Aneurysms of Spinal Arteries Associated with Intramedullary Arteriovenous Malformations. II. Results of AVM Endovascular Treatment and Hemodynamic Considerations

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Purpose: To evaluate the changes occurring in spinal aneurysm (SA) size related to modification of endovascularly treated AVMs. **Methods:** Fourteen patients with an intramedullary AVM and associated SA underwent endovascular treatment of their AVM with particles. Embolization sessions numbered from one to 14 (mean five) in each patient. **Results:** Four patients had SAs with size changes mirroring those of the AVM with embolization: these decreased in size or disappeared after AVM reduction or cure and increased or recurred after AVM recanalization. A second group of patients had SAs that remained unchanged despite AVM changes (six of seven of these were in patients with metameric angiomatosis). **Conclusions:** Results in the first group lend support to the hemodynamic theory of associated aneurysm formation. On the other hand, aneurysms that remained unchanged probably are not AVM flow-related and could be an expression of an extensive vascular disorder such as metameric angiomatosis; however, hemodynamic and developmental factors could be concurrent.

Index terms: Arteriovenous malformations, spinal; Spine, angiography; Aneurysm, arteriovenous; Aneurysm, therapeutic blockade; Fistula, arteriovenous; Fistula, therapeutic blockade; Interventional materials, particles and microspheres

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The association of aneurysms and arteriovenous malformations (AVMs) in the spinal cord, as in the brain, raises questions regarding their genesis and treatment. Cerebral aneurysms associated with AVMs have been separated into AVM flow-dependent aneurysms (located on AVM feeders and with unusual topography) and remote or dysplastic aneurysms (located on arteries remote and hemodynamically unrelated to the AVM and with the usual distribution pattern of isolated cerebral aneurysms) (1, 2) (see part I). There have been reports of disappearance of a flow-dependent cerebral aneurysm after surgical removal (3–8) or endovascular treatment (1, 2, 6) of the associated AVM. These results tend to confirm the hypothesis that high flow to the AVM is the operative factor in the development of an associated aneurysm. In the spinal cord, except for a single report (9), the modification of spinal aneurysm (SA) size after treatment of the associated AVM has not been reported.

The purpose of this study was to evaluate the changes occurring in SA size relative to reduction and recurrence of the endovascularly treated AVM. In our series, all intramedullary AVMs were embolized with particles, which caused a temporary occlusion of the lesion (10). This appeared to be an excellent subset of patients with which to evaluate the hemodynamic theory of associated aneurysm formation.

Materials and Methods

Fourteen patients with an intramedullary AVM and an associated SA underwent embolization with particles in order to treat the intramedullary AVM. There were no

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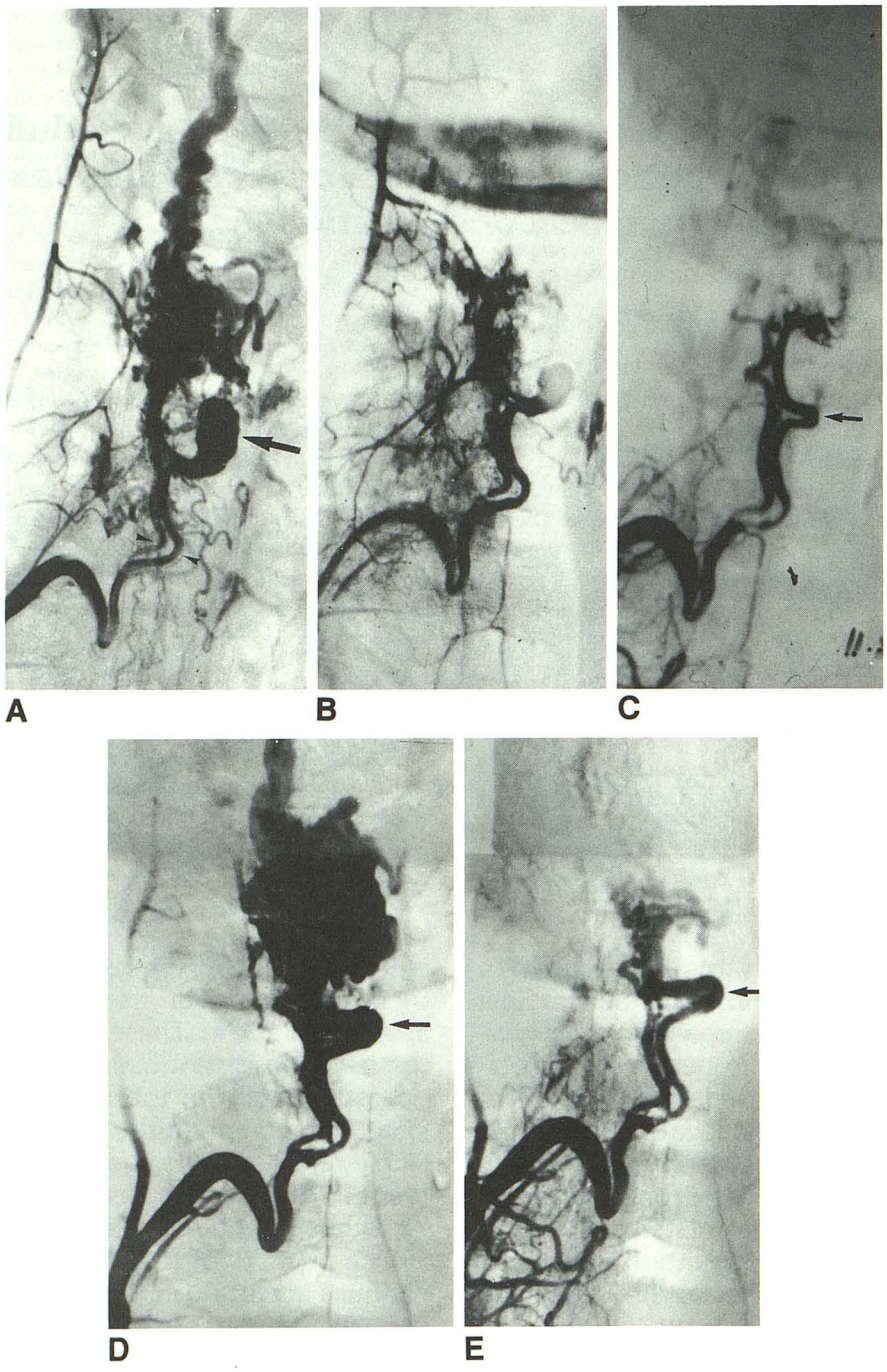


