Ruptured cerebral arteriovenous malformations (AVMs) are associated with significant morbidity and mortality. Up to 18% of patients with bleeding AVMs harbor associated arterial aneurysms, approximately half of which occur on feeding arteries (prenidal aneurysms). Numerous reports have suggested a correlation between AVM-related aneurysms and hemorrhage risk. Some authors have previously noticed that prenidal aneurysm ruptures were frequent when AVMs were located in the posterior fossa. We sought to confirm, in a series of cases involving patients with AVMs who were treated at our institution, that patients with posterior fossa AVMs frequently present with hemorrhage associated with prenidal aneurysm ruptures. We also aimed to review the outcome for patients presenting with such ruptures who were treated by endovascular means.

Abbreviations used in this paper: AVM = arteriovenous malformation; mRS = modified Rankin Scale; PICA = posterior inferior cerebellar artery.

Methods

We conducted a retrospective chart and imaging review of all consecutive cases of AVMs in which patients were treated by means of endovascular techniques in our institution between April 2001 and August 2012. This study was approved by our local ethics committee. Patients were included if their neuroradiology reports (angiography and CT) mentioned hemorrhage and the presence of a prenidal aneurysm. Neuroimaging studies were reviewed by a senior neuroradiologist (D.R.). By definition, hemorrhage secondary to the rupture of a prenidal aneurysm was diagnosed when the hematoma and/or the epicenter of subarachnoid hemorrhage was clearly remote from the AVM nidus but in the vicinity of the aneurysm (Fig. 1). Cases in which hemorrhage could be related to an aneurysm located at the circle of Willis or at usual locations in the posterior circulation were excluded. Using this definition, we recorded posterior fossa AVMs that bled from prenidal aneurysms and looked for cases in which...
the patients had a similar presentation but had supratentorial AVMs. Proportions were compared using the Z-test. Finally, we report the outcome (modified Rankin Scale [mRS] score) for patients with posterior fossa AVMs presenting with prenidal aneurysm hemorrhage (p < 0.01). These data are summarized in Table 1.

**Management and Outcome**

The patients’ age, AVM and aneurysm characteristics, treatment, and mRS scores are summarized in Table 2. In all 9 cases of hemorrhage due to prenidal aneurysm rupture, the aneurysm presumed responsible for the hemorrhage was treated by means of an endovascular approach on an emergency basis with either coils (5 aneurysms) or glue (4 cases). In most cases (7), the aneurysm was occluded along with its parent artery. One patient (Case 7) died of the initial hemorrhage. In 2 cases, parent vessel occlusion caused a small vermian infarct, with only transient symptoms, if any. There was no permanent neurological complication related to the treatment of prenidal aneurysms. In the 8 surviving patients, 3 unruptured prenidal aneurysms (2 in Case 9, 1 in Case 2) and 1 unruptured posterior inferior cerebellar artery (PICA) aneurysm (in Case 4) were treated electively after the acute phase. Curative treatment of the AVM itself, proven by angiography, was performed on an elective basis in 3 cases (all with embolization followed by resection). One patient (Case 9) was treated the following month, and the other 2 (Cases 4 and 6) were treated 5 months and 20 months, respectively, after the initial hemorrhage, when clinical recovery was optimal. In the remaining 6 patients, the AVM nidus was left untreated: 1 patient (Case 7) died of initial hemorrhage; 1 patient, who was 76 years old at presentation (Case 3), was lost to follow-up; and 4 patients declined definitive treatment of the AVM. One of these patients who declined definitive AVM treatment died 4 years later from AVM rupture (Case 5). The mean follow-up period of the 6 surviving and followed patients was 46.8 months (range 19–74 months). At the end of follow-up, 6 of 9 patients had a good outcome. The mRS score was 0 in 2 cases, 1 in 1 case, 2 in 3 cases, 6 in 2 cases, and 1 patient was lost to follow-up.

