Management of arteriovenous malformations: A multidisciplinary approach

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Background: **Management of arteriovenous malformations (AVMs) remains challenging because of their unpredictable behavior and high recurrence rate. A multidisciplinary approach based on a new classification scheme and improved diagnostic techniques may improve their management. The purpose of this study was to review our experience with combined embolotherapy, sclerotherapy (embolo/sclerotherapy), and surgical procedures to manage AVMs.**
Methods: **A total of 797 patients with congenital vascular malformations (January 1995 through December 2001) was investigated with noninvasive studies. Once an AVM was diagnosed, all underwent angiographic confirmation as a roadmap for treatment. Embolo/sclerotherapy and surgical procedures were instituted by the multidisciplinary team with periodic follow-up per protocol. Seventy-six patients with AVMs were reviewed retrospectively to assess the diagnosis and management by a multidisciplinary approach.**
Results: **Seventy-six (9.5% of all CVM) patients had AVMs, mostly infiltrating, extratruncular form (61/76). Embolo/sclerotherapy with various combinations of absolute ethanol, *N*-butyl cyanoacrylate (NBCA), contour particles, and coils were used in 48 patients. Sixteen patients with surgically accessible localized lesions completed preoperative embolism and sclerotherapy through 24 sessions, with subsequent surgical excision with minimal morbidity. Interim results were excellent, with no evidence of recurrence in all 16 patients with a mean follow-up of 24 months. Thirty-two patients with surgically inaccessible lesions (infiltrating) were treated with embolism and sclerotherapy alone. There were nine failures in a total of 171 sessions. Interim results with a mean of 19 months' follow-up of embolism and sclerotherapy alone were excellent in the majority (25/32) and good to fair among the rest (7/32). However, 31 complications, mostly minor (27/31), occurred in 30 sessions. Four major complications occurred, including facial nerve palsy, pulmonary embolism, deep vein thrombosis, and massive necrosis of an ear cartilage.**
Conclusions: **Diagnosis and management of AVMs by a multidisciplinary approach that integrates surgical therapy with embolism and sclerotherapy appears to improve the results and management with limited morbidity and no recurrence during early follow-up. (J Vasc Surg 2004;39:590-600.)**

Congenital vascular malformations (CVMs), including arteriovenous malformations (AVMs),[1-3] remain an enigma despite efforts during the last century to improve their care. They have a wide range of clinical presentation and an unpredictable course. Complicated anatomic, pathologic, physiologic, embryonic and hemodynamic characteristics must be evaluated. Previous classification of CVMs has created confusion in proper diagnosis. High morbidity has been related to both surgical and nonsurgical treatments. There has been an associated high recurrence rate. Among CVMs, AVMs have been particularly confusing because of their unpredictable nature.[4-6]

AVMs behave aggressively as a primitive type of CVM because the majority belong to the extratruncular form as the residual remnants of a developmental arrest in the early stage of embryonic life. They have a tendency to progress with a more destructive potency. The primary effect on the surrounding tissues is by the lesion itself, with compression and erosion. Secondary hemodynamic effects include a potential arterial steal phenomenon. The heart can be affected by high-output cardiac failure. Peripheral tissues can be affected in a wide range of changes from distal ischemia to gangrene, venous stasis dermatitis, and ulcer or gangrene caused by venous hypertension.[2,7-10] AVM, especially its infiltrating extratruncular form, has a high recurrence rate because of its origin from the mesenchymal cells at an early stage of embryogenesis.[11] It retains the evolutional potential to grow, which is often represented clinically as a recurrence.[12,13] Its behavior, therefore, is totally unpredictable, often responding to various stimulations such as injury or surgical intervention, as well as a systemic hormone effect. The result can be explosive growth. Improper treatment often stimulates dormant AVM to grow rapidly, making the condition worse. This recurrence and unbridled growth are the trademarks of AVM.

Complete eradication of the nidus of an AVM is the only potential "cure." But this, however, is often difficult if not impossible.[14] Radical resection to remove the lesion completely, such as the Malan operation, has been described as "demolishing surgery."[6] It is often accompanied by excessive blood loss in addition to serious complications. Thus, incomplete removal of the AVM is a frequent result of attempts to avoid the high morbidity associated with total excision. Adjuvant therapy in the past has included ligation or embolization of arteries supplying the AVM. This approach, however, was based on a poor understand-

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Table I. Hamburg classification of congenital vascular malformation: 1988 with modification

*Species**	*Anatomical form†*
Predominantly arterial defects	Truncular forms
	Aplasia or obstruction
	Dilatation
	Extratruncular forms
	Infiltrating
	Limited
Predominantly venous defects	Truncular forms
	Aplasia or obstruction
	Dilatation
	Extratruncular forms
	Infiltrating
	Limited
Predominantly AV shunting defects	Truncular forms
	Deep AV fistula
	Superficial AV fistula
	Extratruncular forms
	Infiltrating
	Limited
Combined vascular defects	Truncular forms
	Arterial and venous
	Hemolymphatic
	Extratruncular forms
	Infiltrating hemolymphatic
	Limited hemolymphatic
Predominantly lymphatic defects	Truncular forms
	Aplasia or obstruction
	Dilatation
	Extratruncular forms
	Infiltrating
	Limited

AV, Arteriovenous.
*Based on the consensus of the International Society for the Surgery of Vascular Anomalies through the internatoinal workshop on congenital vascular malformation held in Hamburg, Germany, 1988: Capillary malformation was not included.
†Based on the embryologic stage at which the developmental arrest has occurred.

Table II. Diagnosis of AVMs

I. Non- to less-invasive study: essential for the baseline evaluation
Duplex scan (arterial and venous)
Whole body blood pool scan
Transarterial lung perfusion scan
MRI with T1 & T2 image
CT scan with angio-contrast enhancement*
Lymphoscintigraphy*
Ultrasonographic lymphangiography
MR lymphangiography*
Volumetry*
Air plethysmography*
II. Invasive study: essential for the confirmation and proper disposition of AVMs as a road-map for treatment
Selective & superselective arteriography
Direct puncture arteriography
Standard and/or direct puncture phlebography
Direct puncture lymphangiography*

AVM, Arteriovenous malformation; *MRI,* magnetic resonance imaging; *CT,* computed tomography.
*Optional.

