Classification of Arteriovenous Malformation and Therapeutic Implication

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Introduction

Vascular malformations constitute one of the most challenging entities in the history of medicine to diagnose and treat effectively by whatever endovascular or surgical approaches are employed. These congenital vascular lesions can involve any tissue in the body. The rarity of vascular malformations in the population compounds the problem of treating them. If a physician rarely encounters patients with vascular malformations, it is difficult to gain enough experience to optimally treat them and effectively eradicate them. High-flow arteriovenous malformations (AVMs) are extremely challenging to surgically extirpate or to endovascularly cure. The world's literature certainly verifies the extreme challenges in the diagnosis and treatment of AVMs. The purpose of this chapter is to advance a new AVM Classification System that has proven therapeutic implications to effectively treat complex AVMs in any anatomical area. By employing the Yakes AVM Classification System, a physician is now able to accurately classify AVMs and determine specific endovascular treatment strategies to consistently treat AVMs, and patients can enjoy the long-term excellent outcomes. Defining the angioarchitecture of the high-flow AVM determines accurately the endovascular management strategy to best permanently ablate the AVM requiring treatment. Further, employing this new Yakes AVM Classification will lower complication rates in treating these complex congenital vascular pathologies.

Overview

The Houdart Classification of Intracranial Arteriovenous Fistulae and Malformations of high-flow lesions and the Cho-Do Classification of AVMs of the peripheral arterial circulation are strikingly similar despite their anatomic locational differences (CNS vs. peripheral vasculatures) [1–3]. Both authors also suggest similar therapeutic approaches based on their arteriographic classification. Houdart et al. Classification states the following types of AVMs: Type A as multiple arterial connections flow into a large aneurysmal vein with single outflow drainage, Type B as multiple microfistulae into an aneurysmal vein with single outflow vein, and Type C as multiple shunts between many arterioles and venules connected to each other. The Cho-Do et al. Classification based on "nidus morphology" provides the following types: Type I being arteriovenous larger fistulae with no more than

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three separate arteries shunt to the initial single venous outflow component, Type II as "arteriovenous smaller fistulae with multiple arterioles shunt to the initial part of a plexiform appearance" into a single venous component, Type IIIa as "arteriovenous fistulae with non-dilated fistulae with multiple fine shunts are present between arterioles and venules," and Type IIIb being "arteriovenous fistulae with dilated fistulae with multiple shunts are present between arterioles and venules."

Houdart Type A is the same as the Cho-Do Type I; Houdart Type B is the same as the combination of the Cho-Do types IIIa and IIIb. Therapeutic implications are also similar as well. The Houdart Type A and Type B and Cho-Do Types I and II proffer retrograde approaches to occlude the vein aneurysm outflow as being a potential for curative treatment of these AVM types. I proposed and illustrated the retrograde vein occlusion techniques for high-flow malformations first published and three cases illustrated in my manuscript published in 1990 [4]. Later, Jackson et al. published the retrograde vein approach in 1996 [5]. The Do group in Seoul, Korea (also the publishers of the Cho-Do AVM Classification), published the retrograde vein approach in 2008 after collaboration with our group demonstrated its efficacy to them in patients at their Seoul, Korea, Samsung Medical Center [6].

The Yakes AVM Classification System has some similarities to both classification systems and some stark differences. The Yakes AVM Classification System consists of the following: Type I is characterized by a direct arteriovenous fistula, a direct artery to vein connection (e.g., typified by pulmonary AVF and renal AVF). This angioarchitecture type is not described in the Houdart or Cho-Do Classification Systems. Type II is an AVM characterized by usually multiple inflow arteries into a "nidus" pattern with direct artery-arteriolar to vein-venular structures that may, or may not, be aneurysmal. Type IIIa consists of multiple arteries-arterioles into an enlarged aneurysmal vein with an enlarged single outflow vein. Type IIIb consists of multiple arteries-arterioles into an enlarged aneurysmal vein with multiple dilated outflow veins. Type IV comprises microfistulous innumerable arteriolar structures to innumerable venular connections that diffusely infiltrate a tissue (typified by ear AVMs that infiltrate the entire cartilage of the pinna). What is different in this lesion is that there are admixed among the innumerable fistulae capillary beds within the affected tissue. If the affected tissue only had AVFs, the tissue could not survive as capillary beds are required for tissue viability. No other AVM angioarchitecture has this duality [7]. This angioarchitecture is not described in the world's literature.

Comparing Houdart's CNS Classification and the Cho-Do Peripheral Vascular Classification to the Yakes Classification has some parallels, as has been described, but has several distinct differences.

Houdart Type A and Cho-Do Type I are the same and compare to the Yakes Type IIIa. Houdart Type B and Cho-Do Type II are the same and again are placed in the Yakes Type IIIa. Whether the arteriovenous (Type A/Type I) or arteriolar-venular connections (Type B/Type II) are present is not important as the same arterial physiology is present that the "nidus" being present in the vein wall itself, regardless of the size of AVF on the vein wall, as they are both treated endovascularly in the same way. Therefore, the AVF size is irrelevant. Further, even when larger AVF are present, microfistulae are also present as well admixed with the larger connections. It never is purely one microsize only or one macrosize only.

The Houdart Type C is the same as bundling Cho-Do Types IIIa (arteriovenous) and IIIb (arteriolar venular). This is similar to the Yakes Type II. Both authors do not explain in their classifications the Yakes Type IV. The angioarchitecture of arteriovenous and arteriolar-venular innumerable fistulae, totally infiltrating a particular tissue, is another vascular phenomenon that is present that is not explained by the Houdart nor the Cho-Do Classifications. Being that arteriographically these innumerable microfistulae are proven to infiltrate a tissue, one has to also consider that despite the innumerable microfistulae, there is interspersed within these abnormal fistulae vascularity that is normal with capillary beds that is nutrient to the infiltrated tissue as well, or the tissue itself would be

