Spinal juvenile (Type III) extradural-intradural arteriovenous malformations

Clinical article

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Object. Owing to their rarity, demographics, natural history, and treatment, results for spinal juvenile (Type III) extradural-intradural arteriovenous malformations (AVMs) are frequently only provided in case report format.

Methods. A pooled analysis was performed utilizing the PubMed database through April 2013. Individualized patient data were extracted to elucidate demographics, hemorrhage risk, and treatment result information.

Results. Twenty-nine studies describing 51 patients were included. The mean age at presentation was 15.0 ± 10.5 years with a slight male predilection (63%, 1.7:1 sex ratio). Presentation modality included progressive deficits in 35%, hemorrhage in 31%, acute deficits not attributed to hemorrhage in 22%, and asymptomatic/incidental in 12% of patients. The annual hemorrhage rate was 2.1%; statistically significant risk factors for hemorrhage included presentation age (HR 0.39 [95% CI 0.18–0.87]) and associated aneurysms (HR 8.74 [95% CI 1.76–43.31]). Seventy-seven percent of patients underwent treatment; after a mean follow-up of 2.6 ± 3.2 years, 73% were improved, 10% were the same, and 17% were worse neurologically. Of 25 cases with described angiographic results, 8 lesions were obliterated (32%). Of these 25 patients, 8 had AVMs with associated aneurysms, and the aneurysm was obliterated in all 8 patients. Over the course of 57.9 patient-years of follow-up, including 55.3 patient-years for partially treated AVMs, no hemorrhages were described, reflecting a trend toward protection from hemorrhage after treatment (p = 0.12, likelihood ratio test).

Conclusions. Spinal juvenile (Type III) extradural-intradural AVMs commonly present symptomatically. Associated arterial aneurysms increase their hemorrhage risk, and protection from hemorrhage may be achieved from partial obliteration of these lesions, particularly if targeted toward associated aneurysms.

Key Words • spinal AVM • arteriovenous malformation • juvenile • metameric • extradural-intradural • natural history • surgery • embolization

Does targeted therapy, such as targeted obliteration of associated aneurysms, improve their natural history? In this report, we attempt to address these questions by compiling demographic information, hemorrhage risk data, and treatment results for these lesions.

Methods

Study Selection

The PubMed database was queried with the terms “arteriovenous malformation,” “avm,” “juvenile,” “metameric,” “cobb syndrome,” “spinal,” “cervical,” “thoracic,” and “lumbar” through April 1, 2013 (Table 1). No publication date or publication status restrictions were imposed. We incorporated all English-language studies that provided individualized data. To enhance the comprehensiveness of the review, references within studies were pursued and incorporated if they met our inclusion criteria. Re-
Spinal extradural-intradural AVMs