Table III. Treatment indications for AVMs*

Absolute indications
Hemorrhage, major or recurrent minor (16/48)
Gangrene or ulcer of arterial, venous or combined origin (18/48)
Ischemic complication of acute and/or chronic arterial insufficiency (26/48)
Progressive venous complication of chronic venous insufficiency with venous hypertension (30/48)
High-output cardiac failure—clinical and/or laboratory (8/48)
Lesions located at life-threatening vital areas that compromise seeing, hearing, eating or breathing (13/48)
Relative indications
Various symptoms and signs affecting the quality of life; disabling pain and/or functional impairment (43/48)
Lesions with potentially high risk of complications (eg, hemarthrosis) and/or limb-threatening location (34/48)
Vascular-bone syndrome with limb length discrepancy (21/48)
Cosmetically severe deformity with/without functional disability (28/48)

*Demographic data of 48 patients described in the study are included.

ing of the complicated nature of AVM as an embryonal remnant.[15,16,17]

New diagnostic technology, including less invasive imaging, has aided the differential diagnosis of CVM to provide a more precise diagnosis of AVM developed in different stages of embryogenesis. Contemporary diagnosis,[18,19] based on the Hamburg classification,[20,21] provides an opportunity for implementation of the new concept of a multidisciplinary team approach[16,17] for managing AVM. This approach is based on a new classification scheme[20] (Table I) and diagnostic technology (Table II). [18]

Embolo/sclerotherapy is a new therapeutic modality that is accepted as independent therapy, especially for surgically inaccessible lesions.[2-24] It has also been implemented as preoperative or postoperative adjunct therapy. It has helped improve surgical results and to expand the role of surgical therapy.[16,17]

The purpose of this study was to conduct a retrospective analysis of the clinical results of a contemporary management program for AVM that was based on a multidisciplinary approach with full integration of embolo/sclerotherapy and surgical intervention.

PATIENTS AND METHODS

Among 797 patients with CVMs investigated at Samsung Medical Center, Seoul, Korea, from January 1995 through December 2001, 76 patients (9.5%) were confirmed to have AVMs (Fig 1). The AVMs were mostly diffuse infiltrating extratruncular AVMs accompanied by a macro-arteriovenous (AV) shunting nidus (61/76). The majority (23) were in the head and neck region, and the second-largest portion were in the upper extremity (17). Multiple site involvement was common (13). AVM patients were evaluated by the new classifications.[25,26]

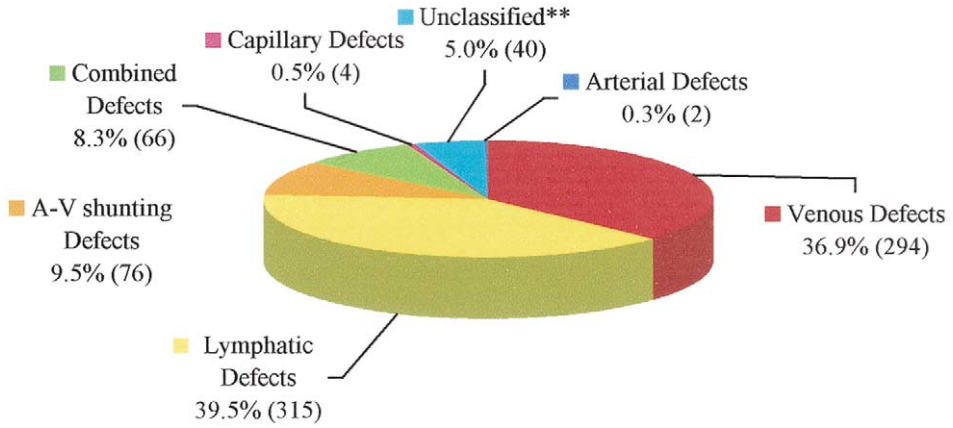

Fig 1. Congenital vascular malformation based on the Hamburg classification for 797 patients registered at CVM Clinic, Vascular Center, Samsung Medical Center (January 1995 through December 2001): 446 females and 351 males; mean age 22.1 years (range, 14 days to 81 years). **Due to various factors including young age, final diagnosis was temporarily deferred until conclusive diagnostic procedures could be included. Unclassified malformations are, however, mostly venous malformations clinically and subsequently confirmed as venous malformations after lymphatic malformations, etc, were properly ruled out. In addition to these deferred diagnostic cases, there were truly unclassifiable malformations from hemangioma as well as capillary malformations clinically and/or histopathologically.

Various combinations of the noninvasive studies based on new diagnostic technologies[27,28] were implemented per protocol (Table II). When the AVM was diagnosed, further review was made by the multidisciplinary team.[16,17] Treatment was approved by consensus (Table III). All AVMs had angiographic evaluation to determine further detailed anatomic and hemodynamic information of the AVM lesion.

Transarterial lung perfusion scan (TLPS) was added as a new, less invasive test to measure the extent of arteriovenous shunting of the AVM. The TLPS was implemented not only for the initial diagnosis but also for follow-up assessment of the treated and untreated lesions. TLPS has been used mostly for AVMs located within the extremity because of the technical limitation of safe access through the femoral or axillary artery for injection. This scintigraphic study used 99m technetium–labeled microsphere albumin. It provided quantitative assessment of the shunting volume to the lung through the nidus of the AVM to calculate the shunting percentage.

Whole-body blood pool scans (WBBPSs) were also included as one of the basic noninvasive tests for AVMs. They used 99m-technetium–tagged erythrocytes to detect abnormal blood pooling through the body. The quantitative assessment was made for the reduction of relative amount, size, and intensity of the blood pooling on the scintigram by the treatment. It was calculated as follows:

$$\text{Reduction percentage} = 1 - \frac{\text{Posttreatment ratio of region of interest/whole body}}{\text{Baseline ratio at the region of interest}}$$

Duplex sonography and magnetic resonance imaging were also included as essential noninvasive tests.

Of the 76 patients with AVMs, 48 patients (extratruncular form, 42 and truncular form, 6; male, 22 and female, 26; age range, 6 to 62 years) were selected for treatment. They were selected for treatment based on a minimum of two indications, described in Table III. The majority of patients had various symptoms affecting their quality of life (43/48), with frequent indications being venous complications (30/48) and potential high risk of complications because of the location (34/48).

