Vascular Embolootherapy
A Comprehensive Approach
Volume 2
Oncology, Trauma, Gene Therapy, Vascular Malformations, and Neck

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Springer
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Vascular Embolotherapy
A Comprehensive Approach

Volume 2
Oncology, Trauma, Gene Therapy, Vascular Malformations, and Neck

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Foreword by
A. L. Baert

With 139 Figures in 368 Separate Illustrations, 26 in Color and 56 Tables

Springer
To my parents
a wellspring of love and support without limit.
I owe you everything.

To my wonderful wife, Elham
and my children Sina and Sadra
Dr. Golzarian

To my wife, Shuzhen, and daughter, Yue
for their selfless support
Dr. Sun

To my wife Lucy, and children Jacob and Evan
Dr. Sharafuddin

To all our teachers
Foreword

Percutaneous image-guided treatment is now well recognized as an effective minimally invasive treatment modality in modern medicine. Its field of application is growing every year due to the availability of more and more sophisticated materials, tools and devices, but also because of the technical progress in reduction of the dose of ionizing irradiation incurred by both patient and radiologist during fluoroscopy.

Vascular embolotherapy is now one of the main forms of endovascular percutaneous treatment of diseases of the vascular system.

The editors of the two volumes of “Vascular Embolotherapy: a Comprehensive Approach”, J. Golzarian, S. Sun and M.J. Sharafuddin, leading experts in the field, were successful in obtaining the collaboration of many other internationally renowned interventional radiologists. I am particularly indebted to Professor Golzarian for his original concept for these books and his relentless efforts to complete the project in good time.

I would like to congratulate the editors and authors on producing these well-written, superbly illustrated and exhaustive volumes covering all aspects of vascular embolotherapy. The readers will find comprehensive up-to-date information as a source of knowledge and as a guideline for their daily clinical work.

These two outstanding books will certainly meet with high interest from interventional radiologists and vascular surgeons. They – and therefore their patients – will greatly benefit from its contents. Also referring physicians may find these books very useful to learn more about the indications, possibilities and limitations of modern vascular embolotherapy.

I am confident that these two volumes will encounter the same success with readers as the previous books in this series.

Leuven

Albert L. Baert
Therapeutic embolization has now become a major part of modern interventional practice, and its applications have become an integral component of the modern multimodality management paradigms in trauma, gastrointestinal hemorrhage and oncology, and the endovascular therapy of vascular malformations and aneurysms. The past decade has also marked the emergence of several new indications for therapeutic embolization, such as uterine fibroid embolization, and the widespread acceptance of embolization therapy as an effective non-operative management modality for major hepatic, splenic and renal injuries that once posed tremendous challenge to the trauma surgeon. Embolization therapy has also become an integral facet of the modern oncology center, offering solid-organ chemoembolization, preoperative devascularization, hepatic growth stimulation prior to resection, and direct gene therapy delivery.

Despite this remarkable growth, there are currently few references available to summarize this major field in vascular interventional therapy. The purpose of our two-volume book was to organize and present the current state of the art of embolotherapy in a comprehensive yet manageable manner. Our goal was to provide a user-friendly, well-illustrated, and easy-to-browse resource to enable both experts and novices in this field to quickly derive high-yield clinically relevant information when needed. In addition to standard applications of embolotherapy, we have also included a number of closely related applications that have become intimately associated with the field of therapeutic embolization, such as stent-graft placement and radiofrequency ablation. The two volumes constitute the combined experience of many of the leading experts in the field and have been generously supplemented with helpful tables, illustrations and detailed imaging material. We have also striven to include insightful discussions and a “cookbook” segment in each topic to provide a quick outline of procedural preparation and technique. We have included a chapter on monitoring and resuscitation of the hemorrhaging patient that should be a “must-read” for the interventionist who is not well versed in surgical critical care. Readers will also find important coverage of pathophysiology and of diagnostic clinical as well as imaging workup.

We hope this reference will meet the needs of physicians providing therapeutic embolization, whether they are trainees, recent graduates or even well-established interventionists who wish to refresh their memory or learn the opinion of some of the field’s renowned experts before embarking on a difficult case or trying a new technique or approach.

