Pan Arab Angiology Journal

International Health Academy

Original Article

INTERVENTIONAL RADIOLOGICAL MANAGEMENT OF EXTREMITIES ARTERIOVENOUS MALFORMATION

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ARTICLE INFORMATION:

Article History
Received: 26 February 2012
Accepted in revised form:
26 November 2012
Published: 26 November 2012

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Keywords:
Arteriovenousmalformations, arteriovenous fistula, Haemangioma.

ABSTRCAT:

Arteriovenous malformations represent a direct connection between the arterial and the venous systems. AVMs are usually present at birth but may not be clinically evident. They become evident in childhood and are often exacerbated during puberty or pregnancy.Closer examination reveals increased temperature, dilated veins, and a thrill that is usually noted at palpation. These lesions can be dangerous when they are in evolution. Cutaneous ischemia with ulceration or infection and haemorrhage are the most common local complications. If the malformation is extensive, high-output cardiac failure can be seen. Vascular malformations of the extremities present a difficult therapeutic challenge. Ligation of feeding vessels may lead to tissue necrosis and limb loss and can make subsequent attempts at transcatheter therapy impossible. The purpose of this study was to review our results with transcatheter and percutanous embolization therapy in symptomatic extremities vascular malformations in 30 patients seen at our interventional unite.

معلومات المقال

تاريخ المقال:
أستلم في:26 نوفمبير 2012
قبل بعد المراجعة في:26 نوفمبير 2012
نشر في: 26 نوفمبير2012

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تشوهات شريووريدية، نصور شريووريدي، ورم وعائي حميد

الملخص العربي:

علاج الأورام الليفيه الرحميه ذات الأعراض بالأنصمام الشرياني الرحمي

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التشوهات الشريوريدية تمثل إتصالاً مباشراً بين الجهازين الشرياني والوريدي. وهذه التشوهات قد توجد من الولادة ولكنها لاتظهر سريرياً إلا بعد فترة أثناء مرحلة الطفولة. عند الفحص الريري يلاحظ إرتفاع في درجة الحرارة، تمدد وريدي و رعشة يحس أثناء الجس. هذه التشوهات قد تشكل خطورة عند إستفحالها. القفار الجلدي مع التقرح أو الإخماج و النزيف هي أكثر المضاعفات الموضعية الممكنة الحدوث. إذا كانت التشوهات الشريووريدية كبيرة فيمكن أن يؤدي ذلك إلى هبوط في القلب بسبب كثرة الضخ تمثل التشوهات الشريووريدية في الأطراف تحدي كبير في علاجها. ربط الأوعية المغذية ربما يقود إلى تماوت نسيجي وربما يؤدي إلى فقدان الطرف كلية، ويجعل إمكانية التداخل العلاجي بالقسطرة غير ممكن. إن الهدف من هذه الدراسة هو مراجعة نتائجنا في علاج 30 حالة من هذه التشوهات عن طريق صمها بإستخدام القسطرة الوعائية في أطراف تعاني من أعراض واضحة.

Citation: Pan Arab J 2012, 1:271011 - http://dx.doi.org/10.5542/LDJ.v3i0v0.271011

INTRODUCTION:

