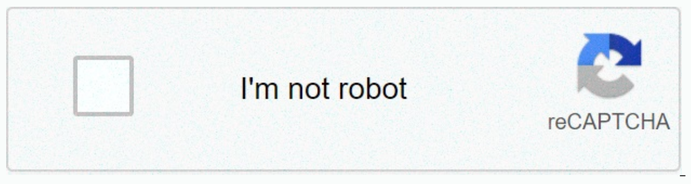

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Arteriovenous malformation pathology outlines skin

Context: Arteriovenous vascular malformations and hemangiomas are benign vascular lesions that are difficult to clinically distinguish from each other. They can also be confused with each other in histopathology. Therefore, histochemical spots for the presence of an artery are often used to distinguish these two substances. Objective: Since it is clinically important to distinguish between arteriovenous vascular malformations and hemangiomas, this study was conducted to examine other diagnostic traces that may assist in the diagnosis and differentiation of these lesions. Design: A total of 167 cases of benign extracranial vascular lesions were obtained from the anatomical pathological file of our institution. These were 66 cases diagnosed as arteriovenous vascular malformations and 101 cases previously diagnosed as hemangiomas. Hematoxylin-eosin-stained glass has been reviewed, Movat pentichrome histochemical spots have been used to identify elastic blood vessels (arteries/arterials), and S100 immunostain has been used to identify nerves in these vascular lesions. For imunohistochemistism, the method of detection of avidin-biotin was used. Results: With the Movat stain, the presence of thick-walled elastic arteries was detected in 12 out of 101 cases previously diagnosed as hemangiomas, and these cases were therefore reclassified as vascular malformations. Using the same criterion, 2 out of 66 cases originally diagnosed as arteriovenous vascular malformations were reclassified as hemangiomas because they lacked arterial structures. Thus, with this strict criterion, we ended up with 91 cases of hemangiomas and 76 cases of arteriovenous vascular malformations. Intralesional nerves have been identified in 91% (69/76) of cases of arteriovenous vascular malformations, including all 12 arteriovenous vascular malformations previously diagnosed as hemangiomas. By contrast, no intralesional nerve was detected in any of the 91 hemangiomas. Conclusions: These results show that nerve bundles are permanently present in vascular malformations and are absent in hemangiomas, so they can be used as a diagnostic guide to distinguish between these lesions. In addition to describing the previously unreported components of vascular malformations, these data further confirm the hamartomatous nature of these lesions. Page views in 2019: 5,738 Page views in 2020 to date: 6,995Cite this page: Chaux A. Arteriovenous Malformations. PathologyOutlines.com a website. . Access to November 18, 2020.Definition/General By Definition, Direct communication is present between arteriology and venules Sites More common in the CNS, intestines, lungs, limbs Very rare in the bladder Etiology Can be congenital or acquired (post-traumatic) Clinical characteristics The most common symptom is hematuria (gross or micro, persistent or intermittent, can be massive) Other symptoms include dysuria, difficulty with voiding and urinary retention Some cases Asymptomatic case reports Treatment Excision is adequate therapy Microscopic (histological) description Of admixtures of damaged blood vessels, such as capillaries, arteries and venulas Sudden changes in the thickness of the middle and elastic layer of blood vessels, abnormal vascular dilation Often advanced diseases of small vessels, bleeding, ulceration (Hum Pathol 1986;17:94) Includes submucose but not muscular proporia May be associated with pseudocarcinomatous epithelial bladder hyperplasia (Am J Surg Pathol 2008;32:92) Microscopic (histological) images hosted byImaimy on other servers : Brain images: arteriovenous malformations back to the top Vascular anomalies are a heterogeneous group of congenital disorders of blood vessels, which are usually referred to as birthmarks. Subcategorised to vascular tumors and malformations, each anomaly is characterized by a specific morphology, pathophysiology, clinical behavior and approach to management. Hemangiomas are the most common vascular tumor. Lymphatic, capillary, venous and arteriovenous malformations make up the majority of vascular malformations. This article evaluates current theory and practice in etiology, diagnosis and treatment of these more common vascular anomalies.1. IntroductionVascular anomalies are congenital lesions of abnormal vascular development. Formerly referred to as vascular birthmarks, vascular anomalies are now classified on the basis of a system developed in 1982 by Mulliken and Glowacki, which takes into account histology, biological behaviour and clinical presentation of these entities [1]. The primary difference is made between a vascular tumor that grows with cellular hyperplasia and vascular malformations, which represents a localized defect in vascular morphogenesis. Due to differences in biological and radiographic behavior, malformations are further divided into slow and fast flow lesions (Table 1). Vascular tumorsVascular malformationsSlow-flowInfantile hemangioma Capillary malformationsSecgenital hemangioma Venous malformationsSuffed angiomymic malformationsKaposiform hemangioendotheliomaFast-flow Arteriovenous malformationsOblastic tumor vessels and malformations can occur anywhere on the body. In short, hemangiomas are vascular tumors that are rarely noticeable at birth, grow rapidly during the first 6 months of life, involuntary over time and do not necessarily infiltrate, but can sometimes be destructive. Vascular malformations are irregular vascular networks defined by their particular type of blood vessels. Unlike hemangiomas, they are present at birth, slowly growing, infiltrative and destructive. Almost all vascular malformations

