

**TITLE: Cutaneous Malignancy**

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## **Epidemiology and Risk Factors:**

Over 800,000 cases of skin cancer occur in the United States annually, with more than 80% of these occurring in the Head and Neck. Basal cell carcinoma is the most common histological type, accounting for about 85% of all skin cancers. Basal cell carcinoma is followed in prevalence by squamous cell carcinoma, at about 10%. Melanoma, which accounts for 7300 deaths per year in the U.S., is beyond the scope of this article. Two thousand deaths per year can be attributed to other rarer forms of cutaneous cancer.

Most basal and squamous cell cancers are seen in patients over 60 years of age in sun-exposed areas, with basal cell carcinomas often occurring in younger individuals. Ultraviolet light is a widely accepted culprit in the etiology of these cancers. The wavelengths of light between 280 and 320 nm cause the greatest amount of sun-damage in laboratory experiments. These wavelengths fall into the ultraviolet B band. This form of ionizing radiation causes mutation of tumor suppressor genes in skin cells, which initiates the path from premalignancy to malignancy. The amount of ultraviolet B radiation that penetrates Earth's atmosphere is inversely proportional to the amount of ozone in the upper-most parts of the atmosphere. With depletion of the ozone layer, an increase in the amount of ultraviolet B radiation will occur. The ultraviolet A band (320 to 400 nm) used widely in tanning salons has recently been implicated in independently producing skin damage and neoplasias as well. Sun exposure, with consequent exposure to ultraviolet radiation, is clearly implicated as a risk factor for skin cancer, as skin cancer rates are highest among Caucasians living in areas of high sun exposure, such as Australia. Childhood and adolescence may be critical periods for establishing adult risk for basal cell carcinoma and may explain why studies have failed to find a large impact from increased cumulative sun exposure in adulthood on the risk of basal cell carcinoma.

Melanin protects the cells of the skin from the effects of ultraviolet radiation. Accordingly, individuals whose melanocytes produce little melanin are at higher risk for skin cancer. The following traits indicate higher risk of sun damage to skin and therefore increased risk of skin cancer risk:

1. fair complexion,
2. light hair,
3. blue/green eyes,
4. inability to tan,
5. history of multiple or severe sunburns,
6. Celtic ancestry.

Other etiologic factors include chronic exposure to chemical agents such as arsenic in Fowler's solution, superficial radiation therapy, burns, scars, and ulcers. Immunosuppression, as in transplant patients, may lead to increased incidence and aggressiveness of cutaneous malignancies. Rare genetic syndromes such as albinism, xeroderma pigmentosa, Bazex's syndrome (basal cell carcinomas, follicular atrophoderma, hypotrichosis, and hypohidrosis or hyperhidrosis), and the nevoid basal cell carcinoma syndrome (Gorlin's syndrome) predispose patients to Cutaneous malignancy. Gorlin's syndrome is a rare autosomal dominant condition in which patients develop multiple basal cell carcinomas, pitting of the palms and the soles of the feet, jaw cysts, spine and rib anomalies, calcification of the falx cerebri, and cataracts.

Cutaneous malignancy of the non-basal/squamous/melanocytic types are often not associated with sun exposure at all. The etiology of these rarer tumors varies widely and is often unknown.

## **Normal Skin Histology:**

The skin is the largest organ of the body and is approximately one sixth of the total body weight. It has four major functions including protection, sensation, thermoregulation and metabolic activity. It varies over regions of the body in thickness, color and presence of appendages such as hairs, glands and nails. Despite these variations, all skin has the same basic structure and organization. The external layer consists of a keratinized squamous epithelium known as the epidermis. The epidermis may be broken down into five layers representing stages of maturation of epidermal cells. The innermost layer is the stratum basale or germinativum. This is the germinal layer of the epidermis. More superficial is the stratum spinosum. Adjacent to this layer is the stratum granulosum in which cells have granules that contribute to the process of keratinization. Above this lies the stratum lucidum, which may only be appreciated in especially thick skin. The most external layer is the stratum corneum which is made up of flattened, fused cell remnants composed mostly of the fibrous protein, keratin.

