

CHAPTER 7

SKIN AND SUBCUTANEOUS LESIONS

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I. BENIGN SKIN LESIONS

- A. Acrochordon (skin tag)
 - 1. Presents as single or multiple small, skin-colored, pedunculated papules
 - 2. Treatment for symptomatic lesions includes cryotherapy, snip excision, or shave excision
- B. Epidermal inclusion cyst (sebaceous cyst, epidermoid cyst)
 - 1. Result from a proliferation of epidermal cells within the dermis, and arise from the infundibular portion of the hair follicle
 - 2. Present as skin-colored to yellow, firm, movable nodules, often with a visible central punctum. Usually asymptomatic, but may become infected/inflamed
 - 3. Well circumscribed by a cyst wall made of stratified squamous epithelium, and communicate with the surface through a small opening, which may contain a keratinous plug or blackhead
 - 4. Treatment of an acutely infected/inflamed EIC is incision and drainage
 - 5. Definitive treatment is surgical excision of the entire cyst (including cyst wall)
- C. Pilar cyst (trichilemmal cyst)
 - 1. Originate from the outer root sheath of the hair shaft, and are lined by stratified squamous epithelium, which undergoes keratinization
 - 2. Present as firm, slow-growing subcutaneous nodules (clinically similar to epidermoid cysts, but they lack the central punctum)
 - 3. Most common cutaneous cyst of the scalp
 - 4. Definitive treatment is surgical excision of the entire cyst (including cyst wall)
- D. Pilomatricoma
 - 1. Benign growth composed of hair follicle matrix cells
 - 2. Classically present as slow-growing, “rock- hard” subcutaneous masses, with a blue hue or ulcerative appearance
 - 3. Bimodal distribution (first and sixth decades), although more common in children
 - 4. Definitive treatment is surgical excision of the entire mass
- E. Dermoid cyst
 - 1. Congenital cysts 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 and the midline of the nose
 - 2. Skin-colored, nontender, noncompressible, slow growing, and can arise in the dermis or subcutaneous tissue, or be fixed to underlying periosteum
 - 3. 40% of midline nasal cysts have intracranial extension through an abnormal foramen cecum, and should undergo MRI preoperatively
 - 4. Treatment is surgical excision of the entire cyst (including cyst wall)

- A. Seborrheic keratosis (verruca senilis, pigmented papilloma)
 1. Present clinically as hyperpigmented, waxy, verrucous papules with a characteristic “stuck-on” appearance
 2. Appear in the fifth to seventh decades of life, usually on the head, neck, or trunk
 3. Arise from the basal layer of the epidermis, are composed of well-differentiated basal cells
 4. Removal is for cosmetic purposes only – cryotherapy, shave biopsy, dermabrasion
- B. Cutaneous horn
 1. Hard, cone-shaped cutaneous projections typically caused by excessive epidermal growth and retention of keratin
 2. 20% are associated with premalignant lesions, and 15% are associated with SCC
 3. Treatment is shave biopsy to exclude malignancy, or excision
- C. Trichoepitheliomas
 1. Neoplasms of follicular origin that presents as multiple, yellowish-pink, translucent papules distributed symmetrically on the cheeks, eyelids, and nasolabial area
 2. More common in women
 3. Can be confused with basal cell carcinoma
 4. Treatment is excision to differentiate from carcinoma
- D. Eccrine poroma
 1. Presents as a solitary lesion (firm papule less than 2 cm in size) usually on the sole of the foot or the palm of the hand in persons older than 40 years. It may also occur on the chest, the neck, or other locations.
 2. Can degenerate into malignant eccrine poroma or porocarcinoma
 3. Treatment is surgical excision
- E. Verrucous nevus
 1. Closely set skin colored, brown, or gray-brown verrucous papules that may coalesce to form well-demarcated plaques, usually in a linear configuration along skin tension lines
 2. Hyperkeratosis, acanthosis, and papillomatosis on histology
 3. Treatment is excision to the deep dermis. For more extensive lesions not amenable to excision, treatments may include laser cryotherapy and electrodesiccation dermabrasion
- F. Desmoid tumor
 1. Benign tumor arising from the musculoaponeurotic layer of the abdominal wall
 2. Treatment is excision with 1cm margins
- G. Keratoacanthoma (Figure 1)
 1. Rapid growth followed by spontaneous regression over several months
 2. Treatment is recommended because it cannot be reliably distinguished from SCCs
- H. Cylindroma
 1. Adult-onset nodules, usually on the face or scalp, with smooth, flesh-colored, possibly telangiectatic surfaces
 2. For solitary lesions, treat with excision or electrosurgery