and almost 40% of hemangiomas eventually require intervention. This document therefore offers pediatricians up-to-date information on recent developments in the diagnosis, management and pathogenesis of vascular anomalies. Due to their complexity, a multidisciplinary approach is often necessary in managing these lesions and a team of experts in pediatric otolaryngology, dermatology, hematology, interventional radiology, surgery, orthopedics and sometimes psychology.2. HemangiomasInfantile hemangiomas are the most common tumor in childhood and occur in approximately 10% of the population. Identifiable risk factors include female sex, immaturity, low birth weight and fair skin [2]. They consist of rapidly dividing endothelial cells. Since their growth is attributed to hyperplasia of endothelial cells, they are classified as, and are the most common, vascular tumors. Hemangiomas are further divided into two types: infantile or congenital. Rare congenital hemangioma is less understood and present at birth. Congenital hemangiomas either quickly involute (rapidly inducing congenital hemangioma (RICH)) for a very short period of time in childhood or never involute (involuntary congenital hemangioma; (THEM)). The rest will focus on more common infantile hemangiomas. The pathogenesis of infantile hemangiomas remains unclear, although current thinking is dominated by two theories. The first theory suggests that hemangioma endothelial cells arise from disturbed placental tissue embedded in amniotic soft tissues during pregnancy or childbirth. Hemangioma markers have been shown to match those found in placental tissue [3]. This is further supported by the fact that they occur more frequently in infants after sampling of chorionic mares, placenta previa and preeclampsia [2]. The second theory arose from the discovery of endothelial progenitor and stem cells in the circulation of patients with hemangiomas [4]. The development of hemangiomas in stem cell animals isolated from human samples supports this theory [5]. However, infantile hemangiomas are most likely to arise from hematopatic progenitor cells (from the placenta or stem cells) in the appropriate environment of genetic changes and cytokines. Abnormal levels of metalloproteinase matrix (MMP-9) and proangiogenic factors (VEGF, b-FGF, and TGF-beta 1) play a role in hemangioma pathogenesis [6]. Genetic errors in growth factor receptors have also been shown to influence the development of haemangiomas [7].2.1. DiagnosisInfantile hemangiomas present shortly after birth most often well-defined, flat, and erythematous red spots. At this stage, hemangiomas can be confused with other red lesions of birth, but rapid proliferation and vertical growth will trigger the diagnosis (Figure 1(a)). Generally speaking, hemangiomas do not scure beyond their original anatomical boundaries. Hemangiomas follow a predictable course with three different developmental phases: proliferation, calmness and involution. In most haemangiomas, eighty percent of proliferation occurs after three months of life, but can last longer [8]. During proliferation, rapid growth can lead to exhaustion of blood supply with the resulting ischaemic, necrosis, ulceration and bleeding. (a) (b) (a) Hemangiomas may be superficial, deep or Superficial hemangioma is red and nodular without subcutaneous component. The deep hemangioma presents itself as a protruding with an insistent bluish tint or telangectasia. Compound hemangiomas have both deep and superficial components (Figure 1(b)). This new nomenclature helps eliminate confusing older terms (Table 2). Old nomenclatureNew nomenclatureStrawberry or capillary hemangiomaSuperficial hemangiomaCavernous hemangiomaDeep hemangiomaCapillary cavernous hemangiomaKomper hemangiomyproliferation, hemangiomas enter slower or no stages of growth, known as quiescence. This stage usually lasts from 9 to 12 months of age. The last and unique phase of the hemangioma life cycle is involution. This stage is marked by greying of the skin and shrinkage of deeper components (Figure 1(b)). Historical reports suggest that involution of 50%, 70% and 90% hemangioma occurs at the age of 5, 7 and 9 years with some variability [9]. In the final stages of involution, fibrofatty protrusion may remain (Figure 1(b)). Another subclassification of hemangiomas is focal versus segmental disease. Focal hemangiomas are localized, non-licular lesions that adhere to the stages of growth and involution. There is also multifocal hemangiomatosis, and