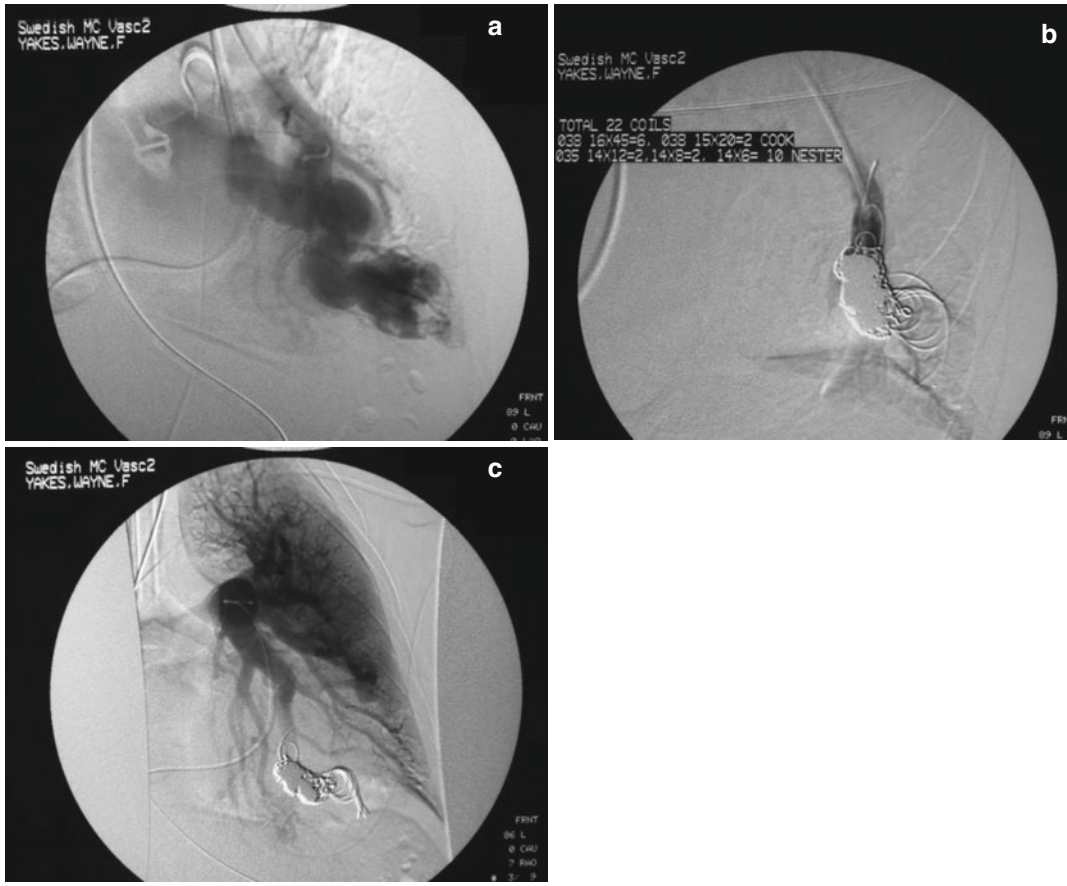

Fig. 33.1 Yakes Type I AVM (AVF) typified by a single inflow artery connected to a single outflow vein. (**a**) Ventilator Dependent 30 Year-Old Female with HHT and Massive Left Pulmonary AVM Causing O2 Sats of 35% on 100% Oxygen Through the Ventilator; Patient Sent By Air Ambulance Emergently For Treatment. Left Pulmonary Artery angiogram demonstrating a massive AVF shunt with single aneurysmal vein drainage. This single arteriovenous connection is Yakes Type I AVM (AVF). (**b**) Post-embolization selective Left Pulmonary Artery angiogram after placement of 22 fibered coils of .038 & .035 sizes in the AVF totally occluding the massive AVF. (**c**) Main Left Pulmonary Artery angiogram demonstrating closure of the massive AVF post-coil placement. Mechanical closure devices will permanently close and treat this Yakes Type I AVM (AVF)

devitalized and forced to necrose. Normal capillaries must be present admixed with the innumerable AVF in the infiltrated tissue, or it would not be viable and could not survive. Venous hypertension is usually the culprit in the injury that occurs in that infiltrated tissue, and this phenomenon as a vascular etiology for pathologic tissue changes was first elucidated by Jean Jacques Merland, M.D., and Marie Claire Riche, M.D [8]. Thus, the "normal" vascularity with capillary beds in the infiltrated tissue to allow it to exist is not discussed in the Houdart or in the Cho-Do Type Classifications or is the angioarchitecture characteristics described.

The Yakes Type I Classification is a direct AV macro-connection that is characteristic of pulmonary AVF and renal AVF, but can also occur in other tissues. This direct AV connection is not described in the Houdart Classification or in the Shin-Do Classification. The Yakes Type I AV connection can also be present and interspersed in complex AVMs as well (Fig. 33.1).

The Yakes Type II Classification possesses an angioarchitecture synonymous with the classical "nidus" pattern commonly seen in AVMs with multiple inflow arteries of varying sizes coursing toward a "nidus" (a complex tangle of vascular structures without any intervening capillaries and

exiting from this "nidus" into multiple veins from this "nidus"). The Houdart Type C and the Cho-Do Type IIIa/Type IIIb most resemble this angioarchitecture pattern. Thus, the Yakes Type II and Yakes Type IV further define the Houdart Type C and Cho-Do Type IIIa/IIIb patterns (Fig. 33.2), much more specifically.

As an aside, the term "nidus" is rampant in the medical literature (AVM nidus, nidus of infection, etc.). Unfortunately, the initial author was only partially familiar with the Latin language. "Nidus" means "nest" in Latin, and indeed it does. However, "nidus" with the ending "us" denotes male gender. In the Latin language, the true term meaning "nest" is, in fact, "nidum." The ending "um" denotes the neuter gender which a "nest" truly is. Thus, the original author accurately describing "nest-like" conglomeration of vascular structure was woefully inaccurate penning the words as "nidus" (masculine) instead of the true word "nidum" (neuter). Being rife in the literature for decades, there is no possibility of any correction of this term.

In summary, Yakes Type I is the simplest macro direct AV connection. Yakes Type II is the common "nidum" (nest-like) AV connection. Yakes Type IIIa has multiple AV connections (arterial and arteriolar into an aneurysmal vein: "nidum" is in the vein wall) with single outflow vein physiology (Fig. 33.3). Yakes Type IIIb has multiple arterial inflow connections (arterial and arteriolar) into an aneurysmal vein ("nidum" is in the vein wall) with multiple outflow veins that is more difficult to treat by retrograde vein approaches (Fig. 33.4).

Yakes Type IV angioarchitecture has innumerable micro-AV connections (with lowered vascular resistance) infiltrating an entire tissue but with concurrent normal vascular structures possessing nutrient capillary beds (with normal