Fig. 1. Case 1.

A, Aneurysm of the anterior spinal artery (ASA) (*arrow*) associated with a cervical intramedullary AVM. Anteroposterior view of the right, deep cervical artery angiogram. Note the presence of a double ASA (*arrowheads*).

B, During first embolization, dura mater emboli are recognizable in the aneurysm sac.

C, Angiographic control 3 months after embolization shows persistent decrease in the size of the AVM and disappearance of the aneurysm (*arrow*).

D, Angiography 5 years after the start of endovascular therapy and before the fifth embolization shows complete recanalization of the AVM and partial reappearance of the aneurysm (*arrow*).

E, Angiographic control after the fifth embolization. AVM as well as the aneurysm (*arrow*) reduced in size.

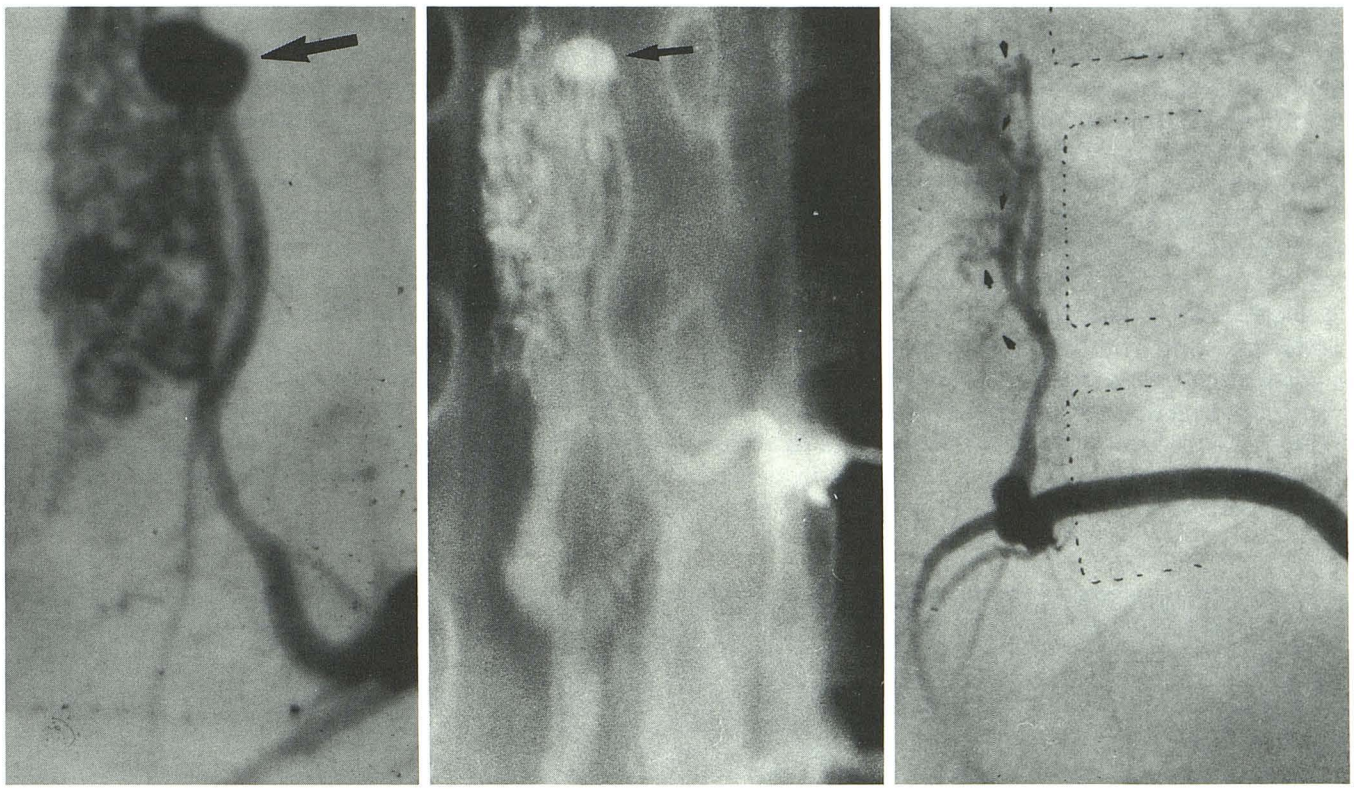


Fig. 2. Case 10; aneurysm of the radiculo-medullary junction of the anterior spinal artery (ASA) (*arrow*) associated with a thoracic intramedullary AVM. Anteroposterior (*A*), anteroposterior angiotomogram (*B*), and lateral (*C*) view of the left ninth intercostal artery angiogram. The large sulco-commissural arteries are well-visualized (*arrowheads*).

