Traumatic intracranial aneurysms

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Traumatic intracranial aneurysms are rare, occurring in fewer than 1% of patients with cerebral aneurysms. They can occur following blunt or penetrating head trauma and are more common in the pediatric population. Traumatic aneurysms can be categorized histologically as true, false, or mixed, with false aneurysms being the most common. These aneurysms can present in a variety of ways, but are typically associated with an acute episode of delayed intracranial hemorrhage with an average time from initial trauma to aneurysm hemorrhage of approximately 21 days. The mortality rate for patients harboring these aneurysms may be as high as 50%. Prompt diagnosis based on arteriography and aggressive surgical management are associated with better outcome than conservative treatment. The authors describe a classification scheme for traumatic aneurysms based on their anatomical location and conclude that 1) post-traumatic aneurysm must be considered in patients with acute neurological deterioration following closed head injury; 2) they can occur following mild closed head injury; 3) they occur more commonly in children than in adults; and 4) surgical clipping and/or endovascular occlusion is the definitive treatment.

KEY WORDS • aneurysm • blunt trauma • intracranial hemorrhage • subarachnoid hemorrhage • traumatic injury

Intracranial aneurysms that develop following closed head injuries present the clinician with both diagnostic challenges and surgical difficulties. Traumatic intracranial aneurysms are rare, comprising 1% or less of all cerebral aneurysms. 10,27,60,63 They can occur after even mild or seemingly trivial head trauma, and are associated with significant morbidity and a mortality rate as high as 50%.14,19,27,44,61 Although found in patients of all ages, intracranial aneurysms are more common in the pediatric population12,21,32,38,42,59 and may occur as the result of either blunt or penetrating trauma.1,12,27,28,30 Although they occur infrequently, these lesions are well described in the literature.1,5,6,8,12,15,17,18,28,49,59

REVIEW OF TRAUMATIC INTRACRANIAL ANEURYSMS

Causes of Traumatic Intracranial Aneurysms

Traumatic intracranial aneurysms may result from various causes. They have been reported in association with both blunt and penetrating trauma, with the former being more commonly described.12,30,63 Of the penetrating injuries, stab wounds appear to have the highest probability of producing traumatic aneurysms;27,28 in one series involving patients with 109 stab wounds to the head, in 11 (14.9%) of the 74 patients who underwent angiography developed posttraumatic aneurysms.28 In contrast, in a series of 223 patients who suffered high-velocity missile injuries and underwent angiography, there were only eight (3.6%) traumatic aneurysms.1 Some authors have described iatrogenic traumatic aneurysms that developed after a variety of procedures, including endoscopic ventriculostomy, intranasal procedures, intracranial surgery, and repeated subdural taps.15,34

Histological Types

Histologically, traumatic aneurysms can be categorized as true, false, or mixed. True aneurysms involve disruption of the intima and variable involvement of the internal elastic layer and media, which leads to localized weakening of the vessel wall and aneurysm formation with the adventitia of the native vessel intact.27,29,30 This phenomenon is presumably secondary to flow dynamics against the weakened vessel wall. False aneurysms are considered to be the most common histological type.1,29,33 These lesions result from disruption of all three layers of the vessel wall with formation of a contained hematoma outside the vessel. A false lumen then develops, creating an aneurysmal dilation.5,12,27 These are presumably the histological type associated with penetrating injuries.5,25,62 The third histological type, the mixed aneurysm, is initially a true aneurysm that subsequently undergoes a contained rupture forming hematoma and false lumen.1,6,12,29,63 Some authors have used the term “mixed aneurysm” to describe saccular aneurysms that occur in association with dissection of...
the parent vessel (Fig. 1). The relative incidence of these histological types is not known, as most case reports contain little or no histological data. The histological type is not particularly relevant in terms of clinical management because intervention is required regardless of the type or mechanism.  

**CLASSIFICATION OF TRAUMATIC INTRACRANIAL ANEURYSMS**

Traumatic aneurysms can be classified into 1) those that involve the vessels proximal to the circle of Willis, and 2) those that occur distal to the Circle of Willis (Table 1).

This classification is based on both the anatomy and mechanism of traumatic aneurysm formation. The majority of aneurysms occur in the supraclinoid segment of the carotid artery and along the anterior cerebral artery and its branches, particularly the pericallosal and callosomarginal arteries (Figs. 2 and 3).