![Fig. 1. A: CT scan showing inferior vermian hematoma with blood inside the fourth ventricle. B: CT angiogram (sagittal view) showing AVM feeding artery aneurysm in the epicenter of a hematoma (long arrow) with the AVM nidus (short arrow) clearly remote from the hematoma. C: Left vertebral artery angiogram, lateral view, confirming the CT angiography findings. D: Superselective injection of the PICA branch harboring the aneurysm. E: Left vertebral injection after glue embolization of the aneurysm and parent vessel.](image)

**TABLE 1: Proportion of bleeding AVMs with prenidal aneurysm rupture**

<table>
<thead>
<tr>
<th>AVM Location</th>
<th>Total No. of AVMs</th>
<th>AVMs w/ Hemorrhagic Presentation</th>
<th>Hemorrhage Due to Prenidal Aneurysm Rupture*</th>
</tr>
</thead>
<tbody>
<tr>
<td>supratentorial</td>
<td>208</td>
<td>107 (51.4%)</td>
<td>5 (4.7%)</td>
</tr>
<tr>
<td>infratentorial</td>
<td>25</td>
<td>22 (88%)</td>
<td>9 (41%)</td>
</tr>
</tbody>
</table>

* Percentages calculated based on number of AVMs with hemorrhagic presentation. 

**Results**

**Presentation and Incidence of Prenidal Aneurysm Rupture**

We identified 233 cases of AVMs involving patients treated between April 2001 and August 2012 in our institution. The patients’ mean age was 44 years (range 11–80 years). Of the 233 AVMs, 208 were supratentorial (89%), and 25 (11%) were located in the posterior fossa. Hemorrhage occurred in 107 (51.4%) of the supratentorial AVMs compared with 22 (88%) of the 25 posterior fossa AVMs. In 9 (41%) of the 22 cases of posterior fossa AVMs, the patients presented with a hemorrhage related to a prenidal aneurysm, whereas only 5 (4.7%) of 107 patients with supratentorial AVMs presented with a prenidal aneurysm hemorrhage (p < 0.01). These data are summarized in Table 1.
Management of prenidal aneurysm rupture in posterior fossa AVMs

TABLE 2: AVM characteristics, management, and clinical outcomes in patients with posterior fossa AVM and prenidal aneurysm rupture*

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Pt Age (yrs)</th>
<th>AVM Size (cm)</th>
<th>AVM Location</th>
<th>Aneurysm Size (mm)</th>
<th>Aneurysm Location</th>
<th>Treatment</th>
<th>Complication</th>
<th>AVM Nidus Treatment</th>
<th>Outcome</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>58</td>
<td>3.0</td>
<td>superficial right hemisphere</td>
<td>10</td>
<td>right PICA</td>
<td>PVO coils</td>
<td>infarct, asymptomatic</td>
<td>no</td>
<td>43 mos, mRS 0</td>
</tr>
<tr>
<td>2</td>
<td>57</td>
<td>2.5</td>
<td>superficial right hemisphere</td>
<td>4</td>
<td>right PICA</td>
<td>PVO glue</td>
<td>infarct, asymptomatic</td>
<td>no</td>
<td>51 mos, mRS 1</td>
</tr>
<tr>
<td>3</td>
<td>76</td>
<td>1.5</td>
<td>superior vermis</td>
<td>10</td>
<td>right SCA</td>
<td>PVO coils</td>
<td>none</td>
<td>no</td>
<td>lost to follow-up</td>
</tr>
<tr>
<td>4</td>
<td>48</td>
<td>2.5</td>
<td>superficial right hemisphere</td>
<td>6</td>
<td>left SCA</td>
<td>selective coils</td>
<td>none</td>
<td>yes</td>
<td>23 mos, mRS 2</td>
</tr>
<tr>
<td>5</td>
<td>58</td>
<td>1.7</td>
<td>superior vermis</td>
<td>5</td>
<td>left SCA</td>
<td>PVO coils</td>
<td>none</td>
<td>no</td>
<td>hemorrhage 4 yrs later, mRS 6</td>
</tr>
<tr>
<td>6</td>
<td>21</td>
<td>2.4</td>
<td>superior vermis</td>
<td>3</td>
<td>right SCA</td>
<td>PVO glue</td>
<td>none</td>
<td>yes</td>
<td>19 mos, mRS 2</td>
</tr>
<tr>
<td>7</td>
<td>62</td>
<td>1.3</td>
<td>superior vermis</td>
<td>13</td>
<td>left SCA</td>
<td>selective coils</td>
<td>none</td>
<td>no</td>
<td>mRS 6, acute phase</td>
</tr>
<tr>
<td>8</td>
<td>63</td>
<td>1.7</td>
<td>superior vermis</td>
<td>5</td>
<td>left PICA</td>
<td>PVO glue</td>
<td>none</td>
<td>no</td>
<td>74 mos, mRS 0</td>
</tr>
<tr>
<td>9</td>
<td>45</td>
<td>3.1</td>
<td>superior vermis</td>
<td>multiple aneurysms</td>
<td>left PICA</td>
<td>PVO glue</td>
<td>none</td>
<td>yes</td>
<td>71 mos, mRS 2</td>
</tr>
</tbody>
</table>