Selection of surgical intervention or embolo/sclerotherapy, either as independent therapy or as adjunct therapy before surgical therapy, depended on the type, location, and extent of the lesions. Anticipation of associated morbidity after the therapy was always considered. Embolo/sclerotherapy alone using various embolo/sclerosants[29-34] was implemented mostly to manage the surgically inaccessible AVMs or those that were considered to be at high surgical risk. Surgical procedures were preferred for all accessible AVMs with acceptable risk to attempt a cure. However, a combined approach with preoperative embolo/sclerotherapy[17,35,37] was implemented for surgically accessible lesions whenever feasible to reduce surgical morbidity. Full integration of various surgical and nonsurgical treatment modalities was implemented for complex AVMs through the multidisciplinary approach.[18]

Extratruncal (ET) AVMs, which consist mostly of nonfistulous lesions with a treatable nidus, were assigned for treatment with ethanol as sole therapy when the lesion was not surgically amenable because of diffuse infiltration. Surgically amenable, limited (localized) ET AVMs were treated with preoperative embolo/sclerotherapy and subsequent surgical therapy. Truncular (T) AVMs, which were mostly fistulous lesions without an adequately treatable

direct connection between an artery and a vein, were assigned to surgical excision combined with preoperative embolo/sclerotherapy when the lesion was surgically amenable as a superficial fistula. Independent embolo/sclerotherapy was assigned to the surgically inaccessible, deep-seated fistula, which was the usual experience.

As embolo/scleroagents, absolute to 80% ethanol, *N*-butyl cyanoacrylate (NBCA), various types of coils, and/or contour particles such as ivalon were used in various combinations, simultaneously or in stages, depending upon the location, severity, and extent of the AVM. Absolute to 80% ethanol was used as the main agent in the surgically inaccessible lesions. It was given via transarterial, transvenous, or direct puncture injection. This depended on the anatomic or hemodynamic status of the individual AVM. NBCA glue was used mainly for the surgically excisable lesions as preoperative embolo/sclerotherapy to reduce the morbidity during the subsequent surgical therapy.[18] We did not use it as permanent agent to control AVM because there is a lack of convincing evidence that this induces permanent damage to the endothelial cells. The treatment response as well as interim or final results were assessed periodically by the multidisciplinary team per protocol.[22,24]

Clinical assessment was based on the subjective improvement of clinical symptoms on a scale of 0 to 10 and on objective evidence of improved clinical signs such as a healed ulcer, cessation of bleeding, reduction of swelling, or improved range of motion on a scale of 0 to 5, to classify the response as "excellent," "good," or "fair."[22,24] Laboratory assessment was based on various combinations of noninvasive to less invasive tests, such as duplex scan, WBBPS, TLPS, magnetic resonance imaging (MRI), especially the interim assessment during multisession therapy. However, the angiographic finding was the gold standard for the ultimate assessment of the treatment response. At the conclusion of each treatment as well as at the completion of multisession therapy, "excellent" was the rating for complete control (disappearance), "good" was the rating for near-complete control with negligible evidence of residual lesions, and "fair" was the rating for substantial control with significant residual lesion that warranted further close observation. Similar criteria were applied to the various noninvasive to less-invasive tests for the treatment result assessment. For example, the duplex scan evaluation was based on the hemodynamic status along the lesion. Complete cessation of the hemodynamic activity at the treated nidus was "excellent." Near-complete cessation, but with some suspicion along the feeding artery and draining veins, was "good." Drastic reduction, but with substantial evidence of the residual activity of the treated nidus, was "fair." For WBBPS, TLPS, and MRI, the same criteria for arteriographic and duplex interpretation of the treatment response were implemented with some modification.

Periodic follow-up evaluation of the treatment results was made based on the duplex scan, WBBPS, TLPS, or MRI in the majority, especially during the multisession therapy. Proper combination of these noninvasive to less invasive tests was adequate to replace most of the role classically played by angiography, not only for the interim treatment response assessment but also for the follow-up assessment of the AVM. However, arteriography has remained the ultimate gold standard for AVM management. It has been included especially for the final confirmation of the treatment results and subsequent follow-up biannually, as routine protocol for AVM follow-up.

RESULTS

Sixteen (extratruncular, 14; truncular, 2) of 48 patients with AVMs were selected for preoperative embolo/sclerotherapy. These patients had surgically accessible lesions with localized, noninfiltrating AVMs. Fifteen patients had multiple sessions of preoperative embolo/sclerotherapy to facilitate subsequent surgical excision. The sessions used mainly NBCA glue with or without additional ethanol (13/16 patients; NBCA glue only, 9; NBCA glue and ethanol, 4; ethanol only, 2; and ethanol and ivalon, l). All 16 patients had subsequent surgical excision within 2 to 4 weeks after the embolo/sclerotherapy. This reduced morbidity, through selective excision of the lesion only. There was minimal blood loss averaging 200 mL, in contrast to massive blood loss with the more conventional surgical therapy alone (Fig 2). One patient had a pulmonary embolism after the use of NBCA glue. All 16 patients had excellent interim results with no evidence of recurrence during a follow-up averaging 24.3 months; these results were confirmed angiographically.

Thirty-two patients (extratruncal, 28; truncal, 4) with surgically inaccessible lesions, mostly diffuse infiltrating AVMs (buttock, thigh, and calf muscles), were indicated for the independent embolo/sclerotherapy. There were 171 sessions with 9 failures. Figures 3 and 4 identify the combinations of agents. Ethanol was combined with coils, glues, or contour particles (158/162 of the successful sessions), and ethanol was used alone in 132 sessions. The majority (25/32 patients) were considered to have an excellent result during follow-up averaging 13.6 months. There was good to fair control of the activity of the nidus among the remaining seven. All had angiographic follow-up. There was no evidence of recurrence during a follow-up of 19.2 after the study protocol. There were, however, 31 complications with the majority, 27 being minor involving the skin. This usually followed treatment using ethanol. Three of the 4 major complications developed after ethanol therapy: 1 transient facial nerve palsy, 1 deep vein thrombosis, and 1 massive ear cartilage necrosis. There was 1 pulmonary embolism after the NBCA glue embolotherapy to a truncal AVM of the fistulous type. These major complications developed in high-flow conditions, whereas the majority of minor complications developed among the extratruncular AVMs. One patient with a high-flow AVM fistula involving bone had cessation of treatment despite what was considered to be good progress of the therapy. Subsequently, the patient required forearm amputation to arrest recurrent massive bleeding and high-output cardiac