infants with more than 5 lesions should undergo an examination to exclude visceral impairment. Segmental hemangiomas are more diffusely plaquey and can lead to undesirable functional and aesthetic results. The limb and face are common places for the disease (Figure 2). Lesions of the head and neck often coincide with the distribution of the trigeminal nerve. The distribution of the vein is associated with subglotic hemangioma 60% of the time [10]. Regardless, it should be assumed that a stridulous child with foal or segmental hemangioma has a subglottic disease until proven otherwise. (a) (b) (b) Patients with segmental hemangiomas should also undergo an examination to rule out PHACES syndrome (posterior fossa cerebral malformations, facial hemangiomas, arterial cerebrovascular anomalies, cardiovascular anomalies, eye anomalies and chest defects or supraumbilical raphe) [11]. Diagnosis of hemangioma is best made by clinical history and physical examination. In cases of unclear diagnosis, the best radiographic methods of use are either Doppler ultrasound or MRI.2.2. ManagementHistorically, hemangiomas have been managed with careful observation during their life cycle [9]. However, research suggests that nearly 40% of children require further intervention due to bleeding, ulceration, visual obstruction, airway obstruction, high-output heart failure, or the risk of permanent disfigurement [12]. With new treatment options, as well as a better understanding of the disease, observation decreases as the only means to treat hemangiomas. However, inconspicuous lesions are still best treated by observation itself. Medical and surgical options are available for the treatment of problematic Medical procedure involves one or more systemic therapies. Corticosteroids, interferon and vinkristin have been successful in massive and life-threatening diseases [13-15]. These substances have also been used for multifocal disease, visceral impairment, segmental distribution, airway obstruction, and periorbital lesions. However, significant side effects accompany systemic treatment and have even led to the rejection of certain substances as treatment options. Surgical treatment includes excision, laser treatment or both. Intralesional steroid treatment is also an option for focal hemangiomas mumps, nasal tip, subglottis, and eyelid. Repeated treatment is often necessary, but systemic side effects are limited [16]. Excision is suitable for localized lesions of fibrofatty residues (residuum) involuted hemangiomas. Optional subtotal excision of massive proliferation proliferating hemangiomas can be used to maintain the aesthetic boundaries of the face. Small remnants of the disease are then left for involution. Residual erythema and telangiectomy often remain in involuted hemangiomes and are best treated with selective photothermolysis using a lightning pulse laser (FPDL). Similarly, ulcerative lesions during proliferation can be treated with FPDL to infer healing and new epidermal growth.2.3. PropranololA paradigm shift has occurred in connection with the treatment of hemangiomas in the last few years. In 2008, propranolol, β non-seamant antagonist and adrenergic, was discovered to cause regression of proliferating hemangiomas in newborns treated with cardiovascular disease [17]. Numerous studies followed, demonstrating the success of propranolol for shrinking hemangiomas [17-19]. In fact, more than ninety percent of patients have a dramatic reduction in the size of their hemangiomas as early as 1-2 weeks after the first dose of propranolol (Figure 2(b)). Treatment of propranolol in the treatment of hemangiomas is recommended 2-3 mg/kg divided into regimens two or three times a day [20]. These doses are significantly lower than those used for cardiovascular conditions in children. Thus, the reported side effects of propranolol for hemangiomas were minimal. However, serious concerns about hypoglycaemia and lethargy that may occur with this medicinal product should not be sidelined [21, 22]. To address these concerns, parents are instructed to take propranolol with food, report any unusual drowsiness, and not administer it during infections. Early and frequent visits to assess vital signs are recommended in young infants during treatment. An exacerbation of esophageal reflux may result due to beta-receptor blockage in the lower esophageal sphincter [18]. Monitoring of propranolol administration varies between institutions and experts. A uniform approach has not yet been established. However, optional admission with cardiovascular monitoring may be required. Outpatient administration with a close was also successfully implemented [23]. However, an electrocardiogram must be reviewed by a pediatric cardiologist before administration. Cardiopulmonary conditions at risk of treatment with propranolol, such as heart block or reactive respiratory disease, should be carefully considered before administration. Consensus on patient monitoring and best dose regimens needs to be determined, but prospective research is underway. Propranolol is currently employed for problematic hemangiomas, those that