**Mechanism of Injury**

Several mechanisms have been proposed in the formation of traumatic aneurysms, all of which involve either direct injury to the vessel or stretching of the vessel by adjacent forces. The mechanism of injury is closely related to the anatomical location of the involved artery. Infraclinoid carotid and basilar artery aneurysms are commonly associated with basilar skull fractures, which is not surprising given the intimacy of these vessels with the skull base.

In the supraclinoid segment, the carotid artery transitions from a relatively fixed structure in the skull base and cavernous sinus to a relatively mobile structure as it ascends in the cisternal spaces. It is believed that either movement of the supraclinoid segment against the anterior clinoid process or stretching of the carotid artery at this transition zone leads to the formation of an aneurysm. Distal subcortical aneurysms occur predominantly along the anterior cerebral artery and its branches. The proximity of these vessels along much of their length to the falx cerebri has led some authors to the hypothesis that traumatic movement of the brain and vessels against the relatively fixed falx cerebri can lead to aneurysm formation. Likewise, posterior cerebral artery aneurysms are thought to be the result of trauma of the vessel against the tentorium. Distal cortical aneurysms occur in association with linear or depressed skull fractures and dural lacerations, commonly involving the middle cerebral artery or ACA. It is believed that momentary or prolonged herniation of the cortical vessel up into the fracture defect leads to direct injury to the vessel wall.

**Clinical Presentation**

Traumatic aneurysms are more common in children; in one review the author estimate that 30% of all traumatic aneurysms occur in patients younger 20 years of age. In addition, there appears to be a consistent male predominance, with reported male/female ratios ranging from just over 1:1 to as high as 12:1. Most authors have concluded that this discrepancy reflects a higher likelihood that behavior leads to blunt trauma among males in this age group.

Traumatic aneurysms have varied clinical presentations (Table 2). The most common symptoms include an acutely decreased level of consciousness, seizure, or focal neurological deficit. Computerized tomography scanning usually...
demonstrates acute intracranial hemorrhage, which may be subarachnoid, intraparenchymal, intraventricular, or subdural. The average time from initial trauma to aneurysmal hemorrhage is approximately 21 days and is associated with a mortality rate as high as 50%. Patients with infraclinoid carotid artery aneurysms can present with cranial nerve deficits, diabetes insipidus, recurrent or massive epistaxis, unilateral blindness, or symptoms of a cavernous-carotid fistula. Patients with supraclinoid carotid artery lesions can present with headache, memory disturbance, and progressive visual loss prior to rupture; such symptoms have been reported to occur for as long as 7 years prior to diagnosis.

At least three cases have presented with hydrocephalus, two involving pericallosal aneurysms and the third with a distal cortical lesion. Distal cortical aneurysms have unique properties that may make them more likely to be diagnosed prior to the occurrence of hemorrhage. These aneurysms can lead to the development of a growing skull fracture that becomes physically palpable months to years after the injury. Buckingham and colleagues found 11 reported cases of distal cortical aneurysms associated with blunt trauma; seven of these patients (63.6%) presented without hemorrhage and were diagnosed primarily with either on routine radiographic follow up or by evaluation of growing skull fractures. However, only 20.5% (of 44) of blunt traumatic aneurysms were diagnosed prior to hemorrhage in more proximal locations.

Clinicians in the early part of the century did not have the benefit of neuroimaging modalities such as CT and MR imaging. The definitive diagnosis of a traumatic aneurysm could only be made in the angiography suite, in the operating room, or at autopsy.
ing, the use of emergency CT scanning has allowed clinicians to diagnose delayed intracranial hemorrhage due to traumatic aneurysmal rupture more rapidly and more frequently than previously possible. Table 3 represents an overview of the more recent literature regarding traumatic aneurysms following blunt trauma since 1970. Earlier definitive diagnosis in this era seems to be associated with a trend toward more aggressive surgical treatment, and better outcomes are reported than in earlier cases.