specific treatments for the SAs. We retrospectively reviewed all spinal arteriograms of these patients to evaluate the SA size changes in relation to AVM modification after endovascular treatment. One patient (case 8) had two SAs; consequently, fifteen SAs were studied. Except in one case, all SAs were located on a pedicle feeding the AVM. The only SA remote from the associated intramedullary AVM (case 14) arose from the intercostal (dural radiculo-medullary) artery feeding a vertebral angioma (the patient had metameric angiomatosis: intramedullary AVM, dural angiomatosis, vertebral angioma).

Clinical and angiographic findings are reported in part I.

Regarding intramedullary AVM embolization, various particulate emboli including dura mater, polyvinyl alcohol, and gelatin foam with or without alcohol were used. The size of the particulate embolic agent was based upon the angioarchitecture of the AVM. In general, no matter what embolic agent was used, the AVM tended to recanalize. The number of embolization sessions for each patient ranged from one to 14 (mean five). Follow-up from initial treatment was 1 to 15 years (mean 6) in 11 patients and less than 1 year in three patients. Angiographic investigations were repeated annually, even without clinical aggravation, to evaluate the evolution of the AVM nidus, and reembolization was performed if necessary. Previously, we have reported in detail our long-term results and technical considerations in the endovascular treatment with particles of intramedullary AVMs (10).

