Common Pediatric Skin Lesions: A Comprehensive Review of the Current Literature

Faryan Jalalabadi, MD¹ Jeffrey G. Trost, BS¹ Joshua A. Cox, MD¹ Edward I. Lee, MD¹
Crystal Y. Pourciau, MD²

¹ Division of Plastic and Reconstructive Surgery, Baylor College of Medicine, Houston, Texas

² Department of Dermatology, Baylor College of Medicine, Houston, Texas

Address for correspondence: Crystal Y. Pourciau, MD, 6701 Fannin St., Suite 670.00, Houston, TX 77030
(e-mail: cypourci@texaschildrens.org).

Semin Plast Surg 2016;30:91–97.

Abstract

Keywords

- dermatology
- pediatrics
- cyst
- hemangioma
- nevus

The timely diagnosis and treatment of dermatologic disease in the pediatric population can be challenging. A basic, yet comprehensive knowledge of common lesions is essential for a successful practice in plastic surgery. In this article, the authors describe vascular, cystic, and pigmented cutaneous lesions that are commonly encountered in the pediatric population. Epidemiology, pathogenesis, clinical course, and management options are discussed for each.

Vascular and soft tissue lesions vary widely throughout the pediatric population. Vascular anomalies are broadly categorized either as tumors or malformations: the former grows by cellular hyperplasia whereas the latter is a morphogenic abnormality. A complete discourse on vascular anomalies is beyond the scope of this article and is covered elsewhere in the literature.^{1–3} However, we will briefly discuss hemangiomas, as they are the most commonly seen tumor in infancy. Additional skin lesions encountered in children include developmental anomalies, subcutaneous cysts, pigmented lesions, and hamartomas. Developmental anomalies, such as cysts and pits, are typically noted at or shortly after birth; they are thought to be remnants of structures that were meant to obliterate in the embryologic period. Pigmented lesions vary in the timeline of their presentation and can degenerate into malignancy, thus making their proper identification and treatment of significant importance.

Hemangiomas

Hemangiomas are the result of hyperplastic endothelial cells. They are benign and are rarely capable of significant disruption of the surrounding tissue. Risk factors for developing hemangiomas are fair skin, female sex, low birth weight, and prematurity.^{4,5} Hemangioma is further divided into infantile and congenital hemangioma.

Infantile Hemangiomas

Infantile hemangiomas are the most common tumor found in infants and are seen in up to 10% of the population.¹ These vascular lesions present shortly after birth and often rapidly expand for the first 6 months of life before starting to involute. It was previously postulated that this type of vascular tumor originates when endothelial cells migrate from disrupted placental tissue and become imbedded in fetal soft tissue during gestation.¹ Infants whose mothers have undergone chorionic villus sampling, or suffered from placenta previa or pre-eclampsia, demonstrate a high incidence of hemangiomas.⁶ However, subsequent immunohistochemical studies have cast doubt on this hypothesis.⁷ Additional theories revolve around interactions between genetically altered hematopoietic progenitor cells and environmental factors such as cytokines, metalloproteinases, and proangiogenic growth factors.^{1,7–10}

Characteristically, infantile hemangiomas present at birth or shortly thereafter as well-demarcated, flat erythematous patches that rapidly proliferate volumetrically (► **Fig. 1**).¹¹ These lesions generally do not spread outside the original borders after the nascent mark has been declared. Growth occurs through three continuous phases: proliferation, quiescence, and involution. In most cases, the proliferative phase is complete by 3 to 5 months of life, as the lesion exhausts its

Fig. 1 Infantile hemangioma.

blood supply.² The quiescent phase follows from 9 to 12 months of age. Involution begins as the overlying skin becomes dull red or gray, and the deep components of the lesion begin to shrink (→**Fig. 2**). Ninety-two percent of infantile hemangiomas have completed involution by the fourth year of life, although fibrofatty protuberant tissue may persist.¹² This newer understanding of the timeline of involution allows families and their physicians to know whether or not surgical interventions for the hemangioma should be pursued before the patient becomes socially cognizant of its appearance.

Anatomically, a superficial hemangioma shows no extension into the subcutaneous tissue, presenting as a red, nodular mass. A deep hemangioma distinguishes itself as a telangiectatic or bluish subcutaneous nodule or protrusion, involving the deep dermis and subcutis. A mixed or combined hemangioma incorporates the characteristics of both.