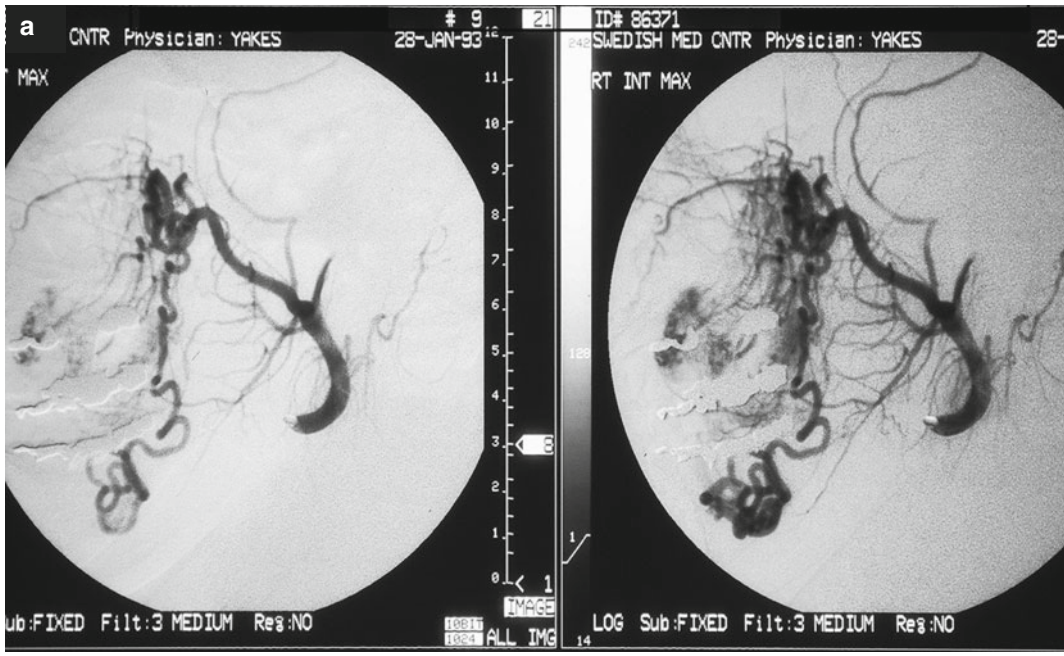

Fig. 33.2 24 year old female with painful right facial AVM also causing right facial swelling. (**a**) Example of Yakes Type II AVM with typical AVM "nidus". This type AVM can be treated by trans-arterial embolization (easiest approach usually), and direct puncture into the nidus (more difficult). Retrograde vein approaches are usually not successful. Lateral Right Internal Maxillary Artery arteriogram demonstrating arterial supply from a terminal Internal Maxillary artery branch arising from the Pterygo-Palatine fossa area. Note the typical AVM "nidus pattern"

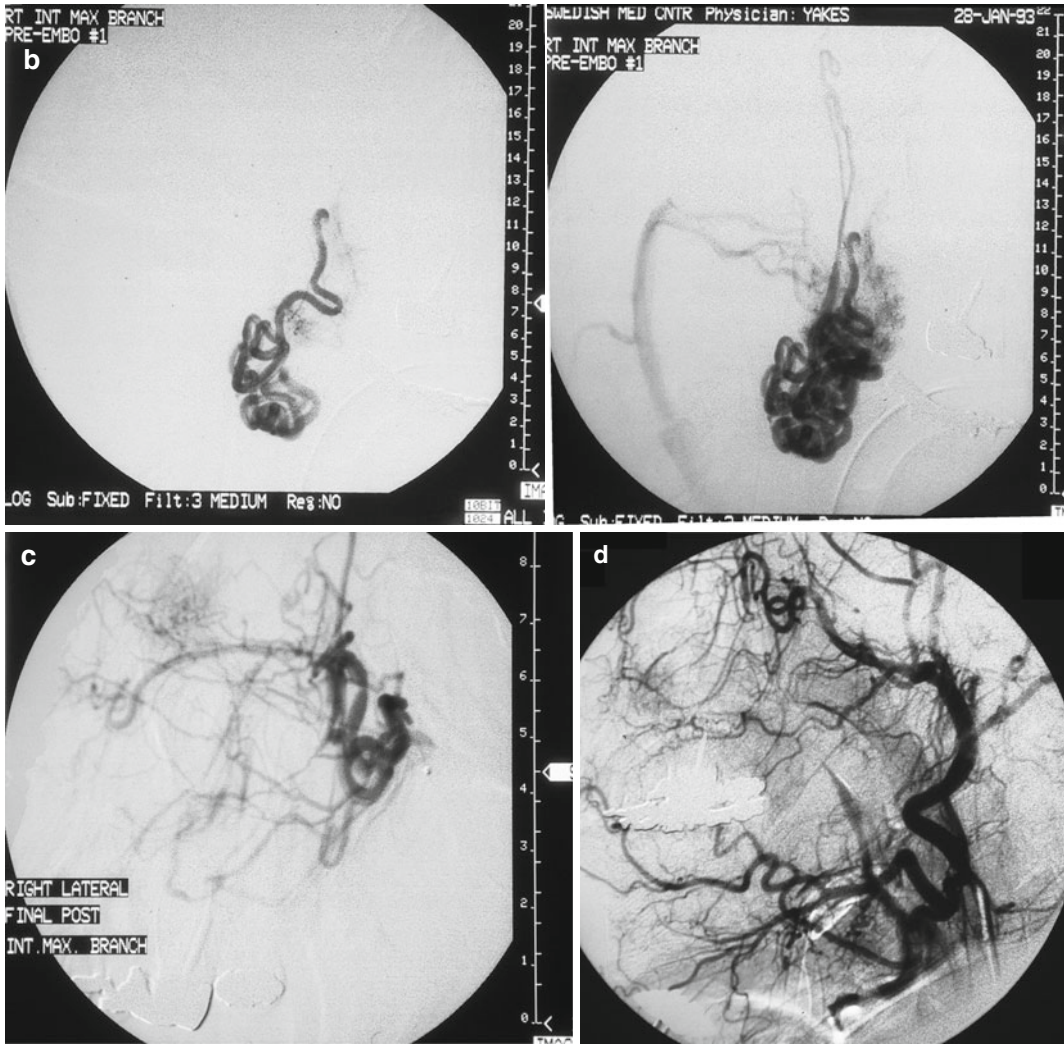

Fig. 33.2 (continued) (**b**) Lateral selective Right Internal Maxillary Artery branch arteriogram pre-embolization. A micro-catheter is required to obtain superselective arterial positioning for ethanol embolization of the AVM. This is required to ONLY embolize the AVM and spare all the normal tissues and capillary beds from ethanol arterial embolization. If not done this way, there will be total tissue devitalization and necrosis that will occur with inadvertant embolization of ethanol of the normal tissues. (**c**) Lateral Right Internal Maxillary Artery arteriogram immediately post-embolization demonstrating total occlusion of the right face AVM with all normal branches remaining intact. (**d**) Lateral Right External Carotid Artery arteriogram at 2 year follow-up. No residual AVM is identified. Note that the normal arterial vascularity remains intact

vascular resistance) to supply and drain the tissue that is diffusely infiltrated to allow this tissue to survive and not be devitalized. The postcapillary veins compete with AVF outflow veins that are arterialized (hypertensive) (Fig. 33.5) and cause the resultant nonhealing pathology. This entity has not been described in the world's literature [9–22].