Deep to the epidermis is the dermis. This layer is composed of fibro-elastic connective tissue that is highly vascular and serves to nourish and support the epidermis. The hypodermis or subcutaneous layer lies deep to the dermis. This layer contains variable amounts of adipose tissue. Hair follicles, sweat glands, sebaceous glands and nails are termed epidermal appendages as they develop from downward growths of epidermis into the dermis and hypodermis.

## **Basal Cell Carcinoma:**

Basal cell carcinoma is a slowly growing malignant neoplasm that originates in the epidermis. The tumor rarely metastasizes, but is capable of causing extensive local destruction, including to the nose, eyes and lips. It derives its name from the fact that the cells appear

histologically similar to the basal cells of the epidermis, though they lack intracellular bridges. Clinically, basal cell carcinoma can be divided into several groups. JL Smith subdivided basal cell carcinoma into nodular/nodulocystic/noduloulcerative, superficial, morpheaform, and pigmented subtypes.

The nodular type of lesion presents as a discrete, raised, circular lesion that appears pink and waxy, with a visible superficial capillary network. Often an area of central ulceration is surrounded by rolled borders of the lesion. The nodulocystic basal cell carcinoma has a more cystic appearance. This lesion is relatively non-aggressive.

The superficial basal cell carcinoma lesion exhibits scarring and atrophy with a threadlike waxy border consisting of one or several red scaling patches. These lesions are uncommon in the head and neck. They tend to spread by radial extension.

The pigmented basal cell carcinoma are uncommon and often resemble a nevus or melanoma. Often mistaken for seborrheic keratosis, melanoma, or dermatofibroma, these lesions behave no different clinically than nodular carcinomas.

The morpheaform basal cell carcinoma is the most clinically aggressive subtype. The lesions often present late because the margins are indistinct and the lesions often look like scars. The lesion is typified by its macular, whitish, or yellowish plaque.

Histologically, the typical basal cell carcinoma cell has a large, oval nucleus with little cytoplasm. The nuclei are relatively uniform. The cells lack intracellular bridges and a connective tissue stroma forms parallel bundles around tumor masses causing palisading of the cells and stromal retraction.

Basal cell carcinoma can be divided into four histological subtypes, with some clinical relevance: solid, keratotic, cystic, and adenoid. The solid subtype shows no cellular differentiation. The keratotic subtype, also known as basosquamous cell carcinoma shows some differentiation towards hair structures. Undifferentiated cells in combination with parakeratotic cells and horn cysts can be seen. Histologically, these lesions show features of both basal cell and squamous cell carcinomas in the same lesion. Keratotic cell carcinoma may be more aggressive than other subtypes of basal cell carcinoma. The cystic subtype shows differentiation towards sebaceous glands, with one or more cystic spaces within the tumor lobules. In the adenoid variety, the tumors display a glandular formation with strands of epithelial cells forming a lacelike pattern.

The metastatic rate of basal cell carcinoma ranges from 0.0028% to 0.55%. Tumors that metastasize tend to be large, locally aggressive, neglected lesions that have recurred despite repeated treatment. The interval from onset to metastasis ranged in one study from seven to 34 years, with a median of nine years; the five year survival rate was 10% (1).

## **Squamous Cell Carcinoma:**

Squamous cell carcinoma usually presents as a crusting, erythematous, ulcerated lesion with a granular, friable base. Some lesions may present as areas of persistent ulceration in the

site of previous trauma, burns, or an old scar (Marjolin's ulcer). Occasionally, squamous cell carcinoma presents as a nodular or exophytic lesion. Squamous cell carcinomas are often preceded by premalignant lesions such as actinic Keratoses or Bowen's disease; and these lesions tend to follow a more benign course than de novo tumors. Actinic keratoses occur in sun-damaged areas and are believed to be a common pathway in the accumulation of DNA damage that leads to squamous cell cancer, with a progression rate of about 20%. They are generally less than 1 cm in diameter and are commonly located on the face, scalp, hands, and forearms. Bowen's disease is carcinoma in situ of the skin, presenting as a well-circumscribed, erythematous scaly patch or plaque with an irregular border. The lesions are particularly common in individuals with a history of chronic arsenic ingestion.