Further subclassification distinguishes focal from segmental lesions. Focal hemangiomas are localized, unilocal, and follow the usual phases of proliferation and involution. Segmental hemangiomas are diffuse, plaque-like, and unpredictable from a functional and aesthetic outcome. Anatomically, they have an affinity for the limbs and face, specifically in nonrandomly assigned segments, which may herald potential underlying complications. When segmental hemangiomas > 5 cm are

Fig. 2 Involved hemangioma.

seen on the face or scalp, it is important to rule out PHACES syndrome, which consists of posterior fossa malformations; hemangiomas; arterial, cardiac, and eye abnormalities; and supraumbilical raphe.

For example, subglottic hemangiomas present with a large cutaneous hemangioma in a beard-like distribution on the face in 60% of patients.¹ The diagnosis is based on clinical history and exam; however, imaging studies, such as Doppler ultrasound or magnetic resonance imaging (MRI), can assist in the diagnosis if the clinical picture is not clear. Close observation and more aggressive management of these lesions are recommended due to their association with potential airway obstruction. Of note, these patients are also at risk for PHACES and should be appropriately screened for that as well.

In general, no intervention is required for inconspicuous and asymptomatic hemangiomas. However, some data suggest nearly 40% of children require intervention.¹³ Bleeding, ulceration, visual axis obstruction, airway obstruction, high-output cardiac failure, and the potential for permanent disfigurement are some of the indications for early intervention.

Medical management is warranted for massive or life-threatening lesions, multifocal disease, visceral or segmental lesions, or those causing airway or visual obstruction. For “problematic” hemangiomas, β blockers, classically propranolol, may be used as a first-line medical treatment.¹⁴ Propranolol is usually dosed 2 to 3 times per day for a total of 2 to 3 mg/kg/d. Treatment usually extends past the anticipated proliferative period and may be dependent on the patient's response to therapy, with most patients remaining on the medication until at least 1 year of life. Corticosteroids, interferon, and vincristine are options for treatment, but their use must be closely monitored for significant side effects.²

Surgical intervention can be used to excise localized lesions or fibrofatty residual tissue. Subtotal resection is an option for early treatment as the residual tissue may involute later in life. Flash pulse dye laser aids in the treatment of remnants or ulcerative lesions during proliferation. Intralesional steroid injection can be used in focal, parotid, nasal tip, subglottic, and eyelid hemangiomas.¹

Congenital Hemangiomas

Unlike their more common counterpart, congenital hemangiomas are, by definition, present at birth. They, in general, have no gender preference, are fully developed at birth, and test negative for the immunohistochemical marker GLUT 1.² They exhibit proportional growth, are usually solitary, and more commonly present on the extremities. Congenital hemangiomas undergo a proliferative phase in utero, which makes it possible to be diagnosed with prenatal imaging. Congenital hemangiomas are later categorized as either rapidly involuting congenital hemangioma (RICH) if they rapidly involute or noninvoluting congenital hemangiomas (NICH) if they fail to involute.¹ Generally, asymptomatic lesions are left alone as medical treatments, such as propranolol, are ineffective for congenital hemangiomas. In rare

instances, RICH can be associated with thrombocytopenia, high-output cardiac failure, or bleeding, which necessitates embolization or excision. Lesions can be excised for cosmesis or if causing local complications.

Developmental Anomalies

Cysts

Cystic developmental anomalies are believed to arise from persistent structural remnants that normally obliterate through the embryologic developmental process. The structural components of a cyst include an epithelium-lined sac filled with gas, a liquid, or a semisolid substance. They exhibit indolent growth and are typically noticed by parents either at birth or shortly thereafter. The most common cysts include dermoid, thyroglossal, and branchial cleft cysts, with dermoid being the most common type.¹⁵

Dermoid Cysts

Embryologically, dermoid cysts originate from the dermis and can therefore contain adnexal structures such as hair follicles and sebaceous glands. In contrast, epidermoid cysts originate from the epidermis and are void of adnexal structures. It is believed that these structures arise from ectoderm that becomes trapped underneath the superficial layer of skin. Anatomically, these congenital cysts are located along lines of fusion in the head and neck region, most commonly along the superior lateral orbital ridge, but also occurring at the scalp, the midline of the nose, and the posterior axis

(**Fig. 3A**).¹⁵ They can be fixed to underlying periosteum, erode into bone, or arise as free-floating structures in a more superficial dermal or subcutaneous layer. These cysts are characteristically skin-colored, nontender, noncompressible, and slow growing.