Therapeutic Implications of the Yakes Classification

Determining a classification system based on the AVM angioarchitecture is of little use without a practical application. For example, the Spetzler-Martin Brain AVM Classification is of importance to determine the surgical morbidity

for treating brain AVMs [23]. The higher the Spetzler-Martin grade, the higher the morbidity. This allows the neurosurgeon to inform his/her patient accurately of the risks for treatment. The Schobinger AVM Classification for peripheral AVMs (non-neuro) is useful to quantify the degrees of symptomatology a patient possesses regardless of the AVM's angioarchitecture. The Yakes Classification is utilized to determine endovascular approaches and embolic agents that will be successful to ablate these peripheral AVMs.

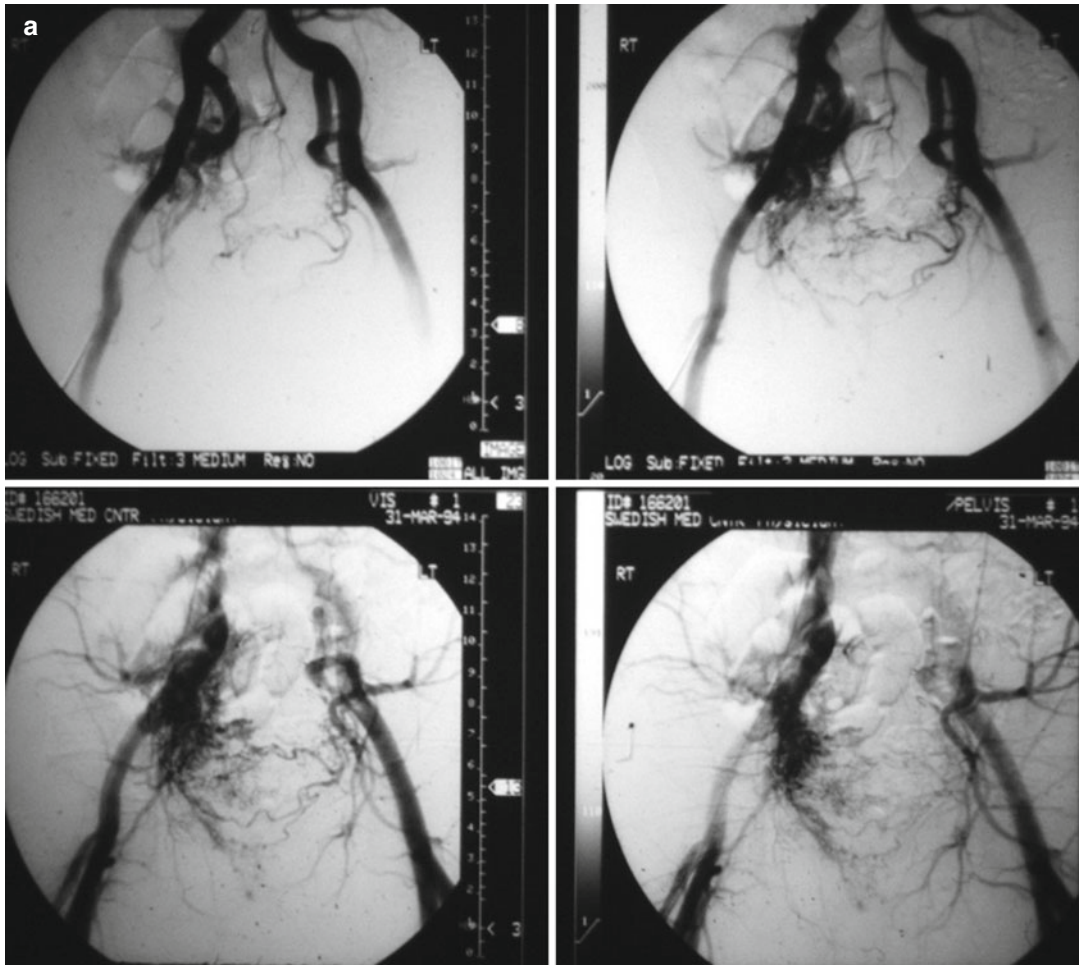

Fig. 33.3 Example of Yakes Type IIIa AVM angioarchitecture with multiple in-flow arteries/arterioles and single out-flow vein physiology. The vein wall is the "nidus" in this AVM type. Multiple Right Internal Iliac Artery branches supply this right pelvic AVM. (**a**) 32 year old male with right pelvic AVM with single outflow vein drainage towards Right Internal Ilaic Vein. Arterial supply is from multiple Right Internal Iliac Artery branches. Because of the diffuse innumerable small arteries suppling the AVM vein aneurysm wall, transarterial ethanol embolization is not possible. Normal structures could potentially be embolized and resultant nerve damage, pelvic organ damage, tissue necrosis, etc., could result

