Case Reports

Traumatic Aneurysm of a Peripheral Cerebral Artery

Review and Case Report

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Cerebral aneurysms due to trauma constitute a very small group. They usually involve the large basal arteries but also occur on the peripheral cerebral arteries. The identification of these lesions is a matter of the greatest urgency because most of them can be easily removed, whereas if they remain unrecognized and rupture, the associated mortality is high.

Case Report

A 14-year-old boy was brought to the accident room immediately following an auto accident in which he had been thrown from the front to back seat striking the right occiput. He was said to have been unconscious for 20 minutes.

Examination. Vital signs were normal, and the patient was alert and well oriented. Comminuted and depressed fragments of bone could be palpated through a 7-8 cm jagged laceration in the right occipital area. Fundoscopic examination, visual acuity, and visual fields (confrontation) were normal. The remainder of the neurological and physical examination was normal. Skull x-rays verified the depressed and comminuted fractures of the right occipital and parietal bones.

First Operation. The occipital laceration was extended and exploratory craniectomy performed. The right transverse sinus was found to contain three large penetrating fragments of bone deeply imbedded in the sinus. Following their removal hemorrhage was controlled by finger pressure over gel foam and paddies. Ligation of the sinus was not considered due to the possibility of total superior sagittal sinus drainage by this route. A new wall for the sinus was constructed from gel foam and a fascia lata graft.

Postoperative Course. The patient regained normal consciousness and vision; the spontaneous central retinal vein pulsations noted on admission were still present. During the first few days the patient's only complaints were occasional dizziness and neck stiffness. On the third postoperative day spontaneous central retinal vein pulsations were no longer present and early blurring of the right disc margin was evident; a percutaneous right brachial arteriogram performed the same day showed an aneurysm of the temporo-occipital branch of the posterior cerebral artery although at the time it was considered normal except for partial obstruction of the right transverse sinus at the point of injury and repair.

The clinical course was stable until the sixth postoperative (and post-trauma) day when the patient suddenly sat up in bed, grasped his head in his hands, and began to shriek because of excruciating headache and then difficulty seeing. Within a few minutes he became lethargic and then comatose, with a pulse rate of 40. The occipital craniectomy site was noted to be bulging; a ventricular needle was passed transdurally through a small scalp incision and 5 cc of bloody fluid under pressure removed. Lumbar puncture revealed grossly bloody cerebrospinal fluid with an opening pressure of 230 mm H₂O and a hematocrit of 3%. Following lumbar puncture the patient improved markedly, and his condition remained stable except for occasional episodes of projectile vomiting. Funduscopic examination revealed the absence of central retinal vein pulsations bilaterally, and 2 diopters of papilledema with flame hemorrhage on the right. Repeated lumbar punctures indicated progressive diminution of pressure and red blood cell content. A right brachial arteriogram repeated on the fourth post-hemorrhage day revealed a definite pea-sized aneurysm arising from the temporo-occipital branch of the posterior cerebral artery; it was now larger than in the...
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postoperative arteriogram performed 7 days previously (Figs. 1 and 2).

Second Operation. On the sixth post-hemorrhage (twelfth post-trauma) day the patient was reexplored through an occipital craniotomy. There was no significant hematoma. The aneurysm was located in an area of contused and scarred occipital cortex adherent to the lacerated transverse sinus; the aneurysm and feeding artery were obliterated by electrocautery.

Second Postoperative Course. The patient made a good recovery. No evidence of the aneurysm was seen on the third right brachial arteriogram performed 9 days after operation (Fig. 3). At the time of discharge the only neurological abnormalities were small left homonymous inferonasal quadrantic field defects and slight blurring of the right disc margin. One year after surgery the patient is well and neurologically intact.

Discussion

A review of related reports has produced only 10 cases sufficiently well documented to justify designation as a traumatic aneurysm of a peripheral cerebral artery. Table 1 presents a brief description of each case. With our case included, the over-all mortality is 54%. None of the cases cited, including our own has been adequately documented by pre-trauma, post-trauma, and postoperative arteriograms as well as operative confirmation and histologic review. Moreover, the possibil-

Fig. 1. Left: 6th post-trauma day. Right brachial arteriogram indicates early evidence of aneurysm (arrow). Right: 12th post-trauma day. Prominent pea-sized aneurysm has clearly developed on temporo-occipital branch of posterior cerebral artery (arrow).

Fig. 2. Anteroposterior simultaneous view of the aneurysm (12th post-trauma day).