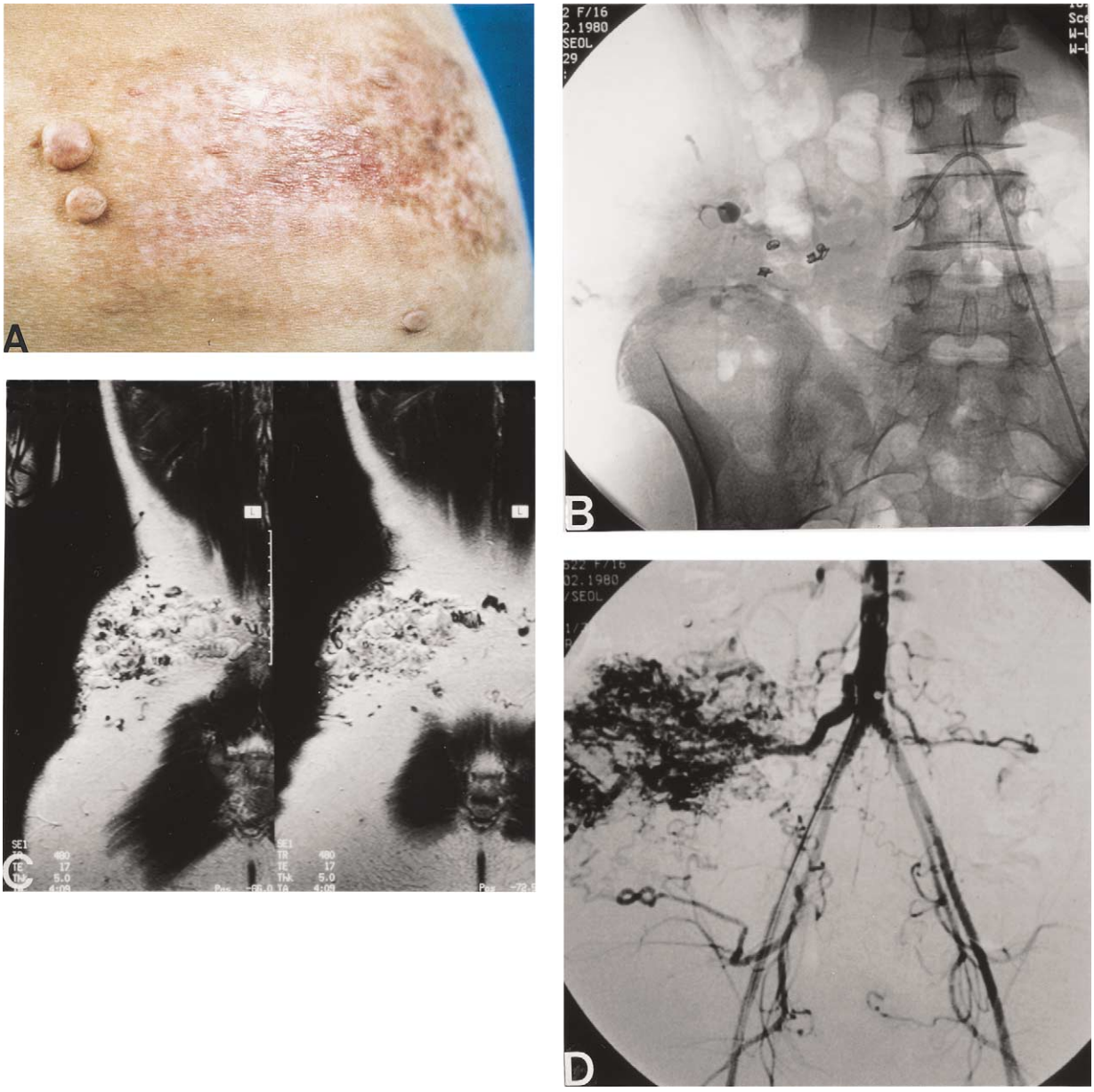

Fig 2. Preoperative embolotherapy with *N*-butyl cyanoacrylate (NBCA), combined with surgical excision of a recurrent AVM lesion. **A,** Clinical appearance of painful tender swelling along the right flank after initially successful coil embolotherapy, done elsewhere. **B,** Plain roentgenogram finding of the scattered coils that were previously used to shut off the feeding artery only. **C,** MRI finding of the AVM lesion, confirmed as a diffuse infiltrating type of the extratruncular form, mostly limited to the soft tissue. **D,** Angiographic finding of the recurred AVM lesion with multiple new or old feeding arteries.

failure as a lifesaving procedure. Unfortunately, the patient eventually committed suicide.

DISCUSSION

Although improved diagnoses based on advanced technology provide adequate information for appropriate treatment, AVM remains the most difficult type of CVM. There is a higher risk of complications and morbidity with AVM. Despite our efforts, complications associated with AVM treatment remain higher than we desire.

Clinically, it is difficult to select the optimal treatment to enhance long-term success in managing AVMs. Surgical excision offers the best opportunity for "cure." However, diffuse excision of infiltrating AVMs of the extratruncular form can be associated with significant morbidity as well as with failure of attempted cure. Embolo/sclerotherapy can