would receive either surgical or other systemic therapies to prevent unwanted side effects. Subglottics, periorbital and massive hemangiomas seem to respond well [24]. Despite the success of propranolol in reducing the size of the hemangioma, adjuvant therapy may be necessary in up to 50% of patients [17]. The mechanism of propranolol in the treatment of hemangiomas remains unclear, but may include the regulation of vascular growth factors and hemodynamic cytokines.3. Vascular malformations OverviewVascular malformations are rare vascular anomalies composed of inappropriately interconnected vasculatures. Any type of blood vessel or a combination thereof can be affected by vascular malformations. These lesions infiltrate normal tissue, which is very difficult to manage. The most common vascular malformations are lymphatic malformations (LM), capillary-venular malformations (CM), venous malformations (VM) and arteriovenous malformations (AVM), which were selected to be included in this document (Table 1). While they differ in their biological and clinical profile, as a whole, vascular malformations do not retreat and continue to expand over time. Periods of rapid growth, infiltration and destruction of soft tissues will stimulate therapeutic approaches that depend on the associated malformation.4. Lymphatic malformationsLymphatic malformations (LM) consist of dilated lymphatic vessels with inappropriate communication, lined with endothelial cells and filled with lymphatic fluid. Their incidence is approximately 1 in 2000 to 4000 live births [25]. Lesions are classified as macrocystic (simple or multiple cysts >2 cm3), microcystic (<2 cm3), or mixed [1]. Previous terminology, which is no longer used, included cystic hygroma and lymphogioma to describe these entities. The etiology of LM is unclear. Although most of them are congenital, there have been reports of LM occurring after trauma or infection. Receptors involved in the formation of lymphatic vascular ducts, such as VEGFR3 and Prox-1, may play a role in the development of this disease [26].4.1. DiagnosisLymphatic malformations can be macrocystic, microcystic, or mixed. Gradual growth and expansion are typical. Approximately half of the lesions are present at birth and 80-90% at the age of 2 years. Local infection converging the course of lymphatic drainage causes LM to swell, protrud, and sometimes become painful. This is the hallmark of LM versus other vascular anomalies The appearance of macrocystic disease differs from the appearance of microcystic. Macrocystic LM present as a soft swelling filled with fluid under normal or slightly coloured skin (Figure 3(a)). Intracystic bleeding or mixed lymphatic venous malformations may result in blue discoloration on the skin ice. Microcystic LMs are soft and incompressible masses with small vesicles covering the skin or mucous membrane above them. These vesicles may cry and occasionally cause pain or minor bleeding (Figure 3(b)). (a) b) (c) (a) b) (c) (c) LM can occur anywhere on the body, and symptoms are determined by the extent of the disease. Most LM are found in the cervical ophthalmosis area and expand to involve the oral cavity or respiratory tract, especially when mixed or microcystic [27]. Symptoms secondary to voluminous diseases often include pain, dysphagia, odynophagia, speech disorders, or in severe cases, airway obstruction. When the skeletal framework is involved in this area, LMs often cause osseous hypertrophy leading to dental or limb abnormalities (Figure 3(c)). Although these malformations can usually be diagnosed by physical examination, MRI is used to confirm the diagnosis, identify the cystic architecture and determine the extent of the disease.4.2. ManagementIn the ideal solution for the treatment of LM does not exist. Several interventions may be required. There have been rare cases of sporadic lesion resolution, although most of these malformations continue to increase with age [27]. Macrocystic lesions are more accessible to treatment and have a better prognosis. Swelling from acute infection is best controlled with a short course of systemic steroids and antibiotics. Final treatment is postponed until disappearance. LM can be detected on prenatal ultrasound and may require special interventions during childbirth. Exit (ex utero intrapartum treatment) procedure provides good airway control of the child if compromise is suspected to occur at birth. Sclerotherapy is often used for lymphatic malformations, especially if they sit deep and difficult to access surgically. It involves injecting the sclerosing agent directly into the lesion leading to fibrosis and eventually cyst regression. Usually several treatments are needed, and after treatment swelling is expected. Macrocystic lesions are easier to treat in this way, but there have been reports of success in microcystic lesions [28]. Several substances have been used for lymphatic malformations, including ethanol, bleomycin, OK-432, and doxycycline [29, 30]. Complications include skin decay, pain, and swelling. Sometimes severe swelling may occur, which can lead to obstruction of the airways