On histologic examination most squamous cell carcinomas consist of irregular masses of epidermal cells that proliferate into the dermis. Keratinization may occur in well-differentiated tumors. There is often a range in the degree of differentiation and anaplasia that may influence management and prognosis. Verrucous carcinoma is a well-differentiated tumor that may show minimal atypia or individual cell keratinization on microscopic examination. Clinically, these lesions often present as white, cauliflower like lesions; and they are uncommon on the skin of the head and neck. Distinction between verrucous carcinoma and other epidermal proliferative processes can be difficult. The spindle-pleomorphic type of squamous cell carcinoma shows little evidence of differentiation. The cells are anaplastic with little keratinization and the spindle cells are intermingled with collagen. Adenoid squamous cell carcinoma is characterized histologically by anaplasia, acantholysis, and dyskeratosis that give a tubular and adenoid appearance.

Histologic risk factors for aggressive behavior include undifferentiated histologic pattern, depth into and beyond the subcutaneous fat, perineural invasion, and lymphatic invasion. Broder classified squamous cell carcinoma by histology in 1932 (see table 1). While most are locally destructive lesions, metastasis does occur in 2 - 5% of all cases overall. Microscopic indicators for increased risk of metastasis are listed in table 2. Squamous cell carcinomas less than 1 cm in greatest dimension rarely metastasize. Less than 1% of grade 1 and 2 lesions metastasize. Metastasis occurs in 3.6% of grade 3 lesions and 17% of grade 4 lesions. Metastasis may occur in poorly differentiated squamous cell carcinoma in as many as 32% of cases.

**Table 1: Histologic Grading of Cutaneous Squamous Cell Carcinoma**

Googe, Paul B., **DermPath Update** Volume 1 Number 4 - December 31, 1995

<b>Broders</b>	<b>UTMCK</b>	<b>Microscopic Appearance</b>
Grade 1	Well differentiated, moderately well differentiated	abundant keratinization, little nuclear anaplasia <

		25% undifferentiated cells
Grade 2	Moderately differentiated	50% keratinizing, nuclear anaplasia present < 50% undifferentiated cells
Grade 3	Moderately to poorly differentiated	less than 25% keratinizing, nuclear anaplasia extensive < 75% undifferentiated cells
Grade 4	Poorly differentiated	extensive nuclear anaplasia, little or no keratinization includes spindle cell and undifferentiated carcinomas > 75% undifferentiated cells

<b>Table 2: Microscopic Indicators of Metastatic Potential</b>	
Size > 2cm	Poorly differentiated (Broders 3 or 4)
Thickness > 2mm	Perineural invasion
Breslow level IV or V	Immunosuppression
Invasion of muscle, bone, or cartilage	Associated
Anatomic site: Ear, lip, genitalia	Locally recurrent

### **Merkel's Cell Carcinoma:**

Merkel cell carcinoma was originally described by Friedrich Sigmund Merkel in 1875 as a tumor arising in presumed mechanoreceptor cells in the snout skin of pigs and moles. The initial human malignant counterpart was described by Toker in 1972 as trabecular cell

carcinoma. Head and neck is the most common site of occurrence (50%), followed by the lower limbs (30%), upper limbs (15%), and trunk (5%). It usually presents as a solitary erythematous to deep purple plaque or nodule of up to several centimeters in size. Light microscopy of MCC shows small, round, basophilic cells arranged in sheets, rests, or trabeculae. The tumor originates in the dermis, and the overlying epidermis is usually spared. As it advances, the tumor infiltrates into underlying subcutaneous fat, fascia, and muscle. Vascular and lymphatic invasion is common. The poorly differentiated tumor cells show rounded nuclei, finely divided chromatin, scant cytoplasm, and a high rate of mitoses. Three histologic subtypes of Merkel's cell carcinoma include a solid type (most common), a trabecular type, and a diffuse type (worst prognosis).

Merkel cell carcinoma is rare and is often seen in the older population, with a roughly equal sex distribution. Management of the primary tumor should include wide excision with negative margins if possible. The optimal radial margin beyond that required for complete resection remains unknown. The effect of adjuvant radiotherapy to the primary site is unknown. Merkel cell carcinoma has a high rate of regional nodal involvement. Therefore, elective lymphadenectomy is often recommended even with clinically negative nodal drainage basins, although the therapeutic effect of elective management of the clinically negative regional nodes, with either surgery or radiotherapy, remains unknown. Merkel cell carcinoma is a radiosensitive tumor. Radiotherapy to the primary skin site after excision is recommended in order to reduce the local recurrence rate. Doses of 45-60 Gy in standard fractions to the surgical site and the regional lymphatic bed are used (6). Sentinel lymphadenectomy may yield prognostically relevant information. Merkel cell carcinoma has a high recurrence rate. The most common sites of initial recurrence are local and regional with median time to first recurrence in 8 months.