Vascular malformations (VMs) are developmental abnormalities of vascular system. They should be differentiated from vascular tumors or hemangiomas, because they have different causes, growth patterns, treatments, and outcomes. Malformations may involve any segment of the vascular tree: arteries, capillaries, veins, or lymphatics. High-flow arteriovenous malformations are associated with shunting of large amounts of arterial blood into the venous system; these lesions can have alarming hemodynamic manifestations, such as venous engorgement, distal limb ischemia, and high-output cardiac failure. Predominantly venous malformations are the most common type seen at vascular clinics; most have a benign clinical course and require no special treatment. In one series, the ratio of venous to arteriovenous malformations was 4:1. [4] Most VMs are mixed, and some complex malformations such as Klippel-Trénaunay syndrome , or Parkes Weber syndrome are associated with developmental abnormalities of other tissues, including bone and soft tissue overgrowth or digital abnormalities [5].Haemangiomas are common and distinguished by endothelial proliferation, characterized by a phase of rapid postnatal growth followed by slow involution. These lesions are rarely treated surgically unless for recalcitrant ulceration or bleeding or if they cause a functional deficit, such as dyspnoea, or obstruction of the upper eyelid. Involution is nearly always complete by ten years of age[6].Vascular malformations have a different origin. They are rare congenital lesions caused by a defect during vascular embryogenesis. By definition they are always present at birth, but sometimes only become clinically evident later in life[7]. They can be divided into either high- or low-flow lesions. Any lesion with an arterial component is considered a high flow lesion. Vascular malformations can be anatomically divided into either capillary, venous, lymphatic or arterial, or combinations of the above. Vascular malformations usually grow proportionally with the child, but sudden progression can be seen secondary to trauma, thrombosis, sepsis, they can be divided into either high- or low-flow lesions. Any lesion with an arterial component is considered a high flow lesion. Vascular malformations can be anatomically divided into either capillary, venous, lymphatic or arterial, or combinations of the above. Vascular malformations usually grow proportionally with the child, but sudden progression can be seen secondary to trauma, thrombosis, sepsis, hormonal changes or surgical intervention [7]. The clinical manifestations of these lesions can vary considerably, ranging from small inconspicuous capillary malformations (port-wine stains), to large arteriovenous malformations causing overflow congestive heart failure. Most present at an early age. Late presentation is a feature of an arteriovenous malformation.Skeletal changes are commonly associated with vascular malformations while they are rarely seen in conjunction with haemangiomas [8]. There are only a few studies which have investigated the clinical symptoms and signs of vascular malformations with associated osseous involvement [9].

Table1. ISSVA Classification of Vascular Anomalies:

Vascular anomalies		
Vascular tumors	Vascular malformations	
Hemangiomas others	Capillary malformation Lymphatic malformation Venous malformation	Arteriovenous fistula Arteriovenous malformation Capillary venous malformation Capillary-lymphatic-venous malformation Lymphatic-venous malformation Capillary –lymphatic-arterio-venous malformation

*ISSVA = International Society for the Study of Vascular Anomalies ISSVA, International Society for the Study of Vascular Anomalies. Based on Scientific Committee of the 11th Meeting of the International Society for the Study of Vascular Anomalies, Rome, Italy, 1996.

MATERIAL AND METHODS:

Between January 2007 to Dec 2010, 30 patients were referred to us from vascular surgeons with vascular anomalies, 63.3% of our patients are male (n=19), and 36.6% were female (n=11), between age of 9 to 42 (mean age 26), 36.6% (n=11) complaint of pain, 60% had palpable lesion (n=18), 50% had palpable pulsation or thrill (n=15), 83% (n=25) complaining of cosmetic reasons, 33.3% (n=10) complaint of pain and cosmetic and 6.6% (n=2) complaint of ulcers. Seventy percent of cases (n=21) seen at lower limb and 30% (n=9) involving upper limb. The vascular anomalies were categorized according to classification of International Society for the Study of Vascular Anomalies (Table1), into haemangiomas (n=5) and vascular malformation

(n-25). Although abnormal vascular channels are presumably present at birth, not all malformations were congenital. Twenty five out of thirty (83.3%) were presented at childhood while five of them manifested at birth. History of trauma which trigger appearance of malformations were noticed in 30% (n=9), while history of previous biopsy or attempted surgical excision were obtained in 16.6% (n=5). Seventy one of cases seen at lower limb (15 out of 21) showed no spared region. All the AVM (n=25) were classified according to Jackson et al and flow related classification, Table2.