While excising these structures, one must be sure to remove the entire lining of the cyst to prevent recurrence (**Fig. 3B**). Cysts in the lateral orbital rim can be removed through an upper eyelid incision to achieve a superior cosmetic outcome, avoid alopecia, and spare the facial nerve.¹⁶ Depending on the size of the cyst, one may find dissection easier around an intact circumferential structure. Use caution not to violate the outer membrane. If the cyst is large, spilling some of its contents and reducing its size may enable a more thorough dissection.

Additional caution must be exercised when addressing midline nasal cysts, as up to 39% of midline dermoid cysts may exhibit intracranial extension through an abnormal foramen cecum.¹⁵ The patient should undergo further imaging to help determine the extent of surgical excision. Computed tomography may be used as an initial screening tool; however, MRI has shown to be more fruitful in delineating soft tissue extension and intracranial involvement.^{17,18} In the presence of intracranial extension, a neurosurgeon must perform a craniotomy. In the absence of intracranial involvement, one may proceed with direct excision and closure with or without an open rhinoplasty. Injection of methylene blue dye into nasal sinus tracts helps ensure complete excision of the epithelial lining.

The differential diagnosis for midline nasal lesions must include nasal gliomas and encephaloceles.¹⁹ Both result from a failure of dural diverticulum to obliterate in the prenasal space.¹⁵ Nasal gliomas are formed from residual extracranial glial tissue after closure of the fonticulus frontalis and may have intracranial extension through a fibrinous stalk. Encephaloceles are composed of meninges, brain tissue, or both. The inclusion of brain tissue differentiates a meningoencephalocele from a meningocele. Encephaloceles often appear clinically as soft, compressible bluish masses that sometimes transilluminate. They can be found on the nose, glabella, or forehead.

One method that may help differentiate these masses from each other on clinical exam is the Furstenberg test. In a positive test, bilateral compression of the internal jugular veins results in enlargement of the encephalocele. Enlargement of the mass with crying is also suggestive of an encephalocele and can help differentiate these lesions from a glioma or dermoid cyst.

Thyroglossal Duct Cysts

Thyroglossal duct cysts are the most common midline congenital neck mass.²⁰ During its embryological migration from the base of the tongue to the pyramidal thyroid, the thyroid may leave behind a trail of tissue contributing to the thyroglossal duct cysts or sinuses. They commonly present in children between the ages of 2 and 10 years. Characteristically, the mass is soft, smooth, and nontender. Anatomically, it is found in the midline at or around the hyoid bone. Due to residual communication with the oropharynx, these masses have a tendency to become infected. Treatment should include complete excision of the cyst as well as the sinus tract to

Fig. 3 (A) Dermoid cyst along the orbital ridge. (B) Removal of a dermoid cyst and its entire epithelial lining.

minimize recurrence.²¹ To ensure total resection, a segment of the hyoid bone, the raphe of the mylohyoid muscle, and a piece of the geniohyoid and geniohyoglossus may be included with the excision. During resection, care must also be taken to protect the hypoglossal nerve, which runs high in the neck and within the proximity of the thyroglossal sinus tract. Well-differentiated thyroid carcinoma, most commonly papillary type, is encountered in approximately 1.5% of thyroglossal duct cysts.²² Thus, it is important to ensure complete removal of the tumor.

Branchial Cleft Cysts

Branchial cleft cysts are the most common congenital neck masses found in the lateral neck.²⁰ Incomplete closure or persistence of the brachial pouches and/or arches is thought to contribute to the formation of a branchial cleft cyst. Characteristically, these masses present as slow-growing, nontender masses that are identified later in childhood or early in the teenage years. Anatomically, they are found between the upper one-third and lower two-thirds of the sternocleidomastoid muscle in the lateral neck. Branchial fistulas and sinuses present a decade earlier and are lined by respiratory epithelium, whereas branchial cleft cysts are lined by stratified squamous epithelium.²³ Regardless of histological differences, treatment of both involves early excision of the mass and tract. Cannulating with a catheter and infusing the sinus tract with methylene blue dye may help ensure complete excision of the structure and its associated parts.

Pilomatricomas

Pilomatricomas are benign structures comprised of hair follicle matrix cells.²⁴ These lesions typically follow a bimodal peak presentation during the first and sixth decades of life, although they are more common in children than adults.²⁴ Another term in the literature for these neoplasms is the calcifying epithelioma of Malherbe. Characteristically, they are slow-growing, “rock-hard” subcutaneous masses, with a blue hue or ulcerative appearance (►Fig. 4A).^{24,25} Anatomically, the majority of these lesions are found on the head and neck; specifically, the scalp, cheek, neck, and the preauricular or periorbital region. These lesions may exist as a solitary neoplasm; however, association with familial syndromes has been reported including myotonic dystrophy, Turner’s syndrome, Gardner’s syndrome, and Rubinstein-Taybi’s syndrome.^{26–28} They are treated by surgical excision (►Fig. 4B).