Iowa City

Jafar Golzarian
Shiliang Sun
Melhem J. Sharafuddin
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Vascular Malformation
1 Percutaneous Management of Hemangiomas and Vascular Malformations

Francis Marshalleck and Matthew S. Johnson

Although the descriptive classification allowed differentiation between benign and more serious forms of vascular malformations, because many different malformations can have similar external appearances, it was limited in its value in differentiating between them. The histopathological classification [1] represented an improvement in the attempt to classify vascular malformations. Its broad use of the word “hemangioma” and lack of clinical correlation limited its usefulness because hemangiomas and vascular malformations differ in pathology and are treated differently. The embryological classification [1] was based on the theory that vascular malformations were due to improper development of various cellular lines (arteries, veins, capillaries, and lymphatics). Although the premise was sound, the embryological classification was not clinically useful to direct treatment. To date, the most pertinent classification of vascular birthmarks has been published by John Mulliken and Julie Glowaki [1–3]. This biological classification separates vascular birthmarks into hemangiomas (vascular tumors) and vascular malformations (malformed vessels) (Table 1.1). Hemangiomas are characterized by a proliferating phase and subsequent involution phase, distinguishing them from vascular malformations which do not spontaneously involute. Vascular malformations may be high-flow lesions (e.g. arteriovenous malformations, arteriovenous fistulae) or low-flow lesions (e.g. venous malformations, capillary malformations, lymphatic malformations, combined or mixed lesions). Vascular malformations are best managed by a vascular anomalies team in a facility equipped and experienced in the management of vascular anomalies. Such a vascular anomalies team might include a vascular interventionalist, dermatologist, plastic surgeon, orthopedic surgeon and/or neurosurgeon, pediatrician, and physiotherapist. The percutaneous management of these lesions including clinical diagnosis, radiological diagnosis, percutaneous treatment (embolization, sclerotherapy) and post-procedure care will be discussed.

1.1 Classification

Vascular birthmarks have intrigued physicians for centuries. Many attempts have been made to classify vascular birthmarks, resulting in much confusion. Historically, various classifications have been developed, each with its own shortcomings. Initially, classifications were largely descriptive [1].

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1.1.1 Hemangioma

A hemangioma is a benign vascular endothelial cell neoplasm characterized by a period of intense cellular and endothelial proliferation resulting in the formation of a cellular mass. During the proliferation phase, there is formation of new feeding and draining vessels similar to that of a high-flow vascular malformation. Proliferation is followed by involution and finally regression. This distinguishes a hemangioma from a vascular malformation.

1.1.1.1 Clinical Presentation

Unlike vascular malformations, hemangiomas are not commonly present at birth but usually become evident during the first month of life. They are more common in Caucasians, females, and premature infants and have a predilection for the head and neck. Hemangiomas are the most common tumor of infancy with a reported incidence of 10%–12% [4, 5].

A hemangioma’s location determines its presentation. When it is superficial, it typically presents as a small red macule or patch which proliferates at a rapid rate during the first 6–12 months of life. A superficial lesion may produce a mass (a “strawberry” lesion) which can grow so large as to become disfiguring. The strawberry appearance is produced by the presence of multiple reddened superficial vessels which result in an irregular raised “pebbly” surface ([4]Fig. 1.1). When the hemangioma is deeper in location, the overlying skin may in fact be normal in color or may show bluish discoloration. The mass is usually warm and may be pulsatile during the proliferative phase. After the first 12 months of life, the majority of hemangiomas undergo an involution phase which can last more than 5 years. Complete resolution of hemangiomas occurs in greater than 50% of children by age 5 years and in over 70% by the age of 7 years [1]. As the hemangioma involutes, it softens, shrinks, loses its red color and becomes dull grey due to its replacement with fibrofatty tissue. Depending on the original size of the hemangioma, the overlying skin may become loose with a “crepe paper”-like appearance. Occasionally, scars or telangiectasias are seen at the site of an involuted hemangioma [4].

Complications of hemangiomas usually occur during the first 6 months of life. The most common complication is ulceration, which occurs in up to 10% of patients, especially when the lips or genital areas are involved [1, 4]. Occasionally, there may be associated bleeding, which is usually not significant. Hemangiomas may also result in congestive cardiac failure (e.g. hepatic hemangioendotheliomas) or platelet consumption (Kasabach-Merritt phenomenon). Both entities will be discussed later in this chapter. When diffuse, hemangiomas may compromise the airway, obstruct vision, or impair hearing [1]. Associated osseous deformities are uncommon [1]. Rarely, hemangiomas may be associated with other anomalies, such as posterior fossa malformations, right aortic arch, coarctation of the aorta, genitourinary anomalies, and spinal dysraphism [6].

