Traumatic intracranial aneurysms

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Eleven cases of traumatic intracranial aneurysms, six saccular and five arteriovenous, are presented with an operative mortality of 22.2%, which compares favorably with the few reports in the literature. These lesions are usually associated with serious head injuries. The diagnosis is often delayed or overlooked as the surgeon's attention is distracted by the presence of an accompanying intracranial hematoma. With increasing replacement of angiography by computerized tomography in aneurysm diagnosis, these aneurysms are even more likely to escape detection. They should be suspected in any patient who deteriorates within 2 weeks of the trauma. Conservative management carries a mortality rate of about 50%. Because of their superficial location, they are amenable to successful surgical extirpation. Improved mortality depends on early recognition and surgical obliteration.

Key Words: traumatic aneurysm, intracranial aneurysm, arteriovenous fistula, false saccular aneurysm

Trauma to intracranial arteries is not rare when one considers the incidence of intracranial hematomas that complicate head injuries. Excluding carotid-cavernous fistulas, however, traumatic saccular aneurysms and arteriovenous fistulas are distinctly uncommon. There are few more than 100 cases recorded in the world literature.1-10,12,18,20,37,39-47,50-62,64-69,71-73

That intracranial aneurysms may occur after trauma to the intracranial arteries has been shown experimentally and confirmed at pathology.14,79 In 1891, Bollinger11 suggested that one mechanism of "delayed apoplexy" following head injury was focal brain softening and delayed hemorrhage from injured blood vessels. With the advent of angiography, it became apparent that some of these cases were due to delayed rupture of a traumatic aneurysm.13

The first traumatic intracranial aneurysm was reported in 1829.62 A young man, after trauma to the left temple, developed an aneurysm of the middle meningeal artery, confirmed at autopsy. Surgical therapy of these lesions was pioneered by Birley and Trotter,19 who, in 1928, reported the case of a posttraumatic aneurysm of the right internal carotid artery (ICA), successfully treated by internal and external carotid artery ligation.

Summary of Cases

In the period from December, 1970, to January, 1978, we have seen 11 cases of posttraumatic intracranial aneurysms; six were saccular aneurysms and five arteriovenous (Table 1). The patients ranged in age from 17 to 65 years (mean 44.7 years), and included nine males and two females.

The etiology of the arterial injuries is listed in Table 2. Scalp damage or a skull fracture confirmed the

TABLE 1
Location of traumatic intracranial aneurysms
in 11 cases

<table>
<thead>
<tr>
<th>Location</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Saccular aneurysms</td>
<td></td>
</tr>
<tr>
<td>Internal carotid artery (supraclinoid)</td>
<td>3</td>
</tr>
<tr>
<td>Middle meningeal artery</td>
<td>2</td>
</tr>
<tr>
<td>Middle cerebral artery</td>
<td>1</td>
</tr>
<tr>
<td>Arteriovenous fistulas</td>
<td></td>
</tr>
<tr>
<td>Middle meningeal artery</td>
<td>3</td>
</tr>
<tr>
<td>Middle cerebral artery</td>
<td>1</td>
</tr>
<tr>
<td>Posterior inferior cerebellar artery</td>
<td>1</td>
</tr>
</tbody>
</table>

TABLE 2
Etiology of injury in 11 cases

<table>
<thead>
<tr>
<th>Etiology</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>Assault</td>
<td>5</td>
</tr>
<tr>
<td>Unknown</td>
<td>2</td>
</tr>
<tr>
<td>Motor vehicle accident</td>
<td>2</td>
</tr>
<tr>
<td>Fall</td>
<td>1</td>
</tr>
<tr>
<td>Iatrogenic (postoperative)</td>
<td>1</td>
</tr>
</tbody>
</table>
D. Parkinson and M. West

FIG. 1. Case 2. Angiograms showing a traumatic arteriovenous malformation (AVM), well anterior to the margin of the craniotomy flap, performed for removal of a recurrent subdural hematoma (arrows). Left: Anteroposterior view showing the superficial location of the AVM, a third recurrence of the subdural hematoma, and the 8-mm shift of the anterior cerebral artery from left to right. Right: Lateral view.

head trauma in the two cases where the nature of injury was unknown.

Table 3 summarizes the clinical signs in this series. Excluding the iatrogenic case, the level of consciousness was depressed in all cases immediately after trauma. The typical history documented post-traumatic coma, with a subsequent lucid interval ranging from hours to days (average 14 days). Seizures were the first evidence of delayed deterioration in two cases. Four patients demonstrated an oculomotor palsy, which in two was presumed to be a direct result of compression by an aneurysm on the ICA, and in the other two, accompanied ipsilateral extracerebral hematomas producing tentorial herniation. The two most severely injured patients were decerebrate from the time of injury.