Pigmented Lesions

Congenital Melanocytic Nevus

Present at birth, congenital melanocytic nevi (CMN) are formed as a collection of melanocytes found in the dermis, epidermis, or both (►Fig. 5). They are found in 1 to 6% of newborn infants.²⁹ Congenital melanocytic nevi may present as a solitary plaque that changes in size in proportion to the child’s growth or multiple lesions with at least one larger in size and surrounded by satellite nevi. The latter may also demonstrate bone and central nervous system (CNS) involvement, specifically, neurocutaneous melanosis (NCM), a rare

Fig. 4 (A) Periorbital pilomatricoma. (B) Appearance of completed excised pilomatricoma.

congenital syndrome that also includes melanocytic neoplasms of the CNS.^{30,31}

Congenital nevi are subdivided into three groups based on their size: small (< 1.5 cm in diameter), medium (1.5–19.9 cm), and giant or large (> 20 cm). Approximately 75% of giant nevi are associated with surrounding smaller satellite nevi, which may be present at birth or become apparent later

Fig. 5 Medium-sized congenital melanocytic nevus.

during childhood.²⁹ Small nevi, by definition, can be closed primarily after excision. Anatomically, they are found in any location on the body. Perifollicular hypopigmentation, or “sparing,” can be seen in CMNs in the scalp as well as other areas of the body. Congenital melanocytic nevi located on acral skin may take a more comma-like appearance.

Although biopsy is not needed in most cases of CMN, it may be warranted in lesions that are demonstrating significant change in appearance, characterized by rapid vertical or radial growth, color variation, or ulceration. Though typically benign, these cells have the potential to transform into melanoma. The lifetime risk of developing melanoma is controversial, but appears proportional to the size of the lesion.^{32–35} The risk of malignant change in small and medium CMN is less than 1% and for large CMN around 5%, with about half of this risk in the first few years of life.²⁹

The risk of developing NCM is associated with axial lesions and is proportional to the number of satellite lesions observed.³⁶ If NCM is suspected, MRI is warranted to rule out CNS involvement, as well as a consultation with a neurologist. Giant CMN in the posterior midline or in the presence of > 20 satellite nevi warrants MRI before the age of 4 months.³⁷ Although the true incidence of NCM, both symptomatic and asymptomatic, is currently unknown, it has been suggested that between 4.5 to 11% of patients with large CMN manifest symptomatic NCM.³⁸ In these patients, hydrocephalus or a mass lesion may contribute to increased intracranial pressure. Symptomatic NCM has a very poor outcome. A review of 39 cases found that death occurred within 3 years of the onset of neurologic symptoms in > 50% of patients, most of whom were younger than 10 years old.³⁸

Histology can help to distinguish congenital from acquired nevi: Acquired nevi are comprised of cells contained within the papillary and upper reticular dermis, sparing the appendages. Congenital melanocytic nevi melanocytes can be found in deep dermal appendages and neurovascular structures including follicles, glands, muscles, and endothelium. These cells may be found in the deep dermis and subcutaneous fat between collagen bundles, and may spare the subepidermal zone.^{39–41}

Treatment depends on size, location, age, potential for malignant transformation, and cosmetic result. As mentioned above, the prophylactic excision of large and giant nevi is controversial. These lesions may extend deep to muscle, bone, and neurovascular structures. Removal of the cutaneous portion does not fully mitigate the risk of transformation. Small and medium-sized nevi can be monitored and removed if it is determined the patient would benefit cosmetically. The anatomical location relative to a vital structure may present a barrier to resection. Staged excision may be used in the realm of a giant CMN. Surgeons may use tissue expansion, grafts, and flaps in their armamentarium to close such defects.^{42–44} Lasers may be used; however, recurrence may occur and the malignant transformation of deeper tissue may persist and become more difficult to detect. It is important to educate your patients on the dangers of ultraviolet light, especially those with giant nevi who are at greater risk for transformation.

