had post-traumatic contusions of the cervical portion of the internal carotid artery.

Franzen et al. reported angiographic demonstration of post-traumatic middle cerebral artery occlusion associated with a filling defect in the proximal internal carotid artery suggestive of a mural thrombus. In our Case 2 we also noted narrowing of the cervical and supraclinoid portions of the internal carotid artery with subsequent clearing.

Verbiest and Calliauw described a patient in whom arteriography, performed the day after neck injury, demonstrated a carotid occlusion in the neck. Two days later angiography revealed the obstruction to be at the upper end of the siphon. There was no filling of the anterior or middle cerebral arteries. The follow-up arteriogram 19 months later was normal. Similar arteriographic findings, recently described by Bladin, and others, were thought to be due to migration of emboli.

2. Spasm. Several authors have emphasized the role of spasm of the carotid artery in the neck as a possible cause of post-traumatic angiographic occlusion of intracranial arteries. Houck et al. explored the cervical portion of the internal carotid artery in cases of occlusion following trauma, and concluded that the obstruction was probably caused by spasm with subsequent thrombosis, since no evidence of actual disruption of the arterial wall was found at surgery.
amples of local and generalized intracranial arterial spasm in patients who underwent angiography following head injuries. Spasm was demonstrated even in the absence of associated intracranial bleeding.

Huber noted areas of arterial narrowing on the cerebral angiograms of patients with head injuries and attributed this finding to spasm. Sedzimer sought to explain spasm of intracranial vessels by movement of the brain during head trauma. He emphasized that the distal segment of the carotid siphon, which is fixed to the brain by the anterior and middle cerebral arteries, moves with the brain, while the more proximal carotid artery remains fixed at the cavernous sinus. Sedzimer believed that overstretching of the artery might cause spasm, and thus give the illusion of thrombosis.

3. Dissecting Aneurysm. The impact of a blow on the head may cause damage to the intracranial arteries with separation of the intima from the media and the outer layers of the vessel wall. The formation of an intramural hematoma results in subsequent narrowing and possible total occlusion of the arterial lumen. Scott et al. preferred the term “dissecting intramural hematoma” to “dissecting aneurysm,” since the dissection occurs within the arterial wall without an increase in the external diameter of the vessel. Cerebral arteries have relatively thin walls and only one elastic membrane. Thus, they are more fragile and probably more susceptible to separation between the intima and media than vessels of similar caliber elsewhere.

A dissecting aneurysm of the middle cerebral artery was demonstrated at autopsy in Duman and Stephens’ Case 10 and in the case reported by DeVeer and Browder. Ritchie and Dratz and Woodhall have also reported autopsy findings of post-traumatic dissecting aneurysms involving the intracranial portion of the carotid artery as well as the middle cerebral artery. Hassen described dissecting aneurysms of intracerebral arteries following electrocution. The mechanism was considered to be the result of sudden violent jarring of the brain and not from the high temperature or electric current per se.

4. Thrombus formation. Mechanical injury may cause thrombosis of vessels by direct trauma to the vessel wall, circulatory stasis or effects on the constitution of the circulating blood. The injury to the intima and a decrease in the volume flow of blood through the injured site are both prime factors in the initiation of the thrombus.

Direct contusion of the middle cerebral artery is probably not common, although it is conceivable that the internal carotid and proximal middle cerebral arteries may be bruised against adjacent bony structures in the base of the skull. Hypotension and slowing of the cerebral circulation, both of which might contribute to circulatory stasis and thrombus formation, have been observed following head trauma. Changes in the constitution of the circulating blood, such as hemoconcentration secondary to shock or intravascular agglutination of blood cells, usually follow severe crushing injuries and cannot be implicated in most cases of mild head trauma.

Prognosis

Four of the 19 reported patients died. Five patients were left with significant neurological deficits. Repeat angiograms, obtained in 3 of these 5 patients, were abnormal in 2 but normal in the 3rd case. One patient, who seemed to have recovered, suffered a recurrence of hemiparesis 15 months later, at which time angiography demonstrated an anterior cerebral artery occlusion. Only 2 patients recovered completely and both had normal follow-up angiograms.

Summary

We have reported 5 cases of post-traumatic middle cerebral artery occlusion, and have discussed the clinical, radiological, pathogenetic and prognostic features of this condition.

References


Post-Traumatic Middle Cerebral Artery Occlusion