## **Pilomatrix Carcinoma:**

Pilomatrixoma, also known as pilomatricoma, and calcifying epithelioma of Malherbe, is an uncommon benign tumor of hair matrix origin. Malherbe and Cheantais first described the lesion in 1880 and originally believed the lesion was a benign tumor of the sebaceous glands. The lesions typically occur on the head and neck and are more common in females. There are two peaks of occurrence with age. The first is before the third decade, with greater than 60% occurring at this time. The second is during the sixth and seventh decades (3). Histologically, pilomatrixomas are characterized by irregularly shaped islands of small basaloid epithelial cells. The basaloid cells generally have small uniform nuclei and scant cytoplasm with indistinct cell borders surrounded by fibrillary material. Also often noted are a variable degree of squamous differentiation and sheets of ghost cells that occasionally are mixed with stroma containing multinucleated giant cells.

Calcifications may or may not be conspicuous. Pilomatrix carcinoma is an exceedingly rare locally aggressive and destructive tumor that usually arises from a pilomatrixoma. Bony invasion and occasional distant metastases have been reported (2). The carcinoma is more common in males than females, with a ratio of 2:1 in a review of 72 cases in the literature (2). Imaging studies such as MRI or CT may be useful to evaluate pilomatrix carcinoma that does not respond to wide local excision with negative margins. Histologically, Pilomatrix carcinoma is characterized by more numerous and atypical mitoses, nuclear atypia, tumor necrosis, and infiltration into subcutaneous fat, blood vessels, nerve and muscle. Aggressive wide local

excision is required for treatment.

### **Dermatofibrosarcoma Protuberans:**

Dermatofibrosarcoma is an uncommon locally aggressive tumor that arises in the dermis and invades deeper subcutaneous tissue. The incidence has been estimated to be 0.8-5 cases per 1 million population per year (4). Histologically, two types of lesions can be identified. In the plaque type of Dermatofibrosarcoma protuberans, slender cells with large, spindle shaped nuclei are embedded in the collagen stroma with sparse mitoses. In the nodular type, findings include high cellularity and irregular, short, intersecting bands of tumor cells forming a storiform pattern. The lesion presents as a large indurated plaque several centimeters in diameter with firm, irregular nodules varying in color from flesh to reddish brown. Mohs micrographic surgery is increasingly accepted as the treatment of choice for this lesion, because of the tendency for subclinical spread (4).

### **Carcinomas of the cutaneous adnexa:**

Cutaneous adnexa include the apocrine, eccrine, and sebaceous glands. The apocrine and eccrine glands are distinct glands, differing in embryological origin and function. Apocrine glands are derived from the pilosebaceous follicles, which also include the hair follicles and the sebaceous glands. In contrast, the eccrine glands are considered the true sweat glands. Carcinomas of the cutaneous adnexa are extremely rare in the head and neck. Sebaceous carcinomas have been reported in the literature.

Carcinomas of the sebaceous gland are rare malignant tumors that usually occur in the sebaceous glands of the eyelids, but they can also occur in the head and neck. The lesion is composed of foamy and pale lipid-containing cells, with Sudan IV staining confirming the presence of lipids. The nuclei are pleomorphic and hyperchromatic. If the tumor is well differentiated, the lesion may contain a peripheral layer of dark-staining basaloid cells, resembling BCCs. The tumor typically spreads by direct extension. Mohs Surgery is an excellent therapeutic modality. Local radiation treatment may be an alternative treatment, especially for patients who do not want surgery.

### **Management of Cutaneous Carcinomas:**

Many modalities exist to treat primary skin malignancies. The following is a discussion of treatment of basal and squamous cell carcinomas. In selected cases, nonsurgical means can achieve cure rates greater than 95%. All of the therapies are aimed at removing the cancer sight and a small but sufficient area of normal tissue. During initial evaluation, a punch biopsy should be taken to assess for depth of tumor invasion if a suspicious lesion is too large to simply resect primarily.

