The incidence of traumatic intracranial aneurysms is extremely low. Most traumatic intracranial aneurysms are located within the anterior circulation, that is, essentially involving the internal carotid artery or the middle cerebral artery. Less than 10% of traumatic intracranial aneurysms are located within the posterior circulation. To our knowledge, only 15 of these cases have been reported in the literature. Among the 15 traumatic aneurysms, two were described as mimicking a tumor of the posterior fossa and the exact diagnosis of aneurysm was generally established during surgery.

We report a rare case of traumatic intracranial aneurysm that developed on the peripheral portion of the superior cerebellar artery (SCA) after closed head trauma and discuss the mechanisms involved in the aneurysm’s formation.

**Case Report**

**History.** This 22-year-old woman presented with a 3-month history of disabling headaches. These headaches were resistant to nonnarcotic analgesic medications. The headaches first occurred 15 days after the patient was involved in a cycling accident in which she lost consciousness for 10 minutes and was taken to the local hospital with closed head trauma. The x-ray films of the patient’s skull were normal. She returned home after 3 days of medical observation. The patient’s medical history showed that she had been healthy with no head trauma prior to this accident. Moreover, her birth had been free of trauma and her parents had no cerebrovascular disease.

**Examination.** The patient’s physical and neurological examinations were normal. Computerized tomography (CT) scanning showed a nodular neoformation that mimicked a meningioma. The aneurysm was identified on magnetic resonance angiography, which showed the SCA as the parent vessel. The parent vessel was trapped, and the aneurysm sac was excised via right temporal craniotomy. Pathological examination of the sac revealed a false aneurysm. The patient’s outcome was excellent. The pathophysiology of traumatic aneurysm at such a location suggests that surgery may be the treatment of choice.

**Key Words** • traumatic aneurysm • tentorial incisure • fusiform aneurysm
by the aneurysm. Because of the absence of a neck, the aneurysm was trapped between hemostatic miniclips and excised along with a 2-mm segment of the SCA.

Pathological Findings
The aneurysm was removed for pathological examination. Histological sections of the excised tissue revealed a false aneurysm formed by fibrous tissue and organized hematoma (Fig. 4).

Postoperative Course. During the immediate postoperative period, the patient was troubled by transitory headaches and instability of gait. She returned home on the 15th postoperative day. A follow-up angiographic CT scan, obtained in the 2nd postoperative month, showed complete exclusion of the sac and occlusion of the SCA. In the 12th postoperative month, the patient was totally asymptomatic and was able to resume her previous work. A routine MR image was obtained 18 months postoperatively and no silent infarction in the distal SCA area was observed (Fig. 5).

Discussion

Diagnosis of Traumatic Aneurysm

Strictly speaking, the definition of traumatic intracranial aneurysm would require proof of its formation after head trauma by comparing pretraumatic and posttraumatic angiographic studies. In practice, an exceptional diagnosis such as the one in this case is supported by the combination of four factors: the history of the trauma, the site of the aneurysm being other than at branching points, the young age of the patient, and the histopathological findings.15,25 In our case, the young age of the patient and the occurrence of head trauma 15 days before the headaches began were two factors that favored the traumatic quality of the aneurysm. The angiographic character of the lesion, the site of the aneurysm, and the neuropathological “false” sac consolidated the diagnosis of traumatic aneurysm. Cockrill, et al.,8 in reporting the case of a traumatic aneurysm that simulated a posterior fossa tumor, rectified their diagnosis at surgery. A traumatic giant aneurysm of the PCA, which mimicked a tentorial edge meningioma on CT scanning, was reported in a 24-year-old patient who presented with grand mal seizures after a head injury.7 In our case, the same differential diagnosis was questionable. The MR image, therefore, was of great interest. It demonstrated the vascular character of the nodule, owing to its early homogeneous enhancement after gadolinium injection. The MR angiography sequences showed the aneurysm sac (Fig. 2 lower) and its partial thrombosis, but could not precisely reveal the parent vessel. Cerebral angiography demonstrated the exact topography and displayed the classic characteristics that differentiate the traumatic aneurysm from its congenital counterpart: filling late in the arterial phase, opacifying to lesser extent, and emptying slowly.5,6,24