requiring intensive care [31]. The risks to local nerves are also real, but usually only result in a temporary loss of function. Laser therapy with carbon dioxide can also be used in limited diseases of the mucous membrane of the respiratory tract and oral mucosa [32]. Macrocystic disease is often cured by surgical extirpation. Surgical excision is also often used for even if it is more aggressive, invasive and difficult to control [33, 34]. Infiltration of normal soft tissue and bones with extensive microcystic LM requires massive resection and local or free reconstruction of the flap. The inability to completely cut out microcystic LM often leads to repetition. Surgery is also used to correct secondary deformities caused by LM, such as bone overprouement of the facial skeleton [34]. Overall, treatment of LM should be aimed at the complete elimination of the disease. If this is not possible, different methods of treatment are combined to control the disease and ensure satisfactory functional results.5. Capillary malformationsCelebral malformations (CM) are sporadic lesions consisting of dilated capillary channels. They occur in approximately 0.3% of children. CMs can pose on any part of the body, but are mostly found in the cervical region. They are categorized as medial or lateral lesions depending on their location. Medial CM gradually lighten with time and eventually disappear. Colloquially they are referred to as stove bites on the nautch and angel kisses on the forehead. Lateral lesions, commonly referred to as port wine stains, have a longer course (Figure 4). Pathogenesis of isolated capillary malformations is unknown. Analysis of whole genome connectivity identified a lodz on the 5q chromosome associated with familiary disease [35]. A rare autosomal dominant hereditary disease consisting of a combination of CM and arteriovenous malformations (AVM) is associated with loss of functional mutation in the RASA1 gene [36]. This prompted further research into the cause of the more common sporadic form of CM.5.1. DiagnosisCMs present at birth as flat, red or purple, skin spots with irregular edges. They are painless and do not bleed spontaneously. Lateral CMs, or port wine spots, usually include the face and present along the distribution of the trigeminal nerve. CMs tend to progress with time as vascular ectasia expands to include deeper blood vessels to subcutaneous tissue levels. This causes the lesion to become darker in color, as well as more elevated and nodular [37]. Although they are mostly solitary lesions, CM can exist as part of the syndrome. The most common of these is Sturge-Weber syndrome (SWS) and is characterized by CM in the area of the eye branch of the trigeminal nerve, leptomeningeal angiomatosis and choroid angiomatosis. Symptoms of SWS are variable between cases and include unmanageable seizures, mental retardation and glaucoma. CM may also be present in Klippel-Trenaunay syndrome (KTS). This syndrome consists of a combination of multiple lymphatic, venous and capillary abnormalities. Diagnosis is usually made only by physical examination. If there are findings incompatible with CM, for example, pain or spontaneous bleeding, an MRI can be performed. Mri of the brain, as well as an annual ophthalmological examination is justified if suspected SWS is present.5.2. TreatmentThe pro cm treatment is laser therapy. FPDL is effective in treating these lesions. Laser slowly causes the redness of the lesion to fade; therefore, many treatments are often counted [38]. Timely treatment of these lesions seems to slow the progression of the disease. Argon, potassium-titanyl-phosphate (KTP) lasers and 755 nm lasers have also been used in more advanced lesions with good results [39]. Surgical excision is also an option for lesions that are not accessible by laser therapy. This is especially true in advanced lesions that have become nodular [37]. 6. Venous malformationsSVenous malformations (VM) are slow flow vascular anomalies composed of ecstatic venous ducts. These anbenal venous connections lead to venous congestion, thrombosis and the gradual spread of these lesions. As a result, virtualchames persist and progress until therapeutic intervention. The occurrence of virtual types is approximately 1 in 10,000 [40]. Virtual currency more often occur sporadically, but research into multifocal diseases and familiary patterns has helped to discover suspected genetic loci involved in their development. There are inherited forms of virtual microorganisms, the cause of which was localized on the 9p chromosome [41]. Recently, a mutation of loss of function in many solitary and multiple sporadic venous malformations was discovered on the TIE2/TEK angiopoetine receptor gene [42]. In addition, upregulation of several factors, including tissue growth factor beta (TGF-beta) and underlying fibroblast growth factor (beta-FGF) has been discovered in patients with venous malformations [26]. Progesterone receptors have been discovered in venous malformations. This probably explains their tendency to grow rapidly during hormonal changes [43]. 