3. For grouped lesions, may need staged excision
- I. Pyogenic granuloma (Figure 2)
 1. Appears in early childhood, usually following minor trauma, as a rapidly growing, small (<1cm) red lesion
 2. Friable and prone to bleeding
 3. Treatment is excision (including the dermis)



Figure 1. Keratoacanthoma demonstrating rapid growth over a 3-month period. From Lee EH, et al. *Benign and premalignant skin lesions. Plast Reconstr Surg* 2010;125(5):188e-98e.



Figure 2. Pyogenic granuloma. These lesions occur most commonly on the face (above). (Below) This lesion on the left supraclavicular fossa shows evidence of recent bleeding (arrowhead). From Li W, et al. *Cysts, pits, and tumors. Plast Reconstr Surg* 2009;124(1 Suppl):106e-16e.

- J. Xanthelasma Palpebrarum
 1. Present as asymptomatic yellow-orange papules and plaques, commonly on the medial eyelids

2. Highly associated with hyperlipidemia
 3. Treatment is surgical excision for cosmetic purposes
 4. Correction of underlying hyperlipidemia is largely ineffective in treating xanthelasma
- K. Syringoma
1. Presents as asymptomatic, skin-colored to yellow papules and plaques commonly found on the eyelids and upper cheeks
 2. Treatment is for cosmetic concerns, and includes laser therapy, cryotherapy, electrodesiccation, and excision
- L. Nevus of Ota (nevus fuscoceruleus ophthalmomaxillaris or oculodermal melanocytosis)
1. Dermal melanocytic hamartoma that demonstrates bluish hyperpigmentation along the ophthalmic and maxillary divisions of the trigeminal nerve
 2. Caused by the failure of complete embryonic migration of melanocytes from the neural crest to the epidermis, resulting in dermal nesting with the resultant dermal melanin causing the Tyndall effect
 3. Primarily affects darker-pigmented individuals, more prevalent in females
 4. Has a bimodal age incidence, with a peak at 1 year of age and a second around puberty
 5. Becomes more prominent with age, puberty, and postmenopausal state
 6. 10% association with ipsilateral glaucoma – ophthalmologic examination recommended
 7. Malignant degeneration to melanoma occurs in approximately 4% of reported cases
 8. Treat with laser therapy – either Q-switched laser with ruby (694 nm), alexandrite (755 nm), or neodymium: yttrium-aluminum-garnet (1064 nm)
- M. Nevus of Ito
1. Large blue-grey lesion that characteristically arises over the shoulder region and areas innervated by the posterior supraclavicular and lateral cutaneous brachial nerves
 2. Treat with pulsed Q-switched laser therapy
- N. Spitz Nevus
1. Benign melanocytic nevus that may resemble melanoma
 2. Most frequent in children, usually seen in the head and neck region
 3. Clinically presents as a well-circumscribed pink papule that rapidly increases in size
 4. Pigmented variant (spindled cell nevus of Reed) is dark brown to black in color with pseudopods at the periphery, giving it a “starburst” appearance
 5. Treatment is surgical excision
- O. Dermatofibroma (benign fibrous histiocytoma)
1. Present as a solitary, firm, hyperpigmented macule or thin papule on the lower extremity
 2. Etiology unknown, 4 times more common in women than in men
 3. Surgical excision only for symptomatic lesions (can be painful or pruritic)
- P. Lipoma