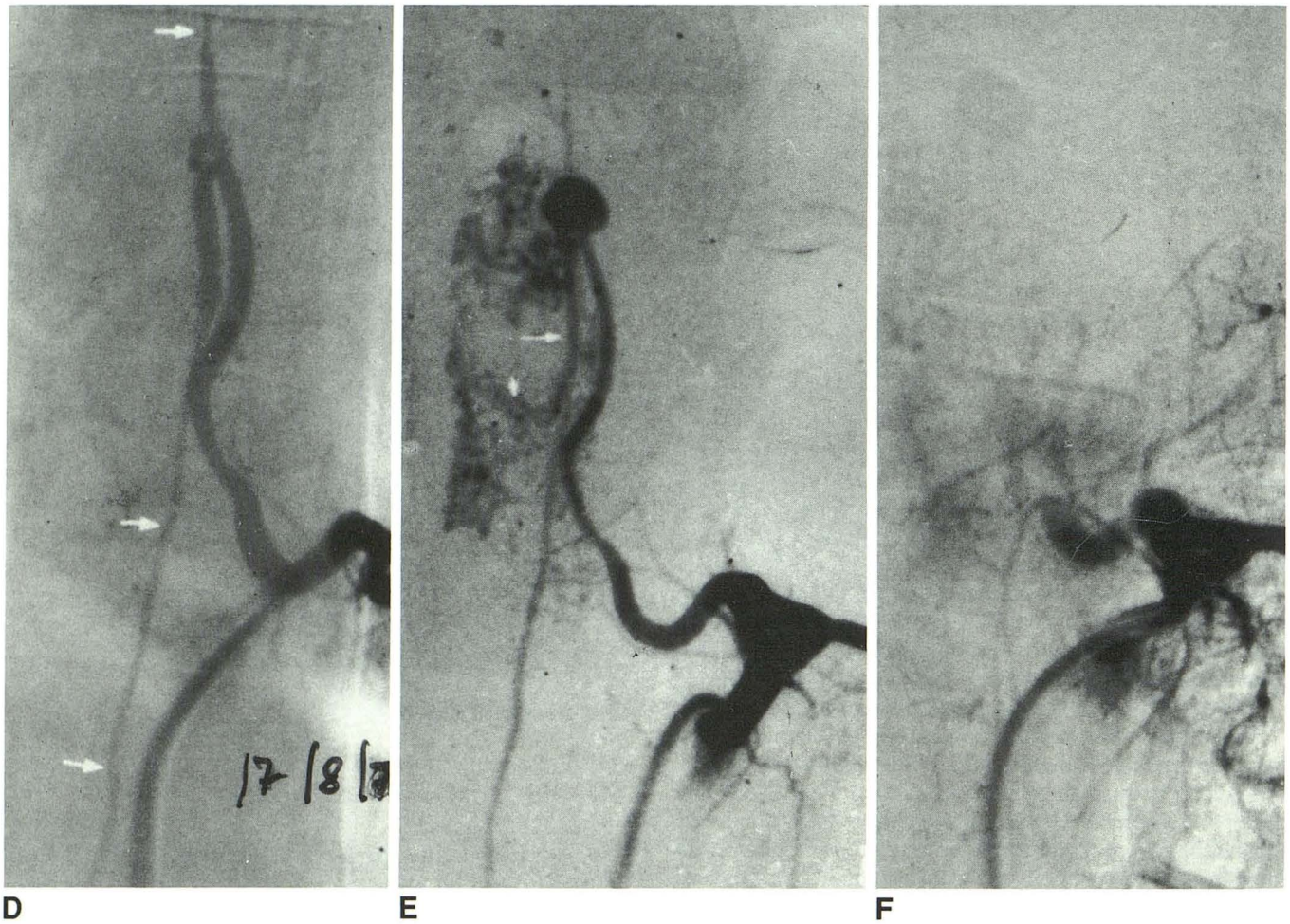
Results

Fourteen intramedullary AVMs were embolized with particles. After the first embolization, 13 out of 14 AVMs were reduced in size or cured (five mild, four moderate, two almost complete AVM reduction, and two AVM disappearance) and one AVM was unchanged. In the only patient (case 14) with the SA remote from the AVM, the vertebral angioma, which can be considered hemodynamically analogous to the AVM, was also reduced in size. In one patient (case 11), the SA appeared during the follow-up 2 years after the start of endovascular therapy. In this case, we considered as the first embolization the session of endovascular therapy in which the SA was identified. In most of the cases, follow-up angiography showed partial or complete AVM

recurrence requiring further embolization. Although the patients underwent multiple embolization sessions, the results after the last embolization were compared to those obtained after the first embolization. Ten AVMs were recanalized (six partial and four complete recurrences), three AVMs showed the same reduction obtained after the first embolization, and one AVM was further reduced. At last angiographic control, 10 patients showed clinical improvement (four mild, five moderate, one marked), one patient remained asymptomatic, and one patient worsened. Clinical follow-up was unavailable from two patients.

The evaluation of the 15 associated SAs showed:

After the first embolization session, no SA modifications were detected in the single case (case 2) in which the AVM remained unchanged; in the 13 AVMs in which endovascular treatment caused AVM reduction or cure, we observed disappearance of the SA in two cases (cases 1 and 10), occlusion of the parent vessel of the SA in two cases (cases 5 and 8b), and reduction of the SA size in two cases (cases 7 and 13). The SA size was unchanged in eight cases (cases 3, 4, 6, 8, 9, 11, 12, and 14). In the patient with two associated SAs (case 8), after reduction of the AVM, one SA remained unchanged, and occlusion of the parent vessel of the other SA occurred. No modifications were detected in the SA size



D E F

Fig. 2.—*Continued. D*, After the first embolization, figure shows complete disappearance of both lesions and preservation of the ASA (*white arrows*).

E, Anteroposterior angiographic follow-up 6 months later. Partial recanalization of the lesions (ASA, *white arrow*; sulco-commissural artery, *white arrowhead*).

F, The next embolization led to the occlusion of the radicular segment of the ASA at T9. No neurologic deficit was detected due to collateral revascularization of the ASA (and AVM) from the left fifth intercostal artery.

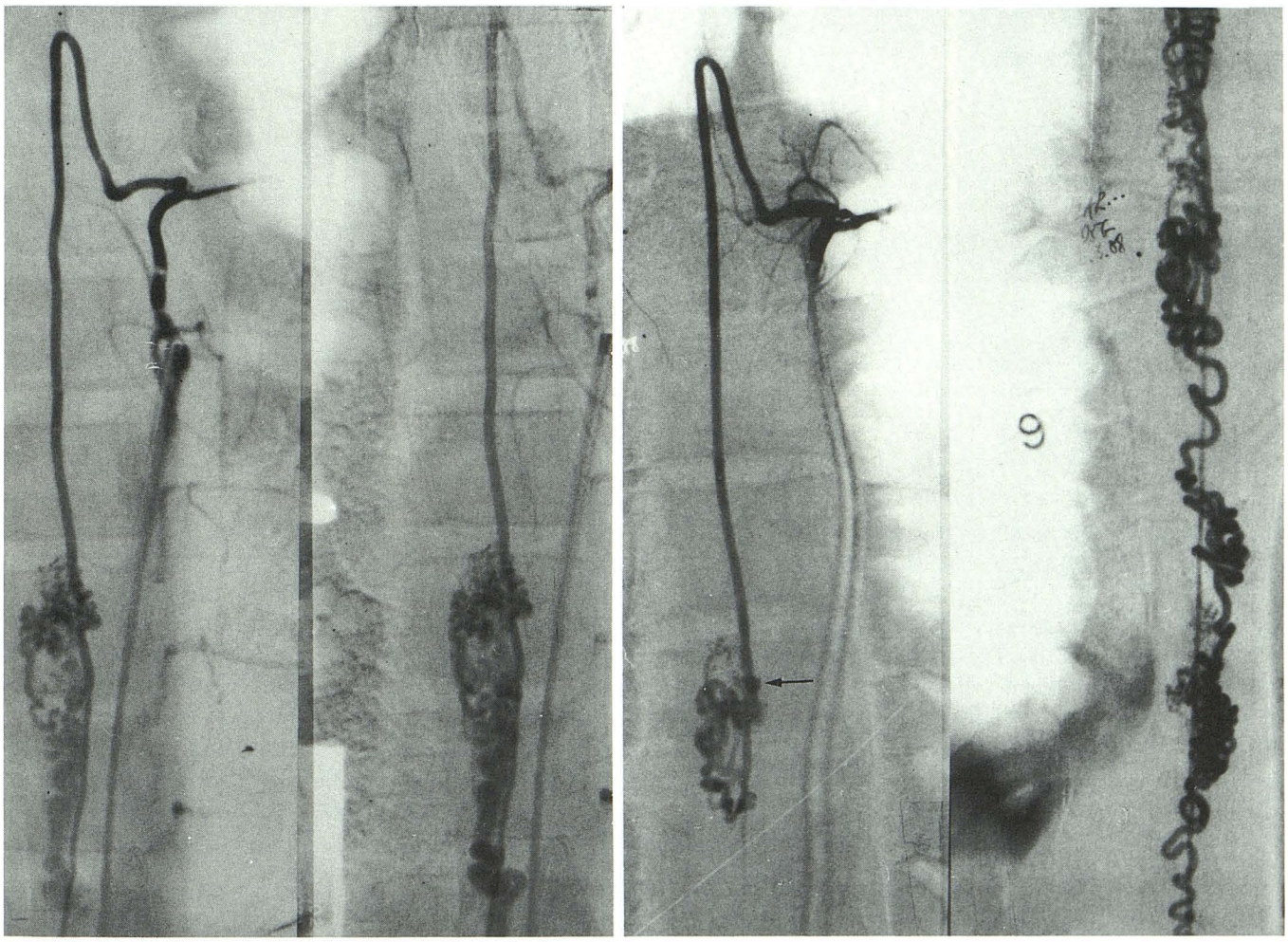
G, Early and late phase anteroposterior views of left fifth intercostal angiogram (1 year after *F*). No aneurysm is seen.