Traumatic Aneurysm of the Vertebrobasilar System

To our knowledge, 15 cases of traumatic aneurysms located within the VB system have been reported in the
Penetrating wounds were responsible for 40% of the traumatic aneurysms located in the VB system compared with 10% of traumatic intracranial aneurysms as a whole. Furthermore, traumatic aneurysms of the VB system are less frequently the consequence of a closed head trauma (60%) than those located within the anterior circulation. In closed head trauma, several factors are considered responsible for the formation of traumatic aneurysms. The contusion of the vertebral artery (VA) caused by rubbing against a bony prominence was reported by Paul, et al. The fracture and its bone margins, located on the clivus or the occipital vault, can lacerate vessel walls. This mechanism is the most frequent of all traumatic aneurysms, whatever the location. In the absence of a fracture or penetrating wound, some traumatic aneurysms have been observed on arteries in relation to the edge of rigid meningeal structures. The tentorium has been implicated for traumatic aneurysms located in both the PCA and the SCA. Casey and Moore believed that a traumatic aneurysm located on the PCA was caused by rapid-deceleration head injury with sudden brain and artery movement, causing the vessel wall to be injured by the free edge of the tentorium. In their case, the traumatic aneurysm was secondary to severe head trauma. Piatt and Clunie reported on an aneurysm of the SCA in which formation could be attributed to birth trauma. During delivery, the distance from the base to the vertex increases while the tentorium resists this type of deformation. Vessels can be damaged by rubbing against the free edge of the tentorium. In our patient,

**TABLE 1**

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>Age (yrs), Sex</th>
<th>Injury Type</th>
<th>Presentation</th>
<th>Location</th>
<th>Histology</th>
<th>Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>Burton, et al., 1968</td>
<td>14, M</td>
<td>penetrating (MVA)</td>
<td>occipital laceration</td>
<td>PCA</td>
<td>false an</td>
<td>trapped, excision</td>
<td>good</td>
</tr>
<tr>
<td>Ferry &amp; Kempe, 1972</td>
<td>21, M</td>
<td>penetrating (fragment)</td>
<td>diplopia, pain</td>
<td>SCA</td>
<td>false an</td>
<td>ligated, excision</td>
<td>good</td>
</tr>
<tr>
<td>Shaw &amp; Alvord, 1972</td>
<td>59, M</td>
<td>closed</td>
<td>coma</td>
<td>BA</td>
<td>pontine infarct</td>
<td>none</td>
<td>death</td>
</tr>
<tr>
<td></td>
<td>30, M</td>
<td>closed</td>
<td>SAH Day 19</td>
<td>BA</td>
<td>false an, swelling, IVH</td>
<td>none</td>
<td>death</td>
</tr>
<tr>
<td>McDonald, et al., 1976</td>
<td>44, M</td>
<td>penetrating (screwdriver)</td>
<td>hemiparesis</td>
<td>SCA</td>
<td>NP</td>
<td>none</td>
<td>good</td>
</tr>
<tr>
<td>Cockrill, et al., 1977</td>
<td>15, M</td>
<td>closed</td>
<td>diplopia, ataxia</td>
<td>SCA</td>
<td>NP</td>
<td>none</td>
<td>good</td>
</tr>
<tr>
<td>Bank, et al., 1978</td>
<td>44, M</td>
<td>penetrating (shotgun)</td>
<td>coma, hemiplegia</td>
<td>BA</td>
<td>NP</td>
<td>cervical clamp</td>
<td>good</td>
</tr>
<tr>
<td>Paul, et al., 1980</td>
<td>8, M</td>
<td>closed (fall)</td>
<td>SAH Day 9</td>
<td>VA</td>
<td>true an</td>
<td>none</td>
<td>death</td>
</tr>
<tr>
<td>Wortzman, et al., 1980</td>
<td>56, F</td>
<td>closed (MVA)</td>
<td>SAH Day 9</td>
<td>PICA (distal)</td>
<td>false an, fracture, dural tear</td>
<td>trapped, excision</td>
<td>good</td>
</tr>
<tr>
<td>Meguro &amp; Rowed, 1985</td>
<td>54, M</td>
<td>closed (MVA)</td>
<td>coma</td>
<td>PICA (proximal)</td>
<td>false an, fracture, midbrain lesion</td>
<td>trapped, excision</td>
<td>good</td>
</tr>
<tr>
<td>Quattrrocci, et al., 1990</td>
<td>23, M</td>
<td>penetrating (antenna)</td>
<td>coma</td>
<td>SCA</td>
<td>false an</td>
<td>none</td>
<td>death</td>
</tr>
<tr>
<td>Morard &amp; de Tribolet, 1991</td>
<td>31, M</td>
<td>closed (fall)</td>
<td>coma</td>
<td>PICA (distal)</td>
<td>false an, fracture</td>
<td>clipped, excision</td>
<td>good</td>
</tr>
<tr>
<td>Piatt &amp; Clunie, 1992</td>
<td>5 days, M</td>
<td>closed (forceps)</td>
<td>seizures</td>
<td>SCA</td>
<td>NP</td>
<td>thromb</td>
<td>good</td>
</tr>
<tr>
<td>Casey &amp; Moore, 1994</td>
<td>24, M</td>
<td>closed</td>
<td>seizures</td>
<td>PCA (proximal)</td>
<td>NP</td>
<td>trapped</td>
<td>good</td>
</tr>
<tr>
<td>Rezai, et al., 1994</td>
<td>42, M</td>
<td>penetrating (nail)</td>
<td>coma</td>
<td>PCA</td>
<td>false an</td>
<td>none</td>
<td>death</td>
</tr>
</tbody>
</table>