6.1. DiagnosisRight malformations are often visible at birth, but can be exposed as deep matter. Protrusiveness can be the only presenting symptom. It is known to grow proportionally with a child with sudden expansion in adulthood. Rapid growth can occur during puberty, pregnancy, or traumatic injury. The virtual system can be well localized or large. Lysing skin may appear normal or have a bluish discoloration. With greater skin involvement, the lesion appears darker blue or purple (Figure 5(a)). Upper aerodigestive wiring is common, and VMs are especially noticeable when the mucous membrane is affected (Figure 5(b)). (a) (b) (a) (b) Virtual images are compressible and swell when the area is dependent or there is an increase in hydrostatic pressure, for example during a valsalva maneuver. With time, there will be pain and swelling in the formation of fleboliths (calcified thrombus), or small clots, secondary to trauma or venous stasis. In very large lesions with significant thrombosis, the risk of distal embolism remains low, but real. D-dimers may be elevated and marker disease [44]. When isolated, virtual systems are generally benign with slow growth. They extend secondary to venous stasis and elastic vascular expansion. Airway obstruction, snoring, sleep apnea may also be present with recumbence [45]. VM can occur anywhere in the body, but is often found in the head and neck, where they relate to the oral cavity, airways, or cervical muscles. MRI is the imaging modality of choice when diagnosing a virtual image and offers an excellent definition of the disease for treatment planning [46].6.2. Treatment No one therapeutic modality is preferred in the treatment of virtual screens and often more than one modality is used [47]. Surgery, Nd : YAG laser therapy, and sclerotherapy (directing vascular injury) are all options for the treatment of VM.Conservative observation of small VM in children may be an option in the knowledge that growth is on the coming. Alifting the participating area can reduce hydrostatic pressure and vascular expansion and can hinder growth. With large lesions, altitude also reduces swelling and improves pain and obstruction of the airways. Similarly, compression clothing is the initial option for advanced lesions of the limbs, which allows to avoid risks from other treatment options. Low molecular weight heparin may improve thrombosis pain [44]. Treatment of larger respiratory tract and multifocal diseases is often justified. Treatment aimed at symptoms is the goal of these lesions. Management techniques usually aim to relieve respiratory symptoms, pain, and/or disfigurement. Surgical resection and sclerotherapy itself can sometimes be therapeutic for minor lesions. Local recurrence can occur years after treatment. Laser therapy provides good control over VM [48]. Use of Nd : Yag and KTP lasers [47, 49] have been described. Yag laser can be used through fiber attached to the endoscope to treat intraoral and respiratory venous malformations. Direct injury of deep venous malformations can also be carried out by passing the laser directly into the lesion (interstitial therapy). The laser causes a reduction in the lesion along with thrombosis. Mass treatment with these lasers offers disease reduction and control [48]. With an interstitial laser, nerve injury may occur. Sclerotherapy, as described above, has been widely used to treat VM [50]. The most commonly used sclerosants include ethanol and sotradecol [51]. Complications of sclerotherapy include skin and mucosal injuries, swelling leading to respiratory compromise, infections, and nerve injuries. In addition, each sclerosant has its own risk profile. Cardiovascular shock may occur in ethanol, shock symptoms with OK-432, interstitial pneumonia or pulmonary fibrosis with bleomycin and discoloration of teeth or electrolyte abnormalities with doxycycline [33]. Surgery remains one of the most excellent treatment options and can offer a cure for localized VM. Excision of complex lesions remains difficult secondary to intraoperative bleeding. Preoperative sclerosant can be used before excision (24-48 hours) to reduce surgical risk. Patients with extensive disease will often require combined modality. Treatment is not common, but disease control for many years is often Arteriovenous MalformationsArteriovenous malformations (AVMs) are congenital high-flow malformations composed of anomalous capillary beds that move blood from the arterial system into the venous system. They are often misdiagnosed at birth as other vascular lesions due to delays in the presentation of characteristic symptoms of malformations. Puberty and trauma provoke the growth of the lesion and the manifestation of its unpleasant symptoms [52]. They are infiltrative causing destruction of local tissue and often life-threatening secondary to massive bleeding. Extracranial AVM differs from their intracranial counterpart and is found in several areas in the cervical region. Little is known about the origin and pathogenesis of AVM. It is believed that a defect in vascular stabilization causes AVM, but it remains unclear whether these lesions are primarily