### **Staging:**

The first step in management of cutaneous carcinoma is staging the tumor, though this step is often skipped in smaller basal cell lesions, because of their low rate of metastasis and high cure rates. The following is the staging system for nonmelanoma skin cancer (basal and

squamous cell):

The primary tumor (T) is classified according to the following categories:

- TX: The primary tumor cannot be assessed.
- T0: There is no evidence of primary tumor.
- T1: Tumor is 2 centimeters or less in greatest dimension.
- T2: Tumor is more than 2 centimeters, but less than 5 centimeters in greatest dimension.
- T3: Tumor is more than 5 centimeters in greatest dimension.
- T4: Tumor invades the deep, extradermal structures (cartilage, bone, or muscle).

The regional lymph nodes (N) are clinically divided into the following categories:

- NX: Regional (nearby) lymph nodes cannot be assessed.
- N0: There is no regional lymph node metastasis.
- N1: Regional lymph node metastasis is present.

The state of metastasis (M) is defined as follows:

- MX: Distant metastasis cannot be assessed.
- M0: There is no distant metastasis.
- M1: Distant metastasis is present.

There are four basic stage groupings within the TNM system, as well as a "Stage 0" classification, which refers to carcinoma in situ:

- Stage 0: Tis, N0, M0
- Stage 1: T1, N0, M0; or
- Stage 2: T2, N0, M0; or T3, N0, M0
- Stage 3: T4, N0, M0; or T(any), N1, M0
- Stage 4: T(any), N(any), M1

In addition to staging, several other characteristics of the tumor should be noted. For instance, it is important to note whether the lesion is recurrent. Recurrence indicates inadequate previous therapy of the previous malignancy, and heralds a poorer prognosis for cure with most forms of treatment. The location of the tumor should be noted because certain areas of the head and neck have a higher risk of recurrence. Swanson (5) concluded that high risk areas include the junction of the ala with the nasolabial fold, the nasal septum, the nasal ala, the inner canthi, the lower eyelids of the periorbital region, the periauricular region extending to the temple, and certain scalp lesions. These areas combine to form an "H" zone on the face.

## **Curettage with Electrodesiccation**

Curettage with Electrodesiccation is one of the most commonly used modalities by dermatologists. It involves the use of various sizes of curettes until normal tissue is encountered over the entire base of the excision. This is possible because of the unusually soft feel of carcinoma when compared to normal tissue. Once normal tissue is encountered at the

base of the excision, electrodesiccation or fulguration of the wound is performed. After two to six cycles, the wound is treated topically and allowed to heal. Although this method is highly expedient and spares normal tissue, it does involve caring for an open wound and a risk of scarring. Usually, electrodesiccation is reserved for histologically and clinically favorable basal cell carcinomas (under 2cm, less aggressive histology). Squamous cell carcinomas are not often approached with this modality because of their more aggressive nature and higher risk of recurrence.

## **Cryosurgery**

Cryosurgery involves the use of a cryogen, usually liquid nitrogen to freeze and consequently kill tumor cells. A temperature of at least  $-30^{\circ}\text{C}$  is required, though many surgeons use a temperature of  $-50^{\circ}\text{C}$ . A thermocouple inserted at the margin of freezing ensures adequate ablation of the lesion, and a small rim of normal tissue is also taken. This technique may be useful in tumors overlying cartilage because the cartilage can be frozen without undergoing necrosis. Also, this technique has high tissue-sparing capabilities and is relatively quick. While the surgery may be especially useful in patients with multiple small lesions, larger lesions lead to a prolonged healing phase and more wound care. Hypopigmentation and scarring can occur with cryosurgery. Its use should be limited to lesions with well-defined borders and it is not useful for higher risk tumors.

## **Radiation Therapy**

Radiation therapy was used extensively in the past because of its high efficacy in curing most skin cancers. It can be used to treat a wide field of tumor and allows avoidance of surgery. However, radiation therapy does not allow surgical staging of tumors. Also it involves a protracted treatment course, it is more expensive than other methods, and it can lead to radiodermatitis and delayed carcinogenesis. It is currently reserved for an adjuvant role in high risk malignancies or in poor operative candidates.