* An = aneurysm; BA = basilar artery; IVH = intraventricular hemorrhage; MVA = motor vehicle accident; NP = not performed; PICA = posterior inferior cerebellar artery; SAH = subarachnoid hemorrhage; thromb = spontaneous thrombosis shown on routine angiography performed 40 days after injury.
the aneurysm sac was located on the free edge of the tentorium, but the underlying parenchyma remained intact preoperatively. Following the mechanism suggested by Piatt and Clunie, we surmise that in our case the head trauma produced a rotary movement of the brain within the skull, tearing an arterial branch of the SCA that passes through the perimesencephalic cistern. Some distal branches of the SCA passed medial to the posterior third of the free edge of the tentorium as they entered or exited the cerebellomesencephalic groove. These arteries can be adherent to the dura mater, or more rarely can be the origin of an arterial twig leading to the dura mater, according to the mechanism of subdural hematoma formation secondary to benign head trauma described by Drake.

Natural History of Traumatic Aneurysm and Its Treatment

Whatever their location, in traumatic aneurysms the major causes of morbidity and mortality are associated cerebral lesions and delayed hemorrhage resulting from the ruptured aneurysm. The natural history of intracranial traumatic aneurysms is difficult to appreciate because the frequently associated cerebral lesions “are the most important factor in determining the prognosis of a particular case.” The rate of mortality varies between 32% and 50% of cases, which decreases to between 18% and 24% after surgical treatment. The probability that a traumatic aneurysm might rupture is high because 40 to 60% have been revealed after a delayed hemorrhage. These results favor surgical treatment of this type of traumatic malformation. One-third of the 15 reported patients with traumatic aneurysms located within the VB system died. The causes responsible were associated lesions resulting from penetrating wounds in two cases, a pontine infarction in one case, and delayed hemorrhage in two cases (Table 1).

In addition to rupture, the sacs of intracranial aneurysms can be the object of angiographic modifications. They can increase in size or, in rare cases, evolve to spontaneous occlusion. The latter has been demonstrated to be the cause of traumatic aneurysms of the pericallosal and middle cerebral arteries. Among the traumatic aneurysms of the VB system, Piatt and Clunie also described the spontaneous disappearance of an SCA aneurysm on follow-up angiography performed 40 days after injury.

The majority of authors argue that intracranial traumatic aneurysms should be treated. Ideally an aneurysm in any location is clipped after parent vessel trapping in the majority of cases. Rarely, however, it is necessary to protect the parent vessel by placing a tangential clip on the neck or repairing it with a microvascular suture. The difficulty in preserving the flux in the parent vessel is the result of the pathological characteristics of this traumatic aneurysm sac. These “false” aneurysms are formed after complete rupture of the vessel wall. They are composed of cavities of encapsulated hematoma that communicate with the artery lumen.

This composition explains the lesser opacification and slower emptying observed on angiography. Some “true”
traumatic aneurysms have been described; these consist of a dilatation resulting from partial disruption of the arterial wall. As regards the VB system, this type of true traumatic aneurysm of the VA has previously been reported. In the posterior fossa, among the seven cases surgically treated, six trapped were performed without inducing ischemia. In our case, the absence of a neck necessitated the occlusion of the SCA. In the immediate postoperative period, our patient complained of headaches and instability of gait, both of which completely disappeared in 10 days. These symptoms probably resulted from infarction in the area of the SCA. The CT scan obtained 2 months postoperatively showed no hypodensity; however, artifacts caused by the microclips prevented an entirely correct interpretation. The MR image obtained 18 months postoperatively demonstrated the absence of a silent infarction in the distal SCA area but raised suspicion of a small infarction in the area of the perforating arteries. The outcome of an infarction in the SCA area is generally benign with normal findings on neurological examination. At present, the occlusion of the parent vessel by embolization can be avoided in cases of false aneurysms. In our case, the endovascular technique was not used because of the existence of a loop in the SCA, which made it difficult to raise.

Conclusions
This type of traumatic aneurysm is extremely uncommon. Although on initial diagnosis it can be mistaken for a meningioma, the use of MR imaging can facilitate the correct diagnosis. In the case described in this report, the causal mechanism was probably a tear in the twig of the SCA. An analysis of the literature clearly demonstrates that these traumatic aneurysms should be treated surgically; however, the risk of sacrificing the parent vessel remains in the majority of cases.

Acknowledgment
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