1. Present as soft, rubbery, nontender, slow-growing subcutaneous nodules that are freely movable on palpation
 2. Consist of mature adipocytes surrounded by a thin fibrous capsule
 3. Treatment – enucleation or surgical excision for symptomatic lesions
- Q. Nevus sebaceous (Jadassohn nevus)
1. Presents as a hairless, solitary, linear, well-demarcated patch or thin plaque that is pink, yellow, orange, or tan in color, usually on the scalp
 2. During adolescence, hormonal changes cause the lesion to thicken and become more verrucous and nodular in appearance
 3. Risk of degeneration to basal cell carcinoma is approximately 15 to 20%
 4. Keratoacanthoma and squamous cell carcinoma may also develop (less frequently)
 5. Because of the risk of malignant transformation, complete excision is recommended prior to puberty

II. PREMALIGNANT AND MALIGNANT SKIN LESIONS

A. Actinic Keratoses

1. Erythematous, rough, scaly lesions, typically in sun-exposed areas
2. 25% chance of progression to squamous cell carcinoma
3. Treatments include topical imiquimod (Aldara), photodynamic therapy with 5-aminolevulinic acid (Levulan), cryotherapy, 5-fluorouracil, retinoids, and diclofenac gel

B. Squamous Cell Carcinoma (SCC)

1. Presents as an erythematous, scaly or verrucous papule or plaque
2. Associated with chronic sun exposure and are more commonly seen with lighter skin, increasing age, and tanning bed use
3. High-risk – poorly defined borders, recurrent lesion, immunosuppressed patient, site of previous radiation/chronic inflammation (Marjolin's ulcer), rapid growth, neurologic symptoms, invasion to fat, size >2cm, or size >6mm in the central face, ears, scalp, genitalia, hands/feet
4. Margins
 - a. Low-risk: 4-6mm
 - b. High-risk: 10mm or Mohs micrographic surgery
5. May metastasize (most often with lesions on the ear or lip, lesions > 2 cm in size, and in the immunosuppressed population)
6. Bowen's Disease – SCC in-situ (full-thickness epidermal atypia that presents as a thin eczematous, erythematous plaque)
7. Erythroplasia of Queyrat – SCC in-situ of the glans penis

C. Basal Cell Carcinoma (Figure 3)

1. Presents as a pink, pearly papule with overlying telangiectasia and rolled borders. Ulceration may be present, giving a characteristic "rodent bite" ulcer
2. Arises on sun-damaged skin of the head, neck, and upper extremities, with an increasing incidence with age, fair skin, chronic sun exposure, and a history of tanning bed use

3. High risk – poorly defined borders, recurrent lesion, immunosuppressed patient, site of previous radiation, peri-neural involvement, aggressive histology (morpheaform, sclerosing, mixed infiltrative, basosquamous, or micronodular), >2cm in the trunk/extremities, or >1cm in the head and neck
4. Treatment options include surgical excision, Moh's, ED&C, cryosurgery, Imiquimod (for <2cm BCCs of the trunk, extremities, or neck), photodynamic therapy, 5-FU, and radiation therapy
5. Margins
 - a. Low-risk: 4mm
 - b. High-risk: 6-10mm (or Mohs micrographic surgery)
6. Vismodegib and Sonidegib (a selective inhibitor of hedgehog pathway activation) are approved for the treatment of metastatic basal cell carcinoma and locally advanced basal cell carcinoma that has recurred after surgery, or in patients who are not surgical or radiation therapy candidates



Figure 3. (Above, left) Superficial basal cell carcinoma. (Above, right) Nodular basal cell carcinoma. (Below, left) Infiltrative basal cell carcinoma. (Below, right) Pigmented basal cell carcinoma. *From Lee E, et al. Benign and premalignant skin lesions. Plast Reconstr Surg 2010;125(5):188e-98e.*