Reflecting the severity of the initial head injury, there was a high incidence of associated intracranial pathology in these cases (Table 4). Seven patients had demonstrable cerebral contusions and/or laceration. Six patients had an associated intracranial hematoma; there were three subdural hematomas, two extradural, and one intracerebral.

Initial investigations included a plain skull radiograph in 10 patients. Seven of the radiographs were abnormal, and demonstrated linear skull fracture in four cases and compound skull fractures in three, each complicated by a cerebrospinal fluid (CSF) fistula. There were no significantly depressed skull fractures.

Angiography was performed in all patients. The diagnosis of traumatic intracranial aneurysm was suspected pre-angiographically in only one case (the postoperative ICA aneurysm). Indications for angiography were 1) evolving coma, usually with focal signs following a head injury; 2) a delayed deterioration in the neurological status after a previously satisfactory course; and 3) failure to improve neurologically after head injury once other causes had been excluded. Angiography satisfactorily demonstrated the aneurysm and any accompanying hematoma.

**Table 3**

Summary of clinical signs in 11 cases

<table>
<thead>
<tr>
<th>Signs</th>
<th>No. of Cases</th>
</tr>
</thead>
<tbody>
<tr>
<td>depressed level of consciousness</td>
<td>10</td>
</tr>
<tr>
<td>hemiparesis</td>
<td>6</td>
</tr>
<tr>
<td>dysphasia</td>
<td>3</td>
</tr>
<tr>
<td>seizures</td>
<td>2</td>
</tr>
<tr>
<td>oculomotor palsy</td>
<td>4</td>
</tr>
<tr>
<td>abducens nerve palsy</td>
<td>1</td>
</tr>
<tr>
<td>decerebration</td>
<td>2</td>
</tr>
</tbody>
</table>

**Table 4**

Associated intracranial pathology

<table>
<thead>
<tr>
<th>Pathology</th>
<th>Cases</th>
<th>No.</th>
<th>Percent</th>
</tr>
</thead>
<tbody>
<tr>
<td>cerebral contusion and/or laceration</td>
<td>7</td>
<td>63.6</td>
<td></td>
</tr>
<tr>
<td>subdural hematoma</td>
<td>3</td>
<td>27.3</td>
<td></td>
</tr>
<tr>
<td>extradural hematoma</td>
<td>2</td>
<td>18.2</td>
<td></td>
</tr>
<tr>
<td>intracerebral hematoma</td>
<td>1</td>
<td>9.1</td>
<td></td>
</tr>
</tbody>
</table>
Traumatic intracranial aneurysms

Of the 11 patients, two died within hours of their injury, as a result of intrinsic brain damage. The nine patients who survived the initial injury were operated on an average of 17 days after trauma. The iatrogenic ICA aneurysm was treated by clipping the aneurysm intracranially. In the remaining cases, the lesion was coagulated and excised. Arteriovenous fistulas in the dura (three cases) were treated by clipping or embolizing the feeding vessel, followed by coagulating and/or excising the involved vessels. In five cases, associated extracerebral hematomas were evacuated. The traumatic aneurysm was recognized and obliterated at the time of hematoma evacuation in all but one case. Five patients underwent intraoperative or postoperative angiography, which demonstrated satisfactory obliteration of the lesions in all cases.

There were two postoperative deaths, (constituting 22.2% mortality). One patient, with a posttraumatic arteriovenous fistula of the posterior inferior cerebellar artery (PICA), died on the eighth day following posterior fossa craniectomy. Autopsy disclosed a mucus plug occluding the trachea and both main-stem bronchi. The central nervous system was intact. The patient with the iatrogenic ICA aneurysm died 4 months after clipping of the artery.

Case Reports

Case 1
This 58-year-old man was admitted on December 6, 1970, after an assault and 5 to 10 minutes of unconsciousness. He later became drowsy and dysphasic. A skull x-ray film demonstrated a right parietal skull fracture, crossing the posterior middle meningeal groove. Left carotid angiography demonstrated a chronic subdural hematoma, which was evacuated. Four days postoperatively, two generalized seizures occurred. A right carotid angiogram then demonstrated an arteriovenous fistula beneath the fracture site between the middle meningeal artery and the superior sagittal sinus. The fistula was obliterated without incident, and the postoperative course was uneventful. Unfortunately, the angiograms have been discarded.