TABLE 1: Summary of incorporated studies*

<table>
<thead>
<tr>
<th>Authors &amp; Year</th>
<th>No. of Pts</th>
<th>Mean Age (yrs)†</th>
<th>M/F</th>
<th>Presentation</th>
<th>Level</th>
<th>Tx</th>
<th>Obliteration</th>
<th>Condition at Follow-Up After Tx</th>
</tr>
</thead>
<tbody>
<tr>
<td>Alomari et al., 2011</td>
<td>4</td>
<td>8</td>
<td>2:2</td>
<td>1 progressive ND, 1 acute ND, 1 asymptomatic</td>
<td>3 thoracic, 1 lumbar</td>
<td>2 combo</td>
<td>0/1</td>
<td>1 worse</td>
</tr>
<tr>
<td>Baraitser &amp; Shieff, 1990</td>
<td>1</td>
<td>13</td>
<td>1:0</td>
<td>hemorrhage</td>
<td>thoracic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Biondi et al., 1992**</td>
<td>5</td>
<td>17</td>
<td>3:2</td>
<td>5 hemorrhages</td>
<td>2 cervical, 3 thoracic</td>
<td>5 embo</td>
<td>1/5</td>
<td>5 improved</td>
</tr>
<tr>
<td>Cullen et al., 2006</td>
<td>2</td>
<td>3</td>
<td>2:0</td>
<td>1 acute ND, 1 asymptomatic</td>
<td>1 thoracic, 1 lumbar</td>
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</tr>
<tr>
<td>Clark et al., 2008</td>
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<td>8</td>
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<tr>
<td>Dilmé-Carreras et al., 2010</td>
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<td>12</td>
<td>1.0</td>
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<tr>
<td>Eldridge et al., 1989</td>
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<td>12</td>
<td>1:0</td>
<td>hemorrhage</td>
<td>lumbar</td>
<td>surgery</td>
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<td>same</td>
</tr>
<tr>
<td>Ferch et al., 2001</td>
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<td>21</td>
<td>1:0</td>
<td>progressive ND</td>
<td>thoracic</td>
<td>combo</td>
<td>0/1</td>
<td>worse</td>
</tr>
<tr>
<td>Johnson &amp; Petrie, 2009</td>
<td>1</td>
<td>24</td>
<td>1:0</td>
<td>acute ND</td>
<td>thoracic</td>
<td>embo</td>
<td>0/1</td>
<td>worse</td>
</tr>
<tr>
<td>Kalani et al., 2012</td>
<td>2</td>
<td>11</td>
<td>1:1</td>
<td>1 acute ND, 1 progressive ND</td>
<td>1 cervical, 1 thoracic</td>
<td>1 combo, 1 surgery</td>
<td>2/2</td>
<td>2 improved</td>
</tr>
<tr>
<td>Kähärä et al., 1997</td>
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<td>16</td>
<td>1:0</td>
<td>progressive ND</td>
<td>thoracic</td>
<td>embo</td>
<td>0/1</td>
<td>same</td>
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<tr>
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<td>0:1</td>
<td>acute ND</td>
<td>thoracic</td>
<td>combo</td>
<td>0/1</td>
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</tr>
<tr>
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<td>1</td>
<td>1:0</td>
<td>hemorrhage</td>
<td>lumbar</td>
<td>surgery</td>
<td>0/1</td>
<td>improved</td>
</tr>
<tr>
<td>Kerber et al., 1978</td>
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<td>47</td>
<td>0:1</td>
<td>acute ND</td>
<td>thoracic</td>
<td>combo</td>
<td>1/1</td>
<td>improved</td>
</tr>
<tr>
<td>Konan et al., 1999</td>
<td>2</td>
<td>18</td>
<td>1:1</td>
<td>2 hemorrhages</td>
<td>1 cervical, 1 thoracic</td>
<td>2 embo</td>
<td>0/2</td>
<td>2 improved</td>
</tr>
<tr>
<td>Linfante et al., 2012</td>
<td>1</td>
<td>9</td>
<td>0:1</td>
<td>progressive ND</td>
<td>thoracic</td>
<td>embo</td>
<td>1/1</td>
<td>improved</td>
</tr>
<tr>
<td>Martin et al., 1995</td>
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<td>15</td>
<td>1:0</td>
<td>progressive ND</td>
<td>thoracic</td>
<td>surgery</td>
<td>1/1</td>
<td>improved</td>
</tr>
<tr>
<td>Matsumaru et al., 1999</td>
<td>10</td>
<td>16</td>
<td>8:2</td>
<td>7 progressive NDs, 2 hemorrhages, 1 asymptomatic</td>
<td>2 cervical, 6 thoracic, 2 lumbar</td>
<td>6 embo</td>
<td>1/1</td>
<td>1 same, 1 worse</td>
</tr>
<tr>
<td>Matullo et al., 2007</td>
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<td>11</td>
<td>1:0</td>
<td>hemorrhage</td>
<td>thoracic</td>
<td>embo</td>
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<td>improved</td>
</tr>
<tr>
<td>Menkü et al., 2005</td>
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<td>1:0</td>
<td>progressive ND</td>
<td>cervical</td>
<td>combo</td>
<td>1/1</td>
<td>improved</td>
</tr>
<tr>
<td>Ommaya et al., 1969</td>
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<td>11</td>
<td>1:1</td>
<td>1 hemorrhage, 1 progressive ND</td>
<td>1 thoracic, 1 lumbar</td>
<td>1 embo</td>
<td>0/1</td>
<td>1 improved</td>
</tr>
<tr>
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<tr>
<td>Rudy &amp; Woodside, 1983</td>
<td>2</td>
<td>10</td>
<td>0:2</td>
<td>1 acute ND, 1 progressive ND</td>
<td>2 thoracic</td>
<td>1 combo, 1 surgery</td>
<td>2 improved</td>
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<td>Schirmer et al., 2012</td>
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<td>combo</td>
<td>0/1</td>
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<tr>
<td>Shim et al., 1996</td>
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<td>19</td>
<td>0:1</td>
<td>acute ND</td>
<td>thoracic</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Soeda et al., 2003</td>
<td>1</td>
<td>newborn</td>
<td>0:1</td>
<td>acute ND</td>
<td>thoracic</td>
<td>embo</td>
<td>0/1</td>
<td>improved</td>
</tr>
<tr>
<td>Spetzler et al., 1989</td>
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<td>32</td>
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<td>improved</td>
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<td>43</td>
<td>0:1</td>
<td>acute ND</td>
<td>thoracic</td>
<td>combo</td>
<td>1/1</td>
<td>improved</td>
</tr>
<tr>
<td>Wang et al., 2005</td>
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<td>18</td>
<td>2:0</td>
<td>2 acute NDs</td>
<td>2 thoracic</td>
<td></td>
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<td></td>
</tr>
</tbody>
</table>