D. Melanoma (Figure 4)

1. 3% of all skin cancers, but 65% of all skin cancer deaths
2. Risk factors include fair hair/skin, history of sunburns/sun exposure, and family/personal history of melanoma
3. ABCDE signs of melanoma include
 - a. Asymmetry

- b. Border irregularity
- c. Color variegation
- d. Diameter (>6 mm)
- e. Enlarging or evolving
- 4. Superficial spreading melanoma
 - a. Most common subtype of melanoma
 - b. Occur particularly on sun-exposed skin and often arise in preexisting nevi
 - c. Have a prolonged radial growth phase before developing a vertical growth phase
 - d. Initially flat, but can become irregular or raised as the lesions grow
- 5. Nodular melanomas
 - a. Second most common form of melanoma
 - b. Commonly seen on the trunk, head, and neck, males > females
 - c. Domed-shaped, dark, and may resemble a blood blister
- 6. Lentigo maligna melanoma
 - a. Rare subtype seen in only 4 percent of melanomas
 - b. Often arise from pre-existing lentigo maligna lesions, which can be present for many years, growing in a slow, radial fashion, before the vertical growth phase develops
 - c. Women>men, often located on the face, head, and neck of older individuals
 - d. Commonly present as large, tan lesions with convoluted patterns and multiple amelanotic patches
- 7. Acral Lentiginous Melanoma
 - a. Rarest form of melanoma in Caucasians but 30-60 % of melanoma in dark-skinned individuals
 - b. Commonly occur in the palms, soles of the feet, and under the nails
- 8. Amelanotic
 - a. Lack pigment and are often mistaken for other lesions
- 9. Desmoplastic Melanoma
 - a. Has aggressive local growth and less frequent nodal metastases
 - b. Often confused with common nevi, blue nevi, Spitz nevi, pyogenic granulomas, or hemangiomas
- 10. Diagnosis
 - a. Gold standard is excisional biopsy
 - b. Shave biopsy is often performed by dermatologists but can under-estimate depth, although this hasn't been shown to affect prognosis/survival
- 11. Treatment is surgical excision, margins are dictated by Breslow depth
 - a. Melanoma in-situ (MIS) – 5mm
 - b. < 1mm – 1cm
 - c. 1.01-2mm – 1-2cm (generally 2cm where allowable such as the trunk and extremities, and 1cm in more aesthetically sensitive areas like the head and neck)
 - d. >2.01mm – 2cm
- 12. Sentinel Lymph Node Biopsy (SLNB)
 - a. Indicated for intermediate thickness (1-4mm) melanomas

- b. May also be indicated for high-risk thin melanomas 0.75-1mm thick and thick melanomas
- 13. Completion Lymph Node Dissection (CLND)
 - a. Currently indicated in cases of a positive SLN, or a clinically palpable node
- 14. Medical treatment for melanoma is currently used for advanced melanoma only (usually Stage 3 or 4) and consists of immunomodulation and targeted molecular therapy toward mutations found in melanocytic lesions
 - a. Vemurafenib and dabrafenib (BRAF inhibitors, improved survival but develops rapid resistance with associated relapse)
 - b. Interleukin-2 (immunomodulator that activates the host immune system to attack malignant cells, severe side effect profile)
 - c. Ipilimumab (monoclonal antibody that suppresses CTLA-4, small but durable response, significant immunologic side effects)
 - d. Nivolumab (anti-PD-1 monoclonal antibody)