Table2. Classification of surface vascular lesions

A: Traditional classifications
- capillary hemangioma
- strawberry hemangioma
- strawberry nevus
- port wine stain
- Flame nevus
- Cavernous hemangioma
- Venous angioma
- Lymphangioma
- Arteriovenous malformation
B: Classification of jackson et al
- Hemangioma
- Vascular malformation
- Low-flow lesion
- High-flow lesion
- Lymphatic malformation
C: Flow related classifications and recommended treatment
- Slow-flow lesion: Sclerotherapy
- Intermediate-flow lesion: Sclerotherapy (plus embolization).
High-flow lesion: Embolization (plus sclerotherapy

into fast flow type 72% (n=18) and low flow type 28% (n=7), depending on MRI findings, malformations with arterial components are considered high-flow lesions which also evolved with prominent thrills and bruits, and those without arterial components are considered low-flow lesions. Pre-embolization planning with color Duplex was used to confirm the presence of fast flow anomalies and to separate haemangioma from malformation. MR imaging was done for all our patients for classification and to delineate the anatomic relation between the vascular lesion and adjacent organs, nerves, tendons, and muscles. Pre embolization diagnostic conventional angiography was done for all patients with malformation , images were obtained at the rate of 2-3 frames/s. Non-ionic contrast agents (UltravistR [iopromid] 370 IU per 100 ml, Schering-Germany, and Omnipaque R [iohexol] 350 IU 100 per ml, Nycomed, Ireland) were administered using an Angiomat 6000 (Liebel-Flarsheim Company, Cincinnati, OH, USA) automatic injector. After administration of general anesthesia, a 5F or 6F introducer was placed in the femoral artery with a modified Seldinger method using a single wall needle. Bentson (Boston Scientific, Cedex, France) or Glide Wire (Kimal, Middlesex, England) guide wires were used. Diagnostic angiography was performed using a standard vascular angiography catheter, based on lesion location. Vascularity and localization of feeding arteries, relationships to neighboring vascular formations, and the existence of an arteriovenous fistula (AVF) nidus were angiographically evaluated. For all high flow AVM (n=18) distal catheterization was achieved using a diagnostic catheter, super selective catheterization was performed with micro catheter (Rebar by Micro Therapeutics, Inc. ev3, California, USA). Embolization of all AVM were performed with non-adhesive liquid embolic agent (Onyx 34, by Micro Therapeutics, Inc. ev3,California, USA) which is consistent of ethylene vinyl-alcohol copolymer , it was administered carefully and slowly with hand injection under road map and fluoroscopy control, avoiding any back flow and for better delineation and controlling of onyx filling through AVM. Additional angiography after completion of the procedure was performed for all cases to evaluate technical success of embolization. We considered embolization technically complete when there filling and opacification of AVM nidus with onyx, Fig1.