(case 14) located on the intercostal artery feeding a vertebral angioma, despite reduction in both the vertebral angioma and the AVM.

At intermediate follow-up, in three cases (cases 2, 5, and 8b) occlusion of the SA parent vessel during AVM embolization did not permit the future evaluation of aneurysmal size changes. Four SAs (cases 1, 7, 10, and 13) "followed" the AVM changes: they reduced or disappeared after AVM reduction or cure and they increases in size with AVM recurrence (Figs. 1–3). One SA (case 11), which appeared 2 years after initial angiography, remained unchanged during follow-up despite AVM changes. Seven SAs (cases 3, 4, 6, 8, 9, 12, and 14) remained unchanged after AVM reduction or recurrence.

In two patients (cases 1 and 6), embolic fragments were recognizable within the SA during one of multiple embolization sessions. In both cases, the aneurysm decreased in size. In one case (case 1), shrinkage of the SA, when no embolic fragments were detectable in the aneurysmal sac, was still apparent on follow-up angiography after reduction of the intramedullary AVM. In the other case (case 6), the aneurysm had remained unchanged despite AVM reduction from previous endovascular treatment. The aneurysm itself decreased in size only after fragments of embolic material were noted in the aneurysmal sac itself. We have no further follow-up for this patient (Fig. 4).

At final follow-up after last embolization, four



G H

Fig. 2.—*Continued. H*, Early and late phase angiogram of the same artery as in (*G*) 7 years later, after four further embolizations. A small aneurysmal dilatation at the same site as the original aneurysm (*arrow*) and recurrence of the AVM is seen.