Case 2
This 39-year-old man was found unconscious on the street with a partial right third-nerve palsy. He underwent two evacuations of acute subdural hematomas, and then the bone flap had to be removed because of progressive brain swelling. Angiography was repeated because of persistent deterioration and bulging of the craniotomy wound, and disclosed a recurrent subdural hematoma and an arteriovenous aneurysm of the ascending frontal branch of the middle cerebral artery well anterior to the limit of the previous craniotomy (Fig. 1). On the 36th postoperative day, through a fresh right frontal flap, the fistula was coagulated and a postoperative angiogram confirmed its obliteration. The subsequent course was uneventful.

Case 3
This 65-year-old alcoholic man was admitted on December 4, 1974, in a stuporous state. Skull film demonstrated a vertical linear occipital fracture. Bilateral brachial angiograms demonstrated an "arteriovenous aneurysm" of the left PICA, lying directly beneath the fracture line (Fig. 2 left). Operation was delayed due to the patient's moribund condition, and absence of an accompanying intracranial hematoma. On December 20, a sudden deterioration in the patient's level of consciousness prompted repeat cerebral angiography, which showed no change in the aneurysm. Because of a temperature elevation, a lumbar puncture was performed revealing bloody, xanthochromic fluid. On January 3, 1975, a posterior fossa craniectomy was made. An arteriovenous fistula, surrounded by a small area of old hemorrhage, was...
observed on the posterolateral aspect of the left cerebellar hemisphere. The fistula was coagulated and excised. Postoperative angiography confirmed its obliteration (Fig. 2 right). The patient died on the eighth postoperative day due to aspiration of a mucus plug. At autopsy, the distal PICA was thrombosed with no evidence of residual fistula.

Case 4

This 27-year-old woman was rendered unconscious by a blow to the head the evening before admission. Within 15 minutes of the injury, she was lucid and was put to bed by her family who found her unconscious the next morning. On admission she was decerebrate, with a fixed, dilated right pupil and irregular respirations. An immediate right carotid angiogram demonstrated an aneurysm of the right supraclinoid carotid artery, accompanied by spasm of the ICA. She died within 24 hours. Autopsy disclosed massive basilar subarachnoid hemorrhage secondary to a 1.2 × 0.5 mm tear in the right supraclinoid internal carotid artery, 1 mm proximal to the posterior communicating artery. The angiograms have been destroyed.

Case 5

This 45-year-old man was admitted on March 22, 1977, in a stuporous state, with extensive ecchymoses of the scalp and face, a left Battle’s sign, left hemotympanum, dysphasia, and a right hemiparesis. Skull film revealed an extensive left parieto-occipital fracture. A left carotid angiogram demonstrated an arteriovenous fistula between the left middle meningeal artery and the sagittal sinus (Fig. 3). On April 6, a left parietal craniotomy was performed. The arteriovenous fistula and surrounding dura were coagulated and excised. Intraoperative angiography confirmed the obliteration of the fistula. The postoperative course was uneventful and the patient was discharged with a very mild weakness of the right leg.

Case 6

This 56-year-old man was admitted on August 7, 1977, unconscious after an assault. He had evidence of multiple trauma to the left face and head, the chest, and abdomen, and developed a total left third-nerve palsy and a right hemiparesis within hours of admission. Skull films demonstrated a comminuted left temporal parietal fracture, crossing the middle meningeal groove. A laparotomy was performed for a ruptured spleen; a burr-hole was made in the left temporal region and 30 cc of subdural clot drained. The patient’s neurological status did not improve. A left carotid angiogram was performed on August 16, revealing a traumatic aneurysm of the left middle meningeal artery and a large subtemporal extradural hematoma (Fig. 4). A left temporal craniectomy was made and the extradural hematoma was evacuated. The foramen spinosum was plugged and the middle meningeal artery was obliterated. The postoperative course was uneventful. Six months later, the left third-nerve palsy and right hemiparesis had completely resolved.
Traumatic intracranial aneurysms

Case 7

This 48-year-old alcoholic man was admitted on August 18, 1977, after being struck by a moving vehicle and rendered immediately unconscious. There was fresh blood and CSF draining from the right ear, and a left hemiparesis. Skull films demonstrated a linear vertical right parietal skull fracture, extending into the base of the skull. Four days after the injury, the patient was able to speak. He continued to improve until August 31, when he became drowsy and complained of headache. A right carotid angiogram was performed (Fig. 5), demonstrating a traumatic aneurysm of the distal middle cerebral artery, directly underlying the skull fracture. A right parietal craniotomy was performed. When the bone flap was removed, it was evident that the aneurysm along with contused brain had herniated through a laceration in the dura. The aneurysm ruptured during dissection and was coagulated. Postoperative angiography confirmed the obliteration of the aneurysm (Fig. 6). The postoperative course was uneventful. When he was seen again 6 months later, the patient's hemiparesis had completely resolved.