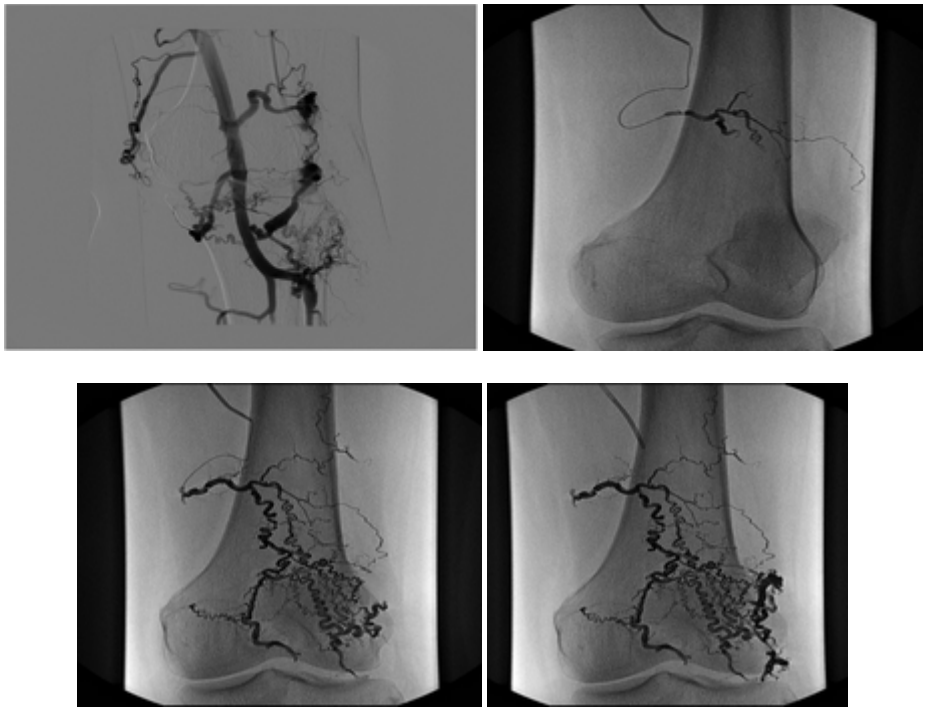

Figure1. A. Left lower limb angiogram showing AVM with multiple feeders. B. Coaxial selective cannulation of one of the feeders with rebar microcatheter. C. Gradual injection of ONYX with partial opacification of nidus of AVM. D. Full obliteration of AVM nidus.

ALL low flow AVM (N=7) and four cases of haemangioma underwent percutanous sclerotherapy, procedure was done under general anesthesia, guided with ultrasound and color Doppler scanning, 20 G short spinal needle was directly inserted through into mass lesion (haemangioma) or nidus in low flow AVM after applying tourniquet distal to lesion, phelebography was done, by slowly injection of contrast under DSA scanning till opacification of draining vein, amount of injected contrast was calculated and replaced with the same volume of sclerosant material, seven cases of low flow AVMs and two cases of haemangiomas were injected with (dehydrated alcohol, injection USP, 100% v/v; Sandoz Canada, Inc., Boucherville, Quebec, Canada), we used ethanolamine oleate (Oldamin for injection; Grelan Pharmaceutical, Tokyo, Japan) for the other two cases of haemangiomas ,Fig2. All cases were followed by MIR scanning after one, 6, 12, 18 and 24 months in correlation with clinical data

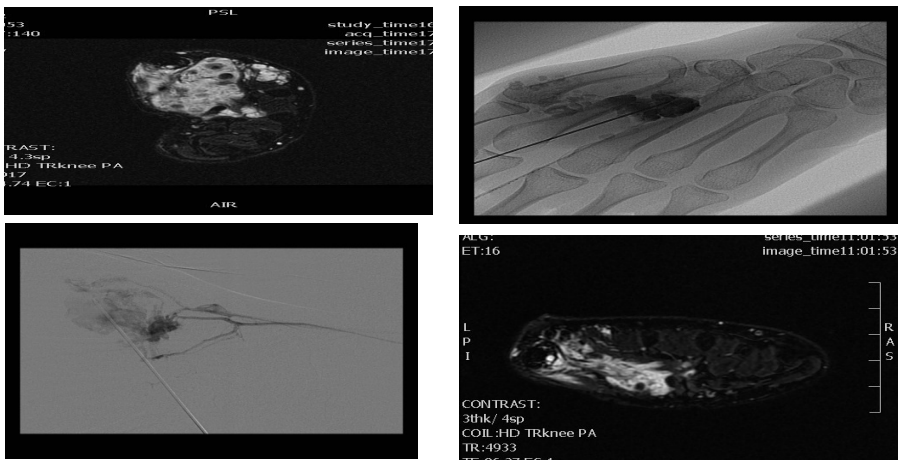

Figure2. A. Axial MRI FSAT T2W image showing sizable thenar and hypothenar haemangioma. B. Percutanous phelebography. C. DSA phelebography before embolization. D. Post embolization MRI showing significant reduction of haemangioma size.

RESULT:

Forty percent of high flow AVM (n=10) needed one session of embolization, 32% (n=8) required 2 session, 20% (n=5) required three sessions, and only two patients with extensive AVM needed more than three sessions. Two cases of haemangioma and one case of low flow AVM needed two sessions of embolizations. The repeated session were done after clinical and MRI assessment with at least one month intervals between sessions. The other two cases of haemangioma acquired only one session of percutanous embolization with 45% and 53% reduction in mass volume at follow up MRI, however significant clinical improvement were achieved and no further embolization was required.Ten patients complained of local skin redness and swelling accompanied by a burning sensation. One patient developed a blister-like change in the skin, two patient showed back flow of onyx with non target embolization of muscular branches, however No major complications were encountered.