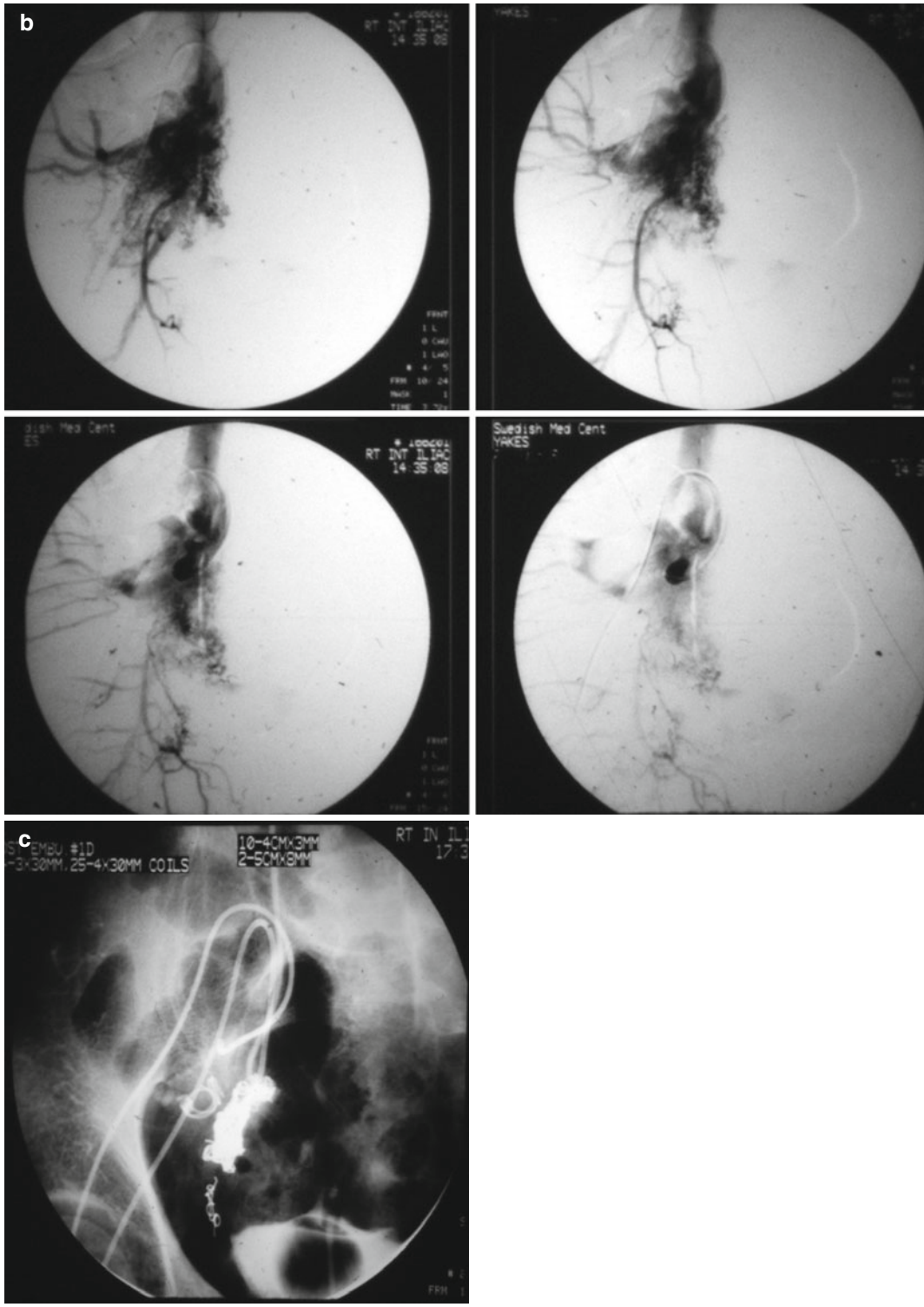

Fig. 33.3 (continued) (**b**) AP selective Right Internal Iliac Artery arteriogram demonstrating innumerable small arterial connections to the single out-flow aneurysmal vein. Superselective catheter positioning for transarterial embolization is not possible. A retrograde venous approach must be employed to treat this Yakes Type IIIa AVM. (**c**) AP pelvis spot film demonstrating arterial catheter in Right Internal Iliac artery, and the retrograde vein catheter placed centrally within the AVM vein aneurysm with the resultant deposition of multiple coils in the vein aneurysm to treat this Yakes Type IIIa AVM

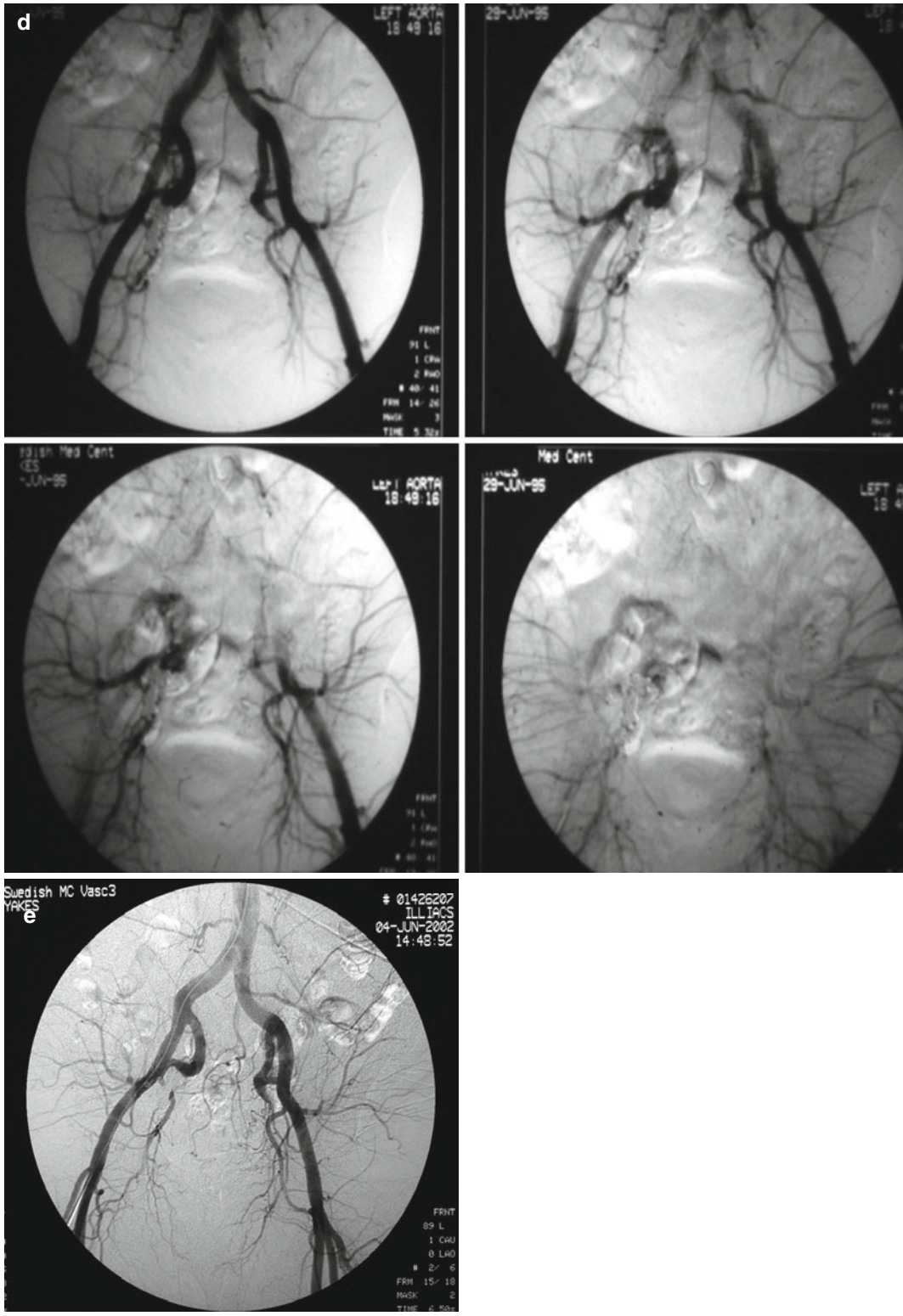

Fig. 33.3 (continued) (**d**) AP pelvis arteriogram immediately post-coil embolization demonstrating total occlusion of the right pelvic AVM. No residual arteriovenous shunting is present. All normal arteries remain intact post-coil embolization without complication. (**e**) AP pelvis follow-up arteriogram 7 years post-retrograde vein coil embolization demonstrating long-term cure of the right pelvic AVM. Again, this technique is curative in Type IIIa and Type IIIb AVMs