1.1.2 Diagnostic Imaging

Hemangiomas, when superficial, are easily diagnosed clinically as previously discussed. Appropriate treatment of a symptomatic hemangioma,
however, requires delineation of its extent. Diagnostic imaging is also useful when the diagnosis is in doubt. On CT and MR imaging, hemangiomas are well-circumscribed lobulated masses that demonstrate intense parenchymal enhancement following the administration of intravenous contrast (Fig. 1.2a,b). During the proliferating phase, dilated vessels representing feeding arteries and draining veins are seen. MR is the optimal modality for the diagnosis and evaluation of hemangiomas [5]. The vessels are seen as flow voids on T1- and T2 (spin echo)-weighted MR images. A proliferating hemangioma is hypointense to muscle on T1-weighted images and hyperintense on T2-weighted images. During involution, there may be a preponderance of fat (high signal on T1-weighted images) with lack of flow voids. If a lesion lacks the classic clinical and imaging findings already discussed for a hemangioma, then a biopsy should be performed to exclude other potentially more serious tumors such as rhabdomyosarcoma, infantile fibrosarcoma, or neurofibroma.

1.1.1.3 Treatment

About 75% of hemangiomas will regress on their own without treatment [1, 4]. Multiple factors will determine whether a hemangioma requires treatment, including the child’s age and emotional needs, the location of the lesion, and symptomatology. When hemangiomas are small or are already decreasing in size before the child enters school, observation and reassurance are all that is needed. When treatment is deemed necessary, systemic corticosteroids have been the therapeutic mainstay, with a nearly 90% response [8]. Side effects of systemic steroids include gastrointestinal symptoms, weight gain, hypertension, immunosuppression, and growth retardation [7–9]. Intralosional corticosteroids have been used to treat rapidly growing hemangiomas with the dose limited by the size of the hemangioma [7–9]. Intralosional corticosteroids have been used to treat rapidly growing hemangiomas with the dose limited by the size of the hemangioma [7–9]. When steroids fail to cause adequate response, alpha interferon, chemotherapeutic agents, and radiotherapy have also been used [10–12]. The use of α-interferon is now limited to refractory cases due to its effects on the central nervous system such as spastic diplegia [13]. Laser therapy has been used to treat areas of ulceration, bleeding, telangiectasias, and skin discoloration [14]. Surgical removal becomes warranted in cases of ocular hemangioma unresponsive to medical therapy and for airway compromise. Cosmetic needs may dictate surgical removal depending on the parents’ and patient’s wishes especially for head and neck hemangiomas. After involution, surgical resection may be required to remove excess skin and fibrofatty tissue [4]. In the minority of cases in which a hemangioma fails to involute (noninvolving hemangioma) despite medical management, surgical resection of the lesion, if possible, is indicated [15]. Percutaneous embolization prior to surgical resection has also been successful [16]. Rarely, arterial embolization is required to treat life-threatening hemorrhage, high-output cardiac failure, or platelet consumption (Kasabach-Merritt phenomenon) ([17], Fig. 1.3a–c).

1.1.2 Kaposiform Hemangioendothelioma

Kaposiform hemangioendothelioma is an infiltrative variant of pediatric hemangioma. It commonly affects the trunk and extremities, producing an edematous mass of variable size with purple skin discoloration (Fig.1.4a,b). It proliferates and involutes like a typical hemangioma but persists, infiltrates, and consumes platelets (Kasabach-Merritt phenomenon) resulting in hemorrhage [18, 19]. Rarely, it may resemble a classic hemangioma [20]. Platelets decrease to low levels (< 5000) despite repeated transfusions. Management involves a multidisciplinary approach [21]. Chemotherapy, ste-
Fig 1.3. a Giant hemangioma of the trunk resulting in Kasabach-Merritt phenomenon. b CT image demonstrating parenchymal enhancement with contrast administration. c Angiographic images demonstrating multiple enlarged feeders. This lesion was embolized with PVA particles prior to surgical resection.

Fig 1.4. a Kaposiform Hemangioendothelioma presenting as an edematous purple discoloration of the trunk. b Angiographic findings in the same patient demonstrating a diffuse parenchymal blush with multiple enlarged feeders. (Images provided by Dr. Phillip John, MD)