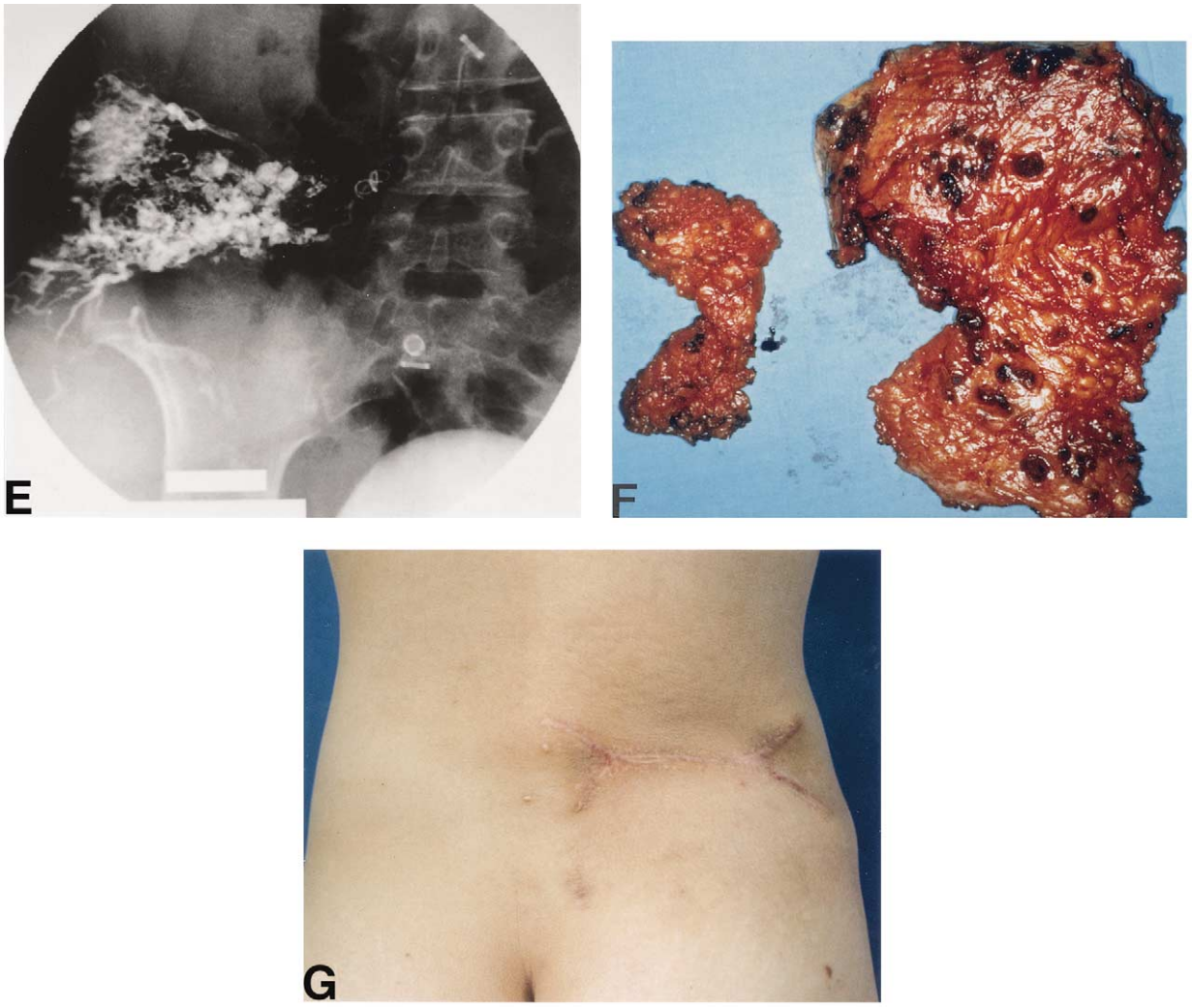

Fig 2 (continued). E, Radiographic finding of the NBCA glue-filled AVM lesion after preoperative embolotherapy for the subsequent surgical therapy. **F,** Gross finding of the surgical specimen containing glue-filled vessels. **G,** Clinical result of well-healed surgical wound after the en bloc resection of the recurred infiltrating lesion at the flank with minimum blood loss by the preoperative glue embolization.

be successful as a second choice in treating AVMs. We adopted absolute ethanol reluctantly as a major scleroagent despite the high complication rate reported elsewhere.[22,24] Until a better agent for sclerotherapy is identified, this approach has been associated with the fewest recurrences of AVMs.

Sclerotherapy alone using absolute ethanol to treat surgically inaccessible AVMs is associated with complications; however, major complications were reasonably low in our series (in 4/186 sessions, complications arose in 4/48 patients). We do not recommend absolute ethanol in AVM treatment unless there is a specialized team approach, such as in our referral center. We recognize that serious consideration of significant risk continues to be the challenge of many AVMs. For surgically inaccessible AVMs, we used embolo/sclerotherapy.

Recurrence remains a challenge, particularly with extratruncal AVMs. Nevertheless, inadequately treated lesions represent greater problems than the evolutional potential for recurrence. Because of this, our clinical approach for AVM concentrates on the hemodynamic aspects of the AVM. The high-flow status of the fistulous type of truncal AVM makes treatment extremely difficult, if not impossible, without additional morbidity (deep vein thrombosis and pulmonary embolism). Temporary control of inflow or outflow of the lesion with balloon catheters has helped implement control of the high-flow status. There is less risk for subsequent therapy with this approach. Ethanol sclerotherapy to high-flow fistulous lesions is usually contraindicated because of the high risk of early wash into the systemic circulation.