DISCUSSION:

Vascular (arteriovenous) malformations of the extremities constitute some of the most difficult diagnostic and therapeutic challenges. In 1982, Mulliken and Glowacki proposed hemangioma and vascular malformations as two major categories of lesions based on histopathologic findings and clinical features of the lesion ,Table 3. This system divides vascular anomalies into hemangiomas, which are neoplastic lesions with endothelial hyperplasia, and vascular malformations, which are congenital lesions with normal endothelial turnover [10]. In 1993, Jackson et al [11] proposed another system for classifying hemangiomas, vascular malformations, and lymphatic malformations on the basis of vascular dynamics. A classification system for vascular anomalies based on cellular features, flow characteristics, and clinical behavior was updated during the meeting of the International Society for the Study of Vascular Anomalies [12]. In 1996, Kawa nabe et al [13], reported a system for practical classification of vascular lesions in which the treatment procedure is selected according to the characteristic flow within the lesion [14].

Table3. Classifications of vascular anomalies by Mulliken and Glowacki

Vascular anomalies	
Tumors	Malformations
hemangioma	Capillary
Pyogenic granuloma	Lymphatic
Kasopiform hemangioendothelioma	Venous
Other rare tumors	Combined

The prevalence of vascular malformations is estimated to be 1.5% in the general population, most commonly involving the head and neck, followed by the lower extremities and trunk [15]. AVMs mostly involve the skin and intramuscular AVMs are rare we reported in our series 5 out of 30 patients with intramuscular AVM and three cases with intraosseous AVM, Fig3. There is no reported gender difference in incidence. Because intramuscular AVMs are rare, to our knowledge, there are no reported data on their exact incidence. The natural history of these lesions follows four stages as described by Schobinger: quiescence, expansion, destruction and decompensatio[16].Clinical examinations and patient history are usually adequate for an accurate diagnosis of these lesions. Diagnostic tests should focus on evaluating the type and extent of the malformation; the presence or absence of any arteriovenous shunting must also be established. Physical examination of limb and pelvic lesions should be complemented by segmental systolic limb pressure measurement and establishment of the ankle-brachial index. Pulse volume recording is helpful in patients with arteriovenous shunting. Placement of a tourniquet on a limb with a high-flow, high shunt arteriovenous malformation and occlusion of the fistula will increase systolic blood pressure, followed by a slowing of the heart rate due to a vagal response in the baroreceptors in the aorta and carotid arteries (bradycardia sign).