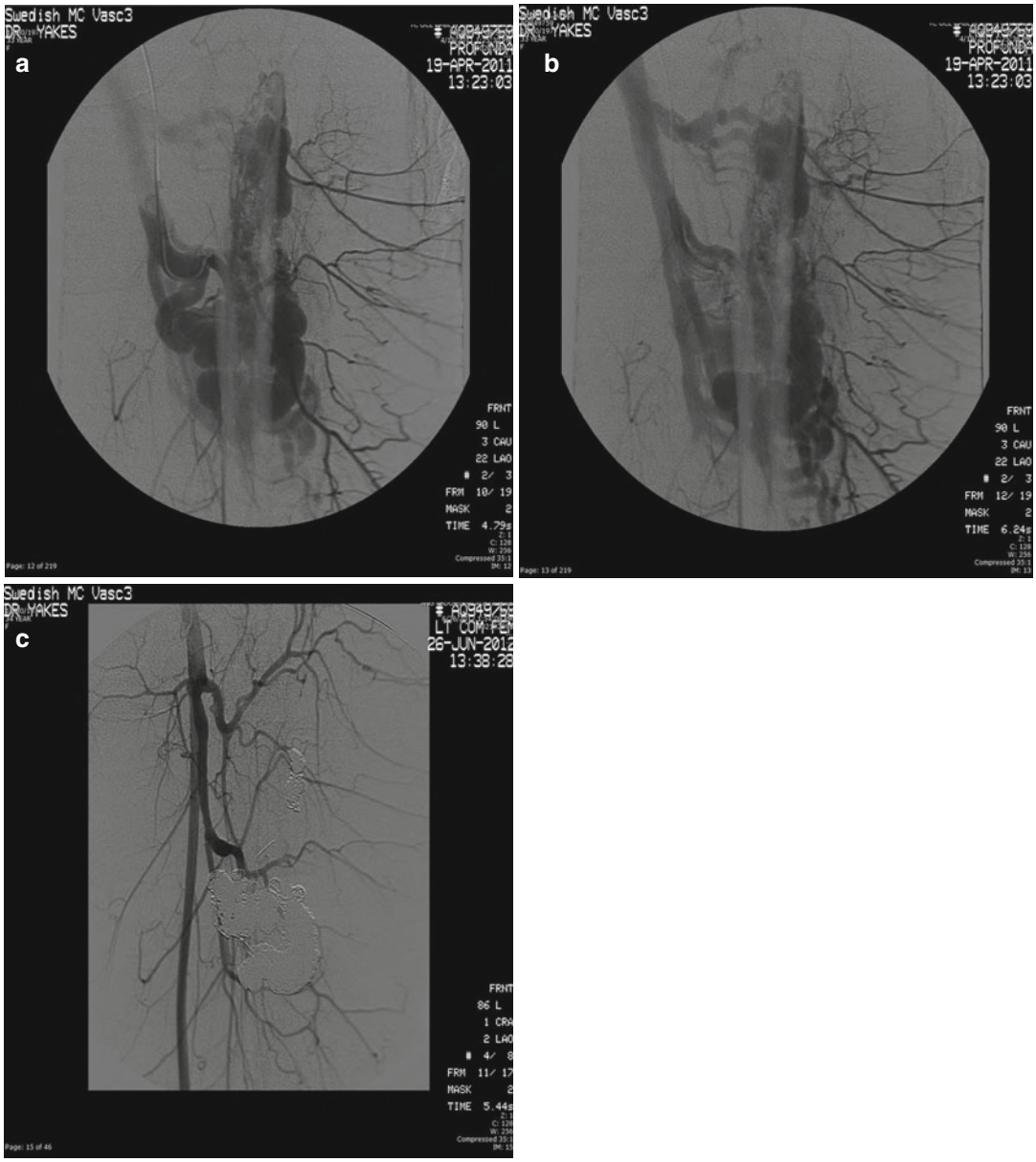

Fig. 33.4 Example of Yakes Type IIIb AVM typified by multiple inflow arteries/arterioles shunting into the aneurysmal vein with multiple out-flow veins. The "nidus" is the vein wall with the innumerable AV connections. (**a**) Left soft tissue and intraosseous left Femur AVM. Multiple arterial inflow branches from the Left Profunda Femoris Artery into the AVM. The arterial/arteriolar inflow has many parenchymal branches and also provides vascular supply to the AVM. (**b**) Venous phase of the Left Profunda Femoris arteriogram demonstrating the vein aneurysms and multiple out-flow vein physiology. To treat this Yakes Type IIIb AVM, multiple veins must be occluded to completely treat this AVM. Transarterial embolization is difficult to perform in that tissue necrosis could occur due to the many parenchymal arterial branches also arising from these multiple AVM feeding branches. (**c**) Left Common Femoral arteriogram at over one year follow-up demonstrating cure of the soft tissue and intraosseous AVM components. Note the multiple coil placements required to treat the multiple out-flow vein compartments that are present in Yakes IIIb AVMs