Case 8

This 44-year-old alcoholic man was admitted with alcohol withdrawal syndrome on May 1, 1978. The neurosurgical service was consulted on May 3, after the patient had become drowsy and dysphasic. A left carotid angiogram demonstrated evidence of elevation of the left middle cerebral artery with an avascular area in the left temporal region. There was an arteriovenous fistula in the anterior left middle fossa, fed by the middle meningeal artery. Following the angiogram, a left temporal craniectomy was made; a small subdural hematoma (30 cc) and a larger intratemporal hematoma (60 cc) were evacuated. The patient recovered well from this procedure. On May 23, the left external carotid artery was exposed and cannulated. With the aid of intraoperative angiog-
Case 8

D. Parkinson and M. West

FIG. 7. Case 8. Left: Intraoperative angiogram of the left external carotid artery during the second craniotomy (May 23), demonstrates arteriovenous fistula along the middle meningeal artery (arrow). Right: Postoperative angiogram showing obliteration of the fistula (arrow) accomplished by embolization and excision.

raphy (Fig. 7 left), the arteriovenous fistula was embolized with small shavings of Gelfoam. When the fistula was exposed, it was noted to be surrounded by old hemorrhage. The fistula and surrounding dura were coagulated. Postoperative angiography confirmed complete obliteration of the fistula (Fig. 7 right). The postoperative course was uneventful. Follow-up studies 3 months later revealed no demonstrable neurological deficit.

ruptured and bled profusely. The site of rupture was trapped between two Heifetz clips placed across the carotid artery. Postoperatively, the patient remained in a persistent vegetative state until the time of his death on September 10. Autopsy disclosed bilateral basal ganglion infarctions.

Case 9

This 43-year-old man was admitted on May 5, 1978. A CT scan demonstrated a pituitary tumor with suprasellar extension. Bilateral carotid angiograms demonstrated displacement of the proximal supraclinoid carotid arteries laterally, with displacement of the proximal A1 segments of the anterior cerebral arteries superiorly. On May 12, a right frontal craniotomy was performed. During dissection of the tumor capsule, a small medial branch of the ICA was avulsed. Hemorrhage was controlled by packing with Gelfoam and Surgicel. On the ninth postoperative day, the patient became drowsy, with a left sixth-nerve palsy and mild right hemiparesis. A left carotid angiogram demonstrated a multilobed false aneurysm of the left ICA (Fig. 8). The ICA and anterior cerebral artery were displaced to the same extent as preoperatively. The craniotomy was reopened and hematoma evacuated from the operative site. As the hematoma was sucked out, the false aneurysm ruptured and bled profusely. The site of rupture was trapped between two Heifetz clips placed across the carotid artery. Postoperatively, the patient remained in a persistent vegetative state until the time of his death on September 10. Autopsy disclosed bilateral basal ganglion infarctions.

Traumatic intracranial aneurysms

Case 10

This 11-year-old boy was admitted on July 30, 1978, 2½ hours after a motor vehicle accident. He was in a decerebrate condition with a fixed dilated right pupil, extensive facial fractures, and bloody rhinorrhea. Right carotid angiography revealed extensive fracturing of the base of the skull and a supraclinoid carotid aneurysm (Fig. 9). The patient died within 24 hours of injury. Autopsy disclosed an area of complete disruption of the carotid artery wall, 1.0 × 0.5 mm in size, correlating with the angiographic site of the false aneurysm.

Case 11

This 57-year-old alcoholic woman was found unconscious with bright red blood in the right ear canal and a left hemiparesis. X-ray films revealed a right linear temporoparietal skull fracture, with a shift of the calcified right choroid plexus from right to left. Immediate right carotid angiography demonstrated a large temporoparietal extradural hematoma, associated with a false aneurysm arising from the posterior branch of the right middle meningeal artery. (Fig. 10). At craniotomy, 200 cc of clotted extradural hematoma was removed. The two false aneurysms on the posterior branch of the meningeal artery were coagulated. The postoperative course was uneventful, with complete resolution of the left hemiparesis within 10 days.