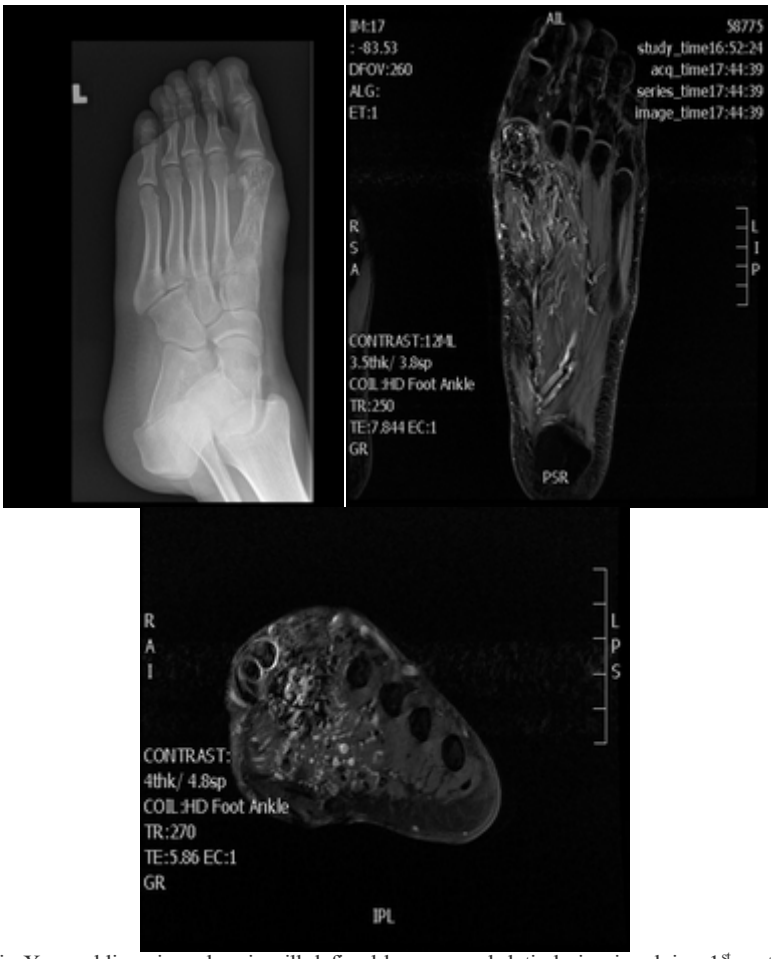

Figure3. A. left foot plain X-ray oblige view showing ill defined honey comb lytic lesion involving 1st metatarsal bone with slight expansion of the bone with cortical MRI FSAT T2W image with contrast showing evident AVM with significant bony involvement.

The two noninvasive imaging techniques that are most useful in the examination of vascular malformations are MR imaging and sonography. In our clinic, MR imaging is the primary imaging technique in the evaluation of suspected vascular malformations**.** The primary goals of imaging vascular malformations or hemangiomas include characterizing the lesion and discovering the anatomic extent of disease[17]. Knowing which tissues the vascular malformation involves and whether adjacent vital structures, such as neurovascular bundles, are involved by the lesion is important. Such information is vital to planning surgery or imaging-guided procedures. When the physical examination and clinical history are diagnostic or highly suggestive of a vascular malformation, the most important characterizing feature is whether the lesion is a high- or low-flow vascular malformation. Most information needed to examine the lesion is available from a combination of T1-weighted, fat-saturated T2-weighted, and gradient-echo (flow-weighted) MR images. Our MRI protocol for those cases included, Cor STIR, Cor T1, Axil T2 FS, Axial T1, AxialT1 SPGF FS without contrast, Axial-Coronal-Sagittal T1 SPGF FS with contrast, all of our cases were evaluated by sub-specialized musculoskeletal consultant before planning of therapy. MR imaging of proliferating hemangiomas often shows a discrete lobulated mass that is hyperintense to muscle on T2-weighted images and isointense to muscle on T1-weighted images. Typically, prominent draining veins will be identified as both central and peripheral high-flow vessels. Hemangiomas usually enhance diffusely with gadolinium involuting hemangiomas can indicate areas of fibrofatty tissue with associated high signal intensity on T1-weighted images and less contrast enhancement than that of proliferating hemangiomas[18].The appearance of a low-flow vascular malformation on MR imaging depends on the composition of lymphatic and venous components. The venous portions of a malformation will appear as a collec-

tion of serpentine structures separated by septations. These serpentine structures represent slow-flowing blood in the venous channels and appear as high signal intensity on T2-weighted images and intermediate signal intensity on T1-weighted images. Phleboliths may be present and appear as round, low-signal-intensity lesions on MR imaging. Gadolinium-enhanced T1-weighted images may show enhancement of the slow-flowing venous channels. Lymphatic components of the malformation may contain cystic structures of various sizes ranging from macrocystic to microcystic. These cystic structures typically appear as high signal intensity on T2-weighted MR images and do not exhibit central enhancement with gadolinium, fluid-fluid levels are often present [19]. Any lesion that has arterial components is considered a high-flow malformation. These include arteriovenous malformations (AVM) and arteriovenous fistulas. During the proliferating stage, infantile hemangiomas may also be considered high-flow lesions. On MR imaging, the lesions appear as a tangle of multiple flow voids that indicate high flow on gradient-echo images. Although the lesions can be associated with surrounding edema or fibrofatty stroma, usually no focal discrete soft-tissue mass is found[20].Other potential imaging sequences that have been used in the evaluation of vascular malformations include MR angiography, venography, and lymphangiography[21].