* combo = combined surgery and endovascular treatment; embo = embolization; ND = neurological deficit; Pts = patients; Tx = treatment.
† Age at initial clinical presentation.

view articles and studies without individualized patient information, those not involving human subjects, or those evaluating only different spinal AVM subtypes were excluded. Patients described as having “juvenile” and/or “metameric” lesions that had only intramedullary glomus AVMs or arteriovenous fistulas (AVFs) were excluded.

Data Extraction

From all studies, we extracted individualized demographic, symptomatic, and AVM angioarchitectural data including patient age, sex, presenting neurological deficits, hemorrhage on presentation, AVM location, and associated arterial aneurysms. For each patient with at least 1 month of untreated follow-up, the time in years from original clinical presentation until treatment or last clinical follow-up was noted. Hemorrhages over this time period were noted. For treatment results, we extracted information detailing the surgical or endovascular approach, obliteration rates, and neurological condition at follow-up.
**Statistical Analysis**

Statistical analysis was performed using R version 2.11. Pooled rates were calculated for demographic information, annual hemorrhage rates, and results of intervention. Comparisons between pooled rates for treatment subgroups were performed using the Fisher exact test for categorical variables and with a 2-tailed t-test for mean follow-up periods. For our analysis of hemorrhage risk prior to treatment, AVM presence was assumed since birth. The Cox proportional hazards regression model was calculated using the survival package in R. Hemorrhage was used as the censoring event, and multiple events per subject were taken into account. To explain the variability in the primary outcome, we defined 4 a priori variables: age, sex, presence of an aneurysm, and location.

**Results**

After an initial screening of 358 studies, 125 full-text articles were assessed for eligibility, and 29 reports with 51 patients met inclusion criteria (Table 1). \(^1\)\(^-\)\(^4\),\(^9\),\(^10\),\(^12\)\(^-\)\(^14\),\(^20\)\(^-\)\(^25\),\(^29\),\(^32\)\(^-\)\(^37\),\(^39\),\(^41\)\(^-\)\(^43\),\(^45\),\(^48\),\(^50\) For these 51 patients, the mean age at presentation was 15.0 ± 10.5 years (± SD), and there was a slight male sex predilection (63% male, 1.7:1 male/female ratio) (Table 2). Presentation modalities included progressive neurological deficits in 35% of cases, acute hemorrhage in 31%, an acute neurological deficit without hemorrhage in 22% of cases, and incidental discovery in the remaining 12%. Arteriovenous malformations were most commonly thoracic (63%); 20% were cervical and 18% were lumbar. Across 23 cases where angiarchitectural description was provided, associated arterial aneurysms, 48% of AVMs had at least one associated aneurysm.