Discussion

The rarity of these lesions has already been alluded to. Other than carotid-cavernous fistulas, no case of posttraumatic intracranial saccular or arteriovenous aneurysm was recorded in the Cooperative Study. Benoit and Wortzman recorded four cases in a series of 850 intracranial aneurysms. Ferry and Kempe recorded two cases in a series of 2187 cases of penetrating wounds of the brain.
Traumatic aneurysms are mainly of two types; either true or false. Other types include mixed or dissecting aneurysms.

Histological study of a "true" traumatic aneurysm demonstrates that the normal structures of the arterial wall, namely, intima, elastica, and media, have been disrupted, leaving only an intact layer of adventitia. A "false" traumatic aneurysm results from laceration of the full thickness of the arterial wall, which is occluded by a hematoma. Subsequent fibrous organization and hemodynamic excavation of a hematoma result in aneurysm formation. The aneurysm wall therefore contains none of the normal arterial wall structures. Mixed aneurysms result from the posttraumatic rupture of a true aneurysm producing a secondary false aneurysm. Dissecting aneurysms result from the formation of a false lumen between the intima and internal elastica.

Traumatic aneurysms have been reported following both blunt and penetrating head trauma. Blunt trauma includes trauma to the pericallosal artery by the edge of the falx or to the middle cerebral artery by the sphenoid ridge. Fifty to 98% of cases have associated skull fractures. Angiograms in these cases have, on occasion, illustrated trapping of a traumatic aneurysm or arteriovenous fistula. Agents causing penetrating injuries include a diverse category of missiles, ranging from bullets to umbrella tips. Fifty to 98% of cases have associated skull fractures. Angiograms in these cases have, on occasion, illustrated trapping of a traumatic aneurysm or arteriovenous fistula. Iatrogenic trauma to intracranial arteries during intracranial surgery is also a cause that is well recognized.

Reflecting the severity of the initial head injury, traumatic aneurysms are frequently accompanied by intracranial pathology such as vascular spasm, cerebral contusion and laceration, and intracranial hematomas. As a rule, the associated pathology is the most important factor in determining the prognosis of a particular case.

Most traumatic aneurysms are located on the supratentorial circulation, at sites other than branching points. Rarely, they occur on the infratentorial circulation or are multiple. Aneurysms and arteriovenous fistulas of the middle meningeal artery have only been mentioned in scattered case reports. Other than the syndrome of delayed deterioration following head injury, there are few clinical findings that point to the diagnosis of a ruptured traumatic aneurysm (excepting carotid-cavernous fistulas). Consequently, common carotid angiography is the only reliable method of establishing the diagnosis and guiding further surgical management. Serial angiography most often demonstrates enlargement of the lesion, but occasionally may demonstrate a decrease in size, or spontaneous disappearance. Features that aid in differentiating the traumatic aneurysm from its congenital counterpart are 1) delayed filling and emptying of the sac; 2) a peripheral location, at a site other than a branching point; 3) irregular contour of the sac; and 4) the absence of a neck.

The increased use of the CT scan in the evaluation of the head-injured patient may produce an artificial reduction in the incidence of traumatic aneurysms. The CT scan will demonstrate an intracranial hematoma, but may miss an associated aneurysm.

Early diagnosis is most important. Patients who are diagnosed following rupture of the aneurysm have a mortality almost three times as high as those who are diagnosed before rupture. The surgical mortality rate recorded in the literature averages 24%. The diagnosis should be considered in every case of delayed deterioration following head injury, as in untreated cases the mortality rate approaches 50%.

Most authors agree that surgical treatment of traumatic aneurysms is indicated because of their very poor natural history. The superficial location of the majority of these lesions reduces the operative risk compared to that of the congenital aneurysms. Possibly in the future, some of these lesions will be amenable to excision with direct repair of the cerebral artery by graft or end-to-end anastomosis.

Summary

Eleven cases of traumatic aneurysm have been reported and the literature reviewed. Because of the superficial location of these lesions, the operative mortality should be close to zero, and the overall mortality is that of the associated brain damage. Unfortunately, they are rarely recognized until their presence is heralded by delayed deterioration, at which time the salvage rate is markedly decreased. Earlier recognition can only be accomplished by more frequent use of angiography following head injuries, and/or more sophisticated scanning.

References

Traumatic intracranial aneurysms


68. Wolman L: Cerebral dissecting aneurysms. Brain 82:276-291, 1959


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