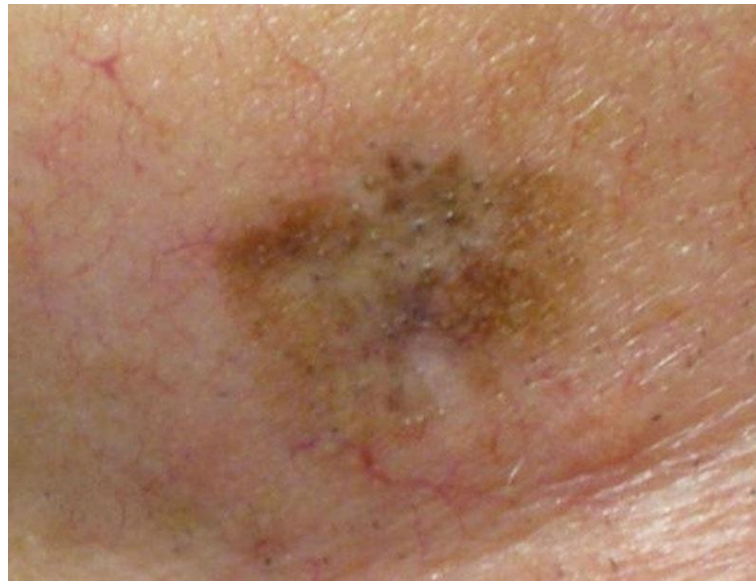


Figure 4. ABCDE signs of melanoma. A 75-year-old man with a left cheek lesion presenting with asymmetry, borders irregularity, color variation, and diameter of 2.5 cm evolving over an 8-year period. *From Dzwierzynski W. Managing malignant melanoma. Plast Reconstr Surg 2013;132(3):446e-60e.*

E. Merkel Cell

1. Presents as a firm, painless nodule (up to 2 cm in diameter) or a mass (>2 cm in diameter), usually in the head and neck region, classically red in color, but may be flesh-colored or blue, and often enlarges rapidly
2. Risk factors include exposure to sun and ultraviolet light, immunosuppression, and the Merkel cell polyomavirus
3. Treatment is surgical excision (1-2cm margins down to investing fascia) and sentinel node biopsy, combined with adjuvant radiation therapy to decrease local recurrence rates

F. Verrucous carcinoma

1. A variant of squamous cell carcinoma – requires wide local excision with negative margins for treatment
- G. Paget's Disease of the Breast
1. Presents with eczematous skin changes of the nipple areolar complex
 2. Often associated with ipsilateral breast cancer
- H. Extramammary Paget Disease
1. Intraepithelial carcinoma involving the vulvar, perianal, perineal, scrotal, and penile regions
 2. Presents as well-defined, moist, erythematous plaques associated with pruritis
 3. 7 to 40% rate of associated malignancy – treated with wide local excision
- I. Dermatofibrosarcoma protuberans (DFSP)
1. Malignant mesenchymal tumor that arises in the dermis and is characterized by latency in its initial detection, slow infiltrative growth, and local recurrence if not adequately treated
 2. 90% of DFSP tumors have the chromosomal translocation t(17;22) that fuses the collagen gene COL1A1 with the platelet-derived growth factor gene
 3. Most common on the trunk followed by the proximal extremities
 4. Treatment is wide surgical excision (2-3 cm margins). Mohs can be used
 5. Molecular targeted therapy with imatinib mesylate (Gleevec) is indicated for unresectable, recurrent, or metastatic DFSP
- J. Angiosarcoma
1. Appears as a purple plaque, commonly found in the face and scalp in older Caucasian men
 2. 50% in the head and neck, and also commonly found in the breast and extremities, particularly in patients with a history of lymphedema or radiation therapy
 3. Treatment is wide local excision, but it is frequently multifocal, and local recurrences are common
- K. Stewart-Treves Syndrome
1. Lymphangiosarcoma in post-mastectomy patients
 2. Diagnosis is via incisional biopsy
 3. Treatment includes WLE if possible with margins of at least 1cm, or isolated limb perfusion with tumor necrosis factor and melphalan

III. VASCULAR ANOMALIES

Vascular anomalies may be divided into hemangiomas and vascular malformations. Hemangiomas are vascular tumors characterized by increased cellular proliferation. Classically, they exhibit rapid growth and slow regression. Approximately 80% of hemangiomas are noted in the first month of life, and 60% occur in the head and neck region. Vascular malformations are present at birth and grow slowly.