**Hemorrhage Risk**

Assuming AVM presence since birth, a total of 825.7 patient-years until treatment or last clinical follow-up were tallied for the 51 patients analyzed. Over this period, 17 hemorrhages occurred, corresponding to an annual rate of 2.1%. Hazard ratios for hemorrhage risk factors after univariate and multivariate Cox proportional hazards regression are summarized in Table 3. After univariate analysis, lumbar/conus location (HR 3.66 [95% CI 1.19–11.22], \(p = 0.02\)) and associated aneurysms (HR 8.09 [95% CI 1.73–37.82], \(p = 0.008\)) were significant risk factors for hemorrhage while increasing age at presentation was associated with a lower risk of hemorrhage (HR 0.82 [95% CI 0.74–0.91], \(p = 0.0002\)). After multivariate analysis, associated aneurysms remained a significant risk factor for hemorrhage (HR 8.74 [95% CI 1.76–43.31], \(p = 0.02\); Fig. 1) while increasing age at presentation remained associated with a lower risk of hemorrhage (HR 0.39 [95% CI 0.18–0.87], \(p = 0.02\)).

**Treatment Results**

Treatment information was provided for 45 patients. Ten patients did not undergo surgery or embolization (22%). Twenty underwent embolization (44%), 11 underwent embolization followed by resection (24%), and 4 underwent resection alone (9%). Treatment results for those undergoing surgery with or without embolization and those undergoing embolization alone are provided in Table 4. Angiographic obliteration after treatment was described in 25 cases overall; in 8, complete obliteration was achieved (32%). Rates of obliteration did not significantly differ between treatment modalities, although angiographic results at follow-up were rarely addressed. Of note, of 8 cases with associated aneurysms described angiographic results, the aneurysm was obliterated endovascularly in all cases. The overall mean follow-up was 2.6 ± 3.2 years, corresponding to a total of 57.9 patient-years, including 55.3 patient-years of follow-up for partially treated lesions. Over this time period, no cases of hemorrhage were described, reflecting a trend toward protection from hemorrhage after treatment (\(p = 0.12\), likelihood ratio test). Neurological condition at follow-up was described for 30 patients—22 were improved (73%), 3 were the same (10%), and 5 were worse (17%). This did not significantly differ between treatment modalities.

**Discussion**

Although a modern perception of spinal “juvenile metameric” AVMs is that of a formidable extradural-intradural lesion,\(^27\),\(^44\) the terms “juvenile” and/or “metameric” historically encompass a broader subset of spinal vascular malformations. Juvenile spinal vascular arteriovenous shunts have also referred to intramedullary AVMs in some reports.\(^11\),\(^34\) Some of the earliest descriptions of spinal AVMs likely referred to diffuse intramedullary spinal AVMs as the juvenile or “Type III” lesion in contrast to more compact lesions referred to as “Type II.”\(^16\) Most now consider both compact and diffuse intramedullary AVMs as Type II lesions or as, in the Spetzler classification, in the group of intramedullary AVMs.\(^27\),\(^44\) These lesions group well together angiarchitecturally in contrast to formidable extradural-intradural lesions that are better considered as a separate Type III subgroup (Fig. 2).
Spinal extradural-intradural AVMs

The Type III distinction should not be based on a patient possessing a spinal metanemic/Cobb syndrome, as any spinal arteriovenous shunt5,31,38,51 or cavernous malformation9,10 may serve as the spinal component, not necessarily an extradural-intradural AVM. We thus do not use the term “juvenile metanemic AVM” to refer to these lesions but rather “juvenile extradural-intradural” to more specifically describe these lesions.

Most classification schemes consider 4 spinal dural/intradural arteriovenous shunts: dural AVFs (Type I), glomus AVMs (Type II), juvenile extradural-intradural AVMs (Type III), and pial AVFs (Type IV).5,16–18,49 A comparative analysis of these spinal arteriovenous shunts is provided in Table 5. Our study confirms that patients with extradural-intradural AVMs present at the youngest age. We also now demonstrate a slight male sex predilection for these lesions, somewhat akin to Type I and Type IV lesions17,40,47 but an interesting contrast to glomus AVMs where no sex predilection is seen.66 Angioarchitecturally, Type III AVMs seem to have the greatest prevalence of associated aneurysms. Aside from Type I lesions that essentially do not present a significant risk of hemorrhage,40,47 annual hemorrhage rates for the other spinal arteriovenous shunt subtypes are generally similar.10,17 The similarity of hemorrhage rates among spinal AVMs draws analogy to the modern cerebral AVM maxim that AVM size does not impact hemorrhage rate.15,52