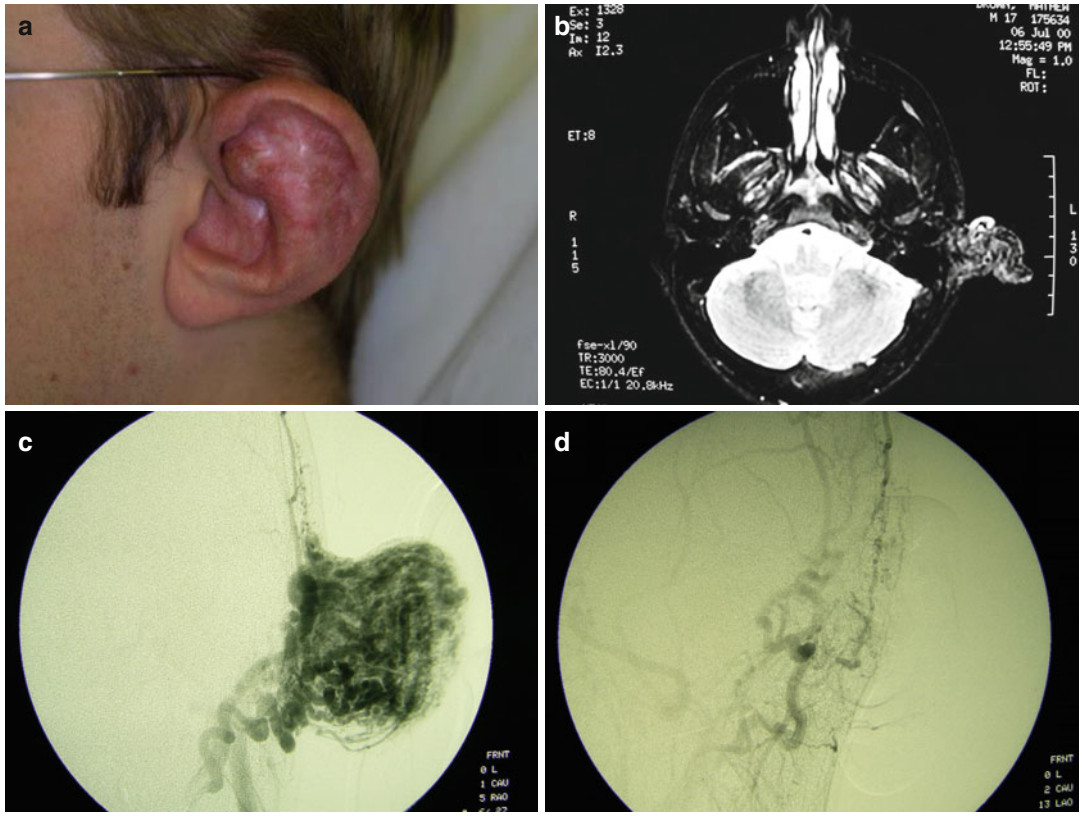

Fig. 33.5 Example of Yakes Type IV AVM typified by total infiltration of a tissue with innumerable micro-fistulas and innumerable outflow-veins. Capillaries are admixed with the innumerable fistulas throughout the tissue involved with this "infiltrative" form of AVM. (**a**) 19 year old male with progressively enlarging left ear over the last 5 years. Now has developed intermittent ulcerations, infections, and hemorrhages (Shobinger III stage). (**b**) T-2 weighted axial MR demonstrating an enlarged left ear with flow-voids totally infiltrating the entire ear and cartilages. The enlargement of the abnormal ear tissues is apparent. (**c**) AP External Carotid arteriogram demonstrating diffuse vascular infiltration of the left ear with innumerable micro-fistulae shunting into abnormal arterialized veins. The arteriogram mirrors the findings noted on the MR with ear enlargement and total micro-fistulous AVM infiltration and AV shunting evidenced by the MR flow-voids. (**d**) AP Left External Carotid arteriogram at 4 year follow-up demonstrating persistent cure of the left ear AVM. Note the normal vascularity that is now present and the total absence of any residual AV shunting post-endovascular ethanol sclerotherapy

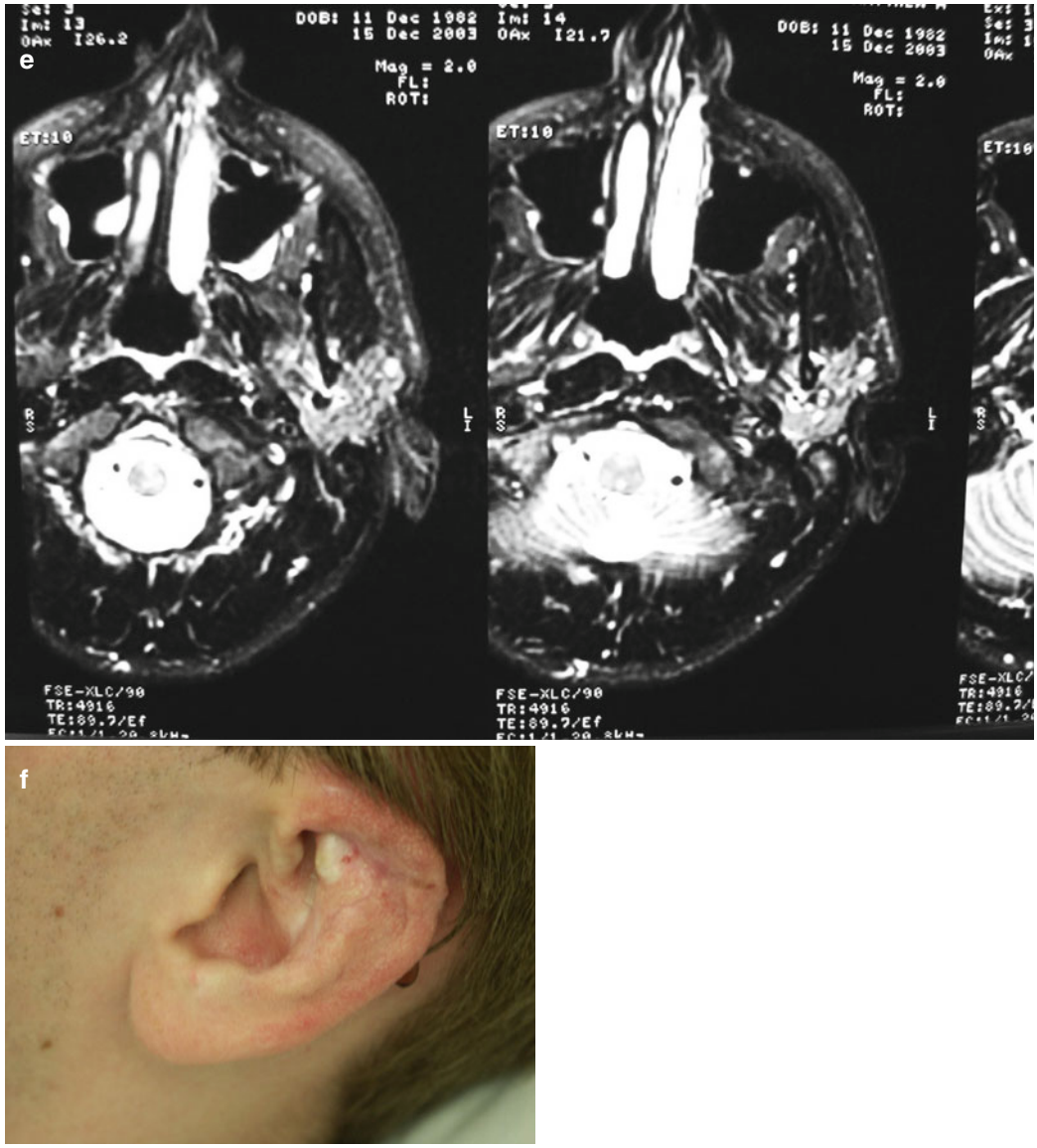

Fig. 33.5 (continued) (**e**) Axial MR T-2 weighted at 3 year follow-up demonstrating shrinkage of the left ear and normalization of the vascularity with total absence of the innumerable flow-voids previously present on the pre-treatment MR. (**f**) 4 year clinical follow-up of the left ear. A successful plastic surgery procedure was performed on the superior aspect of the left ear after the ear AVM was totally ablated and cured. The ear now has a more normal contour. Note the normalization of the skin color, no residual ruborous venous hypertensive skin changes are present

Embolic Agents Employed in the Yakes AVM Classification

Yakes Type I direct AV connections, as typically seen in pulmonary AVF and renal AVF, can be permanently ablated by occluding mechanical devices. Coils, Amplatzer plugs, occluders, detachable balloons, and the like are universally successful to cure Yakes Type I AVMs.