congenital. Most AVMs are present at birth, but there are several caries of these lesions presenting after trauma in adults. Defects in TGF-beta signaling and the genetic two-hit hypothesis are the prevailing theory of pathogenesis [53, 54]. Progesterone receptors were isolated in AVMs explaining their expansion during puberty [43].7.1. Diagnosis Diagnosis of AVM is based on clinical examination and imaging. Growing hypervascular lesions may be present as a slight blush at birth. AVMs are often resting for many years and grow proportional to the child. Occasional expansion suggests a diagnosis [52]. Hormonal changes are thought to affect growth [43]. The characteristic characteristics of AVM will be palpable heat, pulse or excitement due to high vascular flow [55]. The above-bed skin may have a well-defined blush with increased temperature relative to the adjacent skin (Figure 6). The natural course of AVM is early inactivity, late expansion, and eventually infiltration and destruction of local soft tissues and bones. Common sites for occurrence are the middle, oral and limbs [52]. Oral lesions can occur early due to gingival involvement, disruption of deciduous teeth, and abundant periodontal bleeding. Although there are focal (small blood vessels) and diffuse lesions, avm are by far the most severe vascular anomaly that needs to be managed due to the replacement of normal tissue by the vessels of the disease and a very high incidence of recurrence [55, 56]. Imaging is necessary to identify the AVM range. MRI may be useful, but MRA and CTA can give a better outline of these lesions [57]. Numerous hypolucent arterial flow cavities are a hallmark of AVM MRI. CTA allows evaluation of surrounding tissues and bones. Individual arterial feeders can be visualized with this display [58]. The arteriogram, a time-tested approach to AVM diagnosis, will provide a good definition of the central nidu of the affected blood vessels and, if necessary, ensure access to intravascular treatment [59]. 7.2. Treatment AVM treatment consists of embolization, surgical or a combination of these methods. Treatment and timing are often individualized to the patient and the extent of the disease. For example, it is known that avm with small blood vessels are localized and can be reseked with good long-term results [60]. Historically, young children have been closely monitored until the spread of the disease with the concept that treatment should not be worse than the disease. However, this approach is currently being questioned because of the high recidivation rate encountered by avm [61]. Diffuse lesions are a lifelong problem. Long-term monitoring with a dedicated multidisciplinary team is important for AVM management. Intravascular embolization of AVM can be used alone or in combination with surgical excision. Absolute ethanol, polyvinyl alcohol and ONYX were used as AVM inbolising materials [62]. These substances selectively prevent and destroy the treated arteries. Complications of this approach include local ulceration of the skin, soft tissue necrosis, mucosal peeling or nerve injury. Embolization ensures temporary control of the disease, but recurrence is high [61]. This is theoretically due to the provision and recruitment of new vessels to support the undetected part of the nidus. Frequent serial embolization can improve patient outcomes. In general, surgical treatment of AVM requires preoperative suprasenctional embolization, prudent tissue removal and complex reconstructive techniques. Focal lesions have been shown to cure AVM surgical excision [56, 63]. However, diffuse avm have a recurrence rate of up to 93 % [61]. Excision is pre-shaped 24-48 hours after embolization. This helps to control blood loss and define the surgical edges of the lesion. Close postoperative observation with expected local repetition control is required. Recruitment of new vessels occurs even after excision. Basically, AVMs are debilitating vascular malformations that are often misdiagnosed early in life. Despite successful initial therapy, these lesions can be repeated many years later, which is necessary for vigilant management.8. ConclusionsVascular anomalies embody a myriad of abnormalities of blood vessels, which are believed to occur perinatally. The correct diagnosis is necessary for appropriate treatment. The most common vascular anomalies in the order of presentation include hemangiomas, lymphatic malformations, capillary malformations (spots from port wine), venous malformations and arteriovenous malformations. Treatment of vascular anomalies is complex and often involves multiple disciplines and therapeutic options. When considering the treatment of problematic hemangiomas and vascular malformations, the recommendations of the vascular anomaly team are recommended. Copyright © 2012 by Gresham T. Richter and Adva B. Friedman. This is an open access article distributed under the Creative Commons Attribution License, which allows unlimited use, distribution and reproduction on any medium, provided that the original work is correctly quoted. Cited.

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