## **Photodynamic Therapy**

Photodynamic therapy uses a photosensitizing drug that localizes selectively into tumor cells. The drug is applied topically, intravenously, or orally. This drug can then be activated by exposure to light in the form of a laser. Many photosensitizing agents are available, including porphyrin, 5-Aminolevulinic acid, benzoporphyrin derivative monoacid ring A, Tin ethyl etiopurpurin, and lutetium texaphyrin. Each of these agents has a different wavelength for peak absorption of light and the type of laser should be selected to achieve a wavelength closest to the drug's peak absorption spectrum. The efficacy photodynamic therapy in treating cutaneous malignancy is disappointingly low compared to other modalities (with clearance rates as low as 45%). Side effects include local edema and erythema, blistering, ulceration, and occasionally extensive tissue necrosis. This treatment modality is reserved for palliation of advanced carcinoma.

## **Excisional Surgery**

Surgical excision of cutaneous malignancy is the treatment of choice for most head and

neck surgeons. Frozen sections can be used to evaluate the margins of the tumor, decreasing likelihood of recurrence. Surgical excision can be carried out with cold steel or with an operating laser, such as the CO<sub>2</sub> laser. The main disadvantages of the technique are that it is more time consuming, inconvenient, and can be more expensive than other treatments. Although excellent cosmesis can be achieved when primary closure is possible, complete removal of the tumor should never be compromised for the sake of cosmesis. If more than 1/3 of a cosmetic facial unit needs to be excised, consideration should be given to excising the entire unit to achieve better cosmetic outcome.

## **Mohs' Surgery**

The reader is referred to the article "Surgery and Reconstruction after Mohs Surgery," in the 2003 section of the Quinn Grand Rounds Archive. This article contains a discussion about reconstruction of defects cause by tumor removal.

## **Treatment of Lymphatics**

Treatment of the regional lymphatics is indicated in certain cases of SCCa and BCCa of the head and neck. This is more often the case with advanced tumors that invade deep tissues such muscle, bone, cartilage or nerve. Elective parotidectomy is indicated for the complete removal of deeply invasive tumors of the periauricular region or when any lymphadenectomy is to be performed for tumors arising superior to the mandible. Larger SCCa (>2 cm), recurrent tumors and those arising in scarred areas (Marjolin's ulcer) all behave more aggressively and are more likely to require regional lymphadenectomy.

When dissection of the lymphatics is required, the nodal groups that must be removed are dependent on the location of the primary tumor. For tumors arising in the periauricular region, the preauricular, parotid and postauricular nodes are all at risk. The preauricular nodes receive drainage from the anterior parietal scalp, forehead, temple, ear, eyelids, skin overlying the zygoma, and in some cases the nasal ala. The parotid nodes drain a similar area and may be located superficial or deep to the facial nerve. If both are involved, a total parotidectomy may be required to removal all nodes. The facial nerve is spared unless involved with tumor. The postauricular nodes are rarely involved, but must be remembered in dealing with tumors in the posterior parietal scalp, ear and mastoid.

The suboccipital nodes lie along the occipital artery and may be involved with tumors arising on the scalp posterior to an imaginary line drawn through the external auditory canal. The external jugular node lies superficial to the sternocleidomastoid adjacent to the greater auricular nerve and drains the lateral face, ear and upper neck. The facial artery nodal group lies along the nasolabial portion of the facial artery from the nose to the mandible and drains the lateral nose, upper lip, medial face and nasolabial fold. The submental nodes lie between the anterior bellies of the digastric and drain the lips, nasal vestibule and chin. The lymphatics of the posterior triangle of the neck (level V) may be involved with cancers of the neck, ear and scalp. The submandibular nodes (level I) drain the cheek, chin, nose and nasolabial regions. The anterior jugular nodes (levels II-IV) receive drainage from throughout the head and neck and are potentially involved in cancers arising in any location. The supraclavicular nodes located along the transverse cervical artery may be involved in cancers of the lower neck or upper chest.

The same principles of neck dissection for cancers of the upper aerodigestive tract apply in dissection for skin malignancy. This includes the sparing of uninvolved structures, but sacrificing those vessels or nerves that are involved with tumor. Post-operative XRT is indicated for multiple positive nodes, extracapsular spread and lymphovascular invasion.

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