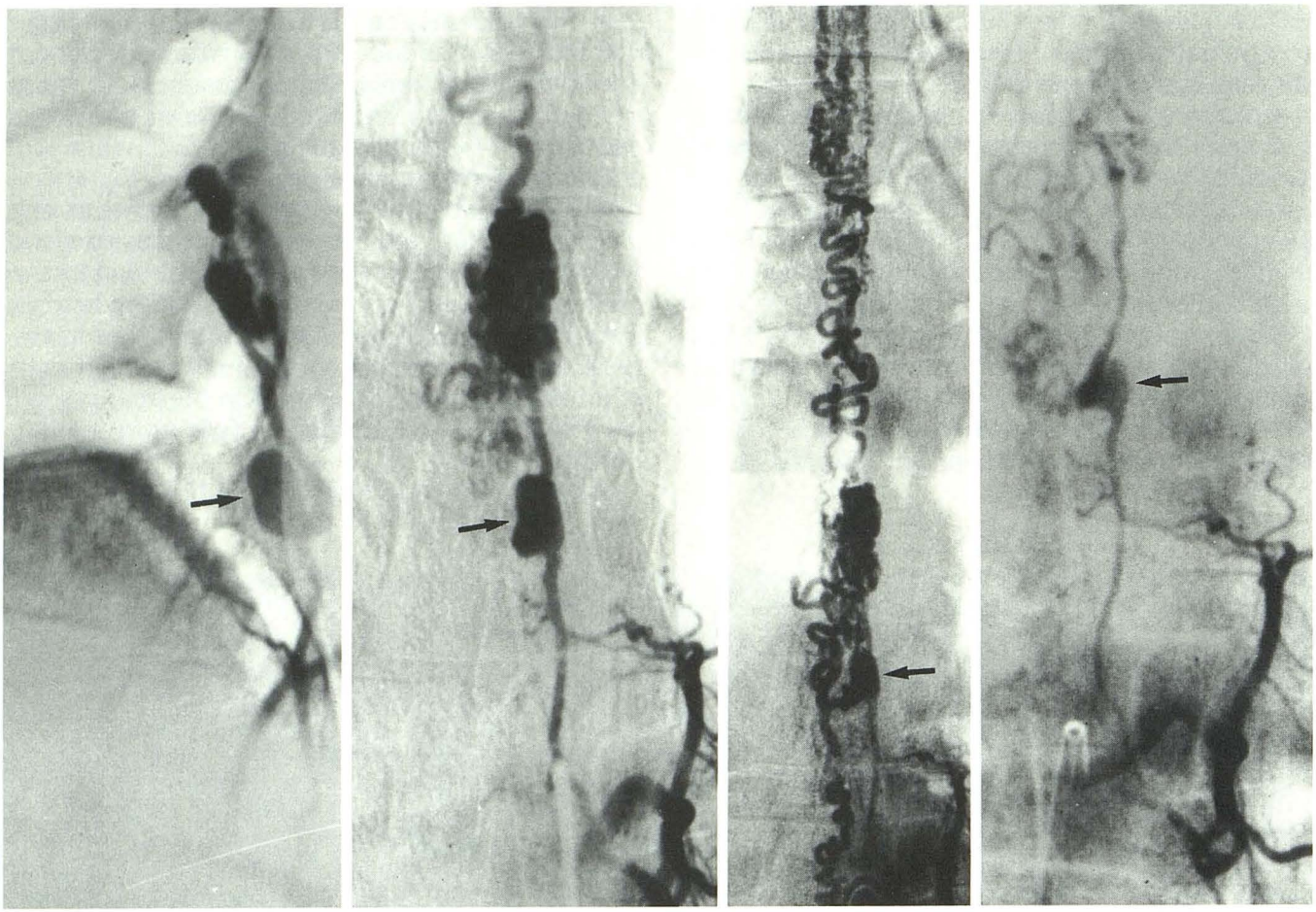


Fig. 3. Case 13; aneurysm of the posterior spinal artery (*arrows*) associated with a thoracic intramedullary AVM. Lateral (*A*), early anteroposterior (*B*), late anteroposterior (*C*), views of the left first lumbar artery angiogram. (*D*), Angiographic control after endovascular treatment of the AVM shows marked reduction in the size of both lesions.

out of 15 SAs were not visualized because of parent vessel occlusion (cases 2, 5, 8b, and 9) and 11 SAs were persistent. In all four nonvisualized cases, the SA parent vessel occlusion was incidental to the embolization of the AVM. Compared with initial appearance, six SAs (cases 3, 4, 8, 11, 12, and 14) were unchanged. The associated AVMs were completely recanalized in three cases (cases 3, 8, and 11) and reduced in the other three cases (cases 4, 12, and 14). These later three patients had metameric angiomatosis. Five SAs (cases 1, 6, 7, 10, and 13) were reduced along with their associated AVMs. The angiographic results are summarized in Tables 1 and 2.

Discussion

The size changes of an associated aneurysm after treatment of an AVM in the brain (1–8) or in the spinal cord (9) gives convincing support for the hemodynamic theory of the genesis of associated aneurysmal lesions. In our series of associated SA, there is evidence that a group of SAs diminished or disappeared after AVM endovascular treatment and increased or recurred after AVM recanalization, and that a second group of SAs remained unchanged despite AVM changes. This later group of seven unchanged SAs included six of the seven SAs found in patients with metameric angiomatosis (one SA in a patient with metameric angiomatosis could not be studied because of early occlusion of its parent vessel).

Cerebral aneurysms associated with an AVM have been found on AVM feeders (flow-dependent aneurysms) and on arteries remote and hemodynamically unrelated to the AVM (dysplastic aneurysms) (1, 2). Unlike cerebral aneurysms, no