Sonography has been advocated as useful in examining soft-tissue masses that are suggestive of hemangiomas or vascular malformations [22]. Vessel density as depicted on Doppler sonography has been used in differentiating other types of masses from vascular malformations [23]. Certainly, the Doppler characteristics of vascular malformations are helpful in differentiating low-from high-flow vascular malformations. We have also found the sonographic depiction of abundant low-flow vascular channels to be a predictor for the potential success of percutaneous sclerosis [24], and we use sonography to guide needle placement during percutaneous sclerosis.Contrast arteriography is reserved for patients who are potential candidates for arterial embolization. Arteriovenous shunting is confirmed by contrast arteriography, which also delineates the feeding arteries and excludes the presence of any vascular tumor. The size of the feeding arteries can be measured, and the size of the arteriovenous shunts (2 mm in large shunts, 100 to 200 μm in small shunts) can be estimated based on the appearance of contrast in the vein. The flow volume is determined by the size and rate of opacification of the feeding arteries, whereas the shunt volume can be estimated with acceptable accuracy by the time and appearance of contrast medium in the veins. Arteriovenous shunting, also can be documented by labeled microspheres. Technetium 99m–labeled human albumin microspheres are injected into the artery proximal to an arteriovenous shunt. Less than 3% of the microspheres should pass through a normal capillary bed. The percentage of the shunted material is calculated based on radioactivity in the lungs, measured after a separate injection of the colloid in a vein of the body [25].

AVMs can prove deceptively problematic and even dangerous to treat. Thus, most authors agree that conservative treatment is preferable in the absence of significant symptoms, we have one of our patient nine years old girl with diffuse left lower limb haemangioma was treated conservatively showing significant improvement on follow up. An elastic garment or bandage, local wound care, compression dressings, special orthopedic footwear, and lifestyle modification may be required to manage daily life and improve limb function. The psychological problems caused by a visible deformity should not be underestimated. Long-term antibiotic therapy may be needed for recurrent cellulitis, and patients with recurrent deep vein thrombosis are treated with lifelong anticoagulants.Surgery was the initial form of therapy to treat AVMs. Embolization followed by surgery has proved beneficial to patients [25]. However, cure rates are almost non-existent with this method unless the AVM is very focal and in a safe anatomic area. In surgically difficult anatomic areas, it is problematic, if not impossible for a complete AVM removal to occur. Proximal ligations of arteries and skeletonization of arteries to AVMs also are documented in multiple surgery articles to fail uniformly in treating AVMs. The remaining nidus that was not removed can grow and be even more symptomatic to the patient compared with its preoperative status, in our series no one our patients was treated surgically or combined embolization with surgery.The treatment of a VM always begins with diagnostic phelebography, which serves as a final diagnostic confirmation of the suspected pathology and also demonstrates the particular malformations morphological characteristics, which, even more so than MR findings, have a strong impact on calculation of sclerosant material dose, technique, complication rate, and outcome[26]. We have done