Our method of assuming AVM presence since birth is a contrast to the other analyses that used lesion follow-up after presentation.15–17,26,46,52 Although our assumption of AVM presence since birth may ostensibly deflate the observed hemorrhage rate, a recent analysis of cerebral AVMs has provided some validation for this approach.26 In this report, it allowed us to additionally evaluate risk factors for hemorrhage, revealing arterial aneurysms and younger age to be associated with a higher hemorrhage risk, similar to glomus AVMs.66 Given that aneurysms are likely acquired, dynamic components of the AVM, our assumption of their static presence since birth is conservative; the fact that they nonetheless remain a significant risk factor for hemorrhage further emphasizes the importance of identifying them and their significant impact on hemorrhage risk.

Not surprisingly, obliteration rates are lowest for extradural-intradural lesions. In fact, our observed obliteration rates are likely an overestimate as we included case reports that may inflate this rate as a result of publication bias of only successful cases. Furthermore, likely in part due to the natural history of partially treated AVMs, worsening at follow-up was seen most commonly among extradural-intradural lesions. The impact of partial treatment of AVMs on outcome thus becomes a particularly important issue to address when considering these lesions. Recent data have suggested a potential benefit on hemorrhage risk for glomus AVMs,66 and this study shows a trend toward protection from hemorrhage after treatment of extradural-intradural AVMs as well (p = 0.12). Given that aneurysms are a known risk factor for hemorrhage and 8 of 8 described aneurysms were obliterated in our reviewed cases, one may postulate that the observed protection from hemorrhage after partial treatment may be in part from securing arterial aneurysms. Thus, although strong treatment recommendations cannot be derived from this report, it does provide preliminary evidence supporting partial treatment aimed at least at securing associated arterial aneurysms to mitigate hemorrhage risk.

Limitations of This Study

Our study encompasses a small, heterogeneous cohort of spinal juvenile (Type III) extradural-intradural AVMs. As a result of extradural-intradural AVM rarity, our pooled results primarily comprise case reports, introducing publication bias of successful or intriguing cases. As mentioned earlier, this likely inflates the already low obliteration rates for endovascular and surgical management; however, treatment goals were frequently not specified in our reviewed studies, limiting our analysis of procedural “success” rates. Unfortunately, as a result

TABLE 3: Hazard ratios for hemorrhage risk factors after univariate and multivariate Cox proportional hazards regression

<table>
<thead>
<tr>
<th>Risk Factor</th>
<th>Univariate HR (95% CI)</th>
<th>p Value</th>
<th>Multivariate HR (95% CI)</th>
<th>p Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>presentation age</td>
<td>0.82 (0.74–0.91)</td>
<td>0.0002</td>
<td>0.39 (0.18–0.87)</td>
<td>0.02</td>
</tr>
<tr>
<td>female sex</td>
<td>1.07 (0.40–2.85)</td>
<td>0.90</td>
<td>1.26 (0.19–8.36)</td>
<td>0.81</td>
</tr>
<tr>
<td>lumbar/conus</td>
<td>3.66 (1.19–11.22)</td>
<td>0.02</td>
<td>1.60 (0.17–14.7)</td>
<td>0.62</td>
</tr>
<tr>
<td>associated aneurysm</td>
<td>8.09 (1.73–37.82)</td>
<td>0.008</td>
<td>8.74 (1.76–43.31)</td>
<td>0.02</td>
</tr>
</tbody>
</table>

FIG. 1. Kaplan-Meier curve of hemorrhage-free survival for AVMs with versus those without associated arterial aneurysms.
of a lack of detailed information, a relatively large series of 15 patients with reportedly “metameric” AVMs was excluded.5

This study largely focuses on the risk of hemorrhage from these lesions, which is only one of several sources of neurological morbidity from these AVMs. Mass effect from growing lesions (or residual lesions after partial treatment) or symptoms from venous hypertension requires better attention in a comprehensive analysis of the natural history of these lesions prior to and after treatment. Although we illustrate similar hemorrhage rates for these lesions that draw analogy to glomus AVMs,6 these larger, more formidable lesions may pose a greater risk of symptomatic deterioration as a result of mass effect or venous hypertension. Such deterioration may be as debilitating as a hemorrhagic event and thus requires better attention in future studies and over a longer period of follow-up. A specific question to address is the feasibility and durability of partial treatment aimed at alleviating venous hypertension.