### Table 2

#### CLINICAL PRESENTATION OF TRAUMATIC INTRACRANIAL ANEURYSMS

- alteration of consciousness
- headache
- seizure
- focal motor/sensory deficit
- cranial nerve deficit
- unilateral blindness
- proptosis
- chemosis
- retroorbital pain
- epistaxis
- diabetes insipidus

- palpable growing skull fracture

### Table 3

#### SUMMARY OF CASE REPORTS OF TRAUMATIC INTRACRANIAL ANEURYSMS AFTER BLUNT TRAUMA SINCE 1970

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Age, Sex</th>
<th>Mechanism of Injury</th>
<th>Presentation</th>
<th>Skull Fracture</th>
<th>Time to Rupture</th>
<th>Location</th>
<th>Management</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Rumbaugh, et al., 1970</td>
<td>18 yrs, F</td>
<td>MVA</td>
<td>delayed SDH</td>
<td>parietal</td>
<td>7 days</td>
<td>distal MCA</td>
<td>clipped</td>
<td>good</td>
</tr>
<tr>
<td>Pathak, 1972</td>
<td>16 yrs, M</td>
<td>MVA</td>
<td>epistaxis</td>
<td>basilar</td>
<td>5 mos</td>
<td>cavernous carotid</td>
<td>observed</td>
<td>died</td>
</tr>
<tr>
<td>Benoît &amp; Wortzman, 1973</td>
<td>9 yrs, M</td>
<td>CHI</td>
<td>visual loss</td>
<td>no</td>
<td>7 yrs</td>
<td>suprACL</td>
<td>?</td>
<td>?</td>
</tr>
<tr>
<td>Thompson, et al., 1973</td>
<td>4 mos, M</td>
<td>CHI</td>
<td>hydrocephalus</td>
<td>no</td>
<td>2 mos</td>
<td>pericallosal</td>
<td>?</td>
<td>?</td>
</tr>
<tr>
<td>Amacher &amp; Drake, 1979</td>
<td>10 yrs, M</td>
<td>fall</td>
<td>ICH</td>
<td>no</td>
<td>4 wks</td>
<td>pericallosal</td>
<td>clipped</td>
<td>died</td>
</tr>
<tr>
<td>Dharker, et al., 1975</td>
<td>17 yrs, M</td>
<td>MVA</td>
<td>MS change, visual loss, hemiparesis</td>
<td>basilar</td>
<td>5 yrs</td>
<td>suprACL</td>
<td>observed</td>
<td>died</td>
</tr>
<tr>
<td>Fleischer, et al., 1975</td>
<td>4 yrs, F</td>
<td>MVA</td>
<td>IVH</td>
<td>yes</td>
<td>4 wks</td>
<td>distal ACA</td>
<td>clipped</td>
<td>good</td>
</tr>
<tr>
<td>Almeida, et al., 1977</td>
<td>6 mos, M</td>
<td>blunt</td>
<td>growing skull fx</td>
<td>parietal</td>
<td>14 days</td>
<td>distal MCA</td>
<td>clipped</td>
<td>good</td>
</tr>
<tr>
<td>Asari, et al., 1977</td>
<td>4 yrs, F</td>
<td>fall</td>
<td>ICH</td>
<td>yes</td>
<td>9 wks</td>
<td>pericallosal</td>
<td>clipped</td>
<td>fair</td>
</tr>
<tr>
<td>Nakamura, et al., 1977</td>
<td>6 yrs, F</td>
<td>MVA</td>
<td>asymptomatic</td>
<td>frontal</td>
<td>?</td>
<td>distal ACA</td>
<td>excised</td>
<td>good</td>
</tr>
<tr>
<td>Laun, 1979</td>
<td>7 yrs, F</td>
<td>MVA</td>
<td>SAH</td>
<td>no</td>
<td>21 days</td>
<td>cavernous carotid</td>
<td>trapped</td>
<td>poor</td>
</tr>
<tr>
<td>Endo, et al., 1980</td>
<td>4 yrs, F</td>
<td>MVA</td>
<td>HCP, growing skull fx</td>
<td>parietal</td>
<td>5 days</td>
<td>distal MCA</td>
<td>excised</td>
<td>good</td>
</tr>
<tr>
<td>Parkinson &amp; West, 1980</td>
<td>11 yrs, M</td>
<td>MVA</td>
<td>CHI</td>
<td>basilar</td>
<td>none</td>
<td>suprACL carotid</td>
<td>none</td>
<td>died</td>
</tr>
<tr>
<td>Paul, et al., 1980</td>
<td>8 yrs, M</td>
<td>fall</td>
<td>SAH, IVH</td>
<td>no</td>
<td>18 days</td>
<td>vertebrobasilar</td>
<td>observed</td>
<td>died</td>
</tr>
<tr>
<td>Shallat, et al., 1981</td>
<td>17 yrs, M</td>
<td>MVA</td>
<td>epistaxis, DI, SAH</td>
<td>basilar</td>
<td>21 days</td>
<td>cavernous carotid</td>
<td>carotid ligation</td>
<td>good</td>
</tr>
<tr>
<td>Pozzati, et al., 1982</td>
<td>17 yrs, M</td>
<td>MVA</td>
<td>VI nerve palsy, hemiparesis</td>
<td>no</td>
<td>?</td>
<td>suprACL carotid</td>
<td>carotid ligation</td>
<td>good</td>
</tr>
<tr>
<td>Nov &amp; Cromwell, 1984</td>
<td>14 yrs, M</td>
<td>MVA</td>
<td>SAH, ICH</td>
<td>no</td>
<td>26 days</td>
<td>suprACL carotid</td>
<td>carotid ligation</td>
<td>good</td>
</tr>
<tr>
<td>Amagasa, et al., 1986</td>
<td>4 yrs, F</td>
<td>MVA</td>
<td>SAH</td>
<td>no</td>
<td>14 days</td>
<td>pericallosal</td>
<td>excised</td>
<td>died</td>
</tr>
<tr>
<td></td>
<td>4 yrs, M</td>
<td>ped/MVA</td>
<td>SAH</td>
<td>no</td>
<td>17 days</td>
<td>calloso-marginal</td>
<td>trapped</td>
<td>good</td>
</tr>
</tbody>
</table>