- A. Infantile hemangiomas
1. May be present at birth (30-50%) but usually appear in the first two weeks of life, with 80% appearing in the first month of life

- a. Proliferating phase – 0-9 months (with most of the growth achieved by 3 months)
 - b. Involuting phase – 9 months to 12 years, but is completed usually by age 4
 2. Involution leaves some scar or discoloration in 50% of patients
 3. Biopsy is rarely indicated, but IHs are GLUT-1 positive on immunostaining
 4. Treatment
 - a. Small (less than 2-3cm) well localized IHs can use intralesional corticosteroid
 - b. Larger problematic lesions can be treated with medical therapy (oral prednisolone or oral propranolol)
 - c. Surgery is indicated in the case of:
 - i. Failure or contraindication to pharmacotherapy
 - ii. A well-localized tumor in an anatomically favorable area
 - iii. If resection will be necessary in the future and the scar would be the same
 - iv. Lesions (of any stage) that are compromising function or destroying vital structures
- B. Congenital hemangiomas
1. Arise in the fetus, are fully grown at birth, and do not have post-natal growth
 2. Red-violaceous with coarse telangiectasias, central pallor, and a peripheral pale halo
 3. More common in the extremities, have an equal sex distribution, and are solitary with an average diameter of 5 cm
 4. Two forms:
 - a. Rapidly involuting congenital hemangioma (RICH)
 - i. Involutates rapidly after birth
 - ii. 50% of lesions have completed regression by 7 months of age; the remaining tumors are fully involuted by 14 months
 - iii. Affects the head or neck (42%), limbs (52%) or trunk (6%)
 - iv. Rarely requires treatment but may leave behind atrophic skin
 - b. NICH (Non-involuting congenital hemangioma)
 - i. Does not regress (remains unchanged with persistent fast-flow)
 - ii. Involves the head or neck (43%), limbs (38%), or trunk (19%)
 - iii. Resection can be considered if the scar will be less noticeable than the lesion
- C. Kaposiform Hemangioendothelioma
1. Presents as a large (>5cm), superficial, and diffuse lesion, with the overlying skin deep red-purple, tense, painful, and shiny
 2. Typically involves the trunk and extremities, 50% present at birth (but can appear in childhood)
 3. Kasabach-Merritt phenomenon (thrombocytopenia <25,000, bruising, and bleeding) is common
 4. Regression seen after age 2, although long-term chronic pain and stiffness can persist
 5. Diagnosis is by MRI
 6. First-line treatment is vincristine

- D. Capillary Malformations
 1. Slow-flowing vascular malformation characterized by ectatic vessels located at various levels within the dermis
 2. Treatment is pulsed-dye laser therapy
- E. Venous Malformations
 1. Most common type of vascular malformation
 2. Present at birth, slowly increase in size as the child grows, change size with position, and are prone to thrombosis (phlebolith formation)
 3. First line therapy is sclerotherapy (STS, ethanol)
- F. Lymphatic Malformations (historically called “cystic hygromas”)
 1. Characterized as microcystic, macrocystic, or combined
 2. Most commonly occur in the cervicofacial region, axilla/chest, mediastinum, retroperitoneum, buttock, and perineum
 3. Diagnosis is by MRI
 4. Treatment is reserved for symptomatic lesions that cause pain, significant deformity, or threaten vital structures
 - a. First line – sclerotherapy (doxycycline, sodium tetradecyl sulfate (STS), ethanol)
 - b. Can also use erbium laser, or surgical excision
- G. Arteriovenous Malformations
 1. High-flow vascular malformations characterized by warmth, pain, bony destruction, discoloration and sometimes ulceration of the overlying skin
 2. Treatment is embolization followed by excision 24-72hrs later

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