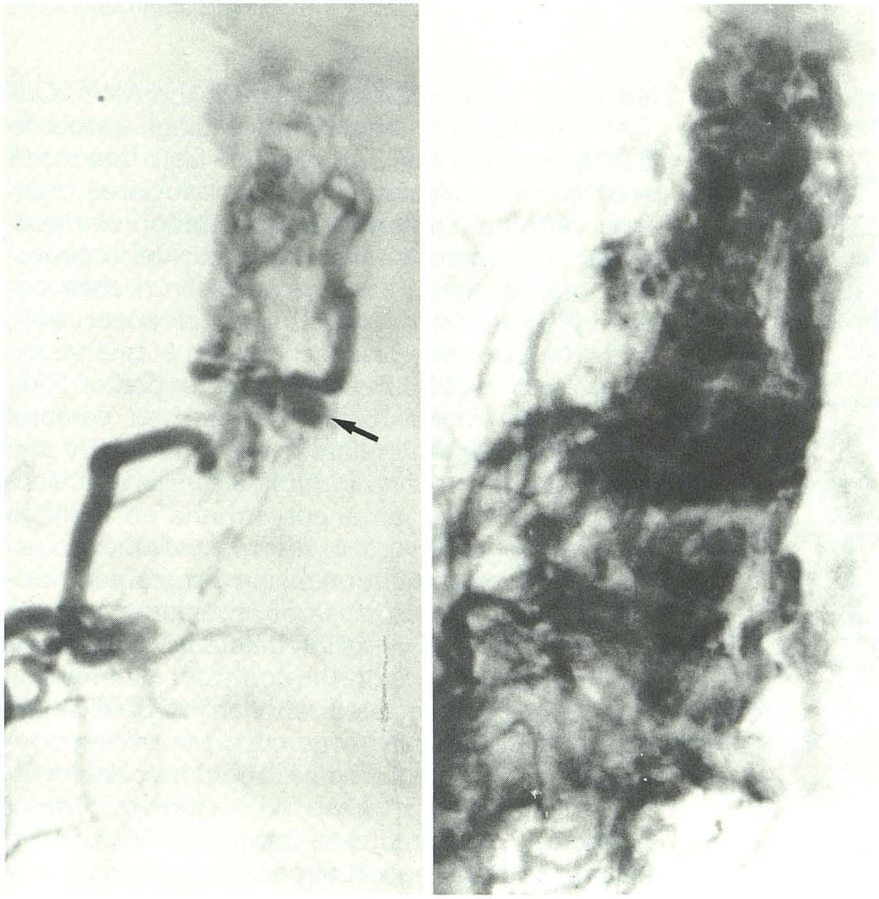


Fig. 4. Case 6; aneurysm of the anterior spinal artery (*arrow*) associated with a cervical intramedullary AVM. Anteroposterior view early (*A*) and late (*B*) phase of right deep cervical artery angiogram.

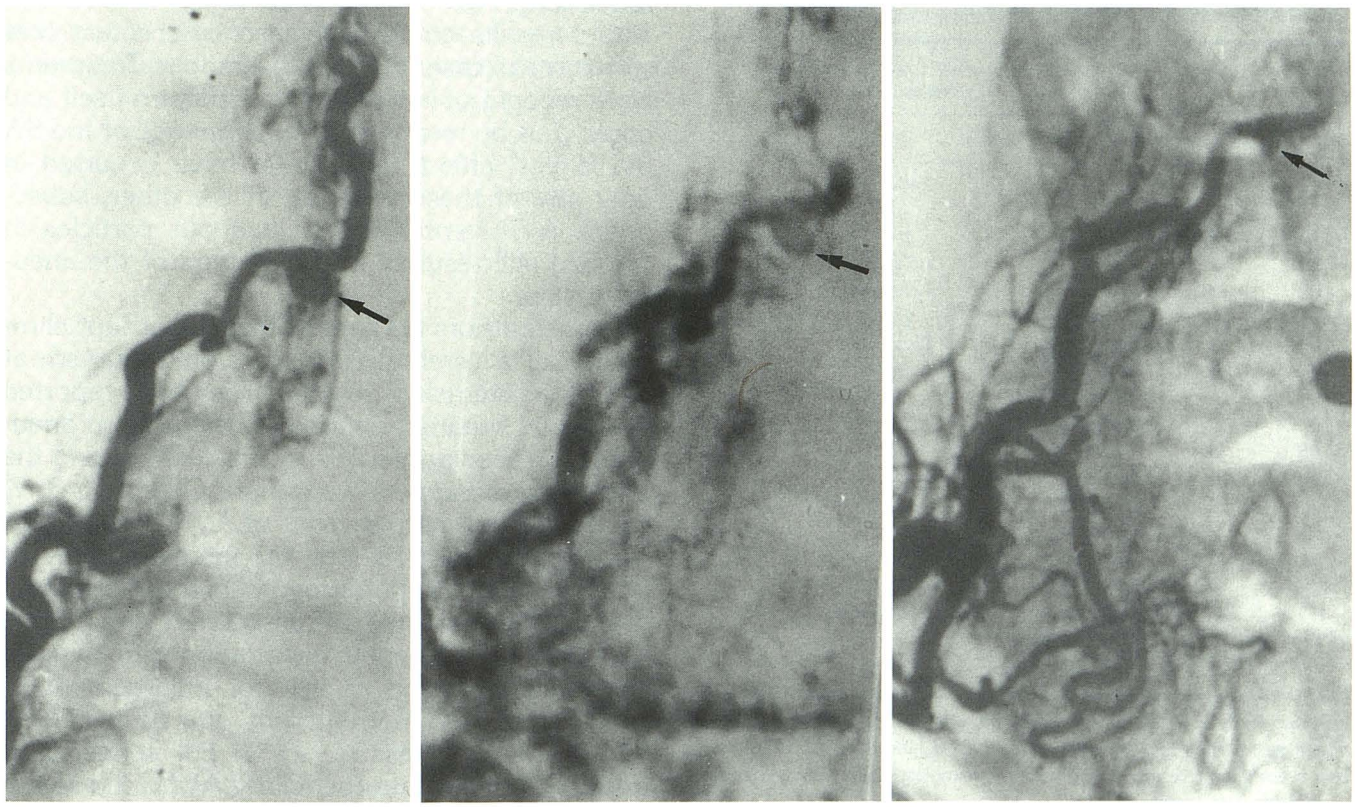


Fig. 4.—*Continued.* *C*, One month after embolization, the aneurysm (*arrow*) is unchanged despite the significant reduction of the associated AVM. *D*, Selective placement of embolic fragments into the aneurysm sac (*arrow*) caused a reduction of the aneurysm (*arrow*) (*E*) which had remained unchanged after previous embolization sessions.