Importantly, our study could not accumulate ample long-term angiographic or clinical follow-up in patients. This is particularly crucial in the context of the high proportion of partially treated lesions and the young age of patients with these AVMs. Only 3 patients in our analysis had more than 5 years of follow-up,5,20,39 and of them, the condition in one deteriorated neurologically without hemorrhage 9 years after treatment.20 Thus, the true impact of treatment on outcome remains to be addressed, and must be considered in the context of not only protection from hemorrhage, but also from durability of alleviated venous hypertension.

Conclusions

Spinal juvenile (Type III) extradural-intradural AVMs affect young patients with a slight male sex predilection. Approximately one-third present with progressive neurological deterioration, another one-third with hemorrhage, approximately one-fifth with acute deficits not attributable to hemorrhage, and the remaining one-eighth with incidental lesions. Interestingly, nearly half have associated arterial aneurysms. Assuming AVM presence since birth, the annual hemorrhage rate for these lesions was 2.1% with age at presentation (HR 0.39 [95% CI 0.18–0.87]) and associated aneurysms (HR 8.74 [95% CI 1.76–43.31]) serving as significant risk factors for hemorrhage (p = 0.02 for each after multivariate analysis). Complete obliteration after treatment was infrequent, although arterial aneurysms were secured in 8 of 8 cases described. After treatment, 17% of patients were neurologically worse after a mean follow-up of 2.6 years. Interestingly, no cases of hemorrhage were described over 57.9 patient-years of follow-up after treatment, perhaps in part due to the beneficial effect

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**TABLE 4: Treatment results**

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Surgery*</th>
<th>Embolization†</th>
<th>p Value</th>
<th>Total</th>
</tr>
</thead>
<tbody>
<tr>
<td>no. of patients</td>
<td>15/35 (43%)</td>
<td>20/35 (57%)</td>
<td>0.34</td>
<td>35</td>
</tr>
<tr>
<td>mean follow-up in yrs (± SD)</td>
<td>2.2 ± 3.6</td>
<td>2.9 ± 3.0</td>
<td>0.59</td>
<td>2.6 ± 3.2</td>
</tr>
<tr>
<td>obliteration</td>
<td>4/11 (36%)</td>
<td>4/14 (29%)</td>
<td>1.0</td>
<td>8/25 (32%)</td>
</tr>
<tr>
<td>condition at follow-up</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>improved</td>
<td>10/13 (77%)</td>
<td>12/17 (71%)</td>
<td>1.0</td>
<td>22/30 (73%)</td>
</tr>
<tr>
<td>same</td>
<td>1/13 (8%)</td>
<td>2/17 (12%)</td>
<td>1.0</td>
<td>3/30 (10%)</td>
</tr>
<tr>
<td>worse</td>
<td>2/13 (15%)</td>
<td>3/17 (18%)</td>
<td>1.0</td>
<td>5/30 (17%)</td>
</tr>
</tbody>
</table>

* Surgery with or without embolization.
† Embolization only.

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Fig. 2. Representative spinal extradural-intradural AVM. This right T-8 segmental artery injection demonstrates supply to an extradural-intradural AVM. The *arrowhead* denotes the extradural component, and the *arrow* denotes the intradural component. Adapted from Linfante et al.: J Neurosurg Spine 16:408–413, 2012, with permission.
of securing associated arterial aneurysms. Although strong recommendations in favor of treatment cannot be made based on our data, the data may support partial treatment aimed at least at securing associated arterial aneurysms, a pertinent finding given their prevalent association with this spinal AVM subtype.

**Disclosure**

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

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**References**

22. Kalani MY, Ahmed AS, Martirosyan NL, Cronk K, Moon K,


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