### Diagnosis

Traumatic aneurysms should be suspected in the setting of acute neurological deterioration following any type of closed head injury. Patients suffering closed head injury should undergo immediate CT scanning, and angiography should be undertaken as soon as possible. Patients with history of trauma and recurrent epistaxis, visual loss, progressive cranial nerve palsy or an enlarging skull fracture should also be evaluated with MR imaging/MR angiography, and if a suspicious lesion is found, the patient should be immediately undergo arteriography. Several cases have been described in which patients with normal arteriograms subsequently present with delayed hemorrhage and have aneurysms that are then revealed on repeated studies. Penetrating injuries, particularly stab wounds, require special consideration, and the authors of several large series recommend routine angiography 2 weeks after the injury to rule out the delayed formation of aneurysm.

### Treatment Options

The goal of treatment is to exclude the aneurysm from the circulation by surgical or endovascular methods. In 1975, Fleischer and coworkers reported a 41% mortality...
rate in patients treated conservatively as compared with an 18% mortality rate in surgically treated patients. In subsequent case reports the authors have also described poor outcomes in patients who underwent conservative treatment.12 We agree that aggressive surgical management is the most appropriate treatment.6,12,15,27,32 Prompt craniotomy with clipping, resection, or trapping of the aneurysm is considered to be the definitive management.

Endovascular techniques such as trapping, in which detachable balloons or embolization with detachable coils are used, have also been successfully performed in the treatment of traumatic aneurysms.12,25,50 Surgical clipping has advantages that in it provides definitive isolation of the aneurysm, allows for the reconstruction of the parent artery if needed, and facilitates removal of mass effect via evacuation of intracranial hematoma and deflation of the aneurysm itself. On the other hand, the use of endovascular therapy avoids prolonged anesthesia, minimizes manipulation of adjacent vessels and structures, and allows diagnostic angiography to be performed throughout the case.35 The wide variation in aneurysm geometry, anatomical location, and comorbid conditions that may be encountered mandates individual consideration as to which technique is most appropriate on a case-by-case basis.

CONCLUSIONS

We conclude that: 1) posttraumatic aneurysms must be considered in patients with acute neurological deterioration after closed head injury; 2) they can occur after mild closed head injury; 3) they occur more commonly in children than adults; and 4) surgical clipping and/or endovascular occlusion are the definitive treatments.

References


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