TABLE 1: Spinal artery aneurysms (SAs) associated with intramedullary AVM: results after the first embolization of the AVM and SA size changes

Case	AVM	SA
1	↓↓↓	Cure
2	Unchanged	Unchanged
3	↓	Unchanged
4[a]	Cure	Unchanged
5	↓	pvo
6[a]	↓↓	Unchanged
7	↓	Reduced
8[a]	↓↓	Unchanged
8b[a]	↓↓	pvo
9[a]	↓↓↓	Unchanged
10	Cure	Cure
11	↓	Unchanged
12[a]	↓	Unchanged
13	↓↓	Reduced
14[a]	↓↓[b]	Unchanged

Note.—↓, ↓↓, ↓↓↓ = mild, moderate, significant AVM reduction; pvo = SA parent vessel occlusion.

[a] Metameric angiomatosis.

[b] Same reduction observed in the associated vertebral angioma (see text).

TABLE 2: Spinal artery aneurysms (SAs) associated with intramedullary AVM: SA size changes during follow-up (after AVM embolization sessions) and results after the last embolization

Case	No. of Emboliza-tions	SA during Follow-up	After Last Embolization		Duration of Treatment (years)
			AVM	SA	
1	6	"followed" AVM changes	↓↓	Reduced	5
2	2	pvo	↓	pvo	<1
3	2	Unchanged	rec	Unchanged	6
4[a]	3	Unchanged	↓↓	Unchanged	3
5	1	pvo	rec	pvo	15
6[a]	5	Unchanged	↓↓	Reduced	4
7	2	"followed" AVM changes	↓	Reduced	<1
8[a]	5	Unchanged	rec	Unchanged	3
8b[a]	5	pvo	rec	pvo	3
9[a]	10	Unchanged	↓↓	pvo	6
10	6	"followed" AVM changes	↓	Reduced	8
11	6	Unchanged	rec	Unchanged	6
12[a]	4	Unchanged	↓	Unchanged	<1
13	4	"followed" AVM changes	↓	Reduced	2
14[a]	14	Unchanged	↓[b]	Unchanged	8

Note.—AVM reduction is compared with AVM size at the initial condition. ↓, ↓↓, ↓↓↓ = mild, moderate, significant AVM reduction; pvo = SA parent vessel occlusion; rec = complete AVM recanalization.

[a] Metameric angiomatosis.

[b] Same reduction observed in the associated vertebral angioma (see text).

SA in our series was remote from the AVM. Our SAs were always located on feeding vessels to the AVM (the only SA "remote" from the AVM was found in a patient with an associated high-flow vertebral angioma—manifestation of metameric angiomatosis). If in the brain the topography of associated aneurysms distinguishes between flow-related and congenital aneurysms, then the presence or absence of metameric angiomatosis could differentiate two types of SAs. One type, similar to AVM-associated cerebral aneurysms, is flow-related and induced by the AVM. For the second type of SAs, we speculate the association of an aneurysm and an AVM to be an expression of a disorder of the spinal vascular development following a metameric distribution. Of course, it is not possible to exclude the development of a flow-related SA in metameric angiomatosis, or, the contrary, a congenital SA could be associated with an isolated spinal AVM. In addition, in some cases, both hemodynamic and dysplastic factors could be coincident. In our series, one SA (case 3), in a patient without metameric angiomatosis, remained unchanged despite AVM changes (the patient underwent only two embolization sessions with subsequent AVM reduction).

Our results could be biased by the fact that sometimes (cases 1 and 6) embolic fragments were recognizable within the aneurysm itself and could thus be responsible for shrinkage of the SA by "direct" effect. This could have occurred in only one of these patients. In the other, subsequent AVM embolizations (without particles in the SA) still resulted in a reduction of the aneurysm size.

Some authors (11) assert that the abrupt elimination of a cerebral AVM puts an aneurysm at an immediate risk of rupture. They have reported disastrous subarachnoid hemorrhage from aneurysms on feeding vessels to the AVM during the immediate postoperative period. Others (1–3, 7) have reported that, in cases of AVM flow-related aneurysms, there is not an increased risk of bleeding after AVM treatment. On the other hand, dysplastic aneurysms not associated with a feeding artery but present in association with an AVM are reported to be at risk for enlargement or rupture after AVM treatment due to increased hemodynamic stress (1, 2). In our series, after treatment of the intramedullary AVMs, no bleeding occurred in any case, including the patients with SAs that did not appeared flow-related.

We have shown (see part I in this issue) that the combination of a SA and an AVM carries an increased risk of bleeding. This result suggests that, in addition to AVM therapy, a selective treatment of the associated SA should be mandatory. However, based on our results, it is impossible to determine whether bleeding occurs from the associated SA or from the AVM. This study is a retrospective review of cases in which the endovascular treatment was performed in order to treat the intramedullary AVM, and not the SA. At that time, the pathologic implications of the presence of the associated SA were unknown.

In conclusion, we have reported results of SA size changes after endovascular treatment of associated intramedullary AVMs. As with AVM-associated cerebral aneurysms we feel that in the spinal cord there may be two different types of SAs associated with an AVM. The results of SAs whose size changes mirrored those of the AVM after endovascular treatment support the hemodynamic theory of the development of associated SA. SAs that did not change with AVM modifications probably are not flow-related. In our series, six of seven cases were associated with metameric angiomatosis. However, hemodynamic and developmental factors may be concurrent. Our results need to be confirmed as the exact relationship of these associated lesions in the spinal cord remains elusive.

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