Nevus of Ota

Also termed nevus fuscoceruleus ophthalmomaxillaris, nevus of Ota is more commonly found in Asian and Black patients.^{45,46} Fifty percent are congenital; however, most of the remaining half arise after puberty.⁴⁷ Nevus of Ota is associated with ipsilateral glaucoma, intracranial melanocytosis, and rarely cutaneous, ocular, or intracranial melanoma.⁴⁸ Histologically, it represents a collection of dermal melanocytes that endow a bluish hue to the overlying skin. The nevus classically involves the first or second branches of the trigeminal nerve and usually includes the ipsilateral sclera. First-line treatment is pulsed Q-switched laser therapy.⁴⁹ Successful treatment with minimal side effects has been reported with the use of Q-switched ruby, alexandrite, and Nd:YAG lasers as well.^{50–53}

Nevus of Ito

Nevus of Ito is an uncommon, large, blue-gray lesion that may arise in patients with nevus of Ota or in isolation. Similarly to nevus of Ota, it typically is present at birth, and most commonly is seen in patients of Asian descent. However, in contrast, nevus of Ito characteristically arises over the shoulder region and areas innervated by the posterior supraclavicular and lateral cutaneous brachial nerves.^{46,54} Just as with nevus of Ota, treatment is with pulsed Q-switched laser therapy.⁴⁹

Spitz Nevus

Spitz nevus is a benign melanocytic nevus that may clinically and histologically resemble melanoma. It most frequently occurs in children, but can arise in adults. The nevi are seen in the head and neck region with some lesions involving the trunk or extremities. Clinically, it presents as a well-circumscribed pink papule that rapidly increases in size. The pigmented variant, or spindled cell nevus of Reed, is dark brown to black in color with pseudopods at the periphery, rendering a “starburst” appearance (→ Fig. 6). Spitz nevus has some unique histological features, but can be further distinguished from melanomas by using immunohistochemistry for BRAF or RAS mutations, which are seen in melanoma only.⁵⁵ In removal, the nevus should be excised completely with adequate margins.⁵⁶

Fig. 6 Spitz nevus.

Nevus Sebaceous

Postzygotic somatic mutations give rise to the mosaic of lesions classified as nevus sebaceous. These are well-defined hamartomatous lesions composed of sebaceous glands. Nevus sebaceous is reported in 0.3% of all newborns and usually presents as a solitary lesion at birth or early childhood, though characteristic features may not present until puberty. Newer studies suggest that this risk is much lower, with the majority of secondary tumors being benign follicular tumors such as trichoblastoma and basaloid hamartoma.^{57–59} Rapid enlargement, ulceration, or development of an exophytic nodule should raise suspicion of malignant transformation.

Anatomically, these solitary, velvety plaques present mostly on the scalp, and on the head and neck regions as a hairless patch; though they may occur anywhere including, rarely, the oral cavity.⁶⁰ Three clinical stages exist. At birth, nevus sebaceous appears as a hairless, solitary, linear, well-demarcated patch or thin plaque that is pink, yellow, orange, or tan in color. The surface may be subtly mammillated or scaled at onset. The lesion typically grows radially in proportion to the growth of the affected individual. However, during adolescence, hormonal changes cause the lesion to thicken and become more verrucous in appearance (► **Fig. 7**). As patients age, the lesions may develop appendageal tumors. A phenotypic variant has been reported with large pink exophytic nodules.⁶¹ Large lesions may be associated with numerous abnormalities including intracranial masses, seizures, mental retardation, skeletal abnormalities, pigmentary changes, ocular lesions, hamartomas of the kidney, and mediastinal lipomatosis.^{62–64}

Histologically, the epidermis shows papillomatous hyperplasia. An increase in the number of sebaceous glands can be visualized in the dermis, whereas the deep dermis exhibits ectopic apocrine glands. The sebaceous glands consist only of immature hair follicles and malformed hair germs represented by buds of basaloid cells.

Fig. 7 Nevus sebaceous.

Although the risk of malignant transformation exists, it is low; therefore, it does not necessitate excision. Nevertheless, patients often desire excision for cosmetic reasons such as its visibility or increased lesion thickening due to aging. Given the risk of transformation, surgical intervention is the preferred treatment modality. Full-thickness skin excision is usually required because any residual tissue carries the risk of recurrence, secondary tumor growth, or malignant transformation. For lesions in the scalp, it is preferred to excise before 1 year of age to utilize the laxity and mobility of the scalp in this age group.

Conclusions

A wide variety of soft tissue lesions and vascular defects exist within the pediatric population. A basic, yet comprehensive knowledge of common lesions is essential, as many of these lesions possess a time-sensitive window for treatment.

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