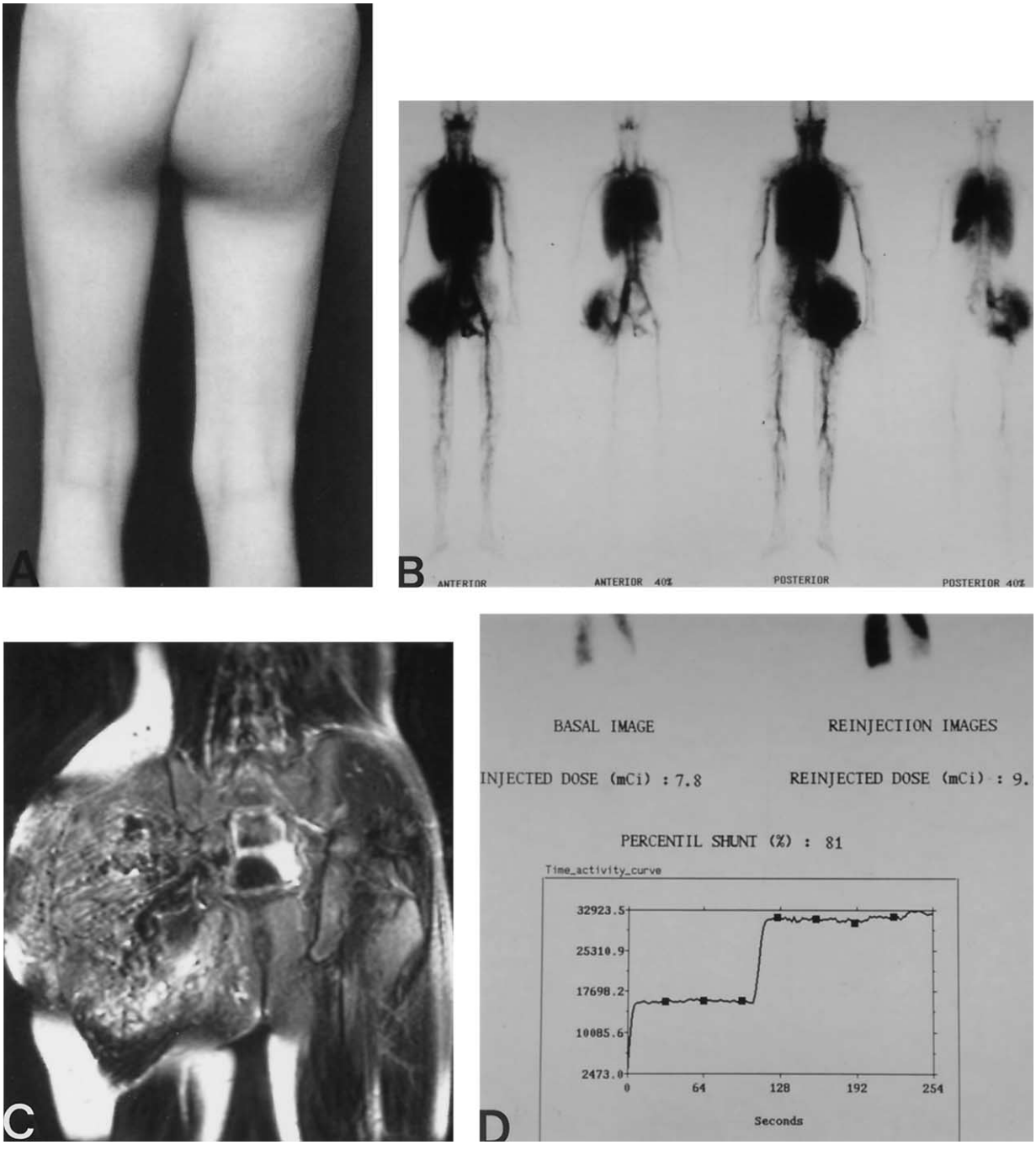

Fig 3. Isolated ethanol sclerotherapy of an AVM. **A,** Clinical appearance of the AVM lesion affecting the hip region with progressive symptoms. **B,** WBBPS finding of the extent of hemodynamically aggressive AVM lesion along the right hip; note the enormous size of right iliac artery equivalent to the size of abdominal aorta. **C,** MRI finding of the AVM lesion, confirmed as infiltrating type of the extratruncular form, affecting entire right hip soft tissue and muscles, and so on. **D,** TLPS finding of the AV shunting status of the lesion (pretreatment baseline study).

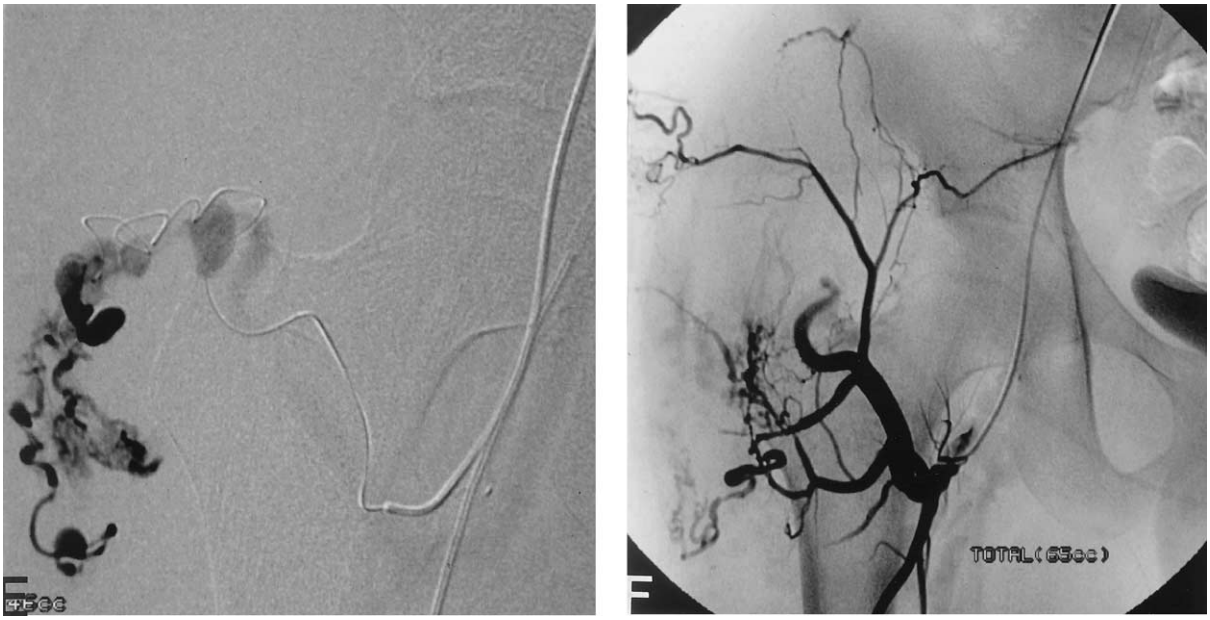

Fig 3 (continued). E, Angiographic finding of the ethanol sclerotherapy directly to the multiple niduses of the extratruncular form of the AVM lesion, reached by superselective catheterization technique. **F,** Angiographic finding of the interim results of the effective control of the niduses by multisession ethanol sclerotherapy, leaving feeding vessels intact.

NBCA embolotherapy is relatively contraindicated in this case as well.