phelebography for all of our haemangioma and low flow malformation patients in the same session before scelerotherapy . Sclerotherapy involves the introduction of a substance into the lumen of the given malformation, which will induce by direct contact the death of the adjacent endothelial lining and initiate a cascade of thrombosis and fibrosis within the lumen and within the lesion as a whole[27]. Several sclerosants are clinically available that vary in their mode of action, relative toxicity, complication rate, and therapeutic effect The most common currently used sclerosants in the literature at present are ethanol (dehydrated alcohol, injection USP, 100% v/v; Sandoz Canada, Inc., Boucherville, Quebec, Canada). Ethanol is very potent, inexpensive, and a readily available sclerosant that exerts its endothelial-cidal effect by causing the endothelial monolayer to dehydrate, precipitate, slough, and denude. However, because of this agent's high toxicity, it must be used with extreme caution to avoid collateral non-target necrosis of adjacent tissues that may occur in the setting of over injection, localized extra-vasation, or reflux either within the soft tissues or skin [28].Other currently used or historically significant sclerosant agents in the treatment of VMs include ethanolamine oleate, sodium morrhuate, and alcoholic solution of zein (Ethibloc, Ethicon, Norderstedt, Germany). There are now increasing reports in the literature describing excellent results with the use of bleomycin A5 (Pingyangmycin) in the treatment of VMs. This agent has always carried with it its long-feared chemotherapy-associated complication of pulmonary fibrosis. However, with reduced dosing (e.g., less than 1 mg/kg per session, more than 2 weeks between sessions, and no more than 5 mg/kg lifetime), this complication has not yet been reproducibly identified[29].

In treating high AVMs endovascularly, super selective catheter placement is absolutely essential. When it is not possible to approach an AVM and access its nidus with an endovascular approach, then direct percutaneous puncture techniques should be employed. If super selective placement in an AVM nidus is not possible, then the use of ethanol must be avoided. Not infrequently, inflow occlusion might be required to induce some level of decreased flow or vascular stasis in the embolized vessel to allow the full contact of ethanol with the endothelium, maximizing the thrombogenic properties of ethanol. Inflow occlusion can be achieved through the use of occlusion balloon catheters, blood pressure cuffs, tourniquets, and the like.

Neovascular recruitment phenomena and recanalizations do not occur after ethanol embolization of AVM because the endothelial cell is destroyed. Following ethanol embolization, the endothelial cells are completely denuded from the vascular wall and their protoplasm is precipitated[30]. Blood proteins are denatured, initiating the clotting cascade. When the vascular wall is denuded of its endothelium and is bare, platelet aggregation occurs with the development of thrombus formation along the vascular wall; the thrombus propagates until total thrombosis of the lumen is noted. This process can take time. Therefore usually waits at least 10 to 20 minutes before a follow-up arteriogram to determine if any additional ethanol is required to complete the thrombosis. With the destruction of the endothelial cell, the cells are no longer able to send chemotactic factor and angiogenesis factor, which may lead to permanent thrombosis and cure. The amount of ethanol used in each endovascular procedure is tailored to the flow and volume characteristics of the individual compartment of the AVM or haemangioma lesion.

No predetermined amount of ethanol is considered. Contrast injections can be practiced prior to ethanol embolization. The amount of contrast needed to completely displace blood and not reflux into the proximal artery estimates the amount of ethanol required for the embolization. It is unusual to cure an AVM in one session. In the larger lesions, we prefer to treat individual compartments serially, eventually effecting total treatment over time. We usually wait at least 4 weeks between procedures to allow the patient to return to a new baseline prior to further treatment of the lesion.Recently, the new liquid embolic agent Onyx has become available for embolization AVMs. Onyx is non adhesive and polymerizes slowly. Onyx is available in several concentrations and the high-concentrated Onyx can be used to slowly occlude large AV shunts in a more controlled way than that achieved by ethanol 20[31]. However, simple Onyx injection is not always feasible: in some very high-flow shunts Onyx may migrate through the fistula into the distal draining veins, all high flow AVMs included in our series were embolized with Onyx. For all percutaneous and endovascular embolization of vascular malformations, post-procedural orders should include frequent-interval skin and neurovascular assessments of the involved regions over several hours. The

patient should be encouraged to resume activity as tolerated as soon as possible. Post-procedural investigations may include complete blood count and urinalysis to document for hemoglobinuria. If there are concerns regarding sclerosant entering the deep venous system, Doppler assessment at the same day or the following day may be helpful to rule out deep venous thrombosis[32].

CONCLUSION:

Vascular malformations are caused by developmental abnormalities of the vascular system. They should be carefully classified based on the predominant vascular structure high or low flow types and the presence or absence of arteriovenous shunting which significantly affect the approach of management. Careful evaluation and management by a multidisciplinary team is required.

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