Yakes Type II AVMs with the "nidum" nest-like angioarchitecture can be permanently ablated with absolute ethanol from a superselective transcatheter/trans-microcatheter arterial approach. Also, a direct puncture into the artery(ies) supplying the

AVM immediately proximal to the AVM "nidum" and distal to any parenchymal arterial branches and then a superselective ethanol injection can be employed to circumvent catheterization obstacles when a transcatheter/trans-microcatheter positioning to achieve the same position to deliver ethanol into the "nidum" is not possible. These two transarterial approaches allow ethanol to sclerose and permanently ablate the "nidum." The "nidum" itself can be directly punctured, and ethanol (undiluted) can be injected to sclerose the "nidum" directly to effect cure in its multiple compartments as well.

Yakes Type IIIa AVMs (multiple inflow arteries into an aneurysmal vein with single enlarged vein outflow) and Yakes Type IIIb AVMs (multiple inflow arteries into an aneurysmal vein with multiple enlarged outflow veins) can be curatively treated by several endovascular approaches. The "nidum" in this type of angioarchitecture with an aneurysmal vein is in the vein wall itself. Superselective transarterial ethanol embolization distal to all parenchymal branches via transcatheter/trans-microcatheter and direct puncture endovascular approaches can be curative. An additional curative endovascular approach for Type IIIa AVMs is to coil embolize the aneurysmal vein itself with, or without, concurrent ethanol injection into the coils within the aneurysmal vein. This is also curative when the aneurysmal vein is totally and densely packed with coils. The aneurysmal vein can be endovascularly approached by direct 18 g needle puncture and by retrograde vein catheterization to achieve the same position within the aneurysmal vein to pack it with coils. The retrograde vein approach to curatively treat high-flow vascular lesions was first published and illustrated in 1990 by Yakes et al. The second article articulating the vein approach to AVM treatment was subsequently published in 1996 by Jackson et al. Cures were documented in these published patient series. Yakes et al. described cures of posttraumatic and congenital high-flow lesions, and Jackson et al. described cures of congenital AVMs by way of the retrograde vein approach in these publications [4, 5].

The Yakes Type IIIb AVMs (aneurysmal vein with enlarged multiple outflow veins) can be cured by transarterial transcatheter ethanol embolization and by direct puncture and retrograde vein coiling techniques. However, the aneurysmal vein portion and the immediate adjacent segments of each outflow vein must also be packed with coils completely to achieve cure. Yakes Type IIIb AVMs are more challenging to cure than the Yakes Type IIIa AVMs due to the more complex vein outflow morphology.

Yakes Type IV AVMs presented a unique challenge to determine curative endovascular treatment. AVMs, by definition, are direct AV connections without an intervening capillary bed (Yakes Types I–IV). Thus, superselective catheter and direct puncture needle positioning distal to *ALL* branches supplying parenchyma and immediately proximal to the AVM itself will obviate tissue necrosis being that the capillary beds are not embolized and only the abnormal AV connections are sclerosed. However, Yakes Type IV AVMs infiltrate an entire tissue, thus termed by the authors as an "infiltrative" form of AVM. Being that the "infiltrated" tissue (e.g., auricular AVMs) is viable proves that capillary beds are undoubtedly interspersed along with the innumerable microfistulae throughout the involved tissue as well. Injection of ethanol by transcatheter/trans-microcatheter and direct puncture approaches will sclerose the innumerable microfistulae, but also would flood the capillary beds with ethanol devitalizing that infiltrated tissue. Necrosis of that tissue would then ensue with occlusion of the capillary beds. Thus, Yakes Type IV AVMs were a conundrum to treat with endovascular approaches. Polymerizing agents would also occlude AVFs, but also capillary beds causing a massive necrosis.

Thinking through this conundrum, one could rightly conclude that the only option is total surgical resection of that entire tissue as the only treatment option. After further reflection, an endovascular option for curative treatment, not palliative treatment, was considered a possibility. Capillary beds have normal peripheral resistance which is a somewhat restrictive vascular flow pattern from artery to capillary to veins. AVMs/AVF has abnormally lowered peripheral vascular resistance with rapid stunting into arterialized veins. The arterialized AVM outflow veins are

hypertensive. In AVMS, the normotensive post-capillary venules compete with the arterialized hypertensive post-AVF veins/venules for outflow of the blood. This then further restricts normal vein outflow, which in turn increases the systemic vascular resistance (SVR) of the normal arterioles immediately proximal to the capillary beds, further restricting arteriolar inflow to the capillary beds. The increased SVR into the capillaries coupled with abnormally low-resistance shunting into the admixed innumerable AVF allows preferential flow into the AVFs.

Mixing nonionic contrast with absolute ethanol changes the viscosity and specific gravity of ethanol in this mixture. Being "thickened" and diluted, this allows for preferential flow to the AVFs and further restricts flow into the capillaries. Despite being 50 % diluted with contrast, the ethanol can still effectively sclerose the innumerable microfistulae, due to the small luminal diameters. This combination of preferential flow into the innumerable AVFs, the increased SVR into the capillaries restricting flow, and the increased viscosity and changing the specific gravity of the contrast and ethanol 50 % mixture all work to spare the capillaries and sclerose the innumerable AVFs. Using pure ethanol would not have this capillary sparing effect, and the AVFs and capillaries would both be sclerosed and occluded. This does cure the AVFs, but devitalizes the tissue itself with occlusion of the capillaries. Use of various polymerizing embolic occlusive agents (NBCA, Onyx) would also cause the same devitalization of the tissues with occlusion of the capillaries. Particulate embolic agents (PVA, Contour Embolic, Embospheres, etc.) cannot permanently occlude the AVFs and will make the capillaries ischemic with the proximal occlusion in the inflow arterioles, but will not devitalize the tissues.

Summary

Yakes Type I: Can be permanently occluded, with mechanical devices such as coils, fibered coils, Amplatzer plugs, and other occluding devices.

Yakes Type II: Can be permanently occluded with undiluted absolute ethanol. At times slowing the arterial inflow in the "nidum" with occlusion balloons, tourniquets, and blood pressure cuffs does allow for less ethanol to be used to treat the AVM compartments. Direct puncture techniques into the inflow artery or AVM "nidum" allow ethanol to embolize the AVM as well.

Yakes Type IIIa: Can be permanently occluded with transarterial embolizations with ethanol of the "nidum" the same way as in the Yakes Type II AVM. They can also be permanently occluded by dense coil packing of the vein aneurysm with or without ethanol embolization. This can be accomplished via direct puncture of the vein aneurysm or by retrograde vein catheterization of the vein aneurysm.

Yakes Type IIIb: Can be permanently occluded via transarterial approach as in Yakes Type II AVMs. They can be permanently occluded by treating the vein aneurysm and the multiple aneurismal outflow veins by coil embolization.

Yakes Type IV: Can be permanently occluded via transarterial superselective 50 % mixture of nonionic contrast and ethanol that treats the micro-AVFs and spares the higher-resistance capillaries. Direct puncture with 23 gauge needles into the microfistulous AV connection itself (thus bypassing any capillaries) with pure undiluted ethanol injections is also curative.

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