The role of NBCA embolotherapy in our clinic is specific. Its role is limited to adjunct therapy for subsequent surgery. Our intention is to remove all NBCA glue, together with the lesion, whenever possible during surgical excision. NBCA embolotherapy helps control bleeding and helps provide an excellent local guide for excision. We do not believe that NBCA can act as a permanent agent to control a lesion effectively because there is no evidence of permanent damage to the endothelium.

The fistulous AVM without a treatable nidus was controlled generally through a staged approach, using a new strategy of coil embolotherapy as a preliminary procedure to slow down the flow, thus reducing the risk of subsequent distal thromboembolism. Ethanol or NBCA glue embolo/sclerotherapy then followed the coil embolotherapy.

Classification of CVM, such as Klippel-Trenaunay and Parkes-Weber syndromes, has been confusing.[6,38,39] A new classification based on a consensus from the workshop of the International Society for the Study of Vascular Anomaly in 1988 has more clinical applicability.[37] Anatomic, pathologic, and physiologic status of developmental failures in various stages of embryogenesis are included.[12,13] There has been further modification of the classification of CVM.[20,21] The basis of contemporary diagnosis of CVM has eliminated the old eponym-based classification.[18,38,39] There is clarification between CVM and the true infantile hemangioma, which is a vascular tumor.[40-43] Although they are related vascular anomalies, these are two independent entities. The new classification of CVM has provided critical support for improved management of AVMs based on the new diagnostic technology developed for CVM in general. Various noninvasive to less-invasive tests were introduced to assess detailed hemodynamic status of AVM.[41] Precise evaluation of AVM has been a result. WBBPS and TLPS have helped to evaluate the initial lesion as well as the interim treatment results of AVM during multisession therapy. Subsequent long-term outcome assessment has also been aided in cases of either treated or untreated AVMs. TLPS is important in screening for hidden micro-AV shunting in AVMs before using invasive arteriographic evaluation.

Duplex ultrasonography has been of value in assessing AVMs. It also helps evaluate inflow arteries, outflow veins, and collateral vessels of the AVM. It is important to assess the interim results during each session of the multisession therapy as well as to assess the long-term outcome of the treated and untreated lesions. MRI is a contemporary gold standard for assessing the anatomic status of the AVM. It delineates the lesion as well as the relationship to surrounding tissues and organs, including muscle, tendon, nerve, vessel, and bone. It helps differentiate low-flow and high-flow status of the CVM in general.

All AVMs are potentially limb threatening and even life-threatening. An early aggressive approach to all AVMs is warranted to reduce, if not prevent, the immediate risk of bleeding as well as the long-term risks of cardiac failure and gangrene. Ligating the feeding artery to an AVM, as was done for many years, only leaves the nidus of the lesion