* mRS = modified Rankin Scale score; PICA = posterior inferior cerebellar artery; pt = patient; PVO = parent vessel occlusion; SCA = superior cerebellar artery.

Discussion

We present a retrospective study, showing that patients with posterior fossa AVMs frequently presented with prenidal arterial aneurysm rupture. The occurrence of hemorrhagic presentation with the hematoma or hemorrhage center close to a prenidal arterial aneurysm and remote from the AVM nidus was more frequent in patients with posterior fossa AVMs than in those with supratentorial AVMs (41% vs 4.7%) in our patient population.

Westphal and Grzyska\(^1\) also reported that feeding vessel aneurysms occur more frequently in association with infratentorial than supratentorial AVMs. In the series reported by Schmidt et al.,\(^8\) 30% of posterior fossa AVMs versus 11% of supratentorial AVMs were associated with aneurysms (on feeding vessels within the nidus). The main difference concerned the feeding artery aneurysms (25% vs 5%). Associated aneurysms were the source of bleeding in 10.5% of cases of infratentorial AVMs but only 1.7% of cases of supratentorial AVMs.\(^8\) In the series of posterior fossa AVMs reported by da Costa et al.,\(^2\) the presence of associated aneurysms was associated with worse outcomes.

We acknowledge multiple weaknesses in this study. This is not a natural history study, since only treated patients who had at least 1 endovascular treatment were registered and reviewed. Patients were often referred from other centers, and reasons for referrals and indications for treatments have undoubtedly biased numbers. A systematic imaging protocol was not followed, and not all patients presenting with hemorrhage had an early angiogram to specifically look for aneurysms. We found an almost 10-fold difference between supratentorial and infratentorial AVMs, with respect to the presence of ruptured prenidal aneurysms (41% vs 4.7%). This difference may be related to an apparent increased “fragility” that has been evoked in hemorrhagic dissections and nonmycotic distal aneurysms.\(^6\)\(^7\)

Knowledge of this tendency in posterior fossa AVMs may be useful for the early management of these cases given that the associated aneurysms are often easier to treat definitively than the AVMs themselves. This may be especially true should the AVM be associated with a higher treatment risk due to its location size or patient’s factors.

The frequent association of prenidal aneurysms in bleeding posterior fossa AVMs suggests that they should be systematically searched for. We suggest that urgent treatment of the offending aneurysm with embolization may be indicated to prevent early rebleeding, with treatment of the AVM itself being deferred if desirable.

Conclusions

In patients with posterior fossa AVMs presenting with hemorrhage, the hemorrhage is frequently associated with prenidal arterial aneurysms. Our series and others suggest that this association could be more frequent in patients with posterior fossa AVMs than in those with supratentorial AVMs. Urgent endovascular treatment, most frequently by parent vessel occlusion, was effective in this small series.

Disclosure

The authors report no conflict of interest concerning the materials or methods used in this study or the findings specified in this paper.

Author contributions to the study and manuscript preparation include the following: Conception and design: Roy, Weill. Acquisition of data: Roy, Kouznetsov. Analysis and interpretation of data: Roy, Kouznetsov, Weill, Ghostine. Drafting the article: Roy, Gentric. Critically revising the article: Ghostine, Raymond. Administrative/technical/material support: Gentric.

References


This study was presented at the 51st Annual Meeting of the American Society of Neuroradiology (ASNR), May 20–23, 2013, in San Diego, California, and at the 40th Annual Congress of the Société Française de Neuroradiologie (SFNR), April 4–6, 2013, in Paris, France.

Please include this information when citing this paper: DOI: 10.3171/2014.6.FOCUS14219.

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