Fig. 3. Postoperative right brachial arteriogram 9 days after removal shows no sign of aneurysm.
### Summary of 11 cases of traumatic aneurysm of a peripheral cerebral artery

<table>
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<tr>
<th>Author</th>
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<td>1. Krauland⁵⁰</td>
<td>35 yrs M</td>
<td>Depressed, left frontal skull fracture with unconsciousness</td>
<td>None</td>
<td>No angiogram</td>
<td>No operation</td>
<td>Multiple dural tears, tears of intima of left carotid artery; organized pea-sized false aneurysm on anterior cerebral artery. Aneurysm lumen in communication with anterior cerebral artery</td>
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<td>2. Finmekeyer¹</td>
<td>50 yrs M</td>
<td>3-wk deterioration of memory with somnolence, confusion, and right frontal headache. Right hemiparesis with unsteady gait. Meningioma at base of right frontal lobe totally removed at surgery</td>
<td>16th day</td>
<td>Previous angiogram showed no evidence of aneurysm. Post-operative shift of anterior cerebral artery across midline</td>
<td>Large conglobated hematoma surrounding aneurysm at the temporal pole. Aneurysm clipped</td>
<td>Specimen not reported</td>
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<td>3. Courville⁴</td>
<td>45 yrs F</td>
<td>Shotgun wound to left side of face 10 yrs prior to admission. Occasional focal seizures. Sudden right-sided headache, vomiting, and seizure. Semicoma with meningeal signs and extensor plantar reflexes</td>
<td>10 yrs</td>
<td>Minimal filling of left middle cerebral artery. Slight shift of anterior cerebral artery</td>
<td>No operation</td>
<td>Aneurysm of left middle cerebral artery. Hyalinization of aneurysm wall with absent or defective intima</td>
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<td>4. Isfori²²</td>
<td>19 yrs F</td>
<td>Closed head injury with unconsciousness; 2-day improvement then rapid deterioration. Left temporal skull fracture</td>
<td>None</td>
<td>Pesa-sized aneurysm in left temporoparietal area</td>
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<td>5. Brenner³</td>
<td>17 yrs M</td>
<td>Penetrating head injury. Fragments of bone found extending into lateral ventricle</td>
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<td>33 yrs M</td>
<td>3 yrs of headache and seizures. Papilledema and left hemiparesis. Resection of right frontal tumor</td>
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<td>No aneurysm on pre-operative arteriogram. Post-operative arteriogram revealed aneurysm of right anterior communicating artery adjacent to hemostatic clip</td>
<td>Saccular sessile aneurysm in line of resection</td>
<td>Thin walled sac composed of collagenous tissue, no endothelial layers; tumor infiltration</td>
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<td>7. Hirsch, et al.²⁰</td>
<td>62 yrs M</td>
<td>Head injury with coma and right hemiparesis. History of alcoholism</td>
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<td>Left to right shift of anterior cerebral artery. Aneurysm of left posterior parietal branch of middle cerebral artery</td>
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<td>9. Cressman &amp; Hayes⁷</td>
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<td>Impaired through left eye with car antenna. Unconscious with right hemiplegia and facial nerve palsy</td>
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<td>Small aneurysm of anterior choroidal artery at twenty-four hours—well developed aneurysm at same site</td>
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<td>Laceration of dura and brain affecting uncus, globus pallidus, thalamus, and internal capsule</td>
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**TABLE 1**

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<td>Direct Arterial Injury. Five cases were associated with penetrating injuries involving skull fragments, missiles, or foreign bodies. In each case the resulting aneurysm was found in the immediate vicinity of the penetrating wound. Early arteriography was performed in the patients with a poor neurological status, and the aneurysm was recognized. When the patient did well initially, however, arteriography was not performed or else was delayed until after deterioration. In our case the first arteriogram was fortuitous. It appears that arteriography performed 5 to 7 days following a penetrating head injury would be the most productive in demonstrating a traumatic aneurysm. This is particularly true in the patient with a benign course where arteriography might not otherwise be considered.</td>
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<tr>
<td>10. Overton &amp; Calvin &amp; a</td>
<td>9 mos M</td>
<td>Head injury with &quot;egg shell&quot; fracture and bilateral subdural hematoma. Daily subdural taps. Craniotomy performed when fluid became grossly bloody</td>
<td>5th wk</td>
<td>No angiogram</td>
<td>Thick hematoma membranes, cortical contusion with clotted blood. 4-5 mm diameter aneurysm arising from cortical artery</td>
<td>Saccular aneurysm with defect in elastic tissue and expansion in area of media</td>
<td>Benign course</td>
</tr>
<tr>
<td>11. Burton, et al.</td>
<td>14 yrs M</td>
<td>Head injury with depressed, comminuted fractures of right occipital &amp; parietal bones. Unconscious 20 min. Craniotomy; right transverse sinus wall reconstructed</td>
<td>6th day</td>
<td>Pea-sized aneurysm of the temporo-occipital branch of the posterior cerebral artery</td>
<td>Aneurysm in area of contused, scarred occipital cortex adherent to lacerated transverse sinus</td>
<td>No specimen</td>
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Indirect Arterial Injury. The three cases reported were major closed head injuries associated with significant neurological deficit for which arteriography was performed. Cases 7 and 8 represent closed head injuries associated with subdural hematoma, and Case 4, with cortical contusion. Although Case 4 was a closed head injury the depressed skull fracture and dural laceration imply direct rather than indirect arterial trauma.

Delayed Traumatic Apoplexy (Intercranial Hemorrhage). This entity was first recognized in humans in 1891 by Bollinger, who suggested that a mechanical brain injury caused encephalomalacia and softening with alteration in local blood vessels leading to delayed apoplexy. Symonds felt that there were two varieties of delayed traumatic apoplexy, early and late. In the early type hemorrhage occurred within a few weeks of injury as the result of traumatic weakening of an arterial wall with aneurysmal dilation and subsequent rupture; blood was often found in the ventricles and subarachnoid space. In late apoplexy the interval was several months or years; the mechanism suggested was hemorrhage into a cyst resulting from the intracerebral hemorrhage at the time of injury.

Although Bollinger's original suggestion of apoplexy due to a damaged blood vessel appears valid, it seems more reasonable to expect this as a sequel of major, rather than minor, head injury. The apoplexy following
minor head injury is better explained on the basis of preexisting small vascular malformations. These have been termed “cryptic hamartomas” or “microangiomas.”

Arteriography. By comparing the lateral views of the first and second arteriograms in Case 11 (Fig. 1), one can observe a developing cortical traumatic aneurysm. Tavera et al. believes that a segment of vessel seen “end on” is always denser than the same vessel immediately proximal or distal, and the density of the aneurysmal sac, with few exceptions, is usually less than an artery of the same diameter.

Although the arteriographic presentation of a cortical traumatic aneurysm may be similar to that of a “mycotic” or septic aneurysm, the lack of association with trauma assists in the differentiation. When trauma is present a cerebral traumatic aneurysm may be confused with a traumatic aneurysm of the middle meningeal artery. Middle meningeal aneurysms can be identified by the following characteristics:

1. Lateral view. The aneurysm shadow lies on the floor of the middle fossa.
2. Anteroposterior view. Aneurysm shadow is seen in the far lateral position against the inner table of the skull unless there is associated epidural hematoma in which case the meningeal and cortical vessels are displaced away from the skull.

Arteriographic differentiation of a traumatic aneurysm from a preexisting saucular aneurysm is aided by the usual location of the latter at arterial bifurcations. In a review of 252 cases of saucular aneurysm found at autopsy, Stehbens was unable to find a single one arising at a site other than an arterial bifurcation. In addition, saucular aneurysms, because of their proximal location, tend to fill earlier than those located on peripheral arteries.

In cases of penetrating head injury, the traumatic aneurysm can be expected in the immediate vicinity of the damaged brain. Following closed head injury, peripheral traumatic aneurysms are usually found along the Sylvian fissure, involving branches of the middle cerebral artery.

Pathological Characteristics

General. When an artery enters the cranial cavity it becomes a “cerebral artery” whose histologic appearance differs from that of typical muscular or elastic artery seen elsewhere in the body. The major structural difference involves the elastic tissue which is almost completely concentrated in a thin inner elastic lamina in the cerebral artery. A recent study on the etiology of saucular aneurysms has added further evidence to the importance of atheromatous degenerative changes on the destruction of the inner elastic lamina and weakening of the vessel wall. If degenerative changes can accomplish this, the same result can be expected if trauma caused injury to the inner elastic lamina.

In the event of trauma the local supportive milieu appears to be an important consideration. Meningeal arteries, whose histologic structure changes to that of a cerebral artery upon entering the cranial cavity, have developed traumatic aneurysms. However, only eight traumatic aneurysms of the middle meningeal artery have been reported, whereas traumatic aneurysms of the arteries at the base of the brain are more frequent. The meningeal arteries appear to be better protected by the supporting brain coverings while the thin-walled and relatively unsupported cerebral vessels sustain injury at the moment of trauma. During the same episode of trauma the craniocephalic mass may be deformed, accelerated, and decelerated as well as compressed, torn, or sheared. The shearing component appears the most significant in the relatively frequent basal artery injuries. Confusion between saucular aneurysms of the circle of Willis and its major branches and traumatic aneurysms of the peripheral cerebral artery is unlikely due to their different locations.

Types of Traumatic Aneurysms. Vascular trauma can be separated into that occurring from direct and indirect injury. The type of traumatic aneurysm produced can be true (partial dilatation resulting from partial disruption of the arterial wall), false (cavities of encapsulated hematomas communicating with the lumen of the artery), or mixed (caused by the rupture of a true aneurysm giving rise to a secondary false aneurysm).

True Traumatic Aneurysms, Direct Trauma. When a penetrating injury fails to sever completely or penetrate the arterial wall the damage to the internal elastic lamina appears to allow dilatation and aneurysm formation.
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Histologic examination shows persistence of normal vessel wall constituents, particularly the intimal layer. This is well illustrated in Cases 1 and 10.

True Traumatic Aneurysms, Indirect Trauma. Cortical damage resulting from closed head injury usually results in local brain necrosis with softening. Bollinger related this to alteration in the local blood vessels causing delayed apoplexy. In six of the cases reported delayed apoplexy was present. None of the patients, however, had a closed head injury or a histologically verified true traumatic aneurysm.

False Traumatic Aneurysms, Direct Trauma. When a penetrating injury causes laceration of an arterial wall, the usual result is rapid massive hemorrhage without aneurysm formation. If this hemorrhage is not massive or can be contained by local factors the limited hematoma resulting may undergo fibrous organization, forming a false aneurysm. The enlargement of this fibrous clot (false aneurysm) may then be promoted by hemodynamic factors aided by the laying down of central laminations of clot or autolysis of the fibrin clot caused by contact with the cerebrospinal fluid. Markwalder suggested that this action of cerebrospinal fluid on false aneurysms was one explanation for delayed apoplexy. In true aneurysms the presence of an intimal layer inhibits the lytic action. Histologically the false aneurysm wall is similar to that in Cases 3 and 6.

False Traumatic Aneurysms, Indirect Trauma. In Cases 7 and 8, a subdural hematoma was found in association with a false traumatic aneurysm following head injury. The “communicating” or “bridging” veins between the arachnoid and the dura which generally have a thin fibrous wall and elastic lamina are usually implicated as being those torn when the brain moves within the skull at the moment of injury. Vance has found in his examination of bridging vessels from the subarachnoid space to the dura that some contained only veins while other contained both arteries and veins. Other authors have shown that the arteries on the lateral cortical surface (where most subdural hematomas of arterial etiology are found) have small arterial twigs that come off the outer wall of the parent vessel at right angles just under the arachnoid where they are exposed and more susceptible to injury. How frequently are these arteries involved in head trauma? A number of authors have documented typical subdural hematomas where the source of bleeding was established as having been due to small rents in surface cortical arteries. Cases 2, 6, and 10 represent traumatic aneurysms where the arterial injury was direct and iatrogenic. True and false aneurysms were produced. Experimental aneurysm production in dogs by introducing a needle through the arterial wall indicates that such an injury has its main effect on the internal elastic lamina.

In 1873 Ponfick proposed the theory that traumatic aneurysms were formed by emboli broken from calcareous atheromatous plaques or calcified thrombi, following laceration or pressure atrophy of the inner coats of small cerebral arteries. Although the seven cases he presented were due to emboli it appears that they were septic and related to malignant endocarditis. Theoretically, traumatic aneurysms may be caused by mechanical internal injury from calcified emboli, but we know of no evidence to support this view.

Summary

Traumatic aneurysms of peripheral cerebral arteries are a definite entity associated with a high mortality, but survivors are known and such a case is reported. We have also reviewed all related cases and suspect the prevalence of this lesion is significantly higher than the 10 cases reported. Because the surgical removal of traumatic aneurysms of the cortical vessels is easy, every effort must be made to identify this lesion as early as possible. The diagnosis should be considered in all patients having delayed apoplexy after a significant head injury.

Acknowledgments

We would like to express our appreciation to Dr. Dietrich Blumer for his assistance in translation and to Dr. A. Earl Walker for his review of the manuscript.

References


36. STEVENSON, W. D. Chronic subdural hematoma in adults. (cit. Drake, C. G., see ref. 9.)