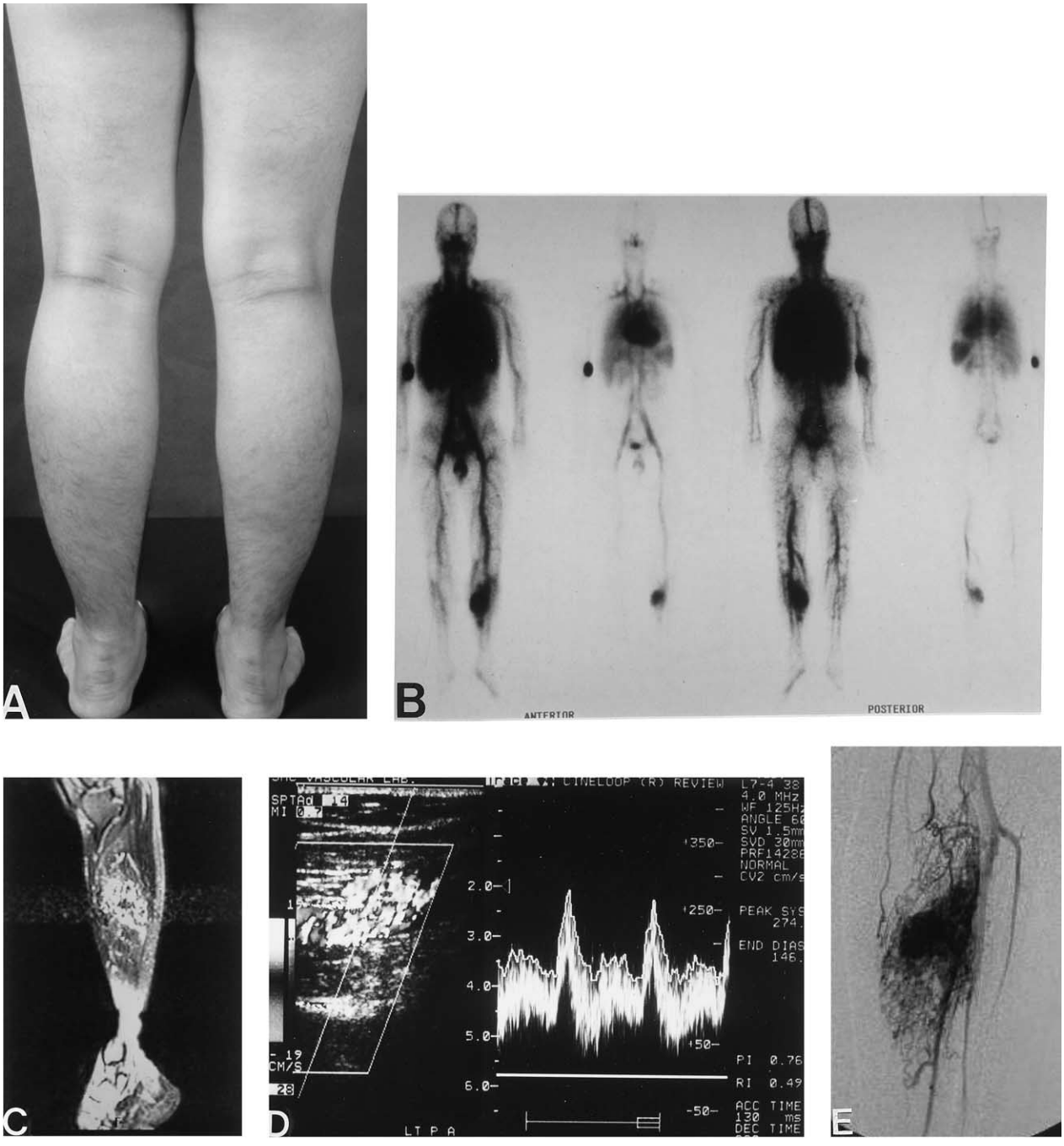

Fig 4. "Staged" embolo/sclerotherapy with absolute ethanol and coil, to a recurred AVM. **A,** Clinical appearance of left calf swelling with increasing pain, after unsuccessful embolo/sclerotherapy done elsewhere. **B,** WBBPS finding of the lesion within the left calf with abnormal blood pool (pretreatment). **C,** MRI finding to confirm the diffuse infiltrating type of extratruncular form of AVM within the calf muscles (pretreatment). **D,** Duplex scan finding of hemodynamically high-flow status along the feeding artery at the popliteal artery level (pretreatment). **E,** Arteriographic finding of AVM lesion to confirm the extratruncular form with the nidus (pretreatment).

intact. This is followed by more aggressive neovascular development to make the AVM more of a risk. Aggressive control of the nidus of the AVM is essential.

Current management of AVMs based on the new concept of multidisciplinary approach[11,16,17] can minimize the morbidity and reduce recurrence. There has been further

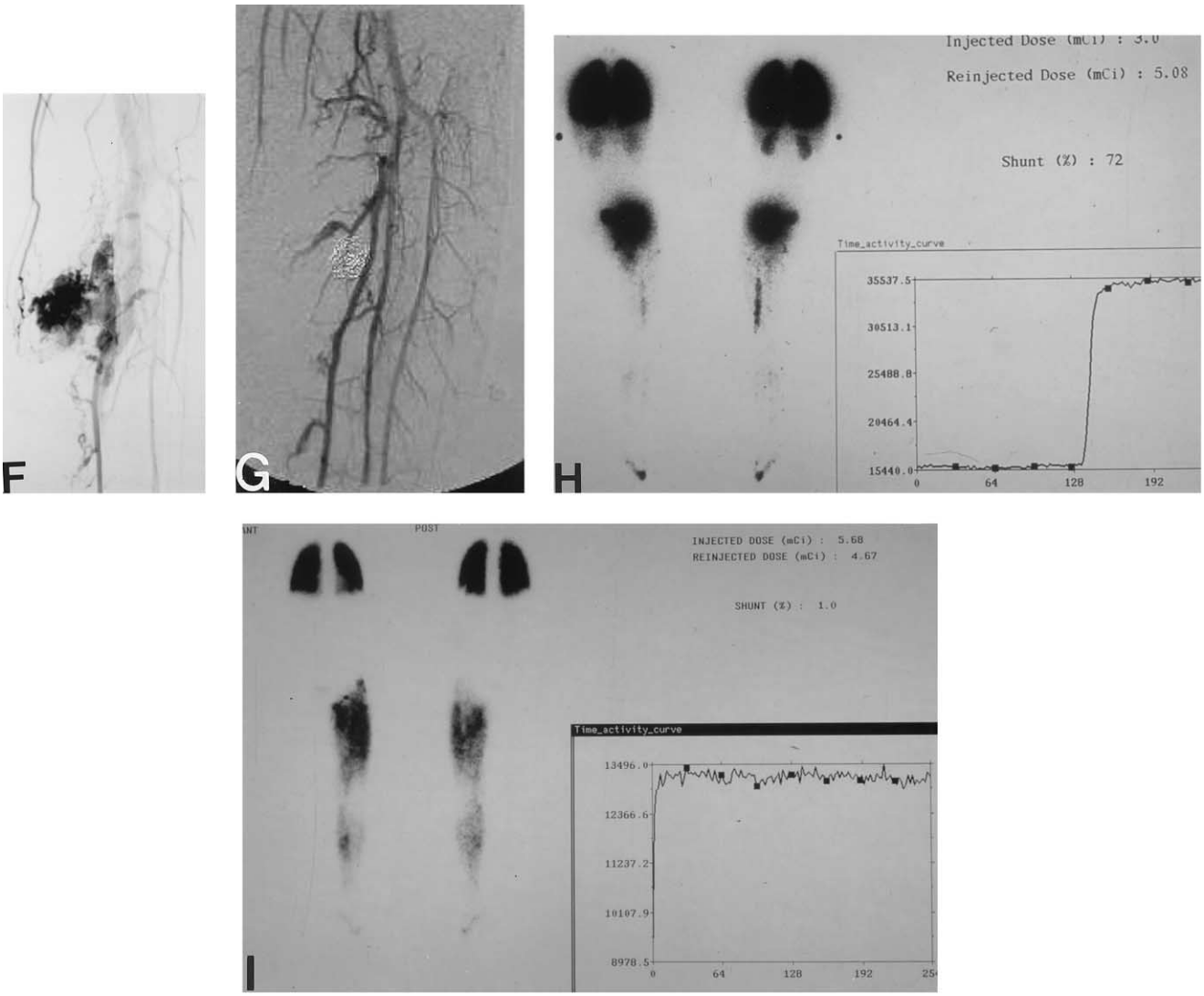

Fig 4 (continued). F, Angiographic finding of the ethanol sclerotherapy to the nidus of the lesion by transarterial and transcutaneous (direct puncture) catheterization technique. **G,** Angiographic finding of the final successful result of ethanol sclerotherapy combined with coil embolotherapy. **H, I,** Transarterial lung perfusion scan findings of the AV shunting status (before and after embolo/sclerotherapy): initial shunting status of 72% range through the AVM nidus before treatment is successfully lowered to the 1% baseline value to confirm the successful therapy.

expansion of the limited role of embolo/sclerotherapy as an adjunctive therapy for conventional surgical resection. This approach has even been helpful in high-risk lesions with high flow status. Multidirectional use of transarterial, transvenous, or percutaneous combinations has been helpful. There must be a positive balance between subsequent morbidity and the treatment gains from an aggressive plan. The importance of careful assessment of the treatment strategy *before* the therapy is instituted, based on the ratio of the benefit versus potential risk, cannot be overemphasized.[16,17] Amputation should not be excluded as one of the practical options, especially when the